Congratulations to the entire field of poster presenters from the ACP Arizona Chapter Medical 2013 Annual Scientific Meeting held on November 15-17, 2013 at the University of Arizona, Tucson, Arizona. It was a record year for submissions, both in quantity and in quality, with a total of 164 posters presented, as well as the oral clinical vignette presenters. It is an honor for ACP Arizona Chapter to celebrate the work of these Resident-Fellow and Medical Student Members and their supporting mentors and educators in this e-publication, ¡Salud! The winners of the Oral Vignette competition, the Research Poster competition, and the Patient Safety/Quality Poster Competition were funded for travel to the Internal Medicine 2014, in Orlando, Florida, for the National ACP meeting, to present their work and represent the Arizona Chapter. Please enjoy these abstracts, and we hope they will inspire you to plan now to participate in the 2014 ACP Arizona Chapter Annual Scientific Meeting at Arizona State University in Tempe, on October 24-26, 2014.

Note Regarding Citation: To cite an abstract from this ¡Salud! publication, we suggest the following format: Author Last Name, Author First Name, (2013) Title of Abstract, ¡Salud!, 4:1, Abstract Page Number.
Congratulations to the ACP AZ 2013 Medical Student and Associate Poster and Oral Vignette winners, set forth here by category. The abstracts from the 2013 ACP Arizona Chapter Annual Scientific Meeting are all included in this publication. We begin with the First through Third Place Oral Vignette winners, followed by the remaining Oral Vignette abstracts in alphabetical order. The abstracts are then organized by PGY-4, PGY-3, PGY-2, PGY-1, or Medical Student, each section in alphabetical order by author’s last name. The poster category winners are identified in place, within each applicable submission year and within the author alphabetical order. To these Resident-Fellow and Medical Student Members, as the physicians whom we entrust with the future of internal medicine, we ¡Salud! your hard work and contributions.

ACP Arizona Chapter, Hamed Abbaszadegan, MD, MBA, Posters/Abstracts Committee Chair 2013

Poster Awards All PGY Years

**Oral Clinical Vignettes:** First Place Oral Clinical Vignette, Natasha Sharda, MD, UACOM at South Campus, *An ImPEEding Treatment*; Second Place Oral Clinical Vignette, Wylie Carhartt, MD, Banner Good Samaritan, *The Perils of Adventure*; Third Place Oral Clinical Vignette, Natalya Azadeh, MBChB, Mayo Clinic Arizona, *Bad Blood*.

**Research Category:** First Place Research, Keri Maher, DO, UACOM at University Campus, *QSOX1: A Desmoid Tumor Modifier Gene in FAP?*; Second Place Research, Robyn Emanuel, MD, Mayo Clinic Arizona, *Impact of Hematocrit on Symptom Burden Among Polycythemia Vera Patients*; Third Place Research, Seong seok Yun, MD, UACOM at University Campus, *Mechanism of Dual mTORC1/mTORC2 Inhibitor-Induced Apoptosis in Human Lymphoid Malignancies*.

**Case Report Category:** First Place Case Report, Arooj Kayani, MD, St. Joseph's Hospital & Med. Ctr., *To Treat Or Not To Treat: A Rare Case of Pseudo-Thrombotic Thrombocytopenic Purpura in a Jehovah’s Witness*; Second Place Case Report (Tie), Seth Assar, MD, UACOM at South Campus, *Ball-Valve Pattern Right Mainstem Bronchus Obstructive Pneumonia Secondary to a Malignant-Transformed Respiratory Papilloma*; Second Place Case Report (Tie), Krystal Renszel, DO, Banner Good Samaritan, *Orbital Apex Syndrome: A Rare Complication of Herpes Zoster Ophthalmicus*.

**Patient Safety/Quality Improvement Category:** First Place PS/QI, Anjali Takyar, DO, UACOM at South Campus, *Improving Compliance With Surviving Sepsis Campaign Resuscitation Bundles At UA - South Campus*; Second Place PS/QI, Leslie Padrnos, MD, Mayo Clinic Arizona, *Increasing Compliance for Human Immunodeficiency Virus Screening in Internal Medicine Residency Medicine Ward Services: A Quality Improvement Project*; Third Place PS/QI, Leigh Anne Rundgren, DO, Banner Good Samaritan, *Interdisciplinary Interventions To Decrease Cystic Fibrosis Exacerbation Length Of Stay*.

Abstracts from the 2013 ACP Arizona Chapter Scientific Meeting - Page 2
**PGY-1 Awards:** First Place PGY-1, Rishi Bhargava, MD, UACOM at South Campus, *Between A Rock And A Hard Place- An Uncommon Cause Of PEA Code Arrest*; Second Place PGY-1, Allon Kahn, MD, Mayo Clinic Arizona, *Disseminated Nocardia Cyriacigeorgica Infection In A Liver Transplant Patient Recipient*; Third Place PGY-1, Kareem Bannis, MD, UACOM at South Campus, *Not Just Any GIST*.

**Medical Student Awards:** First Place Medical Student, Sandeep Bains, UofA College of Medicine/TUC, *Campaign Against Texting and Driving*; 2nd Place Medical Student, Hersh Goel, UofA College of Medicine/PHX, *A case of thrombosed persistent sciatic artery aneurysm resulting in amputation*; Third Place Medical Student, Tiffany Son, UofA College of Medicine/TUC, *Optimization of Porcine Heart Decellularization*.

**ORAL CLINICAL VIGNETTE SUBMISSIONS**

**Oral Clinical Vignette – First Place**

**PROTHROMBIN COMPLEX CONCENTRATE: AN IMPEEDING TREATMENT**

Natasha Sharda MD, UACOM at South Campus

Natasha Sharda MD, Bijin Tajudeen MD

**INTRODUCTION:**

Atypical Hemolytic Uremic Syndrome (aHUS) is a rare thrombotic microangiopathy characterized by hemolytic anemia, thrombocytopenia and acute renal failure. It is caused by the uncontrolled activation of terminal complement which has been linked to a number of etiologies including; autoimmune, genetic mutations, non shiga-toxin producing infections as well as drug toxicity. Eculizumab is a monoclonal antibody which has been used to in treatment of aHUS by inhibiting terminal compliment activation. Here we report an interesting case of drug induced aHUS successfully treated with eculizumab.

**CASE PRESENTATION:**

A 51 yr. old male with a past medical history significant for rheumatic heart disease status post mitral valve replacement 20 years ago on warfarin for anticoagulation, was admitted to the hospital following a mechanical fall. A CT of the head done in the ED showed a subdural hematoma. Laboratory tests on admission reported INR as unable to calculate, PT>100, and serum creatinine 0.8 mg/dL (0.6-1.1 mg/dL). Subsequently patient received pro thrombin complex concentrate (PCC) for reversal of supra-therapeutic INR in view of his subdural hematoma. The next day patient became oliguric and serum creatinine increased to 2.8 mg/dL. Subsequently patient developed severe hyperkalemia and hemodialysis was
initiated. Further evaluation at this time was significant for anemia, thrombocytopenia, elevated LDH, indirect hyperbilirubinemia, low haptoglobin and low complement C3. Peripheral smear showed schistocytes and renal biopsy indicated thrombotic microangiopathy favoring atypical HUS. Upon diagnosis patient received 2 days of plasmapheresis before discharge with eculizumab treatment. Over follow up platelets and LDH stabilized and hemodialysis was successfully terminated within 3 months due to improvement in urine output and solute clearance.

DISCUSSION:

Hemolytic uremic syndrome is characterized by the presence of vascular abnormalities including glomerular endothelial injury and thrombosis. This disease can be divided into two broad categories, typical and atypical. Typical HUS is acquired and triggered by infectious agents such as strains of E. coli (Stx-E. coli) that produce Shiga-like exotoxins, whereas atypical HUS (aHUS) can be genetic, acquired, or idiopathic. The pathogenesis of aHUS involves continuous activation of the terminal complement pathway resulting in endothelial damage which in turn activates the coagulation cascade resulting in thrombocytopenia and anemia. The precipitation of aHUS following exposure to Prothrombin complex concentrate is unique to this case. As per literature drug toxicity associated with aHUS has mainly been associated with chemotherapy agents such as Cyclophosphamide, Cisplatin and Mitomycin. Pathogenesis of PCC as a precipitating factor of HUS in this case correlates with the ability of thrombin to directly activate complement by cleavage of C5. Release of C5b is instrumental in initiation of membrane attack complex and subsequent injury to endothelium and development of thrombosis. Further supporting this pathogenesis includes successful treatment of the patient with eculizumab, a monoclonal antibody that prevents the cleavage of C5. In addition, coagulation factors have also been associated with cleavage of complement factor C3 explaining the low level seen in this case.

References:

5) Loirat C Frémeaux-Bacchi Atypical hemolytic uremic syndrome. Orphanet J Rare Dis. 2011 Sep 8; 6:60.


**Oral Clinical Vignette – Second Place**

**THE PERILS OF ADVENTURE**

**Wylie Carhartt, MD, Banner Good Samaritan Regional Medical Center**

**Introduction**

*Angiostrongylus cantonensis* is the most common cause of an uncommon entity, eosinophilic meningoencephalitis. While it is a condition rarely found in North America, increased international travel commands increased awareness of exotic helminthic infections. Also known as the Rat Lungworm, it is given its name because the mature form of the worm can be found in the pulmonary arteries of rats.
**Case Presentation**

A 29-year-old female with no significant past medical history presented to the Phoenix VA with symptoms of progressively worsening bilateral lower extremity weakness, bowel and bladder dysfunction, and increased somnolence. Over the previous 3 weeks, she had been diagnosed with a urinary tract infection, fecal impaction and traveler’s diarrhea. Her travel history was significant for a trip to Fiji one month ago where she experienced the local cuisine, including the consumption of raw snails. Diagnostic workup prior to admission, including MRI of the lumbar spine and CT of the head, failed to achieve a diagnosis. Her prior labs were significant for a mild leukocytosis with eosinophilia. Fecal leukocytes were elevated but stool culture and ova and parasite exam were unremarkable. On presentation, she was afebrile and vital signs were stable. Her neurologic exam revealed somnolence, confusion, neck stiffness, and asymmetric lower extremity weakness. A lumbar puncture was performed that demonstrated increased intracranial pressure, elevated white blood cells, predominantly eosinophils, with elevated protein and low glucose. CSF fluid analyzed at the Center for Disease Control confirmed the diagnosis of *Angiostrongylus cantonensis*.

**Discussion**

Infection with *A. cantonensis* is a prime consideration in travel medicine when patients present with neurologic complaints. Humans serve as incidental hosts to this parasitic roundworm and transmission can occur from exposure to raw or undercooked snails or slugs, which serve as intermediate hosts, or from contaminated water or vegetables. This helminth is typically found in Southeast Asia and the Pacific basin, but has also been found in the Caribbean and Hawaiian islands. The incubation period is between 1-6 weeks and the self-resolving illness can last upwards of 2 months. Neurologic symptoms typically include headache, nuchal rigidity, paresthesias and weakness. Rarely, severe sequelae such as radicular pain, coma, paralysis and even death can occur. The pathogenesis of disease is multifactorial: from direct invasion of the parasite in parenchymal tissue, toxic byproducts released by dead and living parasites, and pro-inflammatory effects of eosinophils. *A. cantonensis* cannot complete its life cycle in humans and as a result the worms die in the neural tissue, resulting in a severe inflammatory response. Treatment is primarily symptomatic. Lumbar punctures can be performed to relieve intracranial pressure and lessen severity of headache. Combined albendazole and corticosteroid has been suggested to reduce the overall length of infection.
Oral Clinical Vignette – Third Place
BAD BLOOD – A CURIOUS CASE OF RECURRENT HEMOPTYSIS

Natalya Azadeh, MD, Mayo Clinic Arizona

Introduction:
Microcrystalline pulmonary angiopathy as a result of intravenous injection of the insoluble components of oral pain medications is a diagnosis which is not often considered. Self-medication and drug abuse should always be considered in patients with significant psychiatric trauma. The manifestations are variable and can lead to significant morbidity and even mortality.

Case:
A 38 year old male Gulf War Veteran was admitted multiple times for hemoptysis at various hospitals. He had an extensive history including post-traumatic stress disorder (PTSD), significant trauma requiring multiple surgeries and recurrent upper extremity cellulitis. He underwent a prior extensive evaluation which included two bronchoscopies, nasopharyngoscopy, CT angiogram, CT Maxillofacial and esophagogastroduodenoscopy (EGD); all were unrevealing for source of bleed.

On this admission he complained of left sided pleuritic chest pain, and cough with hemoptysis. Physical examination was unremarkable other than mild hypoxia and multiple upper extremity scars. Laboratory testing revealed a new coagulopathy with an international normalized ratio (INR) of 6.09 and a mild normocytic anemia. Coagulation factor levels were checked revealing a pattern of factor deficiency concerning for vitamin K antagonist ingestion. A warfarin level was in the therapeutic range. Repeat CT angiogram of the chest, bronchoscopy and nasopharyngoscopy were again unrevealing.

The patient was requesting intravenous pain medications frequently in a manner felt to be disproportionate to his level of pain. Psychiatry was consulted for and confirmed drug seeking behavior and PTSD. Further discussion with the patient revealed he had undergone a diagnostic video-assisted thoracoscopic surgical (VATS) biopsy at an outside hospital. The biopsy slides were requested and pathology consultation confirmed microcrystalline angiopathy with evidence of microcrystalline cellulose and crospovidone deposition in a perivascular distribution.

Discussion:
Pulmonary disease due to deposition of foreign material has been well reported. Microcrystalline cellulose (200 µm), a dry binder, and Crospovidone (100 µm), a solubility enhancer, are used in oral medications. This includes pain medications and benzodiazepines, which the patient had been
prescribed. Both of these substances can embolize to the lungs when aqueous tablet suspensions are injected intravenously. They both have characteristic histology, refractility, and staining making them readily identifiable and distinguishable from other tablet constituents. In further support of our diagnosis, particles greater than 10-15 μm are not able to reach the distal airways via inhalation. This, in combination with his biopsy, pointed to injection of dissolved oral pain medication leading to perivascular pulmonary inflammation. He was discharged and failed to show up for follow up.

**Oral Clinical Vignettes (continued)**

**WHEN A STROKE IS NOT A STROKE**

Layth Al-Jashaami MD, Maricopa Medical Center

Layth Al-Jashaami MD (Member), Amira Attya MBBCh, Yasamine Al-Kenani MD (Member), Gerges Makar MBBCh (Member), Shaghayegh Abdollahi MD (Fellow), Pedro Quiroga MD

Maricopa Integrated Health System, Phoenix, Arizona

**INTRODUCTION:** Chorea and ballismus can result from a variety of conditions, including cerebrovascular disorders, infections, drugs, metabolic abnormalities, neurodegenerative diseases, immunologic disorders, and tumors, as well as from non-ketotic hyperglycemia in primary diabetes mellitus. The radiographic features of hemichorea-hemiballismus (HCHB) can be reminiscent of a basal ganglia hemorrhage. Since management and work-up of the two conditions is very different, it is important to be aware of this phenomenon.

**CASE PRESENTATION:** A 62-year-old female with history of hypertension, recurrent Bell's palsy, and aseptic meningitis presented following four days of involuntary movements of the right arm. On admission, her blood pressure was 176/100 mmHg. Neurological examination revealed persistent irregular jerky movement of her right upper limb which was exacerbated by voluntary movement, the remainder of the neurological and systemic examination were unremarkable. Blood glucose was 289 mg/dl and glycosylated hemoglobin was 15.3%. Urinalysis was negative for ketones. Brain CT showed a hyperdense lesion in the left caudate and the putamen, sparing the internal capsule. Due to the unusual pattern on CT scan, MRI was obtained to further characterize the abnormality. The MRI showed increased T1-weighted signal in the left caudate, putamen and globus pallidus, again sparing the internal capsule. There was also demonstration of decreased T2-weighted and T2 signal in the same areas without surrounding edema. On diffusion weighted imaging, bilateral basal ganglia demonstrated slight decreased signal, specifically within the right globus pallidus, and left caudate, putamen, and globus pallidus. MRI was initially interpreted as subacute hemorrhage in bilateral basal ganglia. Based on this radiographic pattern, the patient’s presenting signs and symptoms, and elevated glucose level, the diagnosis of HCHB was made. The patient was admitted for hyperglycemic management with aggressive insulin therapy. At one month follow-up, her right-sided chorea/ballismus was worsening, having progressed to involve the right face and leg.

**DISCUSSION:** Hemichorea–hemiballismus is a rare presentation of non-ketotic hyperglycemia. It usually affects elderly females without previous history of hyperglycemia, such as occurred with our patient. It
is characterized by the development of unilateral choreiform and/or hemiballistic movements typically over a period of hours. The key diagnostic feature is the characteristic imaging findings. CT scan usually shows hyperdensities in the striatum. T1-weighted MRI showed hyperdensities in the basal ganglia which usually spare the internal capsule. The differential diagnosis of this MRI finding includes hypertensive hemorrhages, basal ganglia calcification, foreign body, Tay-Sachs disease, tuberous sclerosis and Wilson’s disease.

**CONCLUSION:** Our patient serves to point out that, although rare, non-ketotic hyperglycemic chorea–hemiballisms is something that should be considered in the setting of characteristic radiographic findings in the setting of hyperglycemia. This condition is readily treatable, often with rapid resolution of the abnormal movements, thus making prompt diagnosis and treatment of hyperglycemia essential.

**A CASE OF SURREPTITIOUS SYNCOPE**

Ateefa Chaudhury MD, UACOM at University Campus

Ateefa Chaudhury MD and Joao Paulo Ferreira MD, Department of Medicine, University of Arizona, Tucson, AZ

**Introduction:**

Syncope is a common disorder defined as a transient loss of consciousness combined with an inability to preserve postural tone, followed by spontaneous recovery. Syncope accounts for approximately 6% of hospital admissions each year in the United States and comprises up to 1-3% of all ED visits. Up to 50% of our population will experience a syncopal episode in their lifetime and while the majority of causes are benign, this symptom presages a life-threatening incident in an exiguous subset of patients. This case illustrates the crucial role a thorough history and physical examination combined with extensive workup plays in elucidating a unique and uncommon cause of syncope that can be fatal without early diagnosis and appropriate treatment.

**Case Presentation:**

A 59 year old Caucasian male presented to the ED with complaints of passing out in the kitchen. He felt dizzy and lost consciousness for 1-2 minutes. This unwitnessed event occurred when he rose to a standing position. He denied any incontinence or tongue biting. Review of symptoms was positive for a 40 lb unintentional weight loss over 6 months, poor appetite, generalized weakness/fatigue, blurry vision, mild dysphasia to solids, decreased sense of taste/smell, a viral illness 6 months ago, constipation, hesitancy/dysuria, numbness/tingling in extremities, and erectile dysfunction. He had two prior admissions in community hospitals for syncope and discharged with a diagnosis of adrenal insufficiency. Discharge medications included Midodrine, Fludrocortisone, Lyrlica, Percocet, and Venlafaxine. His vitals were notable for orthostatic hypotension (lying: BP 125/73, P 75 & standing: BP 82/59, P 99). Physical examination revealed only decreased vibratory sense L toe/calf with 1+ L ankle and 0 R ankle reflexes.

He had an extensive diagnostic evaluation based on systems as described (Table 1). His CBC and CMP were normal. He had a lumbar puncture and the CSF revealed: Glucose 49, Protein 36, WBC 2, RBC 82,
and gram stain was negative for organisms. His INR and PT were elevated at 1.3 and 16.4. His UA showed: 500 Protein, 50 Glucose, 11 WBC, and 4 RBC. He required additional workup for further evaluation of his proteinuria (Table 2). After this work up failed to reveal the etiology of his syncope, he was discharged home with a 5-day taper of Venlafaxine, which can cause orthostatic hypotension and outpatient subspecialty follow up with Neurology, GI, Nephrology, and Hematology was arranged. 18 days after discharge he returned to ED with weakness, dizziness and 8/10 pain in his lower extremities.

Two days prior to his readmission he had emesis with dysphagia to liquids, he continued to feel light headed when standing and had 3-4 episodes of syncope. ED vitals were again consistent with orthostatic hypotension (lying BP 114/72, standing BP 65/47) and his physical exam was now notable for anasarca. He developed non-oliguric renal failure with urinary retention (creatinine of 2.5) and continued to have persistent nephrotic range proteinuria. He subsequently underwent a renal biopsy which was consistent with amyloid p deposition in the glomeruli and stained positive for Congo Red. He was diagnosed with generalized autonomic failure and universal autonomic dysfunction from AL Amyloidosis in association with MGUS and Hepatitis C. Per Hematology he was not an autologous stem cell transplant candidate due to HCV & autonomic dysfunction. He was started on Melphalan & Prednisone with a predicted 50% chance of clinical response and median predicted survival of up to 50% in 5 years.

<table>
<thead>
<tr>
<th>Table 1: Differential</th>
<th>Work Up</th>
</tr>
</thead>
<tbody>
<tr>
<td>Metabolic</td>
<td>B12 454, Folate 13.8</td>
</tr>
<tr>
<td>Toxins</td>
<td>Mercury &lt;2, Lead 2.4, Arsenic &lt;2, Copper 46</td>
</tr>
<tr>
<td>Endocrine</td>
<td>Cortisol/Cosyntrpin Stimulation Test: Normal</td>
</tr>
<tr>
<td></td>
<td>TSH 5.01, T4 1.01</td>
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<tr>
<td>Infectious</td>
<td>HIV/Ccci/VDRL: Negative</td>
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<tr>
<td></td>
<td>Hepatitis C Genotype 1a Viral Load: 6,687,790</td>
</tr>
<tr>
<td>Drugs</td>
<td>Comprehensive Urine Toxicology: Oxycodone</td>
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<tr>
<td>Neurologic</td>
<td>CT Head: Negative &amp; Carotid Duplex &lt;50 % Bilateral Stenosis</td>
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<tr>
<td></td>
<td>Anti-ganglioside Antibodies/AchR/Paraneoplastic: Negative</td>
</tr>
<tr>
<td>Cardiogenic</td>
<td>Ekg/Telemetry: Normal</td>
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<td></td>
<td>Echo: EF 60-65% with normal LV RV function</td>
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<tr>
<td>Autoimmune</td>
<td>ANA 8 (1:40), RF&lt;10</td>
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<td></td>
<td>Anti-MAG/ANCA/MP0/PR3 Antibodies: Negative</td>
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</table>

<table>
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<tr>
<th>Table 2: Proteinuria Eval</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Urine Protein to Cr Ratio</td>
<td>15,760 mg/gm</td>
</tr>
<tr>
<td>Renal Ultrasound</td>
<td>Normal</td>
</tr>
<tr>
<td>C3/C4/Cryoglobulin</td>
<td>Normal/Negative</td>
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<tr>
<td>SPEP</td>
<td>IgA kappa 2.27g/dl</td>
</tr>
<tr>
<td>UPEP</td>
<td>No paraprotein</td>
</tr>
<tr>
<td>B2 Microglobulin</td>
<td>5.7</td>
</tr>
<tr>
<td>IgA/IgG</td>
<td>2.101/ &lt;270</td>
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<tr>
<td>Kappa Free Light Chain</td>
<td>20.6</td>
</tr>
<tr>
<td>Lambda Free Light Chain</td>
<td>9.9</td>
</tr>
<tr>
<td>Kappa/Lambda Ratio</td>
<td>2.07</td>
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<tr>
<td>Skeletal Survey</td>
<td>Negative</td>
</tr>
<tr>
<td>Bone Marrow/FISH</td>
<td>Possible cirrhosis, Nonspecific lymphadenopathy</td>
</tr>
<tr>
<td>CT Chest/Abdomen/Pelvis with Contrast</td>
<td>No convincing lytic lesions suggestive of multiple myeloma or a plasmacytoma</td>
</tr>
</tbody>
</table>
Discussion:

AL Amyloidosis is a disease of older adults. Median age at diagnosis is 64, <5% of patients are under the age of 40, and 65 – 70% of patients are male. There is little data regarding whether incidence varies by ethnicity or geography. It is an uncommon disorder & exact incidence is unknown. In the United States, the incidence is 6 – 10 cases per million person-years and <1% of patients with MGUS per year develop signs related to AL Amyloidosis.

Four criteria must be met in order to diagnose AL Amyloidosis: 1) presence of an amyloid-related systemic syndrome, 2) amyloid staining by Congo Red in any tissue, 3) evidence that the amyloid is light chain-related established by direct examination of the amyloid, and 4) any evidence of a monoclonal plasma cell proliferative disorder (presence of a serum/urine M protein, abnormal free light chain ratio, or bone marrow biopsy showing clonal plasma cells).

Amyloid deposition can affect multiple organ systems. 20% of patients will develop mixed sensory & motor peripheral neuropathy (symptoms of numbness, paresthesia, & pain). 15% will develop autonomic neuropathy (orthostatic hypotension due to autonomic nervous system damage that can also cause bowel/bladder dysfunction). The cardiovascular, gastrointestinal, and renal systems are most commonly affected. 60% of patients will develop restrictive cardiomyopathy from amyloid deposition within the myocardium. Patients can have macroglossia (10%), fatigue/weight loss (50%), hepatomegaly/elevated LFTs (25%), and dysphagia/constipation. 50% of patients will develop nephrotic syndrome. Due to interactions of amyloid fibrils with Factor X, 28 – 51% of patients develop bleeding diathesis.

This rare case of syncope secondary to AL Amyloidosis illustrates how the disease can present as a wide range of systemic symptoms, frequently masquerades as other conditions, and documents how early diagnosis of amyloidosis prior to autonomic dysfunction is essential to allow all available treatment options, specifically autologous transplantation.

A TALE OF TWO KIDNEYS: MICROSCOPIC POLYANGIITIS IN AN UNCOMMON PATIENT POPULATION

Jason McCourt MD, St. Joseph Hospital and Medical Center

Jason McCourt MD, Jeff Javed MD, Department of Internal Medicine, St. Joseph Hospital and Medical Center, Creighton University Phoenix Campus

Introduction:

Microscopic Polyangiitis (MPA) is an uncommon form of vasculitis associated with antineutrophil cytoplasmic antibodies (ANCA). The autoimmune disease can affect multiple organs systems, but has also been described as having a renal-limited form. Epidemiology of MPA in the United States appears to most commonly involve Caucasian males with an age of onset in the 5th to 6th decade of life; however, MPA can occur in any population group and in any age range.
Case Report:

A 78-year-old Hispanic male presented to the Emergency Department with family for complaints of weakness and confusion for 2 days. Associated symptoms included weight loss, low back pain, and decreased urination with gross hematuria. Patient denied any cough, hemoptysis, rash, or joint pain. Initial physical exam showed the patient was alert and oriented to person, place, and time, lungs clear to auscultation, a grade II/VI systolic murmur heard best at left sternal border, 3+ edema in bilateral lower extremities,. Initial lab work showed hemoglobin of 9, creatinine 7.2 (prior baseline 0.8), glomerular filtration rate of 7, and presence of blood and sub-nephrotic levels of protein in the urine. With the stated clinical presentation and laboratory findings, the patient received a workup for pre-, post- and intrarenal causes of acute kidney injury. CT of the abdomen showed mild anasarca and no renal abnormalities. Further laboratory workup included complement levels showing decreased C3, positive myeloperoxidase (MPO), and negative proteinase 3 (PR3). These immunologic results, supported by the patient’s age and absence of historical findings of extra-renal complaints or symptoms, led to the suspicion that the patient could be suffering from a renal-limited form of MPA. A renal biopsy was performed for histologic examination, which showed necrotizing crescentic glomerulonephritis with mild-to-moderate interstitial fibrosis. The patient received plasmapheresis, one treatment of cyclophosphamide, and was started on Solu-Medrol 1gm/day. During the hospital stay the patient developed uremic encephalopathy from his renal injury. On the anticipated date of discharge the patient developed an acute GI bleed. EGD showed an actively bleeding duodenal ulcer. After the initial bleed was controlled, the patient developed another bleeding event during the evening, in which the patient received multiple units of blood and eventually the family decided on withdrawing support.

Discussion:

The presentation of disease in this case is quite unique. Studies can be found in the medical literature depicting the common presentation of microscopic polyangiitis. One study reports MPA being far more common in Caucasians. The study quotes from the Glomerular Disease Collaborative Network that 89 percent of patients were white (3). Another study describes the average onset to be at 50 years of age (2). Along with glomerulonephritis, pulmonary capillaritis is typically seen at presentation. Gastrointestinal involvement at presentation is also not uncommon (1). 27 percent of patients in one study looking specifically at gastrointestinal involvement in patients with small and medium-size vessel vasculitides were noted to have gastroduodenal ulcerations diagnosed endoscopically (3). The patient above was of Hispanic descent, nearly 20 years older than mean age at diagnosis, and denied any pulmonary symptoms. The exact cause of the patient’s duodenal ulcer is unknown, but small-vessel vasculitis from MPA can not be excluded as a potentiating factor.

Conclusion:

Studies can be found in the literature detailing microscopic polyangiitis as being most commonly diagnosed in the 5th to 6th decade of life in Caucasian males. Our case illustrates that MPA can present at any age, including those at the extreme ends of the bell curve, and any ethnic background. Physicians need to keep MPA in the differential of all patients regardless of age, gender, or ethnicity if the clinical picture correlates. With the proper history and physical, laboratory studies, and diagnostic workup, a diagnosis of MPA can be efficiently made.
References:


DOUBLE PYLORUS

Iram Qureshi, DO, Sierra Vista Regional Medical Center

Abstract

A rare complication of peptic ulcer disease is double pylorus. It is generally seen in chronic cases of peptic ulcer disease but can also be a congenital malformation. It is characterized by the presence of a fistula from the distal stomach to the duodenal bulb. Its prevalence of this complication is about 0.02% in patients with peptic ulcer disease and in most patients it persists for the remainder of their lives. However in some patients the fistula can merge with the pylorus to form a single channel or spontaneously close.

RARE COMPLICATION FROM A PITUITARY TUMOR

Gina Wu, DO, Verde Valley Medical Center

Pituitary tumors are the most common causes of hypopituitarism in adults. Complications from these tumors can be neurosurgical emergencies and need to be identified and treated immediately. A 44 year old male was recently diagnosed with a pituitary tumor on MRI of the brain at a Phoenix hospital. Since the MRI, he started to gradually develop a headache that continued to increase in intensity. It became so severe with nausea and vomiting that the patient returned to the hospital the next day. He was treated with an analgesic from the emergency department and discharged with no further imaging done. He had minimal improvement of his headache. He decided to go to a different emergency department close to his home. While his wife was driving him to the hospital, the patient suffered a tonic clonic seizure that lasted approximately 8 minutes. The patient was lethargic and confused after the seizure that lasted for the remainder of the day. Neurological examination revealed a right ptosis and ophthalmoplegia with only trace movements of the right eye medially and downwards. The rest of the neurological exam was negative. CT of the head without contrast showed a pituitary tumor measuring 3.9 x 1.6 cm without evidence of hemorrhage. The patient was started on Keppra and did not have another seizure during his hospitalization. Cortisol level was found to be low and IV hydrocortisone was started. He was hypotensive with a sodium level of 123, which was most likely secondary to his adrenal insufficiency. MRI of the brain showed a pituitary macroadenoma measuring 3 cm with extension in the right cavernous sinus with apparent recent hemorrhage.
measuring 1.5cm in diameter in the right pituitary inferiorly. The patient’s lethargy and headache improved. Examination continued to show right ophthalmoplegia with no other focal neurological findings identified. The patient was subsequently transferred to a tertiary center for management of the pituitary tumor with possible neurosurgical intervention.

Complications from pituitary adenomas include acromegaly, cushing’s syndrome, hyperpituitarism, pituitary tumor apoplexy, and central diabetes insipidus. Pituitary tumor apoplexy is an uncommon syndrome resulting often spontaneously from hemorrhage or infarction of a pituitary adenoma. It presents with sudden onset of headache, possible diplopia from compression of cranial nerves within the cavernous sinuses, and acute hypopituitarism. This is a neurosurgical emergency requiring decompression of the pituitary gland. Urgent replacement of glucocorticoids may be necessary because of acute ACTH deficiency.

Identification of complications from pituitary tumors is imperative to ensure that the necessary treatment is given in the appropriate timeframe.

PGY-4 SUBMISSIONS

AN UNEXPECTED CASE OF A PAINFULLY DENUDED ADULT MALE

Monica Guzman-Limon MD, Banner Good Samaritan, PGY-4

Introduction: Staphylococcal scalded skin syndrome (SSSS) is a dermatological condition induced by epidermolytic exotoxins produced by certain strains of Staphylococcus aureus. SSSS can be difficult to distinguish from toxic epidermal necrolysis. SSSS is more commonly seen in younger children due to their inability to effectively excrete exfoliatoxin. Fewer cases have been noted in adults, usually in the setting of renal insufficiency or immunocompromised state. Case description: A 56 year-old homeless male presented with bilateral lower extremity draining wounds. His past medical history included intravenous drug abuse, infective endocarditis requiring tricuspid valve replacement and resultant non-ischemic cardiomyopathy with systolic heart failure. Upon admission, CBC, blood cultures and wound cultures were obtained and he was empirically started on vancomycin for presumed methicillin-resistant Staphylococcus aureus infection (MRSA). On hospital day one, he developed severe septic shock and subsequent oliguric renal insufficiency. His renal insufficiency was thought to be related to compromised renal blood flow in conjunction with the use of nephrotoxic antibiotics. Within the next 12 hours he progressed to develop superficial desquamation of the perineum, erythema with fine pustules over the back and trunk, large radial perioral fissures and periorbital fissures. Greater than thirty percent of his total body surface area was involved. Initially there was concern for toxic epidermal necrolysis (TEN) or other drug related eruption, thus vancomycin was discontinued and daptomycin was started to treat culture confirmed MRSA infection. After obtaining skin biopsies, treatment with steroids and intravenous immunoglobulin was initiated. Despite receiving 4 days this treatment, he had substantial worsening erythema, edema, and superficial sloughing of his skin. Staphylococcal scalded skin syndrome may worsen with the use of IVIG and steroids; this occurrence favored a clinical diagnosis of SSSS rather than TEN. IVIG and steroids were stopped. Pathology eventually revealed findings of epidermal necrosis without full thickness epidermal involvement. He was continued on daptomycin along with wound care.
and pain control. Hemodynamics and fluid status were optimized and he had notable improvement over the next several days as renal function normalized. Discussion: Interestingly, the onset of this patient’s dermatological symptoms corresponds to the onset of his acute renal insufficiency. Although Staphylococcus infections are frequently encountered in all age groups, SSSS is less common, especially in adults. The mortality of adult patients with SSSS is much higher than that of children. This case highlights the importance of optimizing renal function in adult patients with Staphylococcus infections in order to minimize the risk of developing this additional life-threatening complication.

THROMBOTIC STORM REVISITED

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Bijin Thajudeen MD

Renal artery thrombosis is a rare, but serious and often misdiagnosed condition. It can be due to in-situ thrombosis or thromboembolism. Few cases of renal vein thrombosis secondary to pancreatitis have been reported previously however acute necrotizing pancreatitis resulting in bilateral renal artery thrombosis is rare. 66-year-old female presented with abdominal pain, nausea, vomiting and decreased urine output for 5 days prior to admission and was managed as probable acute pancreatitis and acute kidney injury. Significant medical history includes hypertension for 5 years and use of Ibuprofen for low back pain for last three years. CT scan of abdomen and pelvis without contrast revealed normal pancreas, bilateral renal cortical scarring and mesenteric edema. Hemodialysis was initiated because of anuria and hyperkalemia on day 4. Percutaneous biopsy of the left kidney performed due to unclear etiology of acute kidney injury showed normal glomerulus and interstitium. However, there were four interlobular-sized muscular arteries containing intraluminal thrombi with organization without vasculitis suspecting renal artery thrombosis. On day 5, patient had worsening of her abdominal pain and developed severe lactic acidosis. A CT scan of abdomen/pelvis with contrast done showed bowel perforation and bilateral renal artery thrombosis. Extensive pancreatic necrosis was noted on laparotomy and therefore she underwent necrosectomy of the pancreas. Heparin was started 2 days post operatively for renal artery thrombosis. Echocardiogram did not show any evidence of thrombus or vegetation. Doppler studies showed deep vein thrombosis of the lower extremities and internal jugular vein thrombosis. Work up for vasculitis and hypercoagulability were negative. Final impression was acute kidney injury secondary to bilateral renal artery thrombosis from acute necrotizing pancreatitis. There is no sign of renal recovery at 6 months of follow up and patient continues to be on hemodialysis. Virchow’s triad, hypercoagulable state of acute necrotizing pancreatitis and anatomic proximity of renal arteries to inflamed pancreas were the postulated pathophysiology in our patient. The true incidence of renal artery thrombosis is difficult to estimate because of the vague clinical presentation and rarity of the condition. Common lab findings include leukocytosis, proteinuria, hematuria, elevated LDH with minimal or no rise in serum aminotransferase. Elevated LDH usually indicates infarction of the kidneys. The risks and benefits of the use of intravenous contrast with concomitant renal failure have to be carefully assessed as CT scan with contrast can confirm renal artery thrombosis. Anticoagulation with heparin should be initiated for renal artery thrombosis if revascularization by endovascular revascularization or surgical techniques is not possible.
PGY-3 SUBMISSIONS

THE RUM RHYTHM

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Introduction: Alcohol is known to cause conduction disturbances and atrial fibrillation. We describe a case of complete heart block secondary to acute alcohol intoxication.

Case: A 50-year-old man with a history of hypertension and chronic alcoholism was partying with his family members binge drinking alcohol. He suddenly complained of chest discomfort and became unresponsive a few minutes later. The patient was immediately brought to the emergency room. On examination he was unresponsive and pulse was present but at a very low rate of 6 beats per minute. Chest compressions were initiated and atropine was used without any improvement. Cardiac monitor showed complete heart block with ventricular escape rhythm at a very low rate. 1 mg of Epinephrine was given and the heart rate improved to 40-50/min and the patient started to wake up. However one minute later he again became unresponsive and attempts to do transcutaneous pacing were unsuccessful. Glucagon was administered but no response was seen. Epinephrine was repeated and he responded again with increase in heart rate and mental status. Dopamine drip was initiated after multiple doses of epinephrine were tried without sustained response. The patient was taken for temporary pacemaker placement emergently. Temporary transvenous pacer was placed and coronary angiogram was performed which showed angiographically clean coronaries. His urine toxicology screen was positive for THC and benzodiazepines. His blood alcohol level was 370 mg/dl. His renal function, electrolytes including potassium were within normal limits. He did not use beta blockers or calcium blockers. His electrocardiogram (ECG) performed 3 years before admission showed bifasicular block. By day 2 of hospitalization the patient started to have his intrinsic heart rhythm without need of pace maker. Later temporary pacemaker was removed and the patient was stable.

Discussion: This patient had transient complete heart block with spontaneous return of intrinsic rhythm. Ischemia was excluded by angiogram, AV nodal blocker overdose was ruled out by history. Toxicology screen was positive for THC and benzodiazepines but a thorough literature search did not reveal any cases of complete heart block attributable to THC or benzodiazepines. Alcohol is known to cause conduction abnormalities, atrial fibrillation, delay in AV conduction, Mobitz II type AV block and arrhythmias, however complete heart block as a result of alcohol intoxication is a rare occurrence. Given that he had a complete heart block when the blood alcohol level was high with return of intrinsic rhythm after alcohol was metabolized, we attribute this patient’s transient complete heart block to alcohol.

Conclusion: Although alcohol is commonly known to cause atrial fibrillation it is important to be aware of other severe effects of alcohol like complete heart block. This case underlines yet another rare adverse effect of binge drinking.
WHAT WAS THOUGHT TO BE BILIARY COLIC

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Irbaz Bin Riaz MD, Khaled Hamed MBBS, Iyad Mansour MBBS

This is a case of a 68 year old female with a past medical history of type two diabetes mellitus and osteoarthritis involving her spine. She presented with right upper quadrant abdominal pain that radiated to the right flank and chills. Over the three days prior to admission, her pain had become progressively worse and was associated with nausea and vomiting. She was therefore admitted for further evaluation. On physical exam her abdomen was soft and lax with minimal costophrenic angle and right upper quadrant tenderness. The differential diagnosis at that time was biliary or renal colic. Ultrasound of the abdomen showed no abnormalities. Laboratory studies revealed a leukocyte count of 10.4 with 83% neutrophils and a left shift. Hemoglobin was 11.2 and platelets were normal. ESR was elevated at 96 and her kidney and liver function tests were within normal limits, except for alkaline phosphatase of 178. Her blood and urine cultures showed no growth. Upon further questioning the patient reported increasing back pain over the last 6 weeks. Given her history of spinal osteoarthritis, the pain had been treated conservatively with analgesics. A more detailed musculoskeletal exam revealed that she had tenderness at the level of the lower thoracic spine. MRI of the spine was suggestive of osteomyelitis and discitis at the level of the lower two thoracic spines. This was proven by bone biopsy and tissue culture that showed Staphylococcus aureus. The patient was treated with IV Oxacillin for 8 weeks with improvement in her symptoms. Her echocardiogram was negative for vegetations. Spontaneous vertebral osteomyelitis affects mainly older patients with underlying chronic medical conditions, such as diabetes mellitus, and patients who are immunosuppressed. Hematogenous spread is the most common route of infection. The segmental arteries that supply the vertebrae bifurcate and supply two contiguous vertebrae; therefore infection usually occurs in two vertebral bodies and their intervertebral disc. This may cause pain that radiates to the flank or abdomen, especially if the epidural space is involved. Spinal pain is usually insidious and worsens over several weeks. Fever is an inconsistent finding. A review done in 1979 showed that fever occurred in only 52% of patients. A lack of fever combined with attributing the pain to other causes often results in a delayed diagnosis. For example, in a report of ten patients with spontaneous vertebral osteomyelitis, all had been misdiagnosed initially, and half had undergone unnecessary surgery. Blood cultures are positive in only 50-70% of patients. MRI is the most sensitive technique to detect vertebral osteomyelitis. Bone biopsy remains the gold standard test to confirm the diagnosis and to obtain tissue samples for cultures and sensitivities to ultimately guide treatment.

COLLAGENOUS COLITIS AND INFLAMMATORY BOWEL DISEASE: IS IT A COMPLICATION OR SPECTRUM OF THE SAME DISEASE?

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Collagenous colitis is an inflammatory mucosal disorder that results in chronic watery diarrhea without bleeding. It is characterized by a thick subepithelial collagen band and increased lymphocytes in the
lamina propria of an otherwise endoscopically normal-appearing colon. It is generally benign disease, with a course that is characterized by alternating spontaneous relapses and remissions. There have been cases reported in literature in which microscopic colitis preceded Crohn’s disease and ulcerative colitis. It is unclear to date if these cases are only coincidental or represent progression of disease. We report one patient who was diagnosed with collagenous colitis 2 years ago. His prior treatment included budesonide and cholestyramine intermittently. The patient presented with severe abdominal pain, weight loss, anorexia, and bloody diarrhea. Colonoscopy showed mucosal changes suggestive of ulcerative colitis; diagnosis was confirmed with biopsy interpretation. Stool studies were negative. The patient was started on intravenous solu-medrol. His symptoms improved for a few days but then again progressed to severe bloody diarrhea with abdominal pain. A flexible sigmoidoscopy was performed revealing persistent inflammatory mucosal changes. Biopsies also showed cytomegalovirus in the background of ulcerative colitis. Intravenous gancyclovir therapy was added to the medical regimen. Patient’s symptoms resolved within a week and the patient was discharged home on valgancyclovir and prednisone. We report an additional case report, added to the previous six, which augments the theory that collagenous colitis could be a part of the spectrum of inflammatory bowel disease.

**THE HIDDEN ENDEMIC**

**Hammam Alquadan MBBS, UACOM at South Campus, PGY-3**

Bujji Ainapurapu MD

Introduction: There is a wide differential diagnosis for (HIV) patients presenting with Pneumonia like symptoms, although Pneumocystis pneumonia (PJP) remains a common disease in such a group, an alternate infections in certain endemic location should always be considered. Case: 35 year old gentleman with a history of HIV on HAART therapy and Bactrim Prophylaxis, presented with a complaint of productive cough and shortness of breath for one week, Symptoms gradually worsened to the point that he was short of breath at rest, and unable to talk. Patient admitted to fever, chills and night sweats. He denied sick contacts or recent travel he also reported a weight loss of 24 lb over the past six months. On examination patient was afebrile and appeared in severe distress with Tachypnea, tachycardia and hypotension, Lung exam was remarkable for absent air entry at the right side and coarse crackles at the left lung, with the rest of his exam being unremarkable. Initial workup showed a WBC of 23.900/uL with 20% bands, Venous blood gas was PH 7.35; Pco2 of 40, PO2 51, So2 was 80%. LDH: 201IU/L, and CD4 count was 54/uL, the rest of his labs were unremarkable. Chest X-Ray showed bilateral interstitial infiltrate with right pneumothorax. CT Chest showed confluent areas of consolidation with nodular opacifications with cystic and cavitory components. A Chest tube was placed in the ED with immediate relief, and patient was admitted under the diagnosis of PJP that was treated with Bactrim. Bronchoscopy with bronchoalveolar lavage demonstrated spherules consistent with Coccidiodomycosis with negative stain for PJP. Patient was discharged after 2 days with Oral Voriconazole and prophylaxis dose of Bactrim and appropriate follow up was arranged. Discussion: Coccidioidomycosis has a wide spectrum of presentations from asymptomatic infection to an overwhelming pulmonary infection in immunocompromised individuals with mortality rate that reaches 85%. It presents as a diffuse pulmonary reticulonodular infiltrates, this pattern can resemble PJP. PJP and Coccidioidomycosis can coexist, and the negative serology for Coccidioidomycosis doesn’t rule out the disease. This affects the
treatment plan of whether or not to add steroids. All HIV patients with Coccidiodomycosis should be treated, in disseminated disease starting with Amphotericin B and a triazole, with Amphotericin discontinued later on, institution of effective antiretroviral therapy also plays a key role in management. There is no data to support the use of a prophylaxis regimen for HIV patients in Coccidiodomycosis endemic areas. Conclusion: It is important to have a high suspicion for Coccidiodomycosis in HIV patients living in endemic areas, who present with Pneumocystis pneumonia like symptoms, as the approach and treatment in each group will be critically different, at the same time keeping in mind both diseases can coexist in such patients.

RECURRENT ARTERIAL AND VENOUS THROMBOSIS IN A PATIENT WITH LESCH-NYHAM SYNDROME ON TRIPLE ANTITHROMBOTIC THERAPY

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Riaz IB, Husnain M, Bilal J

INTRODUCTION:

Hypoxanthine-guanine phosphoribosyl transferase (HPRT) deficiency is an X-linked defect of purine metabolism. There are few reported cases which suggest association of HPRT deficiency and hypercoagulability. We report a case of Lesh-Nyhan syndrome with recurrent arterial and venous thrombosis despite being on triple antithrombotic therapy.

CASE PRESENTATION:

31 year-old male, known case of Lesch Nyhan Syndrome, with recurrent severe gouty arthritis and end stage renal disease (ESRD) presented with a Non-ST elevation myocardial infarction (NSTEMI), past medical history significant for recurrent pulmonary embolism, ST elevation myocardial infarction (STEMI) status post angioplasty 3 months ago. He has been on triple antithrombotic therapy (high dose aspirin, clopidogrel and warfarin with a therapeutic INR 2-3) since then. During this admission, he underwent a coronary angiography for his NSTEMI complicated by a code arrest. He was resuscitated and needed extracorporeal membrane oxygenation (ECMO) for 2 days. Later in the course he developed right lower extremity pain. A deep vein thrombosis (DVT) wasn’t high on suspicion as the patient was already on warfarin with a therapeutic INR of 2.2. Unexpectedly, the duplex ultrasound of the lower extremity showed an extensive new DVT in the right lower extremity that wasn’t present on the duplex 3 months ago. An extensive thrombophilia work up done several times over the last year was unremarkable. Due to the moderate pulmonary hypertension from the previous pulmonary embolism and complicating cardiac medical issues the decision was made to place an inferior vena cava (IVC) filter and keep the patient on warfarin with a target INR of 3-4. Other anticoagulants couldn’t be used due to the end stage renal disease.

DISCUSSION:

Recurrent and/or unprovoked thromboembolism has been reported in the literature in association with Lesch-Nyhan Syndrome(1,2). Whether this association is related to endothelial dysfunction and prostacyclin deficiency or anticoagulation system defect is not clear yet. As with most cases of recurrent
thromboemolism, interruption of anticoagulation for a few weeks for a comprehensive thrombophilia workup is not an option due to the risk of fatal accidents which makes the research in this area more complicated.

CONCLUSIONS:

Patients of Lesch-Nyhan Syndrome might be at a higher risk for both arterial and venous thromboembolism than the general population. More data is needed to show whether a comprehensive thrombophilia testing and prophylactic antithrombotic therapy is beneficial before the first thromboembolic event occurs.

References:


AMITRIPTYLINE INDUCED AGRANULOCYTOSIS WITH BONE MARROW CONFIRMATION

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Introduction Drug induced agranulocytosis is a life threatening condition caused by an idiosyncratic adverse drug reaction often manifesting as a severe neutropenia with an ANC less than 0.5 x 10/l, leaving patients at serious risk of infection and death. There are a myriad of drugs associated with this reaction and the list is ever growing. We report the second case of Amitriptyline induced agranulocytosis and the first with marrow biopsy confirmation. Case Description A 62 year old Caucasian female with Multiple Myeloma receiving weekly chemotherapy with cyclophosphamide, dexamethasone, and Bortezomib (CYBORD) was admitted for neutropenic fever (ANC of 0.00). Prior to admission she had no previous adverse effects or cytopenias associated with her treatment. Notably, she had been started on amitriptyline at an outside hospital 4 weeks prior for presumed peripheral neuropathy. Differential diagnoses considered included cytotoxic effect of chemotherapy, idiosyncratic drug reaction, and progression of Myeloma. Bone marrow biopsy was obtained and showed a relatively normocellular bone marrow, minimal to no dysplasia, and relative eosinophilia. Importantly, all marrow components were present except for neutrophils, and only 3% plasma cells were seen. Amitriptyline was held on admission and she was supported with broad spectrum antibiotics and Neupogen. Her counts started to recover within 3 days and she was discharged home on day 5 without event. Discussion There have been no reports of agranulocytosis associated with CYBORD or any single CYBORD. (Reeder, 2009 #84) Agranulocytosis thought to be a result of Amitriptyline has been reported once in a case report in 1963. (Gault, 1963 #92) however that case failed to report consistent bone marrow findings. Cytotoxic effects of chemotherapy can cause cytopenias as a direct consequence of their mechanism of action; in this setting multiple cell lines would be affected. This, however, was a case of agranulocytosis secondary to
idiosyncratic drug reaction, as evidenced by disproportionate neutrophil depletion on bone marrow biopsy and resolution, without recurrence, upon withdrawal of the offending agent. Given the ever growing list of medications and the inherent difficulty in diagnosing such idiosyncratic drug reactions, clinicians must have a low threshold to suspect an adverse drug reaction in order to avoid mortality as a result of severe neutropenia and its sequelae.

**CERVICAL CANCER WITH METASTASIS TO BREAST**

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Nedda Alemi MD, Susanna Tan MD, Waqas Arslan MD

Cervical Cancer with Metastasis to Breast Mariya Balon MD, Nedda Alemi MD, Susanna Tan MD, Waqas Arslan MD

Introduction: Metastatic involvement of the breast is very rare and hence the first thought usually involves a diagnosis of primary breast cancer. Findings of a breast mass in combination with axillary lymphadenopathy in a patient with a history of cancer of unknown primary have a high risk of being misdiagnosed. Case presentation: 48-year-old female with no significant past medical history was evaluated for abdominal pain. She was found to have extensive lymphadenopathy surrounding the aorta and causing hydroureteronephrosis. Work-up of lymphadenopathy included chest radiograph, CT scan of abdomen and pelvis, endoscopy, colonoscopy, MRI, PET scan, none of which identified a primary tumor. Pelvic exam, endocervical and endometrial biopsies were also normal. Biopsy of an inguinal lymph node showed metastatic carcinoma of unknown origin. She had bilateral ureteral stents placed to relieve obstruction and was discharged home with oncology follow-up. She presented two months later with worsening abdominal pain. Physical examination showed tender enlarged lymph nodes in the right inguinal, right subclavian, right anterior and posterior cervical regions. She also had a palpable left breast lump at the 6 o’clock position with adjacent axillary lymphadenopathy. Mammogram was done and surprisingly did not reveal any abnormalities. Breast ultrasound showed an irregular 2 cm mass at the 6 o’clock position with increased blood flow. The mass extended towards the nipple with surrounding edema that was attributed to supraclavicular and axillary lymphadenopathy rather than an inflammatory breast cancer. Axillary lymph node biopsy showed poorly differentiated adenocarcinoma. Breast cancer was thought to be the most likely primary. Biopsy from the lymph node was sent for molecular cancer profiling and it showed, with 90% confidence, that it was cervical cancer; breast primary was excluded with 95% confidence. Breast biopsy done later did show metastatic, poorly differentiated carcinoma consistent with cervical primary. Discussion: This case shows us the importance of a complete work-up while trying to identify the primary site when dealing with cancer of unknown origin. In this case, the breast mass with axillary lymphadenopathy could easily have been mistaken for breast primary rather than metastasis. Molecular tumor profiling helped to avoid this mistake. Molecular tumor profiling is a new diagnostic technique that enables prediction of a tissue of tumor origin by detecting site-specific gene expression profiles. This test should be included in the diagnostic evaluation of patients with cancers of unknown primary when clinical and standard pathologic evaluations are not strongly suggestive of a primary site.
LEMIERRE’S SYNDROME

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Lemierre’s Syndrome, or jugular vein suppurative thrombophlebitis, is a condition frequently preceded by pharyngitis, usually in association with tonsilar or peritonsillar involvement. The carotid sheath vessels have infectious involvement with bacteremia. These patients will typically present with antecedent pharyngitis, septic emboli, and persistent fevers despite antibiotic therapy. In my case, the patient was a 26 year old male who was treated with penicillin for a suspected sore throat thought to be a pharyngitis. He felt better for about 4 days. On day 5, he started to have right neck pain, right ear pain, and pain under the right rib cage. His initial chest x-ray showed moderate parahilar and peribronchial interstitial infiltrates. Initial CT neck was ordered and was negative. He was admitted for further evaluation. He had blood cultures and sputum cultures ordered. He was treated for right sided pneumonia with possible sepsis with vaco and zosyn. He had questionable pulmonary nodules so a cocci serology was ordered and patient was placed on TB precautions. While the results of the cultures were pending and the patient being on broad-spectrum antibiotics, the patient kept having persistent fevers three days into treatment. He also complained persistently of right pleuritic pain so a question of PE arose. He had a CT chest which did showed numerous bilateral pulmonary cavitary nodules are identified, the largest measuring 1.7 cm, consistent with an infectious process. He had a pulmonary consult, who subsequently did a bronchoscopy and did a bronchial alveolar leavage. There was also concern of endocarditis, thus an echo was ordered, which was also negative. He still kept having fevers and right neck pain so we did a bedside ultrasound of the right side of the neck. We thought we saw something abnormal so a ultrasound of the right upper extremities was done which did not reveal anything.

THE INCESSANT SECRETORY STORM

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Neuroendocrine tumors have a myriad of clinical symptoms obfuscating the diagnosis and causing significant delay in treatment. Large Cell Neuroendocrine Carcinoma (LCNEC) is an even rarer subset of malignancy particular to the lungs but with the paucity of trials it is still unclear which treatments, if any, may be effective. We present once such case of LCNEC. 67-year-old Caucasian male, smoker with HTN was admitted and evaluated for 6 weeks of large volume diarrhea and hypotension. A work up for infectious diarrhea was negative. However review of his medical records revealed a previous CT finding of an incidental mediastinal mass which had never been evaluated. A trans bronchial biopsy was obtained with resultant pathology demonstrating tissue consistent with LCNEC. The remainder of his hospitalization was complicated by recurrent refractory Atrial Fibrillation, diarrhea with poor response to Loperimide and Octreotide and wheezing that required aggressive therapy. His symptoms were likely from the bioactive amines from this tumor. This patient demonstrated clinical evidence of Carcinoid Syndrome which is unusual given the location of the mass. Eventually patient was stabilized and ready for discharge home, to follow up with the oncology service for chemotherapy. This case highlights a very rare malignancy (LCNEC) which belongs to a neuroendocrine tumor family posing a therapeutic challenge. This case typifies Bronchial Carcinoid Variant Syndrome, which as the name implies,
demonstrates the flushing, wheezing, diarrhea and other symptoms, however in this unusual location. Given the rarity of this disease process, there is still much debate on what is the optimal treatment regimen. Currently many clinicians treat this variant as aggressive SCLC; however evidence shows very poor outcomes regardless of surgery or chemotherapy choice. Clinicians may need to help patient and family decide that due to poor outcomes with current therapies, supportive care should be the front-runner in terms of therapy.

UNIVERSITY OF ARIZONA CANCER CENTER HIGH RISK BREAST CANCER GENETICS CLINIC PERSPECTIVE ON UTILIZATION OF A CUSTOM NEXT GENERATION SEQUENCING PANEL

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Corina Mauss BS 2, Joanne Jeter MD 2, 3, Christina Laukaitis, MD, PhD 1, 2

Background:

One in 8 women will develop breast cancer over the course of her lifetime, making it the most common cancer affecting women and the 2nd leading cause of cancer death. This study will utilize a custom next generation sequencing (NGS) panel to determine if 88 genes of interest thought to play a role in breast cancer susceptibility are found in women with strong family histories of breast cancer enrolled in the University of Arizona Cancer Center High Risk Breast Cancer Genetics Clinic (UACC BCGC).

Methods:

The UACC BCGC has a University of Arizona IRB-approved registry for epidemiologic and medical record information. 182 subjects are currently enrolled. Genomic DNA was captured from 47 BCGC samples using Agilent Technologies HaloPlex protocols to enable analysis of 46 novel custom genes of interest in addition to the University of Washington developed BROCA panel. 88 total genes were sequenced. Captured library DNA was denatured and subjected to cluster amplification on a paired end flow cell in paired-end libraries with ~200 bp insert size, and sequenced on an Illumina HiSeq2000 instrument with 100 bp read lengths. Agilent Technologies SureCall software was utilized for raw sequence alignment, mutation impact analysis, visualization, and categorization of ranked genetic variants.

Results:

19,661 variants were found across 5 SureCall categories in 47 women. 42/47 women have category I mutations that involve truncation of a protein that are pathogenic or likely pathogenic based on the Single Nucleotide Polymorphism Database (dbSNP) or category II mutations that are missense mutations according to dbSNP. 40% (17/42) women have personal histories significant for malignancy (1% gastrointestinal, 12% breast, and 14% gynecologic). Further individual analysis of these 69 category I and 2,644 category II variants will be performed prior to delineating specific mutations to patients. These mutations will be verified in a Clia laboratory prior to dissemination to UACC BCGC patients for clinical use.
Discussion:

Women with strong personal and family histories of breast cancer are routinely tested for BRCA1/2, but other gene testing is not generally available or offered. Development of a custom NGS panel allows us to analyze a greater number of genes thought to play a role in breast cancer susceptibility. Utilizing genetic information increases the chance of identifying cancer early and at a more treatable non-invasive stage by allowing intensive screening, recommending preventive medication, and/or risk reducing surgery to decrease the chance of developing invasive breast cancer in high risk individuals. Population frequency of variation is a major thrust of this project and future studies will probe penetrance. Long terms goals include expanding our recruitment of patients and their family members to determine if these heritable mutations correlate with development of other malignancy through prospective analysis.

NEUROAXIAL INSTRUMENTATION AND INFECTION RISK IN THE ERA OF STERILE TECHNIQUE

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Since the development in the late 19th century of the various measures that today comprise 'aseptic technique,' great strides have been made in reduction of post-surgical infection. Epidural abscess, particularly intracranial, is today quite rare following any neuraxial instrumentation. We report a case of a 40 year old male who underwent lumbar epidural steroid and Sarapin injection for chronic unremitting back pain from prior traumatic compression fractures. There was no mention of complication or breach of sterile technique. He presented to his primary care doctor three weeks later complaining of gradually worsening headache with right eye pain, nausea, vomiting, and subjective fevers. He was admitted to the hospital, found to have right parieto-occipital epidural fluid collection, and taken to the operating room for drainage of the collection. No other focus of infection was found. Culture of the fluid grew Streptococcus mitis, and he was discharged with six weeks of intravenous antibiotic and had an unremarkable post-hospital discharge course. Review of literature regarding CNS infection in patients undergoing regional anesthesia or diagnostic lumbar puncture highlights some of the challenges of diagnosing epidural abscess in general. Compared to patients who develop fulminant meningitis post procedurally, those who are diagnosed with epidural abscess often follow a more indolent course and present with more vague symptoms. Our case is unique in that despite the absence of sinus or oropharyngeal source, our patient's cultures grew Streptococcus mitis. The relative dearth of case reports implicating this organism may be due as much to the difficulty in drawing causal relationships as to differences in definitions and reporting standards. There are studies to suggest that alcohol use can be associated with increased susceptibility to infection. Similarly poor oral hygiene and chronic periodontal disease may also be related to some degree of immune dysfunction, either directly or through shared risk factors. The presence of such host factors, particularly given the lack of studies showing long term benefit of epidural injections for chronic pain, should prompt a re-consideration of the risk to benefit profile. Intracranial epidural abscess as an entity, while a rare complication of epidural injections, should be included in pre-procedural risk-benefit discussions as well as differential diagnoses in patients presenting with any neurological deficit and a history of recent neuraxial instrumentation.

SIMULATION IN INTERNAL MEDICINE EDUCATION: A META-ANALYSIS
Heather Fields MD, Mayo Clinic Arizona, PGY-3

Zhen Wang, Noor Asi, M Hassan Murad, Natalya Azadeh, Darko Vucicevic, Janis Blair, Farouk Mookadam

Introduction: Simulation has been extensively studied in procedural specialties. In the internal medicine curriculum, the role of simulation education is less well defined, but thought to be beneficial. Our aim was to perform a systematic review of the published literature evaluating the use of simulation in internal medicine topics. Methods: A systematic review of PUBMED/EMBASE/MEDLINE, January 1st 1988 - July 31st 2012. Reference lists were scanned and new studies identified. Meta-analysis was performed using a random effects model and the outcome measure was the standardized mean difference (SMD). Inclusion: Eligible studies evaluated the use of mannequins or live patient simulated scenarios for teaching or assessing skills in internal medicine topics: interviewing skills, physical exam, and procedural skills. Results: 27 studies met eligibility criteria and focused on interviewing skills, physical exam, and procedural skills. The use of simulation was associated with improvement in learners’ practical skills (p < 0.001, SMD 2.001, CI 1.37-2.64) and written exam skills (p < 0.001, SMD 2.69, CI 1.53-3.86). Procedural studies evaluating complication rates showed a significant decrease in complications among those subjects that used simulation (OR 0.47, CI 0.27-0.85, p = 0.01). The quality of the studies was fair. Conclusions: Moderate quality evidence suggests that simulation is an effective tool for learning various internal medicine skills.

**NSAID INDUCED DUODENAL STENOSIS - A PINHOLE OF EVIDENCE**

Rene Franco Jr. MD, UACOM at South Campus, PGY-3

Juxiang Huang MD, Imam Imam MD, Raed Sukerji MD, Rosemary Browne MD

NSAID induced Duodenal Stenosis - A Pinhole of Evidence Rene Franco Jr. MD, Jennifer Huang MD, Imam Imam MD, Raed Sukerji MD, Rosemary Browne MD University of Arizona - South Campus

Introduction: Non-steroidal anti-inflammatory drugs are one of the most commonly prescribed medications and they are often used for long periods of time. Despite our thoughts of their "benign" nature, their use does not come without risk. NSAIDS are known to be associated with various forms of gastrointestinal injuries including small bowel ulceration and gastric outlet obstruction which tend to occur with their chronic use. Case: A 30 year old female presented to the ED with complaints of a 6 month history of nausea/vomiting, 60 lb weight loss (unintentional) and abdominal pain in the setting of chronic ibuprofen therapy which she was using for her headaches. The only other significant PMH includes asthma and C-Section 5 months ago. Home medications included Pro-air HFA as well as the OTC Ibuprofen which she used up to 6 times a day. Vomiting was described as constant in nature, occurring three times a day on a daily basis, green/bilious, without evidence of blood, and was not associated with oral intake. Although at time of her presentation, the patient did notice a coffee ground color in her emesis and became more concerned for bleeding. On arrival, she appeared malnourished and hypovolemic due to dehydration. Labs obtained in the ED revealed hypochloremic metabolic alkalosis and microcytosis. An Abdominal Xray did not reveal any evidence of bowel obstruction and a CT Scan with contrast revealed a mildly distended stomach and descending duodenum without evidence of free air, fluid collection, or lymphadenopathy. Initial EGD showed a partially obstructing ulcer at the pylorus with large amounts of food within her duodenum. Repeat EGD did visualize multiple duodenal ulcers with aquired duodenal
stenosis with a small pinhole opening in the 3rd portion of the duodenum. Patient was subsequently taken to operating room for a laparoscopic Roux-en-Y duodenojejunostomy with placement of a feeding jejunostomy tube and decompressive gastrostomy tube. Discussion: NSAID induced duodenal stenosis is a rare cause but clinically significant finding. Ongoing use of enteric coated forms of NSAIDs give both clinicians and patients a false sense of security about their "benign" nature. Reports in previous literature have identified NSAID-induced fibrous daphrags within the duodenum which lead to significant stenosis requiring endoscopic or surgical intervention. An increasing awareness for NSAID induced duodenal stenosis and this clinical phenomenon should prompt an endoscopic diagnosis to be made quickly and allow cessation of the offending agent and appropriate management.

**EVALUATING THE PROGRESSION OF GASTRIC ADENOCARCINOMA**

Lisa Fujima DO, Verde Valley Medical Center, PGY-3

A 70 year old woman presented with anemia on a routine visit and was subsequently found to have a positive hemoccult. She subsequently underwent an upper endoscopy and colonoscopy and was diagnosed with gastric cancer with metastatic disease to the ileocecal valve, liver, lungs, bone, and peritoneal cavity in May of 2011. Since diagnosis, she has continued chemotherapy with irinotecan and cisplatin. In January of 2013, for re-staging purposes, she had a CT of the Chest, Abdomen, and Pelvis, which showed mild wall thickening of the ascending colon, with no other evidence or findings of metastatic disease. Though CT scan did not show signs of carcinoma or metastatic disease, she was due for a repeat EGD and colonoscopy in March of 2013. On observation, there were no obvious signs of carcinoma or metastatic disease. And endoscopy report showed antral gastritis, a gastric polyp, evidence of diverticular disease, and multiple areas of colitis in the colon. However, biopsies were taken from the esophagus, stomach, ileocecal valve, ascending and transverse colon for re-evaluation. Surprisingly, the pathology report from the biopsies showed that the stomach had poorly differentiated carcinoma, consistent with a gastric adenocarcinoma and biopsies from the colon showed metastatic carcinoma. The disease had progressed and was resistant to chemotherapy. In the treatment of gastric adenocarcinoma, even after chemotherapy and what looked like a benign EGD/colonoscopy, biopsies are necessary to re-stage and evaluate disease progression. The standard of care is to re-evaluate patients with gastric adenocarcinoma on a yearly basis.

**APPEARANCES CAN BE DECEIVING**

Jennifer Hill MD, UACOM at University Campus, PGY-3

Introduction: Rowell’s syndrome (RS) is a rare clinical entity consisting of systemic lupus erythematosus (SLE) in association with erythema multiforme (EM)-like lesions and characteristic immunologic findings. First described in 1963 by Rowell et al., there have been approximately 70 cases reported in the literature. Case Description: A 43-year-old Hispanic female with a 15-year history of SLE presented with the acute onset of a widespread pruritic, erythematous skin rash and painful oral ulcerations associated with fatigue and diffuse arthralgias. Physical examination demonstrated erythematous, coalescing macules and papules, some resembling target lesions, on the trunk, arms, and face. Malar erythema was present. Crusted, bleeding erosions were present on the lips and oral mucosa. Complete blood count and comprehensive metabolic panel were unremarkable. C-reactive protein and erythrocyte
sedimentation were elevated at 21 µg/ml and 66 mm/hour, respectively. Autoimmune screening revealed antinuclear antibody with a speckled pattern (1:80), elevated anti-dsDNA antibody, and positive anti-La/SS-B and Anti-Ro/SS-A antibodies. Rheumatoid factor (RF) was negative. Complement levels were reduced. Hepatitis B and C, HIV, cytomegalovirus virus, herpes simplex virus (HSV), and Epstein-Barr virus serologies were negative. Mycoplasma pneumonia antibodies were not detected. Skin biopsy histopathology was consistent with erythema multiforme, with no immune deposition of immunoglobulins or complement on direct immunofluorescence. Clinical and histopathological findings were consistent with RS. She was treated with prednisone 60 mg twice daily and hydroxychloroquine 200 mg daily with slow resolution of cutaneous lesions. Discussion: RS represents a unique clinical association of SLE with EM-like lesions and distinct immunological findings characterized by speckled pattern of ANA antibodies, positive rheumatoid factor, and antibodies to anti-Ro/SS-A or anti-La/SS-B. Misdiagnosis is common given rarity and a broad differential diagnosis including autoimmune bullous diseases, drug reactions, skin manifestations of SLE, and EM. Importantly, classical EM is associated with triggering factors such as infections or drugs and is never associated with specific autoimmune serologic abnormalities. Subacute cutaneous LE (SCLE) lesions may resemble EM clinically and histologically. Therefore, direct immunofluorescence is proposed as a diagnostic criterion for differentiation. Junctional deposition of immunoglobulins and complement are only seen in SCLE. Diagnosis of RS is clinical and requires 3 major and 1 minor criteria. Major criteria include: LE, EM-like lesions, and speckled pattern of ANA. Minor criteria include: chilblains, positive anti-La/SS-B or anti-Ro/SS-A, and reactive RF. Treatment consists of high dose corticosteroids and anti-malarials. Accurate diagnosis is important as the disorder often has a chronic, recurrent course and relapses are common. Conclusion: RS should be considered in all patients presenting with LE and EM-like lesions in the absence of precipitating factors. Familiarity with and early recognition of this syndrome are paramount for institution of appropriate therapy.

**CHOLESTEROL EMBOLI SYNDROME - AN UNUSUAL COMPLICATION OF CARDIAC CATHETERIZATION**

**Alexander Hu DO, Verde Valley Medical Center, PGY-3**

Cholesterol Emboli Syndrome - An Unusual Complication of Cardiac Catherization Alexander Hu, D.O. OGME-3, Verde Valley Medical Center, Cottonwood, AZ A 59 year old Caucasian male presented to the ED with complaints of a new progressive non pruritic lower extremity rash, blue tinged discoloration of his toes and diarrhea. Just two weeks earlier the patient was hospitalized for chest pain and after a full evaluation had successful multi lesional stents placed in the LAD. On this admission he was found to have acute renal failure with a new elevation in creatinine of 4.41 and a rash with the appearance of livedo reticularis. Considerations were mostly of vasculitic disease or atherembolic phenomenon. There are many inherent difficulties in diagnosing atherembolic disease because cholesterol embolism affects multiple organs and the disease progresses slowly and can mimic systemic vasculitis. The specific diagnosis can be made only by demonstrating the cholesterol crystals within vessels. Atheroembolic disease is typically observed after an invasive vascular procedure but can also develop spontaneously. The disease can present as sudden onset of acute renal failure or have a slower course with declining renal function over a period of several months. The kidney is usually involved due to the proximity of the renal arteries to the abdominal aorta where atheromatous plaques are more likely to dislodge and due
to the enormous amount of blood flow through the kidneys. Renal failure is often attributed to other conditions such as radio contrast nephropathy, volume depletion, acute tubular necrosis and drug induced interstitial nephritis. Cholesterol embolization at other sites can lead to cutaneous, musculoskeletal, gastrointestinal, neurologic, and ophthalmic manifestations. The classic cutaneous lesions are livedo reticularis—a purplish rash over the lower extremities and abdominal wall, nail bed infarcts, and purple colored toes. Other symptoms such as abdominal pain, nausea, vomiting, diarrhea, blood loss, myalgias and arthralgias that occur from embolization can add to the confusion. This patient’s skin pathology findings were consistent with embolic cholesterol clefs in the deep dermal arterioles. Autoimmune panel results have been negative for evidence of vasculitis. The patient continues to experience difficulty with severe musculoskeletal pain and lower extremity edema while recovering much of his renal function. Cholesterol emboli syndrome should be part of the differential diagnosis in pts with medical history that includes an invasive vascular procedure regardless of time interval between surgical intervention and time of onset of symptoms. This syndrome remains under diagnosed due to the many manifestations such as deteriorating renal function, cutaneous rash, musculoskeletal, gastrointestinal, neurologic and ophthalmic complications.

**PROPOFOL INDUCED RHABDOMYOLYSIS**

**Imam Imam MD, UACOM at South Campus, PGY-3**

Rene Franco, Jr, MD

Propofol Induced Rhabdomyolysis  INTRODUCTION: Rhabdomyolysis is characterized by muscle necrosis and release of intracellular material into the circulation. Its sequelae include acute kidney injury and related metabolic complications. The common causes are metabolic, thermal extremes, toxins, infections and drugs. Here we present a rare but potentially fatal cause of rhabdomyolysis that occurred with propofol. CASE: A 48-year-old male with past medical history of severe depression presented to our ED with altered mental status. EMS found the patient profoundly confused leading to endotracheal intubation on the scene. On physical exam, initial vitals were stable and GCS was 6 with non-focal neurologic exam. Labs revealed normal CBC, CMP, lactate and negative urine toxicology except for benzodiazepines which patient has received for ETT placement. Patient was subsequently admitted to ICU and started on propofol for sedation at rate of 1.2 mg/kg per hour. Later on the first night, the patient got agitated and propofol was increased to 6 mg/kg per hour. His sepsis workup including blood cultures, urine cultures and lumbar puncture remained negative. However as the patient was still altered and agitated, he remained intubated for the next 2 days. Neurology was consulted for agitation and altered mental status. An MRI of the brain and EEG were normal. On hospital day 3, patient became anuric. Repeat labs revealed a creatinine of 4.1 mg/L and a CPK of 1547 IU/L. Toxicology was consulted and a comprehensive toxicology panel was done which was negative. On review of medications, accumulated propofol was found to be the cause of his symptoms and attributed to propofol infusion syndrome (PRIS). Propofol was discontinued and midazolam was initiated instead. CPK peaked at 10098 IU/L on day 4. The patient was extubated on Day 6. Kidney function improved by discharge with a creatinine of 1.2 mg/L. Rhabdomyolysis developed on the third day of the patient’s propofol infusion. The muscle destruction occurred despite normal blood pressure, absence of convulsions, and normal serum levels of electrolytes. DISCUSSION: Propofol is an IV sedative-hypnotic...
agent for use in the induction and maintenance of anesthesia or sedation. PRIS is characterized by severe unexplained metabolic acidosis, rhabdomyolysis acute renal failure, hyperkalemia, arrhythmias, and cardiovascular collapse. Risk factors for developing PRIS include sepsis, severe cerebral injury, and high propofol doses. The exact pathophysiology of PRIS remains to be determined, impaired tissue metabolism caused by propofol infusion appears to be an important mechanism. The effect of propofol is dependent on dose and duration. Early recognition of this syndrome is important as discontinuation of propofol is critical in the management.

**BONE MARROW STARVATION: A RARE CASE OF SEVERE MALNUTRITION IN AN ADULT WITHOUT SYSTEMIC ILLNESS**

Kahroba Jahan MD, UACOM at University Campus, PGY-3

Alana Morales MD

**BONE MARROW STARVATION: A RARE CASE OF SEVERE MALNUTRITION IN AN ADULT WITHOUT SYSTEMIC ILLNESS**  Kahroba Jahan MD, Alana Morales MD University of Arizona Department of Medicine  INTRODUCTION - Gelatinous Bone Marrow Transformation (GBMT) is a rare disorder of unknown pathogenesis histologically characterized by hypoplasia of the hematopoietic lineages, atrophy of spinal adipocytes, and interstitial infiltration of gelatinous mucopolysaccharides. GBMT occurs almost exclusively in adults. It is most notably found in the setting of severe malnutrition associated with systemic illnesses, including cancer-related cachexia, end-stage AIDS, chronic alcoholism, and anorexia nervosa. This condition is often seen in developing countries and it is rarely noted in patients without an underlying debilitating systemic illness. CASE - We describe a 59 year old male who presented with significant weight loss, chronic poorly healing lower extremity ulcers, leg edema, and fatigue. He was cachectic appearing and found to have very low serum albumin and normocytic anemia requiring multiple blood transfusions. An MRI of the lumbar and thoracic spine was performed and showed irregularities of the bone marrow concerning for a diffuse infiltrative process such as metastasis or myeloma. A bone marrow biopsy did not indicate evidence of an underlying malignancy, however patchy areas of homogeneous, smooth stroma lacking hematopoietic cells were noted. These finding were most consistent with gelatinous transformation in the setting of severe malnutrition. DISCUSSION - Gelatinous marrow transformation of the bone marrow, historically known as “spinal serous atrophy” is a rare condition usually seen in severe, debilitating illnesses. The incidence of GBMT in self-induced malnutrition in Western developed countries is rare. Bone marrow biopsy remains the gold standard method for diagnosis, revealing hypoplasia of the hematopoietic cells and their replacement with gelatinous substances consisting of mucopolysaccharides and hyaluronic acids. All hematologic and morphological alterations disappear rapidly and completely after sufficient refeeding.

**UREA: AN UNCONVENTIONAL TREATMENT FOR AN UNCONVENTIONAL CASE**

Chris King MD, Mayo Clinic Arizona, PGY-3

Irvin M. Cohen

Urea: An unconventional treatment for an unconventional case. Chris S. King, Irvin M. Cohen Department of Internal Medicine, Mayo Clinic Arizona, Phoenix Arizona, 85054, USA. kingcs@mayo.edu

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Introduction: Finding the underlying cause of the syndrome of inappropriate antidiuretic hormone (SIADH) secretion can be difficult, with varying etiologies such as medications, pulmonary disease, central nervous system disturbances or malignancy. Acute treatment depends on the severity of associated symptoms but can include the imposition of draconian fluid restriction and the use of hypertonic saline for patients with seizures or acute mental status changes. The ultimate goal is to alleviate symptoms whilst striving to correct the underlying etiology. Unfortunately, austere fluid restriction and protracted use of antidiuretic hormone (ADH) receptor antagonists can often fail. In our institution, we have started to rediscover an older approach to treating SIADH induced hyponatremia using medical grade urea powder. Case: A 78 year old gentleman with significant idiopathic hyponatremia presented with fatigue, general weakness and pre-syncpe. He reported strict compliance with a fluid restriction goal of < 1000 mL of fluid intake per day. The patient was judged to be euvoletic during our clinical evaluation and was without orthostatic hypotension, evidence of lower extremity edema, or jugular venous distension. His urine osmolality was 421 mOsm/kg, blood osmolality was 256 mOsm/kg, and urine sodium was 73 mEq/L. An extensive cardiac, pulmonary and neurologic workup for his symptoms both at outside hospitals and our own institution was unrevealing. An underlying etiology for his hyponatremia also remained elusive. In order to treat the idiopathic SIADH we started the patient on medical grade urea powder at 30 grams per day. This was quite efficacious in returning his sodium level to the normal range. Our initial goal of sodium above 130 meq/L was easily achieved. Over a period of three weeks his hyponatremia and associated symptoms had resolved and serum sodium had corrected from 123 mEq/L to 142 mEq/L. The patient experienced no side effects and tolerated the treatment well. Discussion: The urea also had the advantage of being quite inexpensive. In this report we discuss the available literature with regard to efficacy, cost and side effects of urea compared with other treatment modalities in overcoming resistant SIADH. Medical grade urea is, in our opinion, an invaluable tool to have in your arsenal to alleviate symptomatic hyponatremia so that the search for root cause might continue.

MICROBIOLOGICAL YIELD OF BRONCHOALVEOLAR LAVAGE SPECIMENS FROM STEM CELL TRANSPLANT RECIPIENTS

Christine Klassen MD, Mayo Clinic Arizona, PGY-3
Sakata KK, Bollin KB, Grys TE, Slack JL, Wesselius LJ, Vikram HR

Background: Infectious complications are a major cause of morbidity and mortality in stem cell transplant (SCT) recipients. Diagnoses of pulmonary infections often require computed tomography (CT) and bronchoscopy with bronchoalveolar lavage (BAL) for diagnostic evaluation. There is a paucity of data on the yield of BAL in SCT recipients. Methods: This is a descriptive, retrospective cohort study in which all SCT recipients who underwent a BAL at our institution between Jan 2009 and Aug 2012 were included. Demographic information, antimicrobials at the time of BAL, radiographic studies, detailed microbiologic, and serologic information were collected and analyzed. Results: A total of 102 patients underwent 137 BAL procedures. 74% had an allogeneic SCT and 26% had an autologous SCT. The median time between allogeneic and autologous SCT to BAL was 132 and 137 days, respectively. 70/137 (51%) BAL specimens yielded positive results. Of those, 60 (43.8% of total) represented true pathogens. Of these pathogens, 39% were bacterial, 47% were fungal, 46% were viral, and 33% were mixed.
Aspergillus (ASP) sp. accounted for 9/14 (64%) of all fungal pathogens isolated. Among these, 7 BALs had a positive ASP culture (137 tested), 4 had a positive BAL ASP Ag (41 tested), and 5 had a positive serum ASP Ag (71 tested). Of the 7 ASP BAL cultures, 5 had concurrent serum ASP Ag tested of which 1 (20%) was positive. None of the positive ASP BAL culture specimens had concurrent BAL ASP Ag tested. Of the 137 BAL procedures performed, 74 had serum Coccidioides (COC) serologies performed of which 3 (4%) were positive. COC PCR was tested in 133 BAL samples and 3 (2%) were positive. None of the 3 patients with positive serum COC serology had a positive COC PCR in the BAL. 1/137 (1%) had a positive BAL COC culture and a positive BAL COC PCR. Cytomegalovirus, Influenza, parainfluenza, adenovirus, and RSV accounted for all BAL viral pathogens. Mortality rate during the same hospitalization was 15%.

Conclusion: BAL remains an essential diagnostic tool in SCT recipients undergoing an infectious work up for pulmonary symptoms or abnormal chest imaging. Our study showed an overall BAL microbiologic yield of 36%.

**First Place – Research Poster**

**QSOX1: A DESMOID TUMOR MODIFIER GENE IN FAP?**

Keri Maher DO, UACOM at University Campus, PGY-3

Corina Mauss, Sarah-Jane Walton, Sue Clark, Christina Laukaitis

Desmoid Tumors (DTs) are locally invasive fibroblastic growths that occur in concert with Familial Adenomatous Polyposis (FAP) with an incidence of 10-15%. With improved treatment of FAP itself, the morbidity and mortality of DTs is increasing and they are now the 2nd-3rd leading cause of death. Previous work suggests the presence of a secondary gene (other than APC) modifying the expression of DTs, however, this gene has not yet been identified. Here, we examined the genetic variants (as identified through next generation DNA sequencing) within an extended FAP-affected family with a relatively high rate of DT formation to search for genes of interest. Data was processed using Broad Institute’s Genome Analysis Toolkit. Variants were sorted based factors thought to increase likelihood of deleteriousness. For example, we searched for those variants that cause non-synonymous substitutions, were relatively rare in the general population (MAF 0.1 or less) and were predicted deleterious by at least one of four functional prediction models. We also narrowed to genes with biological function likely to relate to DT formation. Sanger sequencing from identified genes of interest was used to confirm NGS variants and to search for presence of variants in other individuals. Through this process, QSOX1, which is involved in growth regulation of fibroblasts, has been identified as a gene of interest for further exploration. A particular variant has been identified in a conserved region of the gene with a Minor Allele Frequency of 0.1 and was predicted deleterious by all four models. This variant was present in all sequenced individuals expressing DTs and absent in those who did not, which is consistent with an autosomal dominant with incomplete penetrance inheritance pattern. Further work is ongoing to confirm the potential role of QSOX1 in DT formation and to identify other potential genes of interest.
A TREE HIDING IN THE WOODS: MAKING THE DIAGNOSIS OF GASTRIC ADENOCARCINOMA IN THE PRESENCE OF COMORBIDITIES

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Kirat Gill MD, Emily Schmidt MS 3, Jaya Raj MD

Learning Objectives
A high index of suspicion in the setting of abdominal pain, nausea, and continuous acute worsening of presumed chronic conditions is necessary to identify gastric adenocarcinoma at an early stage and improve the likelihood of a favorable outcome.

Case Presentation
A 69 year-old woman presented to the emergency department (ED) with a month-long history of generalized weakness and fatigue, with accompanying nausea, vomiting, chronic constipation, and crampy abdominal pain. Her past medical history included hypertension, dyslipidemia, poorly controlled type II diabetes mellitus, gastroparesis, and cirrhosis of unclear etiology. Plain x-ray revealed a dilated transverse colon concerning for ileus in a mildly distented abdomen. The patient was medically optimized and by the end of her hospital course denied the symptoms that initially brought her to the hospital. The patient was discharged with a non-tender, non-distended abdomen to a skilled nursing facility per physical therapy recommendation to improve functional mobility. Two weeks later, the patient again presented to the ED for diffuse abdominal pain. She reported that her abdominal pain and distention had been progressively worsening, with associated nausea, vomiting, fevers, inability to void, and lower leg edema. The patient appeared to be in a state of generalized anasarca with new onset ascites. CT scan of the abdomen showed her liver cirrhosis to be significantly different than previous scan. In addition, the scan was also significant for paraesophageal, gastric, and portahepatic lymphadenopathy with a partial small bowel obstruction. Esophagogastroduodenoscopy revealed moderate gastritis with irregularity in the stomach body; biopsies were obtained. Interestingly, gastric biopsy revealed poorly differentiated gastric signet ring cell carcinoma. The patient was diagnosed as stage IV with metastasis to the peritoneum. Discussion with the patient and her family identified a desire for hospice care upon discharge.

Discussion
Diffuse type gastric adenocarcinoma is characterized by signet ring cells that diffusely infiltrate the gastric wall. It is often associated with reactive processes, including desmoplasia or linitis plastica and carcinomatosis. Diffuse type is becoming a more prevalent form of gastric carcinoma, now accounting for about 30 percent of cases. Furthermore, approximately 50 percent of patients with gastric carcinoma present at late, incurable stages. The wide variation of presentation and the similarity in symptoms between gastric carcinoma and other clinical diagnoses requires practitioners to have a high clinical suspicion for diagnosis in order to improve upon generally poor prognoses.
DOUBBLE LYMPHOMA

Anju Nair MD, UACOM at South Campus, PGY-3
Jocelyn Ko, MD, Josephine Taverna, MD, Soham Puvvada, MD

Introduction: Composite lymphomas (CL) represent the occurrence of two distinct lymphomas in the same patient. Often, CL share a common cellular origin, thus representing a unique model to investigate the multistep genetic path leading to lymphomagenesis in general and to the specific development of each distinct lymphoma component in particular. Here, we present the molecular analysis of a case consisting of an unusual Follicular Lymphoma (FL) and a mantle cell lymphoma (MCL), intimately admixed within one another in lymph nodes yet phenotypically distinct, in a patient who first presented with constitutional symptoms and inguinal lymphadenopathy.

Case Presentation: A 65 year-old gentleman presents with fevers, chills, night sweats, unintentional weight loss and generalized lymphadenopathy. Labs were notable for white blood cell count 5.0, hemoglobin 15.5, platelets 229 and a lactate dehydrogenous level (LDH) 157. Computed tomography (CT) scans revealed generalized lymphadenopathy involving the retroperitoneal, mesenteric, axillary, iliac, and inguinal nodal regions. An excisional lymph node biopsy demonstrated grade I follicular lymphoma with mantle cell lymphoma in situ expressing Cyclin D1. Fluorescence in situ hybridization (FISH) analysis demonstrated two discrete translocations: t(11;14) translocation in 35% of tested nuclei and t(14;18) translocation in 65% of tested nuclei. Additionally, cytogenetic analysis detected an abnormal male karyotype 46, XY, t(14;18), q32q21.1, deletion 5 chromosome, q13q22 and Trisomy 8. Bone marrow biopsy revealed a hypocellular bone marrow (20% cellularity), but did not identify a light chain restricted B-cell population consistent with either the patient's previously characterized FL or MCLIS lymphoma. Due to limited stage FL, the decision was made to observe him clinically with close surveillance and serial CT scans.

Discussion: This case illustrates a unique composite lymphoma consisting of both a follicular lymphoma coexisting with mantle cell lymphoma in situ. Most cases of in situ mantle cell lymphoma are caught in the background of reactive follicular hyperplasia, and only rare cases have been reported in association with follicular lymphoma. CL involving MCLIS is rare with only six cases ever reported. We conducted a thorough review of literature comparing the clinical presentations, radiographic findings, pathologic features and molecular analyses in these selected patients. Unlike our patient who presented with nodal disease, cases of extranodal involvement (i.e. orbit, small bowel) can occur. Furthermore, we perform molecular studies including flow cytometry, cytogenetics, FISH in our patient to determine whether the CL represents a separate development from a common precursor or a subclonal evolution of MCL from FL.
KEEP YOUR MIND WIDE OPEN

Muna Omar MD, UACOM at University Campus, PGY-3
iyad Mansour, Naser Mahmoud

introduction: TTP in association with SLE is rare, and the diagnosis may be challenging. Although the etiology of TTP remains elusive, certain autoimmune mechanisms, platelet abnormalities, and fibrinolytic disorders may be shared with SLE and provide the basis for their association. Management requires timely diagnosis and aggressive treatment by therapeutic plasma exchange.

Case presentation: A 30 year old man had 20 years history of systemic lupus erythematosus (+ve ANA, DNA, Thrombocytopenia) treated with Prednisolone, antimalarial medications & azathioprine. Admitted with history of nausea, vomiting followed by loss of consciousness of 1 day duration. Physical exam: Has stable vital signs, afebrile. Cushinoid face, Has petechial rash on the dorsal aspect of her feet, Otherwise was unremarkable.

Labs and imaging: Urinalysis: Red cell: 10-13 Cast, Protein : +2, KFT: Cr 0.8 -> 1.1 WBC: 11.7 (72 PMN, 21% LYM), Hb : 9.5 à 6, Platelets: 26,000 à 5,000 Hospital course: The patient had positive ANA and Lupus anticoagulant, low C3 & C4 with negative Anti-dsDNA. The patient was started on pulse steroid. 2 days later, he developed Fever of 83 C and Pleuritic chest pain. He had Episode of tonic-clonic seizure followed by loss of consciousness, Brian MRI was normal, peripheral smear showed schistocyte, the patient was given 4 units of packed RBCs & platelets, Started on epanutin 100 mg, Q8 Azathioprin 50 mg and plasmapheresis.

Final Diagnosis: TTP.

Discussion: This clinical syndrome may mimic DIC or Catastrophic Antiphospholipid syndrome. There are female predominance of 3:2 & the median age at diagnosis is 35 years. On rare occasions, TTP has been described in association with SLE & occasionally the coarse is fatal. *From Saint Louis University Health Science Center in 1994; Four cases have been reported with SLE-related TTP & 24 have been described from literature review, all were women, 50% had active Lupus anticoagulant were positive in 5 out of 8 cases. SLE with low complement level. *Two cases have been reported in Scand J Rheumatol in 1998. Both were young women, one developed CNS manifestation, & the other developed fever, thrombocytopenia & hemolytic anemia. Both had a very rapid fatal course, although they underwent plasmapheresis. *From the University of Manitoba, in Canada; from 1996-2006. They only reported one male that has been affected by TTP-SLE, as apposed to the other cases which were only females

Second Place – Patient Safety/Quality Improvement Poster

INCREASING COMPLIANCE FOR HUMAN IMMUNODEFICIENCY VIRUS SCREENING IN INTERNAL MEDICINE RESIDENCY MEDICINE WARD SERVICES: A QUALITY IMPROVEMENT PROJECT

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Barr, P., Klassen, C., Fields, H., Azadeh, N., Mendoza, N., Saadiq, R., Pauwels, E., King, C., Chung

Background: In the United States 20% of individuals infected with human immunodeficiency virus (HIV) are unaware of their serostatus. Additionally, many may not have any reportable high risk behaviors. Thus, in 2006, the Center for Disease Control and Prevention (CDC) altered their recommendations to screen all individuals aged 13-64 years old for HIV. A chart review of the Mayo Clinic Arizona Internal Medicine (IM) Ward Service in 2011 revealed only 11% of patients admitted to the hospital had documentation regarding HIV screening. We designed a quality improvement (QI) project to improve adherence to the CDC recommendations on HIV screening for patients admitted to the Internal Medicine teaching services. Methods: The pre-intervention group was identified as all hospital admissions to the IM teach service from August until June of the 2011-2012 academic year. The post-intervention group was identified as all admissions to the IM teach service from August through June of the 2012-2013 academic year. The initial portion of the QI Project focused on identifying and interviewing stakeholders including patients, nursing, administration, social work, and other residents. Next, multiple iterations of Plan, Do, Study, and Act identified barriers to change and opportunities for improvement in IM Medicine Documentation of contemplation of HIV screening. Resident surveys regarding the CDC recommendations, email reminders, education dissemination, and competition between the ward teams were all utilized to improve guideline adherence. Credit was awarded if HIV screening was documented either by referencing the appropriateness of HIV screening in the admission dictation, ordering an HIV screening test, or notation in the resident signout sheet indicating why screening was not performed. Results: The number of admissions to the ward teams were similar in the pre and post intervention groups (559 patients pre, 541 post). Following the intervention, residents were more likely to have screening tests ordered (7.8% vs 55.5%, p= <0.0001) and document contemplation (7.8% vs 46.95%, p=<0.0001). Additionally, the number of HIV screening tests increased from 43 to 240 (p=<0.0001) in the pre and post intervention years. HIV screening documentation in the pre-intervention time period ranged from 0%-16.98%. During the academic year of the QI project, HIV screening ranged from 0%-77.36%. When evaluating for electronic medical record documentation for HIV screening the pre-intervention year demonstrated between 1.89%-16.98% per month. During the academic year of the QI Project these results increased to a range of 22.22%-100% per month.

Discussion: The Mayo Clinic Arizona Internal Medicine residency program demonstrated that HIV screening adherence can be improved through resident education, constructed competition, and system reminders. Possible boundaries for sustainable screening adherence include high resource consumption for a screening test in an acute health care setting and specific provider discomfort with inpatient screening.
A UNIQUE CONSTELLATION

Amitabh C. Pandey MD, UACOM at University Campus, PGY-3

JM Hanna, JP Ferreira

Clinical Case: A 48 year old male presented as a transfer from an outside hospital with an initial complaint of fatigue for several days with ongoing care for acute kidney injury, hydrocephalus, altered mental status and back pain. He had a past medical history significant for suspected pulmonary sarcoidosis, history of thoracic paraspinal mass consistent with non-caseating granulomatous disease on biopsy, status-post limited systemic steroid therapy totaling 3 months that was discontinued 2 months prior to transfer, and numerous admissions over the past two years for back pain, intermittent syncope, scrotal swelling, and persistent headaches. Over the past six months he had experienced progressively worsening back pain with episodes of altered mental status. After presenting to the outside hospital, he developed worsening altered mental status and was transferred to our care.

Hospital Course: Head CT showed non-communicating hydrocephalus. He was found to have acute kidney injury on laboratory analysis; urinalysis was consistent with pyuria without any organisms seen on smear or grown in culture. Pelvic CT showed findings consistent with orchi-epididymitis. Lumbar pucture demonstrated low glucose and lymphocyte predominance; CSF gram stain was positive for acid-fast bacilli. MRI imaging of his spine demonstrated progression of his paraspinal mass. Biopsy of this mass demonstrated acid-fast bacilli on biopsy with tissue culture positive for Mycobacterium tuberculosis, and he was started on antibiotic therapy for cryptic milliary tuberculosis (TB). During his hospital stay, the patient subsequently developed pulmonary TB, which eventually contributed to respiratory failure. The patient’s family ultimately decided to pursue comfort care measures only for the patient, and he died shortly after palliative extubation. The patient was given the final diagnosis of cryptic milliary tuberculosis, manifesting as Pott’s disease, with concurrent pulmonary tuberculosis.

Discussion: The incidence of pulmonary TB in the setting of cryptic milliary TB is rare. Typically extrapulmonary TB does not result in pulmonary TB and vice versa. Furthermore, what confused the clinical picture of this patient was the assumption of sarcoid diagnosis. The case of our patient shows the importance of dedicated chart review and history and physical exam. Furthermore, his constellation of symptoms is classic for TB meningitis, however this was not considered in the differential initially. In retrospect, his multiple admissions with the presenting constellation of symptoms correlate with TB meningitis, recognition of which may have allowed further diagnostics and treatment to be started earlier.

INTERIOR MITRAL VALVE ANEURYSM: A RARE COMPLICATION OF AORTIC VALVE ENDOCARDITIS

Golnar Parvizi MD, UACOM at University Campus, PGY-3

Raj Janardhanan, MD . Brandon Snyder, MD

CASE PRESENTATION: A 41-year-old male presented with three-week history of worsening shortness of breath. He endorsed extreme fatigue, night sweats, and dyspnea during the previous several months. He
had a past history of IV drug abuse. Physical exam revealed a diastolic murmur consistent with severe AR. WBC count was elevated at 19,000. Blood cultures revealed Enterococcus fecalis bacteremia and he was commenced on IV gentamicin and vancomycin. Transthoracic echocardiography demonstrated severe AR and an abnormal anterior mitral valve (AML). 3D Trans-esophageal echocardiography revealed large vegetations on the aortic valve (largest measuring 27 mm). There was mal co-aptation of the aortic valve leaflets resulting in severe AR. The jet was impinging on the AML, which showed aneurysmal dilatation. The 3-D zoom view from the LA side demonstrated the aneurysmal A2 scallop of the mitral valve and the view from the LV side revealed a perforation in the A2 scallop. There was no further perforation of the aneurysm into the LA cavity. Intraoperatively, the resected aortic valve showed several vegetations. There was a large, 2 x 2 cm perforation through A2 scallop of AML. There was a large ballooning piece of tissue seen over the perforation in the A2 scallop of the anterior leaflet, corresponding well to the pre-operative 3D-TEE findings. This was resected along with the valve leaflet. The patient underwent successful AVR and MVR. Cultures of the valves grew entrococcous fecalis. The patient was discharged on ampicillin and streptomycin for 6 weeks duration.

DISCUSSION: In IV drug abusers, infective endocarditis usually involves right-sided valves with staphylococcus aureus being the most common etiologic agent. Interestingly, our patient presented with left-sided endocarditis which is rather unusual. The typical occurrence of mitral valve aneurysm is in the context of aortic valve endocarditis. Complications of aortic valve endocarditis include extension of the infection to the mitral-aortic intervalvular fibrosa producing abscess, aneurysm, or perforation into the left atrium. The development of mitral valve aneurysm is likely due to the infected AR jet striking the ventricular surface of the AML causing physical trauma and possible occult mitral leaflet infection, manifested by valvulitis and the formation of a sac-like outpouching as seen in our case. The incidence of mitral valve aneurysm is very rare. To the best of our knowledge only five cases have been reported in the last 10 years. Early recognition of a mitral valve aneurysm is important because it may rupture and produce catastrophic MR in an already seriously ill patient, or it may be overlooked at the time of AVR. The use of 3D-TEE helped us plan the appropriate surgical approach with a successful outcome in our patient with this rare presentation.

**NOT LITTLE FOR LIDDLE**

**Divya Pati MBBS, UACOM at South Campus, PGY-3**

Bujji Ainapurapu MD

Title: Not little for Liddle  Divya Pati MD, Bujji Ainapurapu, MD. University of Arizona College of Medicine at South Campus.

Brief Introduction: Regarding hypokalemia and when to suspect more. Case: 29 year old male with past medical history of Diabetes, Hypertension was transferred from another facility for evaluation of hypokalemia. Patient presented to that facility for severe muscle cramps for 2 days. He was found to have hypokalemia with potassium of 2.0. Even after replacement, potassium was 1.8 the next day and hence was transferred for evaluation of refractory hypokalemia. On examination patient's blood pressure was elevated at 184/99 and was started on his home medication, Metoprolol 100 mg twice a day. Other vital signs were within normal limits and rest of the examination was normal. His potassium
was 2.2 at presentation with metabolic alkalosis, pH-7.52, PCO2-50.8, HCO3-39, Mg-1.5. Patient’s sodium, creatinine and AM cortisol were within normal limits. He had no history of diuretic therapy or licorice ingestion. He was given potassium and Magnesium replacement. As patient had hypokalemia, hypertension and metabolic alkalosis, evaluation was started for primary hyperaldosteronism. Renin and Aldosterone were ordered. Patient continued to have low potassium during the hospital stay needing replacement and muscle cramps resolved. Renin was low <0.1 and aldosterone was also low <1.6 consistent with Liddle’s syndrome. Genetic testing was planned for outpatient because of cost reasons. He was started on Amiloride. Potassium was normal and blood pressure controlled at discharge. Unfortunately patient lost follow up due to lack of insurance. Liddle syndrome is a rare autosomal dominant disorder associated with increase in the function of the collecting tubule sodium channel called the epithelial sodium channel or ENaC . Patient’s present with triad of hypertension, hypokalemia and metabolic alkalosis. Genetic testing is the most reliable method to establish diagnosis. Treatment consists of potassium-sparing diuretics Amiloride or Triamterene that directly block the collecting tubule sodium channel and correct both hypokalemia and hypertension.

Conclusion: This case illustrates that even though extremely rare Liddle syndrome should be considered in the differential diagnosis of patient with hypokalemia, hypertension and metabolic alkalosis as the treatment of Liddle’s syndrome differs from other forms of essential or secondary hypertension.

AN UNUSUAL PRESENTATION OF MESOTHELIOMA

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Pleural fluid eosinophilia (PFE, also called eosinophilic pleural effusion or EPE ) is defined as pleural fluid with a nucleated cell count containing more than 10 percent eosinophils . It is estimated that approximately 10 percent of exudative pleural effusions are eosinophilic. Although the pathogenesis is unclear, studies have suggested that air or blood in the pleural space may trigger the process. The presence of PFE narrows the differential diagnosis of a pleural effusion. Establishing the cause is dependent upon careful analysis of the clinical presentation and the pleural fluid. The presentation of one such case of eosinophilic pleural effusions will be reviewed here. A 68 year old male presented to the hospital with fevers and worsening shortness of breath for one week prior to admission. He was found to have a very large right sided pleural effusion warranting a chest tube. His pleural fluid revealed significant eosinophilia of almost 60 percent. Leukocytosis with peripheral eosinophilia was found as well. Further imaging revealed a loculated pleural effusion with a cavitary lesion at the base of the right lung. The patient underwent decortication and resection of the cavity and biopsy revealed malignant mesothelioma. The patient was discharged and underwent outpatient chemotherapy as well as radiation therapy. Mesothelioma has been shown to cause an eosinophilic pleural effusion; however, It is unusual for a patient with mesothelioma to present with both pleural and peripheral eosinophilia. Malignancy has been one cause of pleural fluid eosinophilia; however it has been mainly associated with a primary lung cancer rather than a pleural based malignancy. Furthermore, it has been shown that a pleural effusion with less that 40 percent eosinophilia has been associated with malignancy, whereas this patient was found to have almost 60 percent eosinophilia. This is an unusual case of mesothelioma presenting with peripheral eosinophilia as well as an eosinophilic pleural effusion. As in exudative pleural effusions, it is imperative that certain duration of follow-up by clinicians would be strongly
recommended in EPEs in order not to overlook malignancy when an initial work up is unrevealing.


CAUGHT IN THE ACT: A TALE OF ACUTE HIV INFECTION

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Robert Myers, MD , Robert Orenstein, DO

Detection of early HIV infection remains a significant challenge for clinicians despite major advances in the accuracy of diagnostic testing throughout the past few decades. HIV research has transformed a once fatal infection into a chronic disease that can be successfully managed without reduction in life expectancy. Nevertheless, major barriers to early detection still remain. These include a lack of adherence to screening guidelines and failure to recognize the acute retroviral syndrome. Employment of aggressive screening strategies using the latest cost-effective detection assays allows clinicians to diagnose and treat HIV infection earlier in the disease course and achieve better patient outcomes with early anti-retroviral therapy. A 24-year-old otherwise healthy Hispanic male presented to the Maricopa Medical Center emergency department with complaints of several weeks of headache, abdominal pain, nausea, vomiting, sore throat, maligias, anorexia and recurrent fevers up to 40°C. Upon initial examination, he appeared septic with a fever of 39.1°C and tachycardia of 115 bpm. Oral examination revealed diffusely hyperemic oral mucosa, tender left anterior cervical lymphadenopathy and right upper quadrant abdominal tenderness. Laboratory workup revealed an absolute lymphocytosis and transaminitis suggestive of hepatic inflammation. Imaging studies included an unremarkable chest x-ray as well as a normal head and abdominal CT. An acute hepatitis panel was negative for hepatitis A, B and C. Lumbar puncture revealed a polyclonal lymphocytic pleocytosis with elevated protein level of 111 mg/dl and negative Gram stain. A mononucleosis and group A strep screen were both negative. Screening studies for syphilis, Gonorrhea, and Chlamydia were negative, while a serum Quantiferon Gold test was positive. A fourth generation HIV EIA antigen/antibody screening assay was reactive for HIV-1. Flow cytometry of peripheral blood revealed a CD4 count of 1160 cells per microliter with a CD4/CD8 ratio of 0.3. HIV-1 viral load was greater than 10 million copies / milliliter. Given the patient’s severe illness, antiretroviral therapy was initiated with Truvada and Raltegravir. After 24 hours of therapy he reported resolution of his symptoms and labs showed significant improvement of his transaminitis. He was enrolled in the HIV clinic upon discharge from the hospital. This case highlights the critical importance of HIV screening and awareness of acute retroviral symptoms. The latest CDC guidelines recommend HIV screening for patients aged 13 to 64, especially high-risk patients including IV drug abusers and men who have sex with men. The presentation of HIV may vary significantly, making an early diagnosis particularly challenging. Fourth generation HIV EIA screening tests have improved sensitivity and specificity and can identify acute HIV infection earlier than third generation EIA assays.
HIV screening should be offered to patients at primary and urgent care clinics as well as emergency departments regardless of presenting symptoms.

**DRUG REACTION WITH EOSINOPHILIA AND SYSTEMIC SYMPTOMS (DRESS) SYNDROME INDUCED BY VANCOMYCIN**

**Tuan Phan MD, UACOM at University Campus, PGY-3**

Seongseok Yun, Anjali Dixit, Jenifer Huang, Cristian Dominguez, Vijay Hari Chandiramani

Introduction: DRESS is a rare and potentially life threatening syndrome with long latency (2-8 weeks). Diagnostic criteria include drug-induced skin eruption, fever, absolute eosinophil count >1500/mcL or atypical lymphocytosis, and visceral involvement affecting multiple organs. It is frequently associated with latent human herpes virus (HHV) infections.

Case report: We present a case of a 38 year old male with recurrent fever and rash. He was hospitalized for two weeks with MRSA bacteremia and pneumonia, treated with vancomycin, piperacillin/tazobactam, and clindamycin, and discharged with vancomycin for bacteremia. He returned to the ED 3 days later with fever and rash. Vancomycin was replaced with daptomycin and the patient was discharged. He returned 3 days later with continued fever > 38C, BP 86/40 mmHg, HR 120-140 bpm, neck edema, rapidly progressing, non-exfoliative erythematous rash including face, neck, trunk and extremities. Vancomycin was started in the ED then changed to daptomycin on admission. Patient was transferred to the ICU, and intubated for airway protection. Due to concerns of a daptomycin-related drug reaction, ceftaroline was initiated for continued treatment of MRSA bacteremia. Labs showed WBC 40,000/mcL, absolute lymphocytes 24,000/mcL (normal <4000), absolute eosinophil 1600/mcL, AST 243 IU/L, ALT 127 IU/L, alkaline phosphatase 516 IU/L, Cr 1.4 mg/dL, Na 128 mEq/L, pigmented granular casts in urinalysis, and positive EBV-IgG. Chest CT showed significant fibrosis and scarring, axillary, mediastinal, and hilar lymphadenopathy. TEE showed EF 45%, septal akinesis and inferior wall hypokinesis. Skin biopsy was consistent with a bullous drug reaction with no significant IgA, IgM, IgG, C3, C4, or fibrinogen deposit. Lymph node excision biopsy ruled malignancy and non-Hodgkin lymphoma unlikely. The clinical presentations and timeline of exposure to vancomycin were consistent with DRESS syndrome. The patient was started on methylprednisolone and transitioned to oral prednisone. Skin rash and eosinophilia resolved. Liver and kidney function were normalizing.

Discussion: DRESS can cause life threatening multiorgan failure, most commonly acute liver failure, with a mortality rate of about 10%. Forty-four medications have been implicated in 172 cases reported between 1997 and 2009. Vancomycin was reported in about 2% of the cases. Our patient was exposed to a number of antibiotics including vancomycin, daptomycin, clindamycin, piperacillin/tazobactam and azithromycin. Vancomycin was strongly implicated due to the appropriate latent period prior to skin eruption and symptoms exacerbated by a repeat exposure. A literature search did not reveal associations with other antibiotics. Our case was assigned a RegSCAR score of 9, signifying a highly likely DRESS case. Pathogenesis is likely multi-factorial and may involve various immunologic pathways and HHV infection. Initial manifestations are often non-specific and overlap with other drug-induced skin reactions. Early recognition and prompt removal of the inciting medication is important. Treatment may involve intravenous steroids and hemodynamic support.
GEMCITABINE INDUCED SINUS NODE DYSFUNCTION

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Anju Nair, Bujji Ainapurapu

Introduction: Gemcitabine is an analogue of deoxycytidine that specifically targets cells undergoing the S-phase of DNA synthesis and blocks progression of cells through G1/S phase. As a well-established chemotherapeutic agent, we are familiar with its common side effects which include thrombocytopenia, nausea, vomiting, rash, fever, alopecia, and pulmonary toxicity. There are very few cardiovascular adverse effects associated with gemcitabine documented in literature. We present a case of metastatic adenocarcinoma of the gallbladder accompanied by acute sinoatrial dysfunction after gemcitabine infusion. Case Presentation: We present a 75-year-old woman with a history of coronary artery disease and newly diagnosed invasive adenocarcinoma of the gallbladder. After her first dose of Gemcitabine for adenocarcinoma of gallbladder, she developed sudden onset of dizziness, shortness of breath, nausea after which she collapsed. She denied any chest pain, palpitations, or forceful heart beating. She had history of chronic total occlusion of her LAD artery four years ago, with resultant ischemic cardiomyopathy and ejection fraction of 40-45%. She was managed with medical management only. On evaluation, her heart rate was found to be 49. A 12-lead EKG revealed evidence of sinus node dysfunction with bradycardia and slow junctional escape. She was admitted to the hospital, during which time acute coronary events and additional sources of sinoatrial dysfunction were ruled out. Gemcitabine was deemed to be the likely cause of her sinus node dysfunction. After an uncomplicated hospital course, she was discharged to follow up with her outpatient oncologist, and advised close cardiac evaluation during next chemotherapy administration. Discussion: In patients receiving Gemcitabine, there is a predicted 3% incidence of fatal arrhythmias, yet a prior case of sinus node dysfunction has yet to be documented in literature. Cardiac events associated with chemotherapy are most often seen with anthracyclines and multiple alkylating agents, such as cyclophosphamide, ifosfamide, cisplatin, paclitaxel, etoposide, teniposide, vinca alkaloids, fluorouracil. Only one case of myocardial infarction with Gemcitabine has been reported. Multiple mechanisms have been proposed to explain cardiac toxicity associated with chemotherapy. For example, in 1-3% of patients using 5FU are thought to develop coronary vasospasms. The mechanism of gemcitabine induced cardiac toxicity remains unclear. Endothelial dysfunction and coronary thrombosis are potential explanations. Conclusion: As physicians, we must consider the rare complications including acute myocardial infarction and severe arrhythmias in patients receiving Gemcitabine particularly important in patients with known history of cardiac disease. A detailed cardiac examination and recommended investigations should be initiated before chemotherapy, regardless of whether or not the chemotherapeutic agent is considered cardiotoxic.

Third Place – Patient Safety/Quality Improvement Poster
INTERDISCIPLINARY INTERVENTIONS TO DECREASE CYSTIC FIBROSIS EXACERBATION LENGTH OF STAY

Leigh Anne Rundgren DO, Banner Good Samaritan, PGY-3
Introduction: Cystic fibrosis (CF) is the most common lethal genetic disease in Caucasians in the United States occurring in 1/3000 births. CF patients often experience recurrent pulmonary infections that require prolonged hospitalizations. Effective inpatient treatment of a patient with a CF exacerbation requires interdisciplinary teamwork. It is hypothesized that areas of inefficiency in this collaborative process could be optimized to decrease length of stay and, subsequently, hospital cost. Methods: A list of common laboratory tests, medications, and interventions utilized during CF exacerbations was created based on literature review, expert advice, and interdisciplinary collaboration. This data was initially organized into a paper order set in 2011 and provided to the hospital wards to be used by nurses and physicians at the time of admission. This order set was further refined and will be created as an electronic order set in 2013. In addition, weekly meetings of an interdisciplinary team were initiated in 2012. The team is comprised of hospitalists and pulmonologists with specific interest in CF, the adult CF coordinator, case managers, social workers, nurses, and respiratory therapists from two institutions with joint designation as CF centers. Collaboration during these meetings is focused on inpatient management and discharge planning. Average monthly length of stay and cost of hospitalization were obtained for one year prior to these interventions through June 2013. Results: Average monthly length of stay decreased from 11.7 to 10.0 days between June 2010 and June 2013. This is a decrease of 15.5%. Length of stay decreased from 11.7 to 10.3 days after implementation of the paper admission order set, and from 10.3 to 10.0 days after the addition of the weekly CF meetings. Hospital cost did not significantly change. Conclusions: The average length of stay has significantly decreased since June 2010 after the implementation of a paper CF admission order set and weekly interdisciplinary meetings. This is possibly attributed to these interventions; however, there are also other variables that were not measured but are likely contributing to these results. For example, there is an overall increased awareness regarding the significant hospital expense associated with CF exacerbations which likely impacts providers’ care. Also, since 2011, a dedicated CF nurse reviews electronic health records for all CF patients and troubleshoots issues that arise during each hospitalization. The increasing cost of healthcare overall in the past 3 years in the United States may have contributed to the insignificant change in the hospital cost results. Analysis of the above measures will be continued after the implementation of the electronic CF order set this year. We will measure the above outcomes for the subsequent six months, as well as analyze the frequency of admitting provider order set use.

ACQUIRED HEMOPHILIA A IN A 90 YO MALE; MORE QUESTIONS THAN ANSWERS

Victor Sanders MD, UACOM at South Campus, PGY-3

Bashar Domit, MD

Introduction: Acquired Hemophilia A is a rare bleeding disorder caused by IgG autoantibodies against Factor 8, leading to excess bleeding. It occurs in approximately 1.5 cases per million population per year and is commonly associated with age over 50 years, pregnancy, autoimmune disorders, malignancy and drug reactions. However, almost 50% of cases have no underlying disorder and the bleeding is severe enough to constitute a medical emergency. Case Presentation: A 90 year-old male with a PMH of and gingival bleeding presented with nausea and dizziness for one day. It presented suddenly while walking and resolved spontaneously in 1 minute. He denied loss of consciousness, falls, vertigo or preceding chest pain or palpitations. He admitted feeling intermittently short of breath, and an unintentional 35lb...
weight loss. He denied cough, fevers, chills, night sweats or changes in bowel habits. His last colonoscopy was 2 years ago and was normal. His father had lung cancer. On examination, he was comfortable but appeared pale. He was afebrile, hypotensive and pulse oximetry was 88% on room air. He had multiple bruises on his forearms bilaterally, rectal exam was positive for occult blood and his conjunctiva and oral mucosa were pale. The reminder of the physical exam was normal. Blood tests revealed hemoglobin of 8.5, hematocrit 27.1, platelets 79, PT 15.5, PTT 74.8 and INR 1.2. CT of the abdomen and pelvis with contrast showed a mid jejunum mass while the nasogastric lavage and EGD were negative. The mass was resected and turned out to be a blood clot. Post-operatively the patient required 12 units of PRBC’s and 1 unit of FFP. Fibrinogen, LDH, fibrin degradation products and peripheral smear were normal. A mixing study did not correct the PTT, Coomb’s test was negative, von Willibrand Factor antigen and activity were elevated, and lupus anticoagulant and antiphospholipid were positive. Factors 9 and 12 activity were normal however Factor 8 activity was <1% and Factor 8 C inhibitor level was elevated. Urine and serum protein electrophoresis showed lambda and alpha-1 globulin, respectively. Immunofixation electrophoresis showed IgM kappa band suggestive of a monoclonal protein. The patient developed anasarca, additional ecchymoses and an enlarging intra-abdominal hematoma. He received desmopressin and prednisone without improvement; ultimately recombinant human factor 7a was given. Unfortunately his health deteriorated rapidly and ultimately his family withdrew care. Discussion: This case highlights a very rare disease that if not identified in time, has fatal consequences. Early recognition and initiation of treatment is essential and despite treatment, patients remain at risk of life-threatening bleeding. Despite extensive literature, few controlled data are available and management guidelines are predominantly based on case reports, retrospective cohorts and expert opinion. More research into the etiology and the treatment is needed.

THE NOT SO HARMLESS MIGRAINE

Jeffrey Schenk DO, Verde Valley Medical Center, PGY-3

Migraines are a very common disorder and often we think of them as debilitating but otherwise harmless. Here I present a case of one of the rare complications of a migraine. A 50 year old right handed man had a sudden onset bilateral loss of vision that began as a floater in the left upper visual field and rapidly progressed to darkness in both fields of vision. This lasted for 2 hours and then improved with some residual blurring of vision that persisted for 24 hrs and a small left visual field loss. Neurological exam was otherwise unremarkable and physical exam including fundoscopic exam was normal. The patient has no significant past medical history including any cardiac history, takes no medications, and was never a smoker. He was started on an aspirin and worked up for stroke. Initial CT showed no bleeding. The MRI of the brain showed a sub acute right occipital stroke. MRA of the brain is normal, Carotid US and echocardiogram are also negative. Patient had normal full anticoagulation panel, homocysteine level, ESR, CRP, and ANA. Initially patient denies any history of migraine or any family history of migraine. On further questioning about headaches the patient reports that he get frequent “sinus headaches” weekly and has had them for many years now. They are unilateral throbbing headaches preceded by blurring of vision. His parents both have similar sinus headaches. The patient had a similar headache when his original symptoms first occurred. Patient was diagnosed with migraine headaches and an acute migrainous stroke. He was started on oral verapamil and will follow up with neurology for management of the migraines. Migraine is a common disorder that affects 12 %
of the population with a predilection to women. While migraine is common, a complicated migraine with stroke mimicking symptoms is rare and migraine with brain infarction is even rarer. A study published in Neurology in 2011 showed that brain infarction among migraine patients had a prevalence of 0.2%. Neurologists are still unsure what causes the actual infarction but it is hypothesized that severe vasospasm, neuronal glutamatergic hyperexcitability, increased platelet aggregability or endothelial abnormalities are the cause. Migrainous stroke is differentiated from complicated migraine by symptoms lasting longer than 1 hr and evidence of permanent damage to the brain. This is a case of the rare complications of migraines and is ever more interesting that it happened in a male patient. The case shows us how important it is to properly diagnose headaches and treat them accordingly. It also shows how important it is to pay close attention to the details of the patient’s history and diagnose accordingly.

**SMALL BOWEL INTUSSUSCEPTION SECONDARY TO LIPOMA IN AN ADULT**

Ryan Sefcik DO, Sierra Vista Regional Health Center, PGY-3

Small bowel intussusception in an adult secondary to lipoma  A patient in his 50’s presented to the gastroenterologist with a several month history of mid-abdominal pain and weight loss. Laboratory testing including a complete blood count found that patient had a microcytic anemia. The patient underwent both an upper and lower endoscopy which were negative for any pathology. The patient then had a CT enterography of the abdomen completed which was reported as a small bowel mass, with the radiology reading it as possible primary malignancy vs metastasis. Consultation by a surgeon specializing in Oncology was obtained and a laparoscopy was performed for further evaluation. It was discovered during this exam that the patient had a small bowel obstruction caused by an intussusception secondary to a benign lipoma. This case is an example of a rare cause of small bowel obstruction that included atypical symptoms.

**LOST**

Corinne Self MD, UACOM at University Campus, PGY-3

Keri Maher, DO

A healthy 70 year old Hispanic male presented to the emergency department (ED) after a motor vehicle accident and received a CT chest/abdomen/pelvis as part of his evaluation. Imaging incidentally revealed a 6x6x7cm heterogeneous mass on the superior pole of his right kidney concerning for renal cell carcinoma (RCC). The patient had no established primary care provider, therefore an appointment was arranged for soon after ED discharge. Four and a half years later, he was seen by an oncologist outside of our system for a large, movable left chest mass. Mammography was undertaken which revealed a 4x3x4cm lobulated axillary tail mass, BI-RADS 4. Pathology from a core needle biopsy revealed histiocytic, giant cell reaction with inflammation, negative for carcinoma. CT chest/abdomen/pelvis obtained at that time revealed diffuse metastatic disease. He was diagnosed with metastatic carcinoma of unknown primary. Soon after, he presented to the ED with left sided weakness. He was found to have a hemoglobin of 9.8 and a calcium of 11.4. MRI brain revealed extensive metastatic disease with vasogenic edema. Repeat chest mass biopsy at our institution was consistent
with RCC. During the elapsed time the renal mass grew from 6x6x7cm to 8x7x10cm, nearly doubling in volume. It spread to the renal vein, Gerota's fascia, adrenal gland, lung, pectoral muscle, paraspinal muscles, lymph nodes and brain. This case highlights the natural history of RCC with common metastatic sites including lymph nodes, lung and brain. Skeletal muscle metastasis are rare and only described in a handful of case reports. This case also illustrates the importance of seeking outside records from all sights visited by a patient prior to undergoing expensive, invasive procedures as they may prove to be unnecessary. Furthermore, this case highlights the need for universally accessible electronic medical records.

THE NIGHTMARE OF A DERMATOLOGIST

Ismail Tabash MD, UACOM at University Campus, PGY-3

El Ramahi, Razan

Introduction: Leishmaniasis is one of the Neglected Tropical Disease (NTD). That is caused by Leishmania donovani and transmitted by sand flies. More than 90% of VL cases occur in five countries: India, Bangladesh, Nepal, Sudan, and Brazil. Here, we present a case of CLL that was complicated by disseminated leishmaniasis and cutaneous leukemic infiltrates. Case: A 70 year old male patient from Sudan, presented with skin rash of 4 years in duration. HPI: The rash started as a single small elevated skin lesion 2x2cm on the lateral aspect of his right leg. Which ulcerated and didn’t heal. 3 months later, new multiple similar lesions developed and cover the whole body including his face. The rash is painless, non itch, and has no relation to sun exposure. ROS: Significant for weight loss and fatigue. PMH: DM type 2 for 15 years treated with Insulin 70/30. FH & SH: His father had DM. Ex-smoker 4 pack.year, Denied alcohol and drug use. Married, sexually inactive. On PE: Normal vitals, malnourished, pale, normal lungs and heart exam, difficult abomenal exam because of skin rash, and intact peripheral pulses. Skin exam: Diffuse erythematous ulcerated plaques, ranging from 2-5 cm in diameter, with full thickness necrosis. No drainage of pus. Post inflammatory hyperpigmentation changes present. Labs: WBC: 6.5 (N=8 %, L=90 %, E=1%, M=1%, ANC 520) HB:7.5, MCV: 82, Platelets: 137 BUN : 38, CREAT: 0.6, Alb: 2.8, ALT: 5, AST: 8 LDH: 140, ESR: 88, CRP: 81.9, TSH: 3.3, CPK: 23 Peripheral smear: marked neutropenia, 20% Atypical lymphocytes. Ferritin: 324, Retic 0.8% HBS Ag: -ve, HCV: -ve, HIV: -ve, EBV IGM: positive, titer 80, CMV IGM: -ve, ANA: 1:40 Blood cultures: no growth. Abd CT: Splenomegaly. Skin Biopsy: Leukemic infiltration (Leukemia Cutis) and disseminated leishmaniasis. B.M. Biopsy: CLL Discussion: Leishmaniasis is a protozoal disease causing a spectrum of clinical syndromes ranging from cutaneous ulcerations to systemic infections. It is caused by Leishmania donovani and transmitted by sand flies. Rare cases of transmission through needle sharing, transfusions, pregnancy, and sexual intercourse have been reported. It is estimated that 500,000 cases of visceral leishmanias occur annually. Diffuse cutaneous leishmaniasis develops in patients with poor immune response. Infection is characterized by a primary lesion, which spreads to involve multiple areas of the skin. Plaques and ulcers may form over the entire body, resembling leprosy. However, no systemic invasion. Cutaneous leishmaniasis tends to resolve spontaneously. Treatment is indicated if the lesions are disfiguring, painful, infected, or slow to heal. Diffuse cutaneous leishmaniasis respond poorly to treatment, and relapse frequently. Diffuse cutaneous leishmaniasis maybe the presenting sign of immune compromised conditions like HIV3 and CLL as in our case.
ACROSS THE Z LINE. A RARE CAUSE OF GI BLEEDING-ALCOHOLISM RELATED ACUTE ESOPHAGEAL NECROSIS

Varun Takyar MD, UACOM at South Campus, PGY-3
Krunal Patel MD, Bianca Afonso MD

Introduction: Acute esophageal necrosis (AEN) is a rare cause of GI bleeding manifesting as severe fatal hematemesis and shock in most cases. Retrospective data suggests an incidence of 0.28% in endoscopies done for acute GI bleed. Alcoholism is a known risk factor amongst many others. Here, we present one such case and a review of AEN associated with alcoholism. Case and review of literature: A 57 year old female presented to our hospital with 2 day history of epigastric discomfort, nausea and melanic stools. Her medical history was significant for chronic hepatitis C infection and alcoholic liver disease. She admitted drinking alcohol prior to the day of presentation. Physical exam revealed a BP of 78/34 and severe epigastric tenderness with frank melena on rectal exam. Relevant labs included Hgb of 6.2 mg/dL, WBC of 20.5 K/uL and lactic acidosis of 6.0 mg/dL. Despite aggressive resuscitation, the patient remained hypotensive with an elevated lactic acid level. A CT scan of the abdomen showed circumferential thickening of distal esophagus. Upper GI endoscopy found a severe necrotizing, hemorrhagic circumferential esophagitis up to the gastroesophageal junction. Pathology showed extensive necrosis. The clinical course worsened with development of esophageal perforation and mediastinitis leading to an emergent open esophagectomy. The patient was extubated 13 days post-op, however, given her debilitated state passed away 3 months later secondary to disseminated aspergillosis. The known risk factors for AEN include cardiovascular disease, hemodynamic compromise, alcohol ingestion and trauma. Retrospective review of Pubmed and Google Scholar for cases of AEN and Alcoholism and/or liver disease revealed only 25 cases from 1970-2013. Mean age at diagnosis was lower compared to general population (54 vs 67). Male to female ratio was similar to general population (4:1 vs 4.3:1). The postulated mechanism of injury is two fold: vascular insufficiency along with direct mucosal damage. In alcoholic patients, decreased lower esophageal sphincter tone is believed to compound the injury further. Conclusion: Alcoholism-associated Acute Esophageal Necrosis is a rare diagnosis with poorer outcomes and occurs at an earlier age than other causes of this disease. Treatment should be targeted at increasing vascular perfusion to the esophagus while reducing further injury with intravenous proton pump inhibitor and bowel rest.

RED EYE WITH HIV CO-INFECTION

Donovan Williams MD, Banner Good Samaritan, PGY-3

Introduction: Chemosis and pain of a red eye can have a broad differential in a patient who is HIV positive. Ocular manifestations are common in HIV disease with a variety of causes, ranging from a benign HIV retinopathy to sight threatening opportunistic infections. It is important to recognize manifestations of each disease to start appropriate treatment. We present a case of ocular syphilis and orbital infection. Case Presentation: 28 year old homosexual male who had a home HIV positive test presented to the ER with left eye redness, swelling, posterior eye pain, pain with eye movement and light sensitivity for 5 days. Prior to the ER visit, he was seen by an ophthalmologist and had injected him with vancomycin and ceftazidime. In addition, the patient mentioned having non-painful hand and foot
lesions over the past 2 months. Sexual activity involved oral and anal practices. On exam he appeared in pain, sluggish pupil response to light in left eye, left eye chemosis with conjunctival inflammation and yellow discharge. No vesicular lesions on face. Neck was supple. Skin showed macular, erythematous, non-ulcerated lesions on both hands and feet. Laboratory evaluation included WBC 3.4 (normal differential), RPR titer 1:1024, FTA positive, HIV-1 viral load 179,000 copies, CD4 276, CMV IgG/IgM serum reactive, HSV 1 and 2 serum negative, Toxoplasma IgG/IgM serum negative, Coccidioidomycosis IgG/IgM serum negative. PPD skin test negative. CSF evaluation resulted in WBC 12 predominately lymphocytes (82%), CSF VDRL reactive, CSF cryptococcus antigen negative. Ophthalmologic evaluation resulted in 20/20 right eye visual acuity and only light perception with projection in left eye. Slit lamp evaluation showed fibrin in the interior chamber. Dilated fundus exam showed an attached retina, optic nerve swelling, and vitritis. MRI revealed left orbital post septal cellulitis with enhancing fluid is noted anterior and lateral to the globe including lateral rectus muscle. No evidence of intracranial inflammatory extension. Left frontal sinus disease present. Discussion: In this case, there appeared to be two infectious processes. First is an early neurosyphilis with uveitis requiring a lumbar puncture for evaluation. The palmar-plantar salmon colored skin changes provided a clue to syphilis as part of the differential as secondary syphilis. Diagnosis was confirmed with RPR 1:1024, FTA positive, and CSF VDRL positive. Second, there was also an associated left frontal sinusitis contributing to an orbital infection. The MRI showed inflammation of external ocular musculature, which is not consistent with syphilis. The patient did have improvement by treating both for neurosyphilis and bacterial infection of the eye. Conclusion: Patient with HIV can have multiple infections. Ocular syphilis requires a lumbar puncture for evaluation and should be treated as neurosyphilis. Identifying different infections is crucial for a comprehensive management and treatment.

**PROGRESSIVE MULTIFOCAL LEUKOENCEPHALOPATHY (PML) AFTER IMMUNOSUPPRESSION**

Andrew Chung MD, Mayo Clinic Arizona, PGY-3

Donald Northfelt, MD

Introduction: Progressive multifocal leucoencephalopathy (PML) is a demyelinating white matter encephalopathy caused by the JC (John Cunningham) virus, in which progressive neurologic symptoms lead to death. It is caused by reactivation of the JC virus and is almost only seen in patients with underlying immunosuppression. It became increasingly recognized during the HIV/AIDS epidemic, but has also been described in various hematologic malignancies and after the use of certain immunosuppressive and chemotherapeutic agents. This case report describes PML in a patient who had received multiple immunosuppressive agents with an underlying hematologic malignancy. Case: A 52-year-old male with a past medical history significant for Waldenstrom’s macroglobulinemia (WM) presented with complaints of severe gait instability, left-sided weakness, dysarthria, dysphagia, and mental slowing. Symptoms had begun gradually approximately eight months prior. He had been hospitalized as recently as three months prior at an outside hospital with left-sided weakness and slurred speech; MRI then showed no abnormalities and he was diagnosed with a transient ischemic attack. The patient had been diagnosed with WM eight years ago. He was initially treated with two cycles of rituximab three years ago and then had been intermittently receiving single-agent fludarabine.
starting one year ago. His last dose was given four months prior to presentation. On neurologic exam, he had diminished sensation in the distribution of V1–V3 on the left, dense left hemiplegia, extinction, and hyperreflexia. HIV testing was negative. MRI showed extensive white matter lesions, right greater than left, without mass effect, and CSF JC virus PCR returned positive, supporting a diagnosis of PML. The patient declined further treatment and was discharged with hospice services. Discussion: PML was first described in 1958 in a patient with Hodgkin’s lymphoma and the JC virus was first isolated in 1971. Although first described in patients with hematologic malignancies, it became recognized as a major opportunistic infection in HIV patients. However, immunosuppressive agents have also been recognized as a risk factor, such as biologics (rituximab, natalizumab), transplant medicine (mycophenolate mofetil), and chemotherapeutics (fludarabine, cyclophosphamide). In particular, this patient had received fludarabine, which can cause prolonged immunosuppression. Fludarabine’s immunosuppressive effect is thought to be secondary to both absolute lymphopenia and low CD4 count; median time to diagnosis from last dose in one study was 11 months. Rituximab has also been associated with PML, with 57 cases reported from 1997 – 2008. The drug has been associated with PML in both hematologic and non-hematologic diseases. While the actual incidence of PML in patients having received immunosuppressive therapy is low, it is being increasingly recognized. Given the uniformly poor outcomes once diagnosed, providers must have a high index of suspicion for PML in patients receiving such agents who present with new neurologic symptoms.

PGY-2 SUBMISSIONS

ISCHEMIC STROKE AND SIMULTANEOUS NSTEMI AS A PRESENTING FEATURE OF PFO

Saifuldeen Al-Qaisi MD, St. Joseph’s Hospital & Medical Center, PGY-2

Fadi Alrabadi, Feras Alyafi, Raed Al Adham

Ischemic stroke and simultaneous NSTEMI as a presenting feature of PFO. Saifuldeen Al-Qaisi, M.D., Fadi Alrabadi, M.D., M Feras Alyafi, M.D., Raed Al Adham, M.D. Case Report: 65 year old male with past medical history of hypertension and diabetes was admitted to the hospital for persistent dizziness, lightheadedness associated with dysarthria and shortness of breath that started almost 12 hours earlier. BP was 149/86, HR 96, RR 20, Temp. 97 F, O2 saturation was 99% on room air. Physical examination, including cardiac and neurological exam, was unremarkable Initial labs showed sodium of 142mEq/L potassium 4.4 mEq/L, creatinin 1.2mg/dl, WBC 7.7, hemoglobin 13.7, platelets 75000, calcium 9.4mg/, Glucose 159mg/dl, negative cardiac enzymes. Chest x-ray was negative, EKG showed left axis deviation with no st segment elevation or T- wave changes. Ct head w/o contrast was negative. Doppler US of lower extremities showed extensive DVT in the right leg veins. MRI brain showed acute ischemic changes in the frontal lobe and the patient was started on heparin drip. TTE was was inconclusive. The repeated troponin I level was elevated at 1.19, still with no EKG changes. Subsequently, the patient was triaged to the catheterization lab, had Rt and Lt cardiac catheterization as well as Rt and Lt coronary angiography, which showed normal coronaries, as well as a > 1 cm PFO. These findings, together with the presence of DVT and multiple ischemic events led to the conclusion that paradoxical emboli were responsible for the CVA and the cardiac ischemia. The patient underwent a percutaneous closure of the foramen and was started on warfarin treatment before discharge. Discussion: PFO is a very common
condition occurs in about 25-30% of general population. This condition is usually asymptomatic, but it may also present with stroke, TIA or other embolic phenomena. It was also found that the prevalence of PFO was higher in patients with cryptogenic stroke, particularly in those younger than 55, that is why it is so important to consider paradoxical embolism as a cause of unexplained ischemic event so that we can start appropriate management and prevent recurrent episodes. The condition can be identified by agitated saline with ultrasound. Incidental finding of PFO requires no treatment or follow up. Antiplatelet treatment is preferred in most stroke patients with PFO, systemic anticoagulation is indicated in case of associated venous thrombosis or hypercoagulable states. PFO closure appears to be an appropriate choice to reduce the risk or recurrent events in patients who fail medical management, and in a study that included 267 patients in 10 different countries, the use of PFO closure device after stroke or TIA resulted in good clinical results with no recurrent event.

**ISCHEMIC COLITIS IN A YOUNG FEMALE**

Fadi Alrabadi MD, St. Joseph's Hospital & Medical Center, PGY-2

Moustafa Hazin, MD Kareem Shaarawy, MD Saifuldeen Al-Qaisi, MD

Introduction: Ischemic colitis is caused by a reduction in blood flow, which most commonly arises from occlusion, vasospasm, or hypoperfusion of the mesenteric vasculature. It usually affects the elderly and is rare in young patients. Case description: A 21 year old athletic female, with a past medical history of asthma, presented to our emergency room with three days of diffuse crampy abdominal pain, with no relieving or aggravating factors, that was associated with eight to nine episodes of bloody diarrhea, and mild nausea. Patient denied vomiting, fever, recent travel or previous similar symptoms. The patient had completed 10 days of clindamycin after rhinoplasty surgery and was taking Advair, Albuterol, and an oral contraceptive pill (OCP). Physical exam revealed a soft abdomen with diffuse tenderness, but no rebound. Her workup included a clostridium difficile polymerase chain reaction test, stool ova and parasites, and Giardia, all of which were negative. This was followed by a computed tomography of the abdomen and pelvis, which showed diffuse colonic wall thickening with mucosal and serosal enhancement, and submucosal edema consistent with pancoolitis. Colonoscopy was positive for inflammation in the sigmoid and hepatic flexure area, suggestive of ischemic colitis. Colon biopsy was positive for lamina propria hemorrhage and edema with focal collagenization, and patchy acute inflammation, suggestive of early ischemia. Discussion: Bloody diarrhea in a young female can have multiple etiologies, but ischemic colitis, while rare, still needs to be considered in the differential. OCP use is a known risk factor for ischemic colitis. Researchers in Tennessee reviewed 35 cases of ischemic colitis in patients under the age of 40 seen over 5 years in a Memphis hospital; 17 were excluded because of positive culture results for E. coli 0157:H7, severe hypotension, or incomplete colonoscopy examination. Of the remaining 18 patients, 17 were women. Ten (59%) of the 17 women were using low-dose estrogen-progestin oral contraceptives. The study demonstrated a relative risk of 6.31 for ischemic colitis among women using hormonal contraception. The mechanism of estrogen-induced ischemic colitis is not clearly understood, could be related to arterial and venous hyperplasia and an increased incidence of thromboembolism. Exercise-induced intestinal ischemia is rare but has been documented in athletes especially marathon runners; the mechanism may be related to a decrease in mesenteric blood flow during exercise. Conclusion: Although ischemic colitis is a rare cause of lower GI
bleeding and abdominal pain in the younger age group, it still needs to be considered. Suspicions should be raised even higher in the presence of risk factors like vasculitis, sickle cell disease, cocaine abuse, and high risk medications like OCPs, NSAIDs, and sumatriptan.

Second Place – Case Report Poster (Tie)

**BALL-VALVE PATTERN RIGHT MAINSTEM BRONCHUS OBSTRUCTIVE PNEUMONIA SECONDARY TO A MALIGNANT-TRANSFORMED RESPIRATORY PAPILLOMA**

Seth Assar MD, UACOM at South Campus, PGY-2

Tauseef Afaq, MD; James Knepler, MD

Introduction: Squamous cell carcinoma (SCC) transformation is a known but exceedingly rare consequence of recurrent respiratory papillomatosis (RRP). We present a unique case of a patient presenting with an anatomical ball-valve pattern complete obstruction of the right mainstem bronchus and secondary obstructive pneumonia attributed to a malignant-transformed papilloma.

Case Description: The patient was a 22 year old male with RRP who presented with shortness of breath, copious sputum production, and fever. Additionally, he reported an approximate 10-lbs of unintentional weight loss within the last one year. He was referred for admission by his ENT surgeon whom he has seen earlier in the day for follow up of a bronchoscopy and biopsy of a suspicious lung mass two weeks prior. He has had approximately 101 laryngoscopies and/or bronchoscopies in his lifetime for upper airway obstruction. Initial chest x-ray showed a white out of the right lung with a cavitary lesion and an air-fluid level in the inferior aspect of the right upper lobe (Figure 1). After stabilization of the patient’s severe sepsis, he underwent bronchoscopy demonstrating a fungating and exophytic mass originating in the right upper lobe and extending into the right mainstem bronchus. This lesion was excised and sent for biopsy (Figure 2). The patient experienced rapid improvement of his respiratory symptoms with aggressive pneumonia therapy and was discharged home with clinic follow-up for staging and treatment of his lung cancer.

Discussion: RRP occurs by incidence in between 3.8 to 4.3 per 100,000 children with an equal affliction of both sexes. The natural history of RRP in children [particularly] is highly variable. RRP is thought to be exclusively an acquired disease, occurring by vertical transmission during vaginal birth. While complex mechanisms and variables may be present, the overall consensus is that direct mucosal colonization by HPV DNA arising from lesions of the mother lead to this disease. RRP is categorized by age and persistence: there are three subtypes: juvenile, adult, and juvenile-onset with persistence into adulthood. Of the nearly 100 known subtypes of Human papilloma virus (HPV), subtypes HPV-6 and HPV-11 are responsible for the majority of cases or RRP. Bronchial spread of RRP has noted complications of atelectasis, nodular and cystic pulmonary degeneration, and obstructive pneumonia, however the lesions are almost exclusively benign papillomas. Surgical debridement with the aim of preservation of laryngeal function is the mainstay of therapy in children who often present with dyspnea and stridor. Medical adjuvants including alpha-interferon, cidofovir, indol-3 carbinol, as well as other investigational agents have a limited role in therapy.
DON’T BE SUPRISED! JUST NEED TO CONTROL RISK FACTORS!

Thandar Aung MD, St. Joseph’s Hospital & Medical Center, PGY-2

Ischemic stroke in young patients (18-45 years) is relatively rare and cardiovascular risk factors are generally thought to be less important in younger patients. Dyslipidemia and elevated lipoprotein A are associated with higher incidence of ischemic stroke risk in a number of epidemiological studies. 25 y.o male presented with right extremities weakness, right facial droop and slurring of speech. Forty Hours before the admission he noticed his right leg was weak just after he woke from sleep. During the course, he also noticed his right arm was weak and he had a tendency to fall to right. He thought he was too tired from work and ignored it. Four hours before the admission, he had sudden onset of right facial droop and slurring of speech. He was diagnosed with mild hypertension and dyslipidaemia when he was eighteen and was on low dose of HCTZ and statin but non compliance. No significant family history. Vitals were stable. On examination, he was found to have NIHSS stroke scale of 5. He was dysarthria and right upper seventh cranial nerve palsy. Pupils and extraocular movement and visual field were normal. Tongue was deviated to the right side. 3/5 motor strength and 4+ reflex in the right side of the body with positive right pronator drift. No sensory loss. positive babinski response. No cerebellar signs. CT head showed no acute ischemic change nor hemorrhage. MRI and MRA Brain showed acute ischemic changes in left anteromedial pons (Paramedian Basilar Infarct) and left perforator branches of basilar artery were smaller, suggestive of microvascular disease. TEE is negative for signs of chronic hypertension, PFO and valvular anomalies. Hypercoagulable work up, urine toxicology and HbA1c were unremarkable. Total cholesterol - 435mg/dl, LDL - 406mg/dl, HDL-29mg/dl, TAG-70mg/dl. Lipoprotein (a) - 114mg/dl (elevated) Patient was started on ASA, statin and antihypertensive. In young healthy adults, paramedian pontine strokes from atherosclerosis are uncommon. Embolism, arterial dissection and specific conditions of hypercoagulation rather than large or small atherosclerotic arteriopathies are thought to be the most common cause of stroke in young adults. Jukka Putaala had reported that the most frequent risk factors associated with stroke in young adults (15-49) were dyslipidemia (60%), smoking (44%), and hypertension (39%). The unexpectedly high frequencies of modifiable risk factors indicate a need for aggressive management. Patients with unilateral pontine stroke can present with contralateral tongue deviation which has been shown to be associated with supranuclear CN7th Palsy, and contralateral CN lesions and hemiplegia which may pose a particular challenge on diagnosis of stroke subtype. Role of statin in primary prevention of stroke is unclear but recently Jukka Putaala reported that young patients with a first ischemic stroke of undetermined etiology who used statin poststroke had lower rates of new vascular events in long-term followup.

ANALYSIS OF AMPLITUDE SPECTRAL AREA AND SLOPE TO PREDICT DEFIBRILLATION IN OUT OF HOSPITAL CARDIAC ARREST DUE TO VENTRICULAR FIBRILLATION (VF) ACCORDING TO VF TYPE: RECURRENT VERSUS SHOCK-RESISTANT

Amanda Bisla MD, UACOM at University Campus, PGY-2

Madhan Shanmugasundaram, Michael J. Kellum, Gordon A. Ewy, Julia H. Indik

Background: In out-of-hospital cardiac arrest (OHCA) due to ventricular fibrillation (VF), VF may recur during resuscitation (recurrent VF) or fail to defibrillate (shock-resistant VF). While retrospective studies
have suggested that amplitude spectral area (AMSA) and slope predict defibrillation, it is unknown whether the predictive power is influenced by VF type. We hypothesized that in witnessed OHCA with initial rhythm of VF that the utility for AMSA and slope to predict defibrillation would differ between shock-resistant and recurrent VF. Methods: AMSA and slope were measured immediately prior to each shock. For second or later shocks, VF was classified as recurrent or shock-resistant. Cardiac arrest was classified according to whether the majority of shocks were for recurrent VF or shock-resistant VF. Results: 44 patients received 98 shocks for recurrent VF and 96 shocks for shock-resistant VF; 24 patients achieved ROSC in the field. AMSA and slope were higher in recurrent VF compared to shock-resistant VF (AMSA: 28.8±13.1 vs 15.2±8.6mVHz, P < 0.001, and slope: 2.9±1.4 vs 1.4±1.0mVs/#8722;1, P = 0.001). Recurrent VF was more likely to defibrillate than shock-resistant VF (P < 0.001). AMSA and slope predicted defibrillation in shock-resistant VF (P < 0.001 for both AMSA and slope) but not in recurrent VF. Recurrent VF predominated in 79% of patients that achieved ROSC compared to 55% that did not (P = 0.10). Conclusions: In witnessed OHCA with VF as initial rhythm, recurrent VF is associated with higher values of AMSA and slope and is likely to re-defibrillate. However, when VF is shockresistant, AMSA and slope are highly predictive of defibrillation.

ADULT-ONSET NESIDIOBLASTOSIS, A RARE CAUSE OF RECURRENT HYPOGLYCEMIA

Giang Bui MD, St. Joseph's Hospital & Medical Center, PGY-2

Manal Alhakim, Sadia Moinuddin, Alisha Bogus, Laura Knecht and Anna R. Boron

Introduction: Bariatric surgery remains an important therapy option for obesity. Non-insulinoma pancreaticoblastosis syndrome (NIPH) or hyperinsulinemic hypoglycemia with nesidioblastosis has been described in patients with a history of bariatric surgery. Nesidioblastosis is a term that describes pancreatic B-cell hyperplasia, hypertrophic islets, and increased periductular islets. Our patient represented a classic case of NIPH. The patient required both medical and surgical intervention to control hypoglycemia.

Case Report: A 33 year old obese female, with a history of bariatric surgery 6 years ago, presented with recurrent hypoglycemia and symptoms of neuroglycopenia. The hypoglycemia was associated with increased endogenous insulin level. During the 72-hour fast, hypoglycemia was reported during the 65th to 70th hour. The 72-hour fast was suggestive of NIPH. Multiple diagnostic modalities including MRI, Octreoscan and EUS were negative for pancreatic tumor. The calcium stimulation test did not localize tumor. Medical therapy with diet, Acarbose and Octreotide was unsuccessful. Partial pancreatectomy initially helped to de-escalate medical therapies. Histological findings were consistent with nesidioblastosis. However, within 3 weeks, the patient returned with mild hypoglycemic episode which was managed with dietary modification and higher dose of Acarbose and Octreotide.

Discussion: Comparing to insulinoma, NIPH represents about 0.5% to 5% of all pancreatogenous hypoglycemia. In contrast to insulinoma, NIPH is a global histological change consistent with nesidioblastosis. In the past, nesidioblastosis was mostly diagnosed in infancy. With recent availability of bariatric surgery for obesity, reports of adult-onset nesidioblastosis have increased in numbers. For our patient, recurrent postprandial hypoglycemia and the symptoms of neuroglycopenia raised the suspicion of NIPH. However, work-up to rule-out insulinoma was necessary. Multiple diagnostic
modalities were negative for insulinoma. The patient opted for pancreatectomy of the tail and body; however, surgical intervention alone did not resolve recurrent hypoglycemia. The patient still required medical management. It remains unclear when the current therapy will become ineffective for our patient. Total pancreatectomy would be a definitive treatment; however, it would result in IDDM. There are reports of bariatric surgery reversal and resolution of hypoglycemia.

References:


**A CASE OF CURABLE BLINDNESS**

**Umema Burney DO, Verde Valley Medical Center, PGY-2**

Posterior reversible encephalopathy syndrome (PRES) is characterized by altered consciousness, headaches, visual disturbances and seizures. A 61 year old male with past medical history of CAD with stents, hypertension, dyslipidemia, history of TIA, and hepatitis C presented to the Emergency Department with confusion, severe headache and complete vision loss. Interestingly, the visual disturbance was not reported by the patient, who believed he was able to see without issue, but rather by his wife who noted the patient was holding onto walls while moving around. This raised suspicion for Anton’s syndrome. Upon examination, the patient’s blood pressure was 230/120 and a nicardipine drip was started. Given the patient’s symptoms, hemorrhagic stroke was suspected and a head CT was ordered. The head CT was negative for a bleed and the patient was admitted to ICU for continued management. An MRI of the brain was obtained which demonstrated increased T2 signal intensity in the occipital lobes, strongly suggesting PRES. Further titration of the nicardipine resulted in significantly improved blood pressure, and consequent resolution of the patient’s blindness and confusion, which provided clinical confirmation of PRES. Recognition of PRES and timely diagnosis are critical as the condition is reversible. A diagnosis of PRES should be considered in the setting of hypertensive crisis, if renal disease is present, or the patient is on immunosuppressive therapy. If recognized and treated early, most patients will return to their previous state of health.

**GASTROINTESTINAL INVOLVEMENT IN A PATIENT WITH GRANULOMATOSIS WITH POLYANGIITIS**

**Jagman Chahal MD, Maricopa Medical Center, PGY-2**

Carlos Hartmann-Manrique MD, Shannon Skinner MD, Sheetal Chhaya DO, Roselyne Vutien MD

Introduction: We report a case of a middle aged female who presented with upper and lower respiratory symptoms, acute kidney failure and gastrointestinal (GI) bleed, all found to be attributed to
granulomatosis with polyangiitis (GPA, formerly known as Wegener’s granulomatosis). Case Report: A 57-year-old female with no significant past medical history, presented with a two month history of frontal headaches associated with rhinorrhea, congestion, fever and chills. The patient had previously been treated with antibiotics for suspected sinusitis with no improvement. Due to worsening sinus symptoms, a new complaint of right upper quadrant abdominal pain, and intermittent gross hematuria, the patient presented to the hospital for evaluation. On initial evaluation, patient was tachycardic and tachypneic. She had mucosal edema, rhinorrhea, and oropharyngeal exudate. Her initial laboratory evaluation was significant for a white blood cell count of 29,700/µL, hemoglobin of 11.3 g/dL, platelets of 820 K/µL, creatinine of 6.26 mg/dL, and potassium of 6.9 mmol/L. Urinalysis showed gross hematuria and mild proteinuria. C-ANCA was positive. Chest CT showed multifocal consolidation and ground glass opacities in the upper lobes and superior segments of lower lobes. Renal biopsy showed focal segmental glomerulonephritis with a rare cellular crescent (5%), acute tubular injury and mixed interstitial inflammation. Patient was diagnosed with Enterobacter pneumonia for which she received antibiotics and also GPA for which patient was started on pulse cyclophosphamide and pulse steroids. During the course of hospitalization, patient’s renal function improved and she was feeling better. However, after the fourth day of hospitalization, the patient developed hematochezia with a drop in hemoglobin. The patient underwent EGD and colonoscopy. The colonic mucosa showed scattered, at least 10, punched out ulcers with heaped up circumferential margins. Biopsies were taken and showed vasculitic lesions characterized by fibrin necrosis of arteries with rare multinucleated giant cells and peri-arterial inflammation in the submucosa. She was continued on steroids at the time of discharge and has been followed routinely in the rheumatology clinic. Her renal function has improved and she is currently considered to be in remission. Discussion: Granulomatosis with polyangiitis is a systemic vasculitis of medium and small arteries and venules. The mechanism by which it occurs is unclear. It typically involves the upper and lower respiratory tracts along with glomerulonephritis. GI involvement is uncommon and one study detected intestinal involvement in 24% of cases at necropsy and reports of significant clinical manifestations are rare. GI involvement tends to occur after presentation of initial symptoms and GI symptoms can include odynophagia, abdominal pain, nausea, vomiting, diarrhea and GI bleeding. Perforation or ischemia can also occur and can be fatal. Conclusion: Although uncommon, in patients with GPA who develop abdominal symptoms, intestinal involvement due to GPA should strongly be considered.

CATS & BITES: A JOINT VENTURE

Nam Chan MD, Mayo Clinic Arizona, PGY-2
Bryan Hull, MD, Holenarasipur R. Vikram, MD

Introduction Skin and soft tissues are the most common sites of Pasteurella multocida infection after animal bite or scratch. Although uncommon, joint infections and septicemia as complications of Pasteurella infection have been reported. We present a case of P. multocida sepsis complicated by right hip prosthetic joint infection in the setting of rheumatoid arthritis. Case description A 60-year-old Caucasian woman with a history of severely deforming rheumatoid arthritis status post multiple joint arthroplasties, on chronic low-dose prednisone; and T-cell lymphoproliferative disorder with red cell aplasia previously treated with alemtuzumab, presented with three days of fever and worsening right
prosthetic hip pain. On exam she was febrile, tachycardic, her right hip was painful on movement and her left shin was erythematous and warm. Blood cultures were drawn and she was started on empiric vancomycin and piperacillin-tazobactam to cover for cellulitis and possible septic arthritis in an immunocompromised host. Ultrasound of the right hip showed complex fluid collection in the vicinity of the neck of the prosthesis. Fluoroscopy-guided joint aspiration was positive for gram negative bacilli. One of two blood cultures became positive after 12 hours with preliminary report of gram negative bacilli resembling haemophilus. Vancomycin was discontinued. She was taken to the operating room by Orthopedic Surgery on day 2 for incision and drainage of the right hip, debridement, and placement of antibiotic beads. On day 3, final blood culture showed beta lactamase negative Pasteurella multocida, therefore her antibiotic was switched to ampicillin. The next day, right hip joint aspirate culture grew the same organism, as did the intraoperative cultures. On further investigation, she takes care of stray cats and had cat scratches to her left shin one week prior. She was discharged on ceftriaxone to complete a 6-week course, with minimal right hip pain follow-up. She is currently on a 3-month course of oral amoxicillin for suppression. Discussions Septic arthritis is a rare complication of P. multocida infection. In a case series of 20 ICU patients with Pasteurella infection during a 12-year period, there were 6 cases of septic arthritis. A review published in 2010 found 18 cases of total joint arthroplasty infection with P. multocida infection in the preceding decade and identified risk factors such as immunocompromised state, advanced age and diabetes. All but one case was attributable to a distant infectious wound from cat or dog bites, scratches or licks. The knee is the most commonly affected joint. Patients with prosthetic joints who are immunosuppressed may be educated about the risk of serious infection associated with cat and dog bites, scratches, or licks. Post exposure prophylaxis may be considered in high risk individuals such as our patient.

**AN UNUSUAL CASE - FEVER OF UNKNOWN ORIGIN**

_Nathan Copeland MD, UACOM at University Campus, PGY-2_

Corinne Self

Hemophagocytic lymphohistiocytosis (HLH) is an aggressive and life-threatening syndrome of excessive immune activation. It is most common in infants and young children but can affect patients of any age, with or without a predisposing familial condition. Adults have a normal variant called MAS which is along the same spectrum of disease but presents later in life. Both can be associated with autoimmune triggers. 58 year old woman with Systemic Lupus Erythematosis directly admitted by her outpatient rheumatologist to University of Arizona Medical Center for evaluation of recurrent fevers, weight loss and multiple blood transfusion over the past 4 months. Medical record review revealed multiple potential etiologies of the patient’s complaints including ongoing outpatient evaluation for GI neoplasm, long standing rheumatologic disease and chronic immunosuppression. Physical exam notable for Temperature &#8805;38.5°C and Splenomegaly. Initial evaluation and labs reveal anemia of chronic disease, thrombocytopenia, Ferritin of 20548, Fibrinogen- 761, Soluble IL-2- 11,020 (normal 0-1033). Through careful follow up of all abnormalities and careful test selection- an unusual, rapidly life threatening and previously missed diagnosis of hemophagocytic lymphohistiocytosis/ macrophage activating syndrome (HLH/MAS) was confirmed on bone marrow biopsy. Our patient ended up meeting 7/8 criteria (only 7 tested). Patient underwent chemotherapy with etoposide and dexamethasone and is
currently undergoing induction for allogeneic HCT. Presented as an exercise in the systematic evaluation of undifferentiated fever, this case demonstrates investigation of a dauntingly broad differential by etiology and importance of pre-test probability and clinical decision making in this uncommon but very important consideration for FUO. Diagnosis of HLH only requires any 5 of the 8 criteria. Most only meet 3 so low threshold to biopsy of bone marrow and to consider empiric treatment. Untreated HLH has a survival of months due to progressive multi-organ failure.

**BLOODY DIARRHEA FROM COLOVESICULAR FISTULA: A COMPLICATION OF URETERAL STENT PLACEMENT**

Melissa Crawley MD, UACOM at University Campus, PGY-2

Usman Ajaz, MD

Ureteral stent migration is a known late complication of stent placement and may have additional clinical consequences in patients with genitourinary malignancy. A 63-year old male was evaluated for watery, bloody diarrhea (20-30 episodes daily) with tenesmus for two months associated with 50-lb weight loss. He recently was treated with an empiric course of oral vancomycin for possible Clostridium difficile infection with no improvement. He has history of metastatic prostate cancer with radical prostatectomy and more recently transurethral resection of a nodule in the prostatic fossa. He also had obstructive uropathy and had bilateral ureteral stents placed, followed by subsequent right nephrostomy tube placement for obstruction of right ureteral stent. Physical exam was unremarkable except for black stool about the rectum but with no blood on finger. Initial laboratory evaluation showed leukocytosis and iron deficiency anemia, EGD showed salmon-colored mucosa seen in the esophagus, normal stomach and duodenum. No histopathologic abnormalities were seen on biopsy of esophageal or intestinal tissue. On colonoscopy, a ureteral stent was visualized in the rectum; scope was not advanced further. Tissue surrounding the stent was necrotic and inflamed with friable mucosa. A CT of abdomen and pelvis showed rectal wall thickening and a rectovesicular fistula with a diameter of 1.1 cm, interval progression of the local intrapelvic prostatic malignancy and moderate left hydronephrosis and fat stranding concerning for obstruction and pyelonephritis. Left nephrostomy tube was placed for moderate hydronephrosis with 90 ml of pus drained. E. coli susceptible to cefazolin was cultured. He was discharged to interim care on cefazolin. On a later date, he underwent radical cystectomy and pelvic exenteration with creation of end colostomy and ileal conduit urinary diversion. The rectum and bladder were both noted to be involved in a mass. Biopsy of this mass showed both prostatic adenocarcinoma and urothelial carcinoma with extensive squamoid metaplasia. The prostatic adenocarcinoma showed an unusual staining pattern that was different from his previous prostate cancer. The bilateral ureters were also taken for biopsy. The left showed urothelial carcinoma and the right showed prostatic adenocarcinoma. This case illustrates a rare complication of a relatively common interventional procedure. Ureteral stent migration is typically considered a late complication and occurs in up to 4.2% of patients post-procedure. Given the prevalence of obstructive uropathies and prostatic disease in general medicine patient population, fistula formation due to stent migration should be a consideration in patients who have undergone urological intervention and present with diarrhea that is not attributed to an infectious etiology and in particular, patients with malignancies who have undergone radiation therapy due to concern for radiation cystitis and proctitis.
THE DRIER THE BETTER: DOES RISING CREATININE FOLLOWING ULTRAFILTRATION IN ACUTE DECOMPENSATED HEART FAILURE PREDICT READMISSION?

Santosh Desai DO, Banner Good Samaritan, PGY-2
Sarika Desai DO, Amandeep Khurana MD, Akil Loli MD

Introduction: Hospitalizations for acute decompensated heart failure (ADHF) have been increasing and costs related to readmissions account for a majority of total cost of care. Ultrafiltration (UF) is now being considered for ADHF refractory to medical therapy. Purpose: To evaluate clinical outcomes in ADHF among patients treated with UF Methods: A retrospective study was conducted at Banner Good Samaritan and Banner Heart Hospitals between 1/1/2006-11/01/2012 identifying patients with ADHF who received UF. Continuous data are reported as means and standard deviations. A Mann-Whitney U test was used to compare differences in continuous variables. Results: There were a total of 67 patients, 68.7% of whom were male. More than half (55.2%) of the patients were classified as having severely reduced ejection fraction (EF). Of the 67 patients, 43 had chronic kidney disease (CKD) and 22 patients developed acute kidney injury (AKI) during treatment. 19 patients (28.4%) were placed on dobutamine; 14 of these had a severely reduced EF. Serum creatinine increased on average by 0.30 (0.62), mean BNP decreased by 1675 (2897), and mean weight decreased by 7.4 kg (9.6). For our study group, overall 30-day readmission rate was 23%. There were 6 in-hospital deaths. Of the 61 patients who survived their admission, the 30-day readmission rate was 6.2% for patients who developed AKI and 29% for those who did not (p=0.064) [table 1]. Also, of the 61 patients who survived, there was no significant difference in 30-day readmission rate between those with CKD (22.5%) and those without (23.8%). Interestingly, those who died during the index hospitalization had a greater increase in serum creatinine than those who survived [table 1]. Conclusion: An increase in serum creatinine for patients with ADHF treated with UF appears to be a predictor of in-hospital mortality, while a lesser increase seems to be predictive of 30-day readmission.

PASTEURELLA MULTOCIDA BACTEREMIA RESISTANT TO VANCOMYCIN, IN AN IMMUNOSUPPRESSED PATIENT

Nathan Duffin DO, Sierra Vista Regional Health Center, PGY-2

Pasteurella Multocida Bacteremia resistant to vancomycin, in an immunosuppressed patient Nathan Duffin, DO, Associate, Midwestern University Jonathan Mahn, DO; Alfonso Llano, MD Introduction: Pasteurella Multocida is a component of the normal upper respiratory tract flora of fowl and mammals, especially felines. When humans come in contact with these animals through bites, scratches, or licks an infection can ensue. Wound infections typically create pain, swelling, purulent drainage, lymhagitis, and regional lymphadenopathy may also be present. Failure to obtain an accurate history and providing appropriate treatment can result in unwanted complicated outcomes such as sepsis, bacteremia, endocarditis, osteomyelitis, pneumonia, and peritonitis. Case description: A 59 year old Caucasian female with a history of end stage liver disease, and chronic cellulitis presented to the emergency department with a two day history of increasing pain and redness in her right lower extremity. She also had fevers, chills, and generalized malaise. She was found to be in sepsis and acute renal failure. The presumed source was cellulitis. She was started on intravenous vancomycin. The pt. began to
deteriorate further. The blood cultures obtained were positive for Pasteurella Multocida. The antibiotic was switched to intravenous zosyn. The patient was then asked about possible pet exposure. She did admit to having 5 cats that scratch and bite her frequently. MRI of right lower extremity failed to show evidence of osteomyelitis. It is not common to have bacteremia from pasteurella multocida but due to the pt. end stage liver disease and subsequent immunosupression she was found to have bacteremia. Once the appropriate antibiotic was given, the patient gradually improved and was discharged on Augmentin po. She was also counseled to avoid pet exposures due to her immunosupression. 

Discussion: Infections caused by Pasteurella Multocida most likely cause cellulits, but when a patient is immunosuppressed the likelihood of other serious complications increases. Failure to recognize the source of her infection in a detailed history can result in unwanted complications and future recurrences. Pasteurella Multocida is usually resistant to vancomycin. It wasn’t until we put the patient on zosyn that the pt. responded. We also recommended the patient avoid contact of cats and dogs due to her immunosupression and chronic cellulitis.

Second Place – Research Poster
IMPACT OF HEMATOCRIT ON SYMPTOM BURDEN AMONG POLYCYTHEMIA VERA PATIENTS

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Roberto Marchioli, MD; Amylou Dueck, PhD, Holly L. Geyer, MD,Tiziano Barbui, MD, Ruben A. Mesa, MD

Background: Current guidelines suggest that polycythemia vera patients maintain a strict hematocrit less than 45%. The recent CYTO-PV trial (N Engl J Med 2013;368:22-33) found that patients undergoing strict hematocrit control had significantly decreased mortality from cardiovascular death and thrombosis, but suffered from overall increased symptom burden. To date, little is known about the relationship between hematocrit control and PV patient symptom burden.

Methods: PV patient data was analyzed from the CYTO PV trial and the MPN-SAF study (JCO 2012; 30(33):4098-4103). The CYTO PV trial was a randomized, clinical trial assessing the impact of traditional (hematocrit maintained at less than 45%) versus experimental (hematocrit maintained at 45% to 50%) hematocrit control. The MPN-SAF study was an international assessment of MPN symptom burden at the time of an office visit (Cancer 1999;85(5)1186-1196). Surrogate hematocrit was calculated from MPN-SAF data as three times hemoglobin.

Results: Patients and Baseline Symptom Burden: Symptomatic burden was compared between 224 CYTO PV trial patients and 561 MPN-SAF PV patients. Mean age, gender, and distribution of prognostic scoring were similar between the two studies. Among the MPN-SAF cohort, 299 PV patients had hematocrit up to 45%, 89 had hematocrit between 45 to 50%, and 23 with hematocrit greater than 50% (150 no data). Hematocrit Control and Symptom Burden: For both cohorts, end-organ complaints including headache (MPN-SAF 2.1(65%) vs 1.7(54%), CYTO PV 6mo change +0.4 vs +0.0) and cough (MPN-SAF 1.6(48%) vs 1.3(46%), CYTO PV 6mo change +0.3 vs +0.1) were more severe in those with hematocrit greater than 45%. Conversely, strict hematocrit control was associated with increased
problems with concentration (MPN-SAF 2.4(63%) vs 2.1(59%), CYTO PV 6mo change +0.6 vs +0.4) and insomnia (MPN-SAF 3.0(68%) vs 2.8(66%), CYTO PV 6mo change +0.5 vs +0.2). Strict hematocrit control was also associated with increased weight loss (MPN-SAF 1.1(34%) vs 1.0(32%), CYTO PV +0.2 vs -0.3) and night sweats (MPN-SAF 2.1(53%) vs 1.8(49%), CYTO PV 6mo change +0.4 vs 0.0). Hematocrit Control and Fatigue: When comparing MPN-SAF patients, patients with lower hematocrit had significantly worsened fatigue (89% vs 81%, p=0.04). Similarly, analysis of CYTO PV patients undergoing six months of strict hematocrit control had increased worst fatigue (+0.4 vs -0.2, p=0.08) and mean BFI (+0.2 vs -0.3, p=0.04) at 6 months. Conclusions: This study assesses the impact of hemodynamic management among two of the largest prospective evaluations of PV symptom burden to date. Iron deficiency likely contributes to cognitive and fatigue complaints seen among patients with strict control. Conversely, vascular complications may contribute to the end organ complaints seen in those with lenient control. Future studies are needed to further delineate differences in the means of hematocrit control (medicinal vs. phlebotomy) on PV associated symptom burden.

**LACTOBACILLUS: CAN IT BE DEADLY? A RARE CAUSE OF BACTEREMIA**

Rachel Erickson DO, Sierra Vista Regional Health Center, PGY-2

Nusrum Iqbal, MD

Lactobacillus: Can It Be Deadly? A Rare Cause of Bacteremia Rachel Erickson, D.O. OGME-2, Internal Medicine Resident, Sierra Vista Regional Health Center, Sierra Vista, AZ Dr. Nusrum Iqbal, M.D., Department Chair of Internal Medicine, Sierra Vista Regional Health Center, Sierra Vista, AZ Lactobacillus, the Gram positive, facultative anaerobic rod, is part of the normal flora of gastrointestinal and genitourinary regions. However, this organism can be a dangerous cause of bacteremia. If it is not diagnosed early, patients can experience signs and symptoms of sepsis. There are approximately 40-89 cases of Lactobacillus bacteremia on record. While the significance remains unclear, Lactobacillus is becoming more prominent as a source of bacteremia. Risk factors include prolonged hospitalizations, multiple antibiotic uses, an immunocompromised state, history of cancer or Diabetes Mellitus, and recent surgical procedures. In this specific case, an 80 year-old Caucasian male with a history of previous Methicillin Resistant Staphylococcus Aureus in 2012 with prior hospitalizations, s/p aortic valve replacement, and Diabetes Mellitus presented with two day onset of altered mental status and weakness. On physical exam, his temperature was 101.3 with all other vitals stable. Kernig and Brudzinski signs, nor focal neurological defects were seen. An Abdominal/Pelvic CT showed no abdominal abscess or tumor, CT Head without contrast was negative, Chest X-ray was negative for pneumonia, and two electrocardiograms were normal. A urinalysis was negative, and a complete echocardiogram discovered no vegetations, mass, or thrombus. He was started on IV fluid resuscitation, empiric Vancomycin, Zosyn, and close monitoring of mental status. Results of the subsequent blood cultures were tedious to determine, and were eventually positive for Lactobacillus species. As a result, antibiotic treatment was changed to Clindamycin. Approximately four days later, the patient’s mental status had improved. Follow up blood cultures continued to have positive Lactobacillus six weeks after discharge. Antibiotic treatment was then repeated. The case demonstrates that it is imperative that Lactobacillus be recognized as a possible pathogen in bacteremia. In general, while the mortality rates...
have been reported up to approximately 14%, proper identification of Lactobacillus as the causative pathogen could impact this significantly.

**RECURRENT ORALABIAL HERPES ASSOCIATED SEPSIS**

**Sumaya Farran MD, UACOM at University Campus, PGY-2**

Tirdad T. Zangeneh, DO, MA

Herpes viridae- HSV1 (which produces most cold sores) and HSV2 (which produces a variety of illnesses including mucocutaneous herpes) are ubiquitous and contagious. HSV-1 tends to reside in the trigeminal ganglia while HSV-2 tends to reside in the sacral ganglia. The virus can be reactivated by illnesses ranging from stress, common colds to immunosuppression state. Here we describe a 66 years old Caucasian female who was admitted through emergency department for fever of 39°C and lethargy of 1 day duration. She was resuscitated for a working diagnosis of septic shock with fluids, stress dose of hydrocortisone and IV antibiotics (vancomycin and cefepime). Past medical history of Asthma, Hypothyroidism, Addison’s disease requiring treatment with cortisone 5 mg twice a day for 2 years, recurrent oralabial herpes, Parkinson disease and multiple spinal fusion surgeries. No recent illness, sick contacts or travel. Two bedside lumbar punctures were unsuccessful because of multiple fusion surgeries. White blood cell count was 9,800 cells/ml (83% neutrophils). Chest & Abdomen Imaging, Blood cultures for bacteria and fungi were negative at 48 hours. No infectious source could be identified. On day 2, White cell count increased to 12,100 cells/ml (90% neutrophils and 9% lymphocytes). Patient developed two ulcerative lesions on the lower lip and was administered oral Valacyclovir for one day for herpes gingivostomatitis and antibiotics were discontinued. On day 3, patient developed odynophagia. Patient developed extensive ulcerative lesions in the oral cavity and nares. No genital lesions or skin rash was noted. Acyclovir 400 mg Q8h IV started with rapid improvement and tolerance to liquids intake. Swabs from the oral lesions and blood test for HSV1/2 PCR were obtained. HSV-1 was isolated from PCR of oral lesions and serum PCR viral load indicated 300 copies/ml (Normal value-Not detected). This confirmed the diagnosis of HSV-1 gingivostomatitis resulting in viremia and clinical presentation most consistent with sepsis. It was evident that her sepsis was due to reactivation of HSV-1 infection with history of recurrent oralabial herpes resulting in gingivostomatitis and viremia but without any apparent organ involvement on CT scan. The patient had significant clinical improvement and was discharged home with Valacyclovir 1 gram oral Q8h for seven days followed by acyclovir 400 mg oral twice dialy for lifelong suppression of reactivation of orolabial HSV. Patients with HSV viremia have high rate of mortality especially with PCR evidence of viremia. Viral dissemination to blood stream and viscera is higher in immune compromised patients like ours (daily systemic steroids for Addison disease). Physicians should recognize the possibility of sepsis caused by HSV infection with concomitant viremia.

**ISOLATED PULMONARY VALVE ENDOCARDITIS**

**Aaron Fernandes MD, UACOM at South Campus, PGY-2**

Dr. Varun Takyar, MD, Dr. Dr. Rajesh Janardhanan MD

Introduction: Right Sided Infective Endocarditis (RSIE) accounts for 5-10% of all cases of infective endocarditis. Isolated pulmonary valve endocarditis (IPVE) is the rarest form, accounting for 1.5-2% of
all cases. Predisposing factors include IVDA, alcoholism, sepsis and catheter-based infections. Here, we present one such case of IPVE with a review of literature. Case Presentation: A 27 year-old female with known opiate abuse presented with 2 day history of fever, shortness of breath, and back pain. Physical exam revealed a febrile, tachycardic female with bilateral crackles and rhonchi. Labs revealed a normal white cell count, albeit with neutrophilia and lactic acidosis at 4.3 mg/dL. CT of the chest was ordered which showed multiple masses with cavitations. Given the severe sepsis, broad spectrum antibiotics were started and early fluid resuscitation commenced. In light of the clinical picture, an echocardiogram was ordered. A transthoracic echo (TTE), and subsequently a trans-esophageal echo (TEE) confirmed a large, pedunculated, 3.5 cm vegetation on the pulmonary valve with prolapse into the right ventricular outflow tract. Blood cultures grew methicillin-sensitive Staph Aureus for which antibiotics were changed to IV Nafcillin. Given the large size of the bacterial vegetation with a multi-focal pneumonia, cardiothoracic surgery was consulted and took the patient to OR for valvular debridement. She tolerated the procedure well and was subsequently given a 6 week course of IV antibiotics. Patient had an uneventful post-operative course and was discharged with close outpatient follow-up. Given the lack of clinical evidence to support surgical vs medical management, we did a retrospective analysis of all documented cases of IPVE using PubMed from 1960 to 2013. A total of 122 cases of pulmonic IE were found of which 22 cases were IPVE. The most common organisms were Staph. Aureus (45.5 %) and gram negative bacilli (18.1 %). The average size of the vegetation was 2.4 cm. Major risk factors were IVDA and catheter related infections. 41 % of patients had surgical intervention. The most common complication was recurrent septic pulmonary emboli and the mortality rate was 18.1 %. Discussion: Right-sided IE most commonly presents with pulmonary symptoms. IPVE is quite rare and therefore guideline-based support for treatment is lacking. High suspicion and early diagnosis are keys to optimizing outcomes as per published case reports. Sensitivity of TTE is generally low and a TEE is needed for definitive evaluation. Generally accepted indications for surgery are worsening cardiac function, recurrent pulmonary emboli, and persistent febrile episodes despite appropriate antibiotic therapy. As in our case and in agreement with the review of literature presented here, persistent febrile episodes and minimal improvement with large size vegetations make a compelling case for surgical debridement.

**COLONIC LIPOMA MASQUERADING AS A MALIGNANT TUMOR**

Colin Fitterer MD, Mayo Clinic Arizona, PGY-2
Tonia Young-Fadok, MD

Introduction Gastrointestinal lipomas are benign, non-epithelial tumors that are typically small and asymptomatic. The following describes a rare case of a large, symptomatic colonic lipoma that masqueraded as a malignant tumor. Case Description An 84 year-old male presented to our institution complaining of abdominal pain, hematochezia, and unexplained weight loss. He was found via colonoscopy to have a large, fungating cecal mass that was highly suspicious for malignancy. Biopsy of the mass was obtained and pathology was consistent with a hyperplastic polyp. As this was discordant with the colonoscopic findings, it was felt that surgical resection would be the most prudent and definitive treatment. Thus, he underwent laparoscopic right hemicolecctomy. The surgical pathology yielded a 4.5cm polypoid, submucosal lipoma with extensive ulceration and no evidence of malignancy.
Unfortunately, his early post-operative course was complicated by an inferior wall myocardial infarction approximately one week post-discharge from the hospital. He subsequently recovered well with no recurrent gastrointestinal complaints. Discussion Large, fungating masses identified by colonoscopy generally represent colorectal cancer. This case serves to highlight the rarity of large gastrointestinal lipomas masquerading as malignant tumors. Autopsy reports of small, incidental colonic lipomas have shown an incidence of up to 4.4%, but there have been a very limited number of previous case reports of large colonic lipomas (exceeding 2cm in diameter) that produced symptoms. The most commonly reported symptoms have included abdominal pain, hematochezia, and alteration in bowel habits. Colonoscopic findings may be misleading as large colonic lipomas have been found to have varying degrees of fat necrosis and ulceration, suggesting more serious pathology. Therefore, there have been efforts to increase pre-operative diagnoses to determine the most appropriate intervention. One therapeutic option includes endoscopic cautery snare resection. However, some studies have shown a significant risk of perforation with attempted endoscopic removal of gastrointestinal lipomas &gt;800;2cm. Ultimately, surgical resection remains the preferred treatment for most large, symptomatic gastrointestinal lipomas.

**AMBIGUOUS NEUROENDOCRINE TUMOR**

Diana L Franco MD, Mayo Clinic Arizona, PGY-2

Halfdanarson TR

Introduction: Neuroendocrine tumors (NET) are a heterogenous group of rare tumors. They can arise from any anatomic location but are most commonly seen in the gastrointestinal tract and lungs. Most pancreatic NETs (pNETs) are nonfunctional. However, we describe a pNET case that secreted two different hormones. Case: A 59 year old female with past medical history significant for type 2 diabetes mellitus (DM) and metastatic asymptomatic gastrinoma was referred for a case of poorly controlled Cushing’s syndrome. One year prior to the referral the patient underwent an evaluation for lesions in the pancreatic tail and liver. She had an elevated gastrin levels of 7500 pg/ml (&lt;100) but no peptic ulcer disease. A pancreatic biopsy showed a WHO grade 2 well differentiated neuroendocrine tumor (NET) for which she underwent distal pancreatectomy, splenectomy and resection of multiple liver metastases. Three months later, progression was noted in the liver and a hepatic radioembolization with yttrium-90 loaded beads was performed. Octreotide therapy was begun but discontinued due to adverse effects. On presentation the patient complained of mood changes, facial hirsutism, acne, skin darkening; weight gain, weakness and SOB for the last 7 months. Her DM had become more difficult to manage and she became hypertensive. Laboratories revealed gastrin: 24 pg/ml (&lt;100), Adrenocorticotropic hormone (ACTH): 218 pg/ml (10-60 AM) and normal Corticotropin-releasing hormone. A computed tomography of abdomen showed liver metastases. A clinical diagnosis of ectopic ACTH syndrome secondary to progressive and metastatic NET was made. Given her very severe and rapidly worsening symptoms, a bilateral adrenalectomy was urgently performed and long-acting octreotide therapy was begun. Within 2 weeks after the surgery, all of her symptoms had substantially improved. She remains on octreotide and will follow up with serial imaging and tumor markers. Discussion: This unique case illustrates a highly unusual presentation of a NET manifesting as Cushing’s syndrome secondary to ectopic ACTH production where urgent adrenalectomy dramatically improved her symptoms of hypercortisolism, and
the octreotide alleviated the consequences of excessive ACTH secretion. A fascinating aspect of this case is that the initial gastrin producing NET transformed to an ACTH producing tumor and lost its ability to produce gastrin. To our knowledge, this is the first reported case of gastrinoma that converted to an ACTH secreting NET but a case of nonfunctional NET transforming to a gastrinoma has previously been reported.

**HEPATOCELLULAR CARCINOMA IN A YOUNG FEMALE WITH FAMILIAL ADENOMATOUS POLYPOISIS**

**Hyon-he Garza MD, UACOM at University Campus, PGY-2**

Jamie Fleming

Hepatocellular carcinoma in a young female with familial adenomatous polyposis. Hyon-he Garza, MD. Jamie Fleming. University of Arizona Medical Center. Tucson, Arizona. Familial adenomatous polyposis (FAP) is an autosomal dominant disease caused by germline mutations of the adenomatous polyposis coli gene. Extracolonic manifestations are known to be associated with FAP however it is rare for patients to have hepatic neoplasms. Hepatoblastoma and hepatic adenoma are typically the type of hepatic tumors that are associated with familial adenomatous polyposis. There are only a few reported cases of FAP that have been associated with hepatocellular carcinoma [HCC]. We report a case of a 28-year-old woman with known FAP who presented with a diagnosis hepatocellular carcinoma. A 28-year-old African American woman presented with a four-month history of progressively worsening right upper quadrant abdominal pain. She presented to the emergency department as the pain had become intolerable associated with nausea. Her past medical history includes familial adenomatous polyposis which she underwent a total colectomy for at age 12. Her family history includes a strong history of FAP involving her father, two sisters, paternal aunts and paternal grandmother. Physical exam revealed tachycardia and profound tenderness to palpation in the right upper quadrant of the abdomen. Hepatomegaly could not be appreciated as exam was limited due to pain. There was no rebound tenderness or other peritoneal signs. Laboratory tests showed elevated liver function assays, normal alpha-fetoprotein and negative hepatitis A, B, and C. An MRI of the abdomen demonstrated multifocal HCC involving both right and left lobes of the liver and findings of chronic liver disease. To confirm the diagnosis in the presence of normal alpha-fetoprotein, a CT guided liver biopsy was obtained and showed well to moderately differentiated HCC. FAP is known to be associated with extracolonic manifestations however it is rare for patients to have hepatic neoplasms. This case illustrates a possible connection of FAP contributing to development of HCC at a young age. From 1950-2011, only 10 cases have been reported describing the association of FAP and HCC. Of the reported cases of associated FAP and HCC, the youngest patient was diagnosed with FAP at age 15 and subsequently developed HCC at age 28 like this patient. This patient was diagnosed with classical FAP at an early age of 12. She did not have any typical risk factors that would predispose to HCC. The patient was ultimately evaluated for a debulking procedure via transcatheter arterial chemoembolization with Yttrium.
A SIMPLE CASE OF APPENDICITIS, OR SO IT SEEMED...

Carlo Guerrero MD, Mayo Clinic Arizona, PGY-2

Thorvardur Halfdanarson, MD

INTRODUCTION: Appendiceal tumors are relatively rare neoplasms that are often diagnosed incidentally on specimens obtained during otherwise routine appendectomies. They include a variety of histologic subtypes and their management requires such differing modalities as reoperation for hemicolecction, systemic chemotherapy, or simple observation depending on tumor stage and pathology.

CASE PRESENTATION: A 68-year-old female with no significant past medical history presented to an outside emergency department with four hours of severe abdominal pain, nausea, and chills. A contrast-enhanced CT of the abdomen and pelvis revealed a distended appendix 15mm in diameter with an appendicolith contained within its proximal end. Local inflammatory changes were seen, and the final impression was of an acute appendicitis. The patient underwent a laparoscopic appendectomy, which was converted to an open laparotomy due to a retrocecal appendix as well as a complicating phlegmon. No visual evidence of malignancy was observed during the operation. Pathologic evaluation of the appendix revealed a 6.5cm goblet cell tumor spanning the entire length of the appendix and invading the serosa with positive surgical margins. As no regional lymph nodes were collected, the tumor received a pathologic stage of pT4 NX. The patient was seen for a consultation in our oncology clinic, where we recommended referral to surgical oncology for a hemicolecction with lymphadenectomy to complete the tumor staging, followed by adjuvant chemotherapy with a FOLFOX regimen if she were found to have node-positive disease.

DISCUSSION: Appendiceal tumors may present as appendicitis and are found on 1% of all appendectomy specimens. Their growth can precipitate appendicitis by effectively closing off the appendix from the rest of the cecum, as occurred with our patient. They may also be discovered incidentally on abdominal imaging, or when patients become symptomatic from increased tumor burden (e.g. abdominal pain, ascites). They are classified into two main subtypes: carcinoid and epithelial tumors. Carcinoid is the most common subtype, accounting for > 50% of cases. Like other carcinoid tumors, they can produce a classic carcinoid syndrome, which may be the presenting manifestation. These are treated surgically, with octreotide, hepatic regional embolization, or everolimus given for metastatic disease. The epithelial tumor subtype includes adenocarcinoma, mucocoeles, and goblet cell carcinomas. Adenocarcinomas are most aggressive and are treated with surgery and adjuvant chemotherapy. Mucocoeles produce mucin, and when spread to the peritoneum cause pseudomyxoma peritonei, where mucin fills the abdomen causing a characteristic “jelly belly.” Goblet cell tumors, sharing features of both adenocarcinomas and carcinoids, have an intermediate prognosis. The epithelial subtypes are treated with hemicolecction and adjuvant chemotherapy. Aggressive surgical debulking with intraperitoneal hyperthermic chemotherapy (IPHC) is offered as a potentially curative therapy for intraperitoneal disease at selected specialty centers.
RELAPSING POLYCHONDRITIS

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Veenu Gupta MD and Sheetal Chhaya DO

Introduction: Relapsing polychondritis (RPC) is an immune-mediated condition associated with inflammation in cartilaginous structures and other tissues throughout the body, particularly the ears, nose, eyes, joints, and respiratory tract. There is evidence of autoimmunity to extracellular matrix components of cartilage. Onset can be triggered by trauma with equal incidence in both males and females, mostly between the ages of 40 and 60. Auricular involvement is the most common feature with pronounced pain and tenderness, usually misdiagnosed as infection and treated as otitis externa. Recurrent episodes lead to “floppy ear” or “cauliflower” appearance. Inner ear involvement leads to impaired hearing, tinnitus, or vertigo. Eye involvement is in the form of scleritis, keratitis or uveitis. Nasal involvement can lead to “Saddle Nose Deformity”. Large airway involvement can lead to obstructive respiratory disease and post-obstructive pneumonias. Parasternal joint involvement is typical. Heart involvement manifests as aortic and/or mitral regurgitation caused by valve inflammation. Diagnosis is usually made based on the clinical features which, when not obvious, require a biopsy. No form of therapy has been shown to modify the natural history of the disease, although successful suppression of the clinical features can be achieved with NSAID, dapsone or prednisone as the first line treatment followed by cyclophosphamide, azathioprine, cyclosporine, methotrexate, etanercept, infliximab, or adalimumab. The most frequent causes of death are infection, laryngotracheal or bronchial involvement (especially stricture or collapse) with resultant respiratory failure or superimposed pneumonia.

Case: 58-year-old female presented with complaints of tenderness and redness of pinna of the right ear. She was treated for otitis externa. Her symptoms persisted despite multiple trials of antibiotics. Subsequently, she also developed intermittent pain of her nasal bridge, followed by continued chest wall pain mostly of the lower ribs and occasional shortness of breath. She also complained of intermittent hearing loss in the right ear, which usually lasted a few hours. She was referred to our rheumatology clinic. On examination, her vital signs were normal. No rashes, lesions or significant scars were visible on the skin. She had tenderness to palpation of nasal bridge. Her right ear was red, swollen and tender. She also had chest wall tenderness. Lab findings were unremarkable. Based on the clinical criteria, she was diagnosed with relapsing polychondritis. She responded very well to treatment with methotrexate, etanercept and prednisone.

Discussion: Early manifestations of this disease are usually subtle and are often unrecognized for prolonged periods. Hence the diagnosis is frequently obtained only after the emergence of multiple classic features such as auricular inflammation, saddle-nose deformity or costochondritis. There are also no measures to predict the specific manifestation, severity and course of the disease in individual patients.
A CASE OF LETHAL CONSTIPATION

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Aswani Alavala, MBBS, Gordon Carr, MD, Bujji Ainapurapu, MBBS

Introduction: Stercoral colitis is an uncommon condition in which severe constipation leads a situation similar to bowel wall ischemic colitis. We describe a case of stercoral colitis which resulted in a fatal outcome highlighting the importance of early recognition and appropriate management of this rare clinical entity.

Case: A 65-year-old woman with a past medical history of COPD, hypertension, diverticulitis, GERD, chronic constipation and anxiety presented with severe diffuse abdominal pain of 1 day duration accompanied with non bloody non bilious vomiting. Her last bowel movement was 3 weeks ago. On physical examination she was afebrile but severely hypotensive with BP 70/30 mm hg. She had moderate distention and diffuse tenderness of the abdomen without rigidity or rebound. Hypoactive bowel sounds were present. Rest of the examination was normal. Laboratory exam showed metabolic acidosis with (pH 7.19), normal lactate and acute kidney injury. CT abdomen showed extensive stool throughout the colon, fecalization of distal ileum indicating longstanding stasis. After consultation with Surgical service medical management was planned. The patient was initially thought to be in septic shock, but thorough examination including lab work and imaging did not reveal any focus of infection. Meanwhile the patient's condition deteriorated with refractory shock and respiratory failure. A diagnosis of stercoral colitis was made. The patient was given oral lactulose and lactulose enemas and tap water enemas and manual disimpaction was also tried but she did not have a bowel movement. Her lactate continued to rise and her pressor requirement increased, so surgery was reconsulted and surgeons recommended open laparotomy and colonic resection. However patient's family refused to consent for surgery. On day 3 of hospitalization as condition was declining comfort care was opted by her family members and the patient passed away.

Discussion: Fecal impaction leading to colonic obstruction is seen primarily in elderly, less often in young patients who are neurologically impaired. Stercoral colitis is an inflammatory process involving the colonic wall related to fecal impaction. Severe long standing constipation resulting in build up of hard stool in the colon can lead to high pressure on the colonic wall. When this transmural pressure exceeds the perfusion pressure it can lead to ischemia and inflammation of the colonic wall, stercoral ulcers, micro-perforations and sometimes even macro-perforations. This in turn can result in to peritonitis, lactic acidosis, shock and multiorgan failure. Management should be aimed at relieving constipation either by conventional measures or sometimes even with use of colonoscopy. Early surgical intervention is needed in many cases to remove the affected bowel.

Conclusion: Early recognition of Stercoral colitis and understanding it's serious consequences is essential for appropriate management and improved outcomes.
PANCYTOPENIA: DON’T FORGET TO CHECK THE TSH

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Surabhi Amar MD, Harikrishna Dave MD

Introduction: Pancytopenia can have varied causes, including drugs (examples: carbamazepine, phenytoin, sulfonamides), infections (such as viral hepatitis or HIV), myelodysplastic syndromes, or endocrine disorders (such as hypopituitarism). We report a case of severe hypothyroidism presenting with pronounced pancytopenia. Case: A 50-year-old man presented with a few week history of weakness. He had no fever, weight loss, night sweats, hoarseness, skin changes, constipation, dizziness, orthostasis or bleeding. Patient had severe pallor and bilateral lower extremity edema. His white blood count (WBC) was 1.2 x10³/µL, hemoglobin (Hb) of 5.5 g/dL, platelets (PLT) 190 K/µL, thyroid stimulating hormone (TSH) 134.9 mIU/L. Serum folate, serum B12, LDH, liver and kidney functions were normal. 24-hour urine protein was 8 grams. Bone marrow was hypocellular with 35-40% cellularity, trilineage hematopoiesis, and mild dyspoietic changes were noted but there was no evidence of a lymphoproliferative disorder. Flow cytometry revealed a small population of CD55-deficient granulocytes and lymphocytes, consistent with a PNH clone. There was no chromosomal abnormality detected. Kidney biopsy showed focal segmental glomerulosclerosis (FSGS) with 80% effacement of podocytes, without any autoimmune complexes, suggesting primary FSGS. Levothyroxine and high dose steroids were started. Two weeks later, blood counts showed WBC of 3.9 x10³/µL, Hb 11.4 g/dL, PLT 299 K/µL and a TSH of 32.2 mIU/L. A repeat bone marrow biopsy done eight months later showed a cellularity of 50% with active trilineage hematopoiesis; the PNH clone was no longer noted. Proteinuria had improved to 5.2 grams/24 hours. 17 months after initiation of thyroid hormone replacement therapy, patient’s blood counts had normalized with Hb of 16.1 g/dL, WBC 6.2 x10³/µL, PLT 186 K/µL; TSH 27 mIU/L and urine protein of 5.9 grams/24 hours. Patient was subsequently lost to follow-up.

Discussion: Hypothyroidism is associated with normocytic or macrocytic anemia in 20% to 60% of cases. Its role in the hematopoiesis of platelets and white blood cells is less well understood. Thyroid autoimmune diseases are associated with thrombocytopenia in 83% of cases but thrombocytopenia is not usually seen with hypothyroidism. Mouse studies suggest hypothyroidism is associated with impaired B cell maturity, though studies in humans are limited. Pancytopenia has often been reported as a presentation of hypopituitarism (Sheehan’s syndrome—which is associated with hypothyroidism), but rarely as the sole manifestation of hypothyroidism. There is a single case report of a patient with hypothyroidism presenting with pancytopenia and concomitant nephrotic syndrome (NS), both of which resolved with thyroid hormone replacement, but the pathophysiology of this association remains unclear. In our patient the NS was felt to be primary and unrelated to the thyroid disorder. Conclusion: Hypothyroidism is a rare but highly treatable cause of pancytopenia. Treatment of hypothyroidism can reverse bone marrow abnormalities in these cases.
COMMONLY OVERLOOKED SCENARIO WITH DANGEROUS CONSEQUENCES

Juxiang Huang DO, UACOM at South Campus, PGY-2

Uday Kanakadandi MD

Commonly Overlooked Scenario with Dangerous Consequences  Juxiang (Jennifer) Huang, DO; Uday Kanakadandi, MD. University of Arizona College of Medicine at South Campus  Torsades de pointes (TdP) is a polymorphic ventricular tachycardia which commonly occurs in the setting of QT prolongation. QT prolongation can be congenital or acquired. Electrolyte abnormalities as well as many commonly used medications can prolong the QT interval and increase risk of TdP. TdP can degenerate into ventricular fibrillation and lead to death. Early recognition, correction of those electrolyte abnormalities and awareness of the drugs that could prolong QT interval are vital aspects of patient management. A 52 year-old female with past medical history of Diabetes Mellitus type 2 and recent colostomy due to perforated diverticulitis presented to the emergency room with nausea, vomiting and increased stool output through colostomy. Laboratory studies revealed low potassium at 3.6 and low bicarbonate at 11. EKG revealed sinus rhythm with a HR rate of 76 beats/ min and prominent inverted U waves in multiple leads which were fused with the T wave. The QT interval was markedly prolonged at 590 msec (QTc by Bazett’s formula 670 msec), which was not recognized on the automated EKG interpretation. Patient was given on oral metronidazole, IV hydration and ondansetron as needed for her nausea and was admitted to a non-telemetry floor. On hospital day three, she was found unresponsive, in shock. She was resuscitated and intubated. Post resuscitation, patient underwent an emergent cardiac catheterization, which showed angiographically normal coronaries. 2D trans-thoracic echocardiogram showed normal LV function. She was then transferred to the ICU where she was found to have multiple episodes of TdP and QT interval was found to be 550 - 600 msec. Her electrolytes were promptly corrected. Her serum potassium was sustained at greater than 4.5 mEq/L. Ondansetron was discontinued. With these measures, her QT interval came down to 480 msec and the episodes of TdP stopped. TdP is a potentially fatal arrhythmia. The most common causes of TdP are congenital long QT syndrome and drugs. Several routinely used antidepressants, antibiotics and antipsychotics can cause dose dependent prolongation of QT interval. Electrolyte imbalance especially hypomagnesemia and hypokalemia are also known to worsen the QT interval which could lead to TdP. Our patient suffered a major life threatening adverse cardiac event from a preventable cause in spite of not having any primary cardiac pathology. Female patients and patient with electrolyte abnormalities are more susceptible to drug induced QT prolongation as well as TdP.

RARE CAUSE OF BACTEREMIA AND RENAL ABSCESS BY ACTINOMYCES MEYERI : A CASE REPORT

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Sadia Moinuddin, MD, Wesley Ray Shealey MD

Actinomyces meyeri is a rare cause of renal abscess. Actinomycosis has been reported since the late 19th century, with the majority of reported cases involving Actinomyces israelii with orogingival and cervico facial infection. Actinomyces species are normal oral, gastrointestinal and genital tract flora,
having a tendency to invade tissue planes when there is mucosal breakdown. Actinomyces meyeri infection is rarely reported but cases involve pulmonary, soft tissue and skin, gastrointestinal, skin, cardiac, brain or muscle infection. Most cases are non-fatal and require long duration of antibiotic treatment. We present a case of a 29-year-old female with history of left nephrolithiasis status post percutaneous nephrolithotripsy 1 month prior to admission. She presented with fever, chills, nausea, and vomiting for two days. She had a temperature of 39.3 Celsius, a heart rate of 134 bpm, and a respiration rate of 24. Her physical exam was significant for left costophrenic angle tenderness. The patient’s lab demonstrated WBC of 15.3 (3.6-11.1), and a creatinine of 1.22 (0.57 - 1.11). Renal ultrasound report noted a 3.9 cm complex cystic structure in the superior pole of left kidney and Computed Tomography (CT) urogram noted findings of left renal and peri-renal abscesses. The patient was started on intravenous Levaquin. After administration of intravenous antibiotics, her fever, chills, and leukocytosis resolved. She underwent CT guided percutaneous drain of her left renal abscess. The patient’s blood culture and left renal abscess culture grew Actinomyces meyeri. She had a reported penicillin allergy and was discharged home with intravenous doxycycline for seven weeks. After three weeks, her left percutaneous renal drain was removed and repeat CT at the time showed resolution of left renal abscess. After seven weeks of intravenous doxycycline, the patient was started on oral clindamycin in anticipation of total antibiotic treatment of at least 6 months with follow up imaging. In our case, a young female patient who had percutaneous intervention for her nephrolithiasis presented with actinomycosis one month after the procedure. Actinomyces meyeri is a rare causative agent for actinomycosis; less than 40 cases have been reported in English literature since the early 20th century. Additionally, Actinomyces meyeri shows a predilection for dissemination compared to other Actinomyces species. Our case is unique as our patient presented with kidney abscess after percutaneous intervention, compared to majority of reported cases with pulmonary disease. Treatment for the infection involves extensive intravenous antibiotic treatment for extended duration and often involves surgical intervention to remove the source. The infection usually responds well to antibiotic therapy and the mortality rate is low. The antibiotic of choice is penicillin but with penicillin allergic patients, doxycycline or clindamycin are alternative options. The final duration of treatment is tailored based on clinical or radiological response.

**First Place – Case Report Poster**

**TO TREAT OR NOT TO TREAT: A RARE CASE OF PSEUDO-THROMBOTIC THROMBOCYTOPENIC PURPURPA IN A JEHOVAH’S WITNESS**

Arooj Kayani MD, St. Joseph’s Hospital & Medical Center, PGY-2

Rashmi Kumar, Mahesh Seethram, Farzad Sakha

Background: Thrombotic thrombocytopenic purpura (TTP) is a rare microvascular occlusive disorder characterized by systemic intravascular aggregation of platelets, thrombocytopenia and mechanical injury to erythrocytes. A pentad of thrombocytopenia, microangiopathic hemolytic anemia, neurologic abnormalities, renal failure and fever has been associated with TTP. However, a triad of thrombocytopenia, schistocytosis, and elevated lactate dehydrogenase (LDH) levels is often sufficient to suspect this potentially catastrophic diagnosis. We report a rare case of pernicious anemia presenting...
with microangiopathy, thrombocytopenia and neurologic changes, diagnosed initially as life threatening thrombotic thrombocytopenic purpura.

Case: A 46-year-old Jehovah’s witness female presented with nausea, vomiting, diarrhea and epigastric pain for 2 days, and fatigue and parasthesias for 4 weeks. Examination revealed pallor, slow mentation and lower extremity weakness and paresthesias. Initial laboratory evaluation showed severe anemia and thrombocytopenia. MCV, total bilirubin and LDH were elevated. Peripheral blood smear showed schistocytes, macro-ovalocytes and hypersegmented neutrophils. TTP was suspected due to the combination of anemia, neurological symptoms, thrombocytopenia, schistocytes and elevated LDH. Plasmapheresis was offered to the patient but she refused it due to her religious belief. Due to the presence of macroovalocytes and hypersegmented neutrophils, vitamin B12 level was checked and found to be extremely low. Anti-intrinsic factor antibodies and anti-parietal cell antibodies were also positive; hence a diagnosis of pernicious anemia was established. Treatment with intramuscular vitamin B12 was initiated, which resulted in dramatic neurologic and hematologic improvement. The ADAMS-TS 13 level which is used to confirm diagnosis of TTP came back within normal range, arguing against a diagnosis of TTP. Discussion: Vitamin B12 deficiency can lead to elevated levels of homocysteine in the blood. Homocysteine can cause endothelial dysfunction which can lead to formation of microvascular thrombi. Due to this phenomenon, vitamin B12 deficiency can rarely present with schistocytes and thrombocytopenia, which combined with other stigmata of vitamin B12 deficiency, can be misdiagnosed as TTP, and hence called pseudo-TTP.

Conclusion: It is important for clinicians to be aware of unusual presentations of vitamin B12 deficiency since the treatment is simple, and can avoid unnecessary therapies such as plasmapheresis.

**A CASE OF EXTRA-NODAL LYMPHOBLASTIC LYMPHOMA**

**Shubha Kollampare MD, UACOM at University Campus, PGY-2**

Sara Park

Lymphoblastic lymphoma is a type of lymphoid malignancy affecting bone marrow or extra nodal tissues respectively. It is a disease of the young occurring in late teens or early twenties. Abdominal involvement with extra nodal disease is rare. When it occurs, liver and spleen may be involved commonly. There are very few case reports of extra nodal lymphoblastic involvement of the kidneys. Since kidneys do not contain lymphoid tissues, renal involvement is extremely rare. 19 year old male with seizure disorder and ADHD was admitted with high blood pressure and acute kidney injury. Elevated lactate with no evidence of regional or localized hypo perfusion was noted. An ultrasound of the kidneys showed bilateral enlarged kidneys without hydronephrosis with concern for an infiltrative process. A renal biopsy was performed showed monotonous sheet of small to medium sized cells with scanty cytoplasm and effacement of the normal renal parenchyma. Immunohistochemical stains confirmed presence of CD1a, CD3, CD5, CD43 and TdT positive lymph oblasts that co-expressed CD4 & CD8. CD34 was negative. These findings were consistent with lymphoblastic lymphoma involving renal parenchyma. Treatment entails high dose multi-drug chemotherapy in hope of curing the disease. Early detection in combination with systemic chemotherapy may improve the prognosis. Our patient was started on CHOP therapy. Febuxostat to prevent Tumor lysis syndrome. A rapid decrease in lactic
acidosis from 11.5 to 0.5 was observed. Presence of enlarged kidneys with no evidence of hydronephrosis should prompt physicians to obtain biopsy. Although ultrasound, computerized tomography, magnetic resonance imaging, bone scan, bone marrow biopsy help in staging the disease; renal biopsy is the single test in establishing the diagnosis. Flow cytometry for specific cell typing and genetic testing may be considered to document chromosomal abnormalities and may have prognostic implications.

**PANCREATIC NEUROENDOCRINE TUMOR AND THE IMPORTANCE OF ENDOSCOPIC ULTRASOUND**

Eugene Larsen DO, Banner Good Samaritan, PGY-2

Introduction: Pancreatic neuroendocrine tumors (pancreatic NET’s) are rare neoplasms (annual incidence approximately 0.2 per 100,000) that arise from the endocrine tissue of the pancreas. They can secrete a number of peptide hormones resulting in a number of clinical syndromes. However, many are non-functioning and therefore their diagnosis is delayed.

Case presentation: A 78 year old male presented to the Phoenix VA emergency department complaining of a 4 week history of diarrhea. He states that his stool is non-bloody and clay colored. He reports approximately 5-8 bowel movements a day. He has had intermittent abdominal pain for the past 10 years following a colostomy secondary to bacterial peritonitis from a diverticulum rupture. On physical exam the patients abdomen was soft and non-tender with normal bowel sounds and he was found to be jaundiced. The patient denied any travel outside of Arizona or drinking of any well water. In the ED labs were drawn and patient’s total bilirubin was 9.2 with mild elevation of liver enzymes. Patient’s glucose was normal with alkaline phosphatase and lipase both extremely elevated at 438 and 3113, respectively. A CT scan of the abdomen was performed which did not demonstrate any pancreatic mass. However, the common bile duct was dilated at 13mm at the pancreatic head with tapering in the pancreatic head consistent with a mass or radiolucent stone. No obstructing mass was identified near the ampulla. At this time the patient’s symptoms were thought to be secondary to a stone and the patient’s diarrhea was hypothesized to be from pancreatic insufficiency. The patient was admitted to the general medical floor. After evaluation by GI, the patient had a EUS performed which demonstrated a 30 mm hypoechoic heterogenous pancreatic head mass. Pathology of a fine needle aspiration demonstrated malignant cells that stained positive for chromogranin, CD56, and synaptophysin confirming the diagnosis of pancreatic neuroendocrine tumor. After a full body PET scan demonstrated extensive liver, lymph node, and skeletal metastasis, the patient was determined to not be a surgical candidate. He had an external biliary drain placed for palliative purposes. All peptide hormone testing was negative, including vasoactive intestinal peptide, demonstrating that this pancreatic NET was likely non-functioning. The patient was started on pancreatic enzymes which did alleviate his diarrhea.

Discussion: This case demonstrates the wide variety of symptoms that can be associated with pancreatic NET’s. It also demonstrates the widening functionality of endoscopic ultrasound and the capability to perform fine needle aspiration even after patients have undergone other imaging modalities that have greater resolution. Recognition of the numerous clinical syndromes caused by pancreatic NET’s is an excellent tool for any well -rounded clinician to have.
BETWEEN A ROCK AND A HARD PLACE

Adil Lokhandwala MD, UACOM at South Campus, PGY-2

Rene Franco Jr. MD; Magdiel Trinidad MD; Bujji Ainaapurapu MD.

Introduction: May-Thurner Syndrome (MTS) (aka: ilio-caval compression syndrome or Cockett syndrome), is an anatomic variant characterized by Left Lower Extremity (LLE) venous hypertension resulting from venous obstruction of the Left Common Iliac Vein (LCIV) from compression between the overlying Right Common Iliac Artery (RCIA) and underlying L5 vertebral body with or without ilio-femoral deep vein thrombosis (DVT). It can commonly present with unilateral lower extremity pain, swelling, recurrent thrombosis or it may be noted on incidental imaging findings without any clinical presentation.

Case: We present a case of a 69 year old lady with chronic LLE swelling diagnosed two years ago as lymphedema who presented with acute worsening of LLE swelling in the setting of recent left knee replacement three months ago. She had a negative venous doppler of the LLE one year ago. No prior history DVT/PE or coagulation disorders. LLE appeared significantly swollen with 3+ pitting edema, blanching erythema and cyanosis of all five toes and the plantar surface of the foot. Pulses were 1+ at dorsalis pedis and posterior tibial artery. CT Venogram showed an extensive DVT of left common femoral vein extending into the pelvic veins and compression of the LCIV between RCIA and spine. She underwent intraoperative venogram which confirmed an extensive clot burden with minimal flow into the IVC. IVC filter was placed followed by mechanical thrombectomy of clot within the LCIV with Trellis device. Patient was kept on a continuous TPA infusion for 24 hours and taken back to the catheterization lab to assess degree of thrombolysis. Repeat venogram this time showed flow limitation within the LCIV which was confirmed by an intravascular ultrasound. Angioplasty and stenting of LCIV performed without complications. Patient was started on warfarin for anticoagulation. Repeat duplex ultrasound of LLE 10 days post discharge showed widely patent left iliac stent. IVC filter removed 21 days post discharge without thrombus within the filter.

Discussion: MTS is a commonly missed diagnosis and may present as a life threatening totally occluded DVT leading to circulatory compromise which can be triggered by recent lower extremity surgery or trauma to the affected side. Compression ultrasonography has a low sensitivity for pelvic vein thrombosis, especially in obese individuals. If LCIV compression remains a clinical suspicion, MR Venography or CT Venography is a good option. Systemic anticoagulation is not adequate and mechanical thrombectomy with angioplasty and stent placement is definitive treatment with additional surgical bypass options. Conclusion: The intent of this case report is to increase awareness of this commonly missed condition and the need for studies to provide guidelines for identification of these individuals prior to undergoing lower extremity surgery to avoid post-operative lower extremity DVT and life threatening circulatory compromise.
FULMINANT HEPATIC FAILURE FROM RARE PRIMARY ANAPLASTIC SMALL CELL CARCINOMA OF THE LIVER

Scott McShane DO, Banner Good Samaritan, PGY-2
Ruth Franks-Senenedcor MD, Michele Young MD, David Perry DO

Small Cell Carcinoma (SCC) typically occurs as a principle malignancy in the lung. A primary liver SCC is a rare finding which may be difficult to recognize and treat. A 78 year-old man came to the emergency department with concern of altered mental status. In the previous month he had developed lower extremity swelling, somnolence, confusion, and loss of appetite. Symptoms of which had worsened at an alarming rate, in addition to his developing jaundice over the past day. The patient was seen by his primary care physician (PCP) one month prior to presentation and at that time his liver function tests were normal. At that visit, his PCP had changed his simvastatin to lovastatin and began the patient on 500mg of acetaminophen (APAP) up to three times daily for chronic headaches. The patient was in good health with well controlled chronic conditions other than a long history of tobacco abuse. The patients denied alcohol use for more than 30 years and had no knowledge of viral hepatitis. The newly added APAP was reportedly being used 1-2 times per week. His exam revealed a confused, icteric, jaundiced male with distended abdomen and positive fluid wave. His liver function tests were elevated including a total bilirubin of 25.5 and INR of 1.4. CT scan of the abdomen showed ascites and a nodular appearing liver without evidence of biliary dilation. Ultrasound of the liver showed ascites and patent vessels without evidence of blockage. Paracentesis confirmed portal hypertension without infection. APAP toxicity was initially expected; however, his APAP level was low. Despite this, N-Acetylcysteine treatment was initiated as the time-line for ingestion was questionable. The patient was transferred to the ICU; he developed severe shock and his mental status quickly declined in spite of lactulose and therapies provided by the ICU. His family determined that he would not want heroic efforts made to preserve his life. Within 48 hours of admission, the patient died from fulminant hepatic failure. An autopsy was performed to determine the cause of death. The liver was grossly nodular and histological evaluation of the liver revealed anaplastic SCC. No other primary site was discovered. The most common site of SCC is the lung. It has rarely been found at extra-pulmonary sites such as the trachea, larynx, thymus, esophagus, stomach, small intestine, colon, prostate, gallbladder, skin, breast, and uterine cervix. Small cell carcinoma, involving primarily the liver, is extremely rare with few cases reported in the literature.

HEPATITIS C TREATMENT BARRIERS AND SOLUTIONS: PREDICTORS OF OUTCOME FOR TREATMENT OF HEPATITIS C WITH TRIPLE THERAPY IN A SAFETY NET HOSPITAL SETTING

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Keith Brussman, Renee Prevette NP, Andre Valdez PhD, Firoozeh Isfahani, Abdul Nadir MD

Background: Treatment of hepatitis C with protease inhibitors (PIs): boceprevir and telaprevir in combination with PEG-Interferon alfa and ribavirin showed significant improvement of the clinical outcome. However, the excellent results of HCV therapy in the research setting have not been
reproduced in the clinical practice. We present our experience with triple therapy. The aim of our study was to identify the barriers and solutions in treatment of hepatitis C with triple therapy.

Method: We have prescribed triple therapy for 56 patients who were referred to our hepatology clinic at Maricopa Integrated Health System between February 2011 and January 2013. Thirty eight patients (68%) have been followed for a minimum of 12 weeks to determine SVR (sustained virological response).

Results: There were 19 patients who were successfully treated and achieved SVR compared to 19 who could not achieve SVR. 10/19 (53%) and 9/19 (47%) in each group received boceprevir and telaprevir, respectively. 4/19 in SVR group had prior treatment experience in comparison with 9/19 in non-SVR group. 15/19 in SVR group in comparison with 10/19 in non-SVR group were treatment naïve. There was no statistically significant difference between the median age, sex, BMI, platelet count and genotype between SVR and non-SVR groups. Patients with RVR (rapid virological response) and mild fibrosis did better than patients without RVR and advanced fibrosis. 4/19 (21%) had cirrhosis in SVR group compared to 6/19 (32%) in the non-SVR group. Non-SVR patients were more frequently hospitalized; received more blood transfusions and did not have a payor source and received medicines via pharmaceutical company assistance programs. In the non-SVR group, 6/19 (32%) could not complete 12 weeks of treatment compared to 1/19 (5%) in the SVR group.

Conclusion: Hepatitis C treatment failure was predicted by management of adverse effects, limited resources, advanced fibrosis and absence of RVR. Efforts to decrease patient side effects are warranted as are attempts to optimize the outcome of hepatitis C treatment. We suggest that dedicating an RN to HCV therapy clinic can improve patient compliance and ultimately outcome of HCV triple therapy.

**TYPICAL APPENDICITIS WITH ATYPICAL GRAVE CONSEQUENCES**

Kelly Noyes DO, Sierra Vista Regional Health Center, PGY-2

William Elliott, DO; Jodi Jenkins, MD; Ali Madani, MD; Thomas Nabhani, MD

Typical appendicitis with atypical grave consequences Kelly Noyes, DO, Associate, Midwestern University. William Elliott, DO; Jodi Jenkins, MD; Ali Madani, MD; Thomas Nabhani, MD

Introduction: Appendicitis is a diagnosis typically reserved for surgical discussions, but pathology may indicate clear medical indications for intervention as this case describes. Case Description: A 67 year old male presented to the Emergency Department with diffuse abdominal pain, worst in the right lower quadrant. A clinical diagnosis of acute appendicitis was made and the patient underwent emergent appendectomy without complication. Pathology later revealed acute appendicitis, but also revealed invasive, moderately differentiated mucinous carcinoma. The patient returned to hospital and underwent right hemicolecction. Additional pathology did not reveal residual malignancy or lymph node involvement. His course was complicated by perioperative non-ST segment elevation myocardial infarction. After undergoing quadruple coronary artery bypass grafting the patient began radiation to retroperitoneal area where appendix was adherent. Chemotherapy was not initiated due to cardiomyopathy after myocardial infarction. Discussion: Malignancy is found on less than 1% of all appendectomies and mucinous appendiceal carcinoma is present in only 37% of cases. Malignancy is not suspected at the
time of surgery in the majority of cases, highlighting the importance of pathologic examination. Diagnostic and treatment options have frequently favored right hemicolecction, but this case illustrates unforeseen consequences of additional surgery. Lymph node involvement rarely occurs and does not change prognosis, limiting the use of traditional TNM staging and minimizing the need for right hemicolecction for this reason alone. Several treatment options including appendectomy versus right hemicolecction, radiation, systemic chemotherapy and intraperitoneal hyperthermic chemotherapy are available, but due to the rarity of mucinous appendiceal carcinoma large volume studies are limited. This case illustrates the importance of careful patient selection when determining treatment.

PAGE KIDNEY: HYPERTENSION IN THE PRESENCE OF A PERINEPHRIC HEMATOMA

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Page kidney named after Irwin Page refers to the development of hypertension secondary to extra-renal compression of the kidney. The generally accepted pathophysiology of Page kidney dysfunction is that external compression causes hypoperfusion, ischemia, and activation of the renin-angiotensin-aldosterone system. The classic presentation usually involves compression caused by a subcapsular hematoma secondary to blunt abdominal trauma or an invasive procedure such as renal biopsy. Very few cases, however, have been reported of Page kidney-related hypertension developing after spontaneous perirenal hemorrhage. This is the case of a 48 year old Hispanic male whose past medical history consisted only of an incidental finding of C4-5 spontaneous fusion on CT scan resulting in diagnosis of Klippel-Feil Syndrome in 2011. He had no documented history of any genitourinary anomalies that can sometimes be associated with this syndrome. In February 2013, he presented to the emergency department with right lower quadrant abdominal pain. Physical exam revealed a blood pressure of 183/118 and right-sided CVA tenderness. CT scan was performed to rule out appendicitis, and on preliminary read showed an enlarged right kidney with areas of hypodensity as well as perinephric stranding. He was admitted with a diagnosis of pyelonephritis complicated by possible abscess and started on antibiotics. However, overnight his hemoglobin dropped from 14.1 to 8.4, while his creatinine increased from 1.4 to 2.7. Follow-up MRI revealed a focal bleed in the inferior renal parenchyma and anterior cortex of his right kidney with cortical breakthrough resulting in a large perinephric hematoma. Six units of prbc’s failed to stabilize his hemoglobin. At this point, he underwent transcatheter embolization of the right renal artery. Notably, the patient’s elevated blood pressure present on admission persisted and proved difficult to control despite treatment with Norvasc, Metoprolol, and Hydralazine. ACE inhibitors and ARB’s were avoided given his renal insufficiency. With the goal of preserving his kidney, we opted for observation and medical management instead of surgical options like capsulectomy or nephrectomy. He was eventually discharged with Norvasc 10 mg, though his systolic blood pressures remained in the 150’s. He has since been followed as an outpatient and serial imaging has revealed steady improvement in the size of his perinephric hematoma. He has also since been titrated off all blood pressure medications about 4 months out after discharge. This case illustrates that even when the history does not include trauma or renal biopsy, Page kidney should not be excluded as a possible diagnosis when it is supported by the clinical picture. Suspicion must be high as it could mistakenly pass as essential hypertension. Also, despite there being no clear treatment recommendations for Page kidney, this case provides an example of one approach that spared as much renal function as possible.
USE OF IMPELLA 3.5 PERCUTANEOUS LEFT VENTRICULAR ASSIST DEVICE AND EXTRACORPOREAL MEMBRANE OXYGENATION IN SETTING OF SEVERE BETA-BLOCKER TOXICITY: A CASE REPORT

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Akil Loli MD

Introduction: Cardiovascular medications are an increasing category of medications associated with significant substance-related morbidity and mortality in the United States. In setting of severe &beta;-adrenergic receptor blocking (BB) toxicity cardiogenic shock may result. Early aggressive medical interventions including the Impella 3.5 percutaneous left ventricular assist device and extracorporeal membrane oxygenation resulted in survival and positive functional outcomes in our case report. This is the first documented case of the Impella left ventricular assist device being utilized in the setting of severe &beta;-adrenergic receptor blocking toxicity resulting in cardiogenic shock.

Case presentation: A 44 year-old female with a past medical history of depression attempted suicide by ingesting approximately 90 tablets of 50mg Atenolol along with unknown quantities of Chlorthalidone and Lisinopril. Patient subsequently developed cardiogenic shock requiring intubation and multiple inotropic and vasopressor support to maintain mean arterial pressure. After discussion as a multi-collaborative team (cardiology, critical care, toxicology) it was decided to undergo aggressive early intervention due to severity of &beta;-adrenergic receptor antagonist toxicity. Insulin, sodium bicarbonate and calcium gluconate infusion were initiated to counteract the metabolic acidosis and &beta;-blocker toxicity, respectively. Lactic acid at time of presentation was 1.8 with elevation to 5.5 within 12 hours after presentation. Bedside echocardiogram was performed revealing a left ventricular ejection fraction (LVEF) of 65% with normal right ventricular (RV) function. Patient was subsequently taken to the cardiac catheterization laboratory in preparation for Impella 3.5 percutaneous placement for left ventricular support and temporary percutaneous pacemaker placement with swan-ganz catheter placement. Shortly after impella and pacemaker placement, the gastroenterology specialists performed bedside esophagogastroduodenoscopy (EGD) to evaluate for and potentially remove any remaining undigested tablets and for the placement of activated charcoal. Furthermore, nephrology services initiated complete renal replacement therapy (CRRT) in setting of acute renal failure. Despite these aggressive medical interventions, patient continued to require Epinephrine 125mcg/min, Norepinephrine 100mcg/min, Phenylephrine 200mcg/min and Vasopressin 0.4units/min for cardiovascular hemodynamic support. The patient eventually required venovenous extracorporeal membrane oxygenation (ECMO) to maximize oxygenation. After an extensive intensive care management, patient was eventually extubated with full hemodynamic stability and was discharged to an inpatient psychiatric ward.

Discussion: The use of the Impella device has not been documented in severe cardiogenic shock due to severe &beta;-adrenergic receptor blocking toxicity. This case proves that early aggressive intervention (inotropic and vasopressor support medications; insulin, calcium gluconate, and sodium bicarbonate infusions; Impella LVAD; CRRT; ECMO) may result in positive outcomes including increased rates of patient survival and resolution of symptoms to baseline function in this patient population.
FROM SKIN TO BONE: A CASE REPORT

Soyoung Park M.D., UACOM at University Campus, PGY-2

Michael S. Flores, M.D.

Spinal cord compression is a common neurological complication of certain forms of cancer. However, reported cases of osseous metastases of cutaneous squamous cell carcinoma are extremely rare with thus far only one case being reported. Otherwise, the closest data that could be found were in two retrospective analyses of patient records done at Memorial Sloan-Kettering Cancer Center from 1964-1970. It was noted that out of 105 patients presenting with spinal cord compression, 8 patients had head and neck cancer as the primary tumor but it was unclear as to whether any of these cancers presented as squamous cell carcinoma of the skin. We present a rare case of osseous metastases of cutaneous squamous cell carcinoma to the spine. Our patient was a 68-year-old gentleman with a past medical history of coccidiomycoses pneumonia, heart and kidney transplants, and squamous cell carcinoma of bilateral ears that had later metastasized to his left neck area which further was treated with surgical dissection and radiation therapy. He had a subsequent PET scan showing increased activity in the left neck and in the right lower lung field. He presented to us with progressive fatigue, muscle weakness, and increasing diffuse back pain along with an eighteen-pound weight loss and decreased appetite. He denied having fevers or chills and his vitals on admission, other than tachycardia, were unremarkable. Neurological exam was non-focal with bilateral intact deep tendon reflexes. However, an MRI of the total spine for evaluation of back pain showed a pathologic compression fracture of T3 with epidural disease extending from T3 to T5 and severe spinal cord compression. A CT-guided bone biopsy of the spinal T5 was done and showed squamous cell carcinoma. Our patient was then started on radiation therapy for palliative care and discharged to home hospice. Spinal cord compression due to metastases usually will present with a history of progressive pain, paralysis, sensory loss, and loss of sphincter control. Various risk factors that increase the chances of cutaneous squamous cell carcinoma metastases include cancer arising in a previously injured site, in certain locations with increased depth and diameter, and specific host factors such as immunosuppression – some examples are organ transplant recipients, lymphoma and AIDS patients – radiation exposure, or bullous diseases. In our case, our patient had a history of both heart and kidney transplants along with a history of the primary cutaneous squamous cell carcinoma of the ear and local recurrence near the neck, all factors that significantly increased his risk for squamous cell carcinoma metastases. The fact that the site of metastases was a spinal vertebral body provides new insight into understanding the behavior of cutaneous squamous cell carcinoma.

DUODENAL DIVERTICULA: UNUSUAL CAUSE OF A COMMON CONDITION

Krupal Patel MBBS, UACOM at South Campus, PGY-2

Varun Takyar, MD, Adil Lokhanwala, MD, Eugene Trowers, MD

Introduction. Duodenal diverticula are a common occurrence, which have been associated with increased incidence of pancreatobiliary diseases. However there is no conclusive evidence linking the presence of chronic pancreatitis and duodenal diverticula. Case presentation. A 68-year-old female
with past medical history of chronic pancreatitis, colonic diverticulosis and chronic diarrhea presented with nausea, bilious vomiting, abdominal pain and significant weight loss. The pain described was typically epigastric with radiation to thoracic spine and no association with food. Over the last few months, patient has presented with recurrent bouts of acute on chronic abdominal pain. She has been compliant to her medications and takes narcotics to alleviate the abdominal pain on chronic basis. She was non-alcoholic with a history of cholelithiasis and cholecystectomy done many years ago. Physical exam revealed tachycardia, epigastric tenderness and umbilical hernia without signs of incarceration. Labs showed leukocytosis and hypokalemia and elevated lipase levels. CT scan of the abdomen showed signs of enteritis and intra and extrahepatic biliary ductal dilation that were chronic. A follow up MRI showed presence of a large duodenal diverticulum adjacent to the ampulla amongst the other findings. Discussion. Duodenum is the second most common region for diverticula to be present after jejunum, in the small intestine. They occur at the weakest spot in duodenal musculature, such as the point of CBD and pancreatic duct entry. The description is based on its relation to the ampulla. Periampullary diverticula are most common, like in this case, followed by juxtrapapillary diverticula arising 2 – 3 cm away from the ampulla. If the ampulla opens into the diverticula, it is referred to as intradiverticular papilla. Duodenal diverticulae have been associated with pancreaticobiliary disease in multiple retrospective analyses. Although a majority of patients are asymptomatic, few have recurrent attacks of biliary and pancreatic disorders. The risk of having a CBD stone in these patients is approximately 6 times greater with presence of periampullary diverticulae. Proposed mechanisms include sphincter spasm leading to increased biliary pressure and inflammation or sphincter dysfunction causing increased reflux of biliary and enteric contents. The exact cause for the increased prevalence of pancreatic disease however remains unknown. The current management is conservative, requiring resection and treatment in only severe disease. Surgical resection is complicated by retroperitoneal position of the duodenum. Newer endoscopic methods have potential role in treatment of this condition. Laparoscopic resection is recommended owing to improved visualization of the anatomical structures and better localization of the ampulla. Conclusion. Physicians should be aware of the association of periampullary diverticuli in the duodenum with chronic pancreaticobiliary disease. Recognition of this entity is important as resection might be indicated in severe disease with relief of symptoms.

**DISSEMINATED COCCIDIOIDES ASSOCIATED WITH STAT 1 MUTATION**

Snehal Patel DO, UACOM at University Campus, PGY-2

Preethi William

Extrathoracic dissemination of coccidioides species is associated with increased mortality. The organism disseminates in less then 0.5% of immunocompetent patients however up to 5% in immunocompromised patients. Recent studies have shown gain-of-function STAT1 mutation leads to irregular functioning of the IFN-γ; mediated inflammatory pathyway putting patients at a higher risk for disseminated disease. It typically affects the coiled domain, which plays a role in STAT1 nuclear dephosphorylation and dimerization of unphosphorylated STAT1. A 17 year old, now 24 years old, native to Arizona female presented with persistent cough for 8 months associated with fatigue, weight loss, and 3 month history of progressively enlarging non-tender right-sided neck mass. Patient had a CT neck that revealed an approximately 4 x 2 cm abscess in the lower right sternocleidomastoid, right apical pulmonary nodules,
Hepatitis and patient’s antibody

Introduction: CHOP/CHOP duration.

Radhakrishnan alfa.

prognosis admission mentioned maxillary PANDORA’S Case

Lymphoma involving from Gowri

mutations considered such BOX PATHOLOGY

Amphotericin paratracheal

At 7/100,000 and overall disease in Millvany, C6 osteomyelitis, lytic lesions were visualized within the right clavicle, sternum, T5, T6 and T10 vertebral bodies and multiple lesions in the liver and spleen. Given the progressive nature of her disease, patient was started on Amphotericin B, which was changed to Posaconazole. Serology revealed a coccidioidal antigen antibody titer of 1:128 initially which decreased to 1:32. PPD was negative. Despite treatment, patient continued to have further progression of her disease and was noted to have calcified lesions in her kidneys. Since her diagnosis, patient has been on multiple antifungals including the ones previously mentioned and itraconazole, voriconazole, and micafungin. Patient had an immunologic workup at the National Institutes of Health and was found to have a gain-of-function STAT1 mutation. She has been on multiple immunomodulatory agents including interferon gamma, GCSF and most recently interferon alfa. At this time patient’s disease is progressing mildly. She continues to have extensive lung disease and new lesions in the liver and spleen. She is currently on voriconazole and SAMe and is been considered for a possible stem cell transplant. Immunocompromised patients, pregnant women and people of Black or Filipino decent have an increased risk of disseminated disease even though most cases overall are reported in Hispanics. The number of cases reported annually in Arizona has increased from 7/100,000 in 1990 to 155/100,000 in 2009. Patients presenting with symptoms including cough, fever and fatigue should be evaluated for coccidiodes via serology. Infections are typically self-limited and resolve within weeks to months. If treated, it is typically anti-fungals including fluconazole. If patient’s disease continues to progress despite treatment, they should have further evaluations for mutations such as STAT1 deficiency.

PANDORA’S BOX TO THE PARANASAL SINUS-FOLLOWING THE PATH OF A RARE PATHOLOGY

Gowri Radhakrishnan MD, UACOM at South Campus, PGY-2

Josephine Taverna MD, Andrew Kovoor Soham Puvvada MD, Vijay Chandiramani MD

PANDORA’S BOX TO THE PARA NASAL SINUS: FOLLOWING THE PATH OF A RARE PATHOLOGY. Gowri Radhakrishnan MD, Josephine Taverna MD, Soham Puvvada MD, Vijay Chandiramani MD.

Introduction: Plasmablastic lymphoma (PBL) is a rare and aggressive variant of Diffuse Large B cell Lymphoma which was initially reported in Human immunodeficiency virus(HIV) infected persons. The prognosis of PBL is poor since majority of patients succumb to the disease despite treatment with CHOP/CHOP like regimen. Over the recent years, some cases have emerged in HIV negative individuals involving sites like stomach, lung, nasal and oral cavities. Out of them, only 3 had involvement of the maxillary sinus. Here we present the fourth case of Maxillary Plasmablastic Lymphoma in a patient without HIV infection and outline the therapy with a novel chemotherapeutic agent, Bortezomib, a proteasome inhibitor – based on recent Phase II clinical trials.

Case Description: 59 years old male presented with progressive abdominal distention of 1 month duration. Medical history was significant for cirrhotic liver disease with portal hypertension, untreated Hepatitis C, COPD, alcoholism and dental abscess of left jaw-self treated 3 weeks ago. Initial labs revealed
acute renal insufficiency with a creatinine of 2.6 and anemia (hemoglobin -12.9), hepatitis C positivity with normal liver function tests. MRI of abdomen showed extensive retroperitoneal lymphadenopathy, thickening of right colon and soft tissue mass compressing bilateral ureters with hydronephrosis. Bilateral nephrostomy tubes were placed. CT guided retroperitoneal biopsy revealed large lymphoid cells with basophilic cytoplasm which stained positive for CD 138, CD 38 (plasma cell markers) and negative for CD20, PAX-5 (pan B-cell markers) suggestive of Plasmablastic Lymphoma. Cells were positive for EBV by FISH. Maxillofacial CT showed soft tissue mass in the left maxillary sinus which was biopsy proven to be PBL. Bone marrow aspirate and biopsy was normal. Biopsies from gastric mucosa, terminal ileum, colon and rectum were negative for malignancy. Endobronchial ultrasound guided biopsy of station 4R lymphnode was positive for lymphoid malignancy. CHOP plus Bortezomib chemotherapy regimen was begun. Patient tolerated the first cycle well and is planned to complete 6-8 cycles of the same.

Discussion: Plasmablastic lymphoma is an aggressive subtype of diffuse large B cell lymphoma, with characteristics of both a lymphoma and myeloma. It has a predilection for immunocompromised individuals with involvement of the oral cavity. Involvement of the paranasal sinuses is very rare with 3 cases reported so far in the literature. Epstein barr virus infection has been associated with pathogenesis. Due to rarity of PBL, optimal treatment regimens have not been defined. Multiple chemotherapy regimens like Hyper CVAD, CHOP have been tried. Most patients succumb within a year of diagnosis.

Conclusion: This case illustrates an infrequent diagnosis of Plasmablastic lymphoma in a HIV negative individual with maxillary sinus involvement and trial of therapy with Bortezomib.

ACUTE ESOPHAGEAL NECROSIS IN A PATIENT WITH SEVERE LOWER GASTROINTESTINAL BLEEDING: A CASE REPORT AND REVIEW

Preethi Reddy MD, UACOM at University Campus, PGY-2

Laila Abu Zaid, MD

Acute esophageal necrosis (AEN) is a clinical syndrome of exceedingly low prevalence and until 2007 was poorly understood. Though its etiology is unclear, it has been theorized to be multifactorial. AEN has been described in the setting of ischemic insult, chemical injury, insufficient protective barriers and impaired reparative system of the esophagus secondary to a wide range of medical co-morbidities that predispose patients to hypoperfusion, back-flow of gastric contents, malnutrition and immunosuppressed states, respectively. AEN, also known as “black esophagus” due to its black appearance is also commonly referred to as “acute necrotizing esophagitis” and “Gurvits Syndrome.” We report a case of AEN in a patient who presented in a hypoperfused state in the setting of diffuse lower gastrointestinal bleeding. Our patient, who has a history significant for vasculopathy, presented with hematemesis following large volume hematochezia and was incidentally found to have striking circumferential black discoloration of the middle and lower thirds of the esophagus with sparing of the gastroesophageal junction (GEJ), findings consistent with AEN. A literature review of the rarity of a black esophagus is discussed along with the role of ischemia in its evolution and the importance of early recognition and timely in preventing its potentially fatal complications. Overall, the prognosis of black esophagus is poor with 32% of patients in the largest case series to date, dying as result of underlying illness or co-morbidities; one-third of them die from superinfection and sepsis. While males, particularly those in
their sixth decade are typically affected, AEN should be suspected in anyone who presents with signs of upper gastrointestinal bleeding and multiple medical co-morbidities. Fortunately, our patient survived and did not suffer from complications as fluid resuscitation and acid suppression were implemented early. Immediate recognition of patients at risk is crucial and should prompt endoscopy for timely diagnosis of AEN. Underlying illnesses and medical conditions need to be treated. Early supportive care can prevent such complications as perforation for which emergent surgical intervention is imperative in preventing death.

**Second Place – Case Report Poster (Tie)**

**ORBITAL APEX SYNDROME: A RARE COMPLICATION OF HERPES ZOSTER OPHTHALMICUS**

Krystal Renszel DO, Banner Good Samaritan, PGY-2

Leonor Echevarria, MD, Elizabeth Tukan, MD

**Introduction:** Orbital apex syndrome is a rare (seven previously reported cases), but potentially sight-threatening, complication of herpes zoster ophthalmicus (HZO). This syndrome often evades initial diagnosis, delaying prompt referral to an ophthalmologist and possible sight-saving interventions.

**Case Presentation:** We report a case of an immunocompromised 74 year old female who, one month prior to presentation, experienced trauma to her left eye and subsequently complained of left eye pain and erythema. She was treated for presumed bacterial conjunctivitis with antibiotic ophthalmic drops, then temporal arteritis with high dose steroids, and finally corneal abrasion. The characteristic vesicular pattern of herpes zoster appeared one week later in the V1 distribution and she was referred to ophthalmology. Despite outpatient treatment with valacyclovir, the patient’s symptoms escalated to include decreased vision, decreased extraocular movements, and periorbital edema. Given her worsening ocular complaints, she was hospitalized for further work-up. Upon presentation, the patient’s visual acuity was 20/200 in her left eye with total ptosis and complete left ophthalmoplegia. MRI showed left posterior temporal and occipital sulci with subtle leptomeningeal enhancement and lumbar puncture demonstrated lymphocytic pleocytosis, consistent with meningitis. Taken together with her ocular findings, a diagnosis of OAS secondary to HZO was made. The patient was started on IV acyclovir and IV methylprednisolone with stabilization over the following days. Due to a national shortage of IV acyclovir, she was discharged on oral valacyclovir and prednisone. At six month follow up, her left ophthalmoplegia had resolved, although she still had mild residual ptosis. Her visual acuity was back to baseline. The patient has initiated a steroid taper and will continue valacyclovir until the taper is completed.

**Discussion:** Comparison of the seven previously reported cases revealed a trend toward improvement with combination therapy of steroids and antivirals and history of ocular trauma and preceding steroid use as possible precipitating events. Treatment with oral valacyclovir in conjunction with steroids can result in marked return of function.

**Conclusion:** This case illustrates how early accurate diagnosis and treatment of this condition can result in a favorable outcome.
OVERLAPPING META-ANALYSES: TRANSCATHETER CLOSURE VS. MEDICAL THERAPY OF PFO IN PATIENTS WITH CRYPTOGENIC STROKE

Irbaz Riaz MD, MM, UACOM at University Campus, PGY-2
Husnain M, Alkashman A, Bilal J, Pasha A, Anderson KL

Objective:

Meta-analyses provide the highest level of evidence in medical research. The number of meta-analyses published per year has increased 17-fold from 1991 (n=334) to 2011 (n=5861). In recent years there has been increasing tendency of overlapping meta-analyses. After the publication of the RESPECT and PC Trials, multiple meta-analyses were published to resolve the controversy surrounding PFO closure in patients with cryptogenic stroke. We present the meta analyses done on the above topic as an example of overlapping meta analyses. Further, we want to critically evaluate if doing multiple studies provided any added information on this topic.

Method:

We performed a comprehensive literature search using Medline (using PubMed and Ovid SP), Embase, Web of Science, Cochrane Library (Cochrane Database of Systematic Reviews, Cochrane Central Register of Controlled Trials) and conference proceedings using the following search terms: Patent foramen ovale, Inter atrial shunt, Transcatheter closure and Cryptogenic stroke.

Results:

Our search resulted in 5 meta-analyses. All of them included the same three randomized controlled trials (PC, RESPECT, and CLOSURE). The primary outcome in four out of the five meta-analyses was composite end point of death, stroke, or TIA. Results were reported as intention to treat (ITT) or per-protocol meta-analysis. ITT analyses failed to show significant benefit of PFO closure in all five meta-analyses.

Conclusion:

The results of these meta-analyses failed to answer the basic question and still there is no closure on the question of PFO closure. The increasing tendency of overlapping of meta-analyses producing similar results is a futile effort and a waste of resources and energies. We recommend better communication between reviewers and registration of protocols for systematic reviews and meta-analyses to avoid these wasteful efforts.

PROGRESSIVELY WORSENING HEADACHE IN AN IMMUNOCOMPROMISED PATIENT

Brentin Roller DO, UACOM at University Campus, PGY-2
Ibrahim Taweel, Snehal Patel, Irbaz Riaz, Kirsten Cooper

Mucormycosis, an emerging invasive fungal infection seen in patients with diabetes, can opportunistically present in other immunocompromised hosts. The responsible fungi are ubiquitous in
nature often infecting patients via inhalation of spores through the nasal turbinates. Subsequent direct angioinvasion results in spread of the infection with potential to progress to rhino-orbital-cerebral mucormycosis (ROCM) and fungemia. A 29 year-old Hispanic female with history of type 1 diabetes, Systemic Lupus Erythematosus, Lupus Nephritis, status-post kidney transplant on Tacrolimus and Prednisone, presented with headache and photophobia with an initial diagnosis of acute exacerbation of chronic sinusitis. Initial findings from CT sinus/orbits and MRI brain showed mucosal thickening of the maxillary and sphenoid sinuses and frontal air cells. The patient was initially started on Augmentin later switched to Vancomycin for presumed bacterial sinusitis. The patient’s headache worsened during her hospital course so a right endoscopic complete ethmoidectomy, frontal recess dissection, maxillary antrostomy with mucous membrane removal and sphenoidotomy were performed on hospital day 8. Purulent fluid was located near the middle meatus on the skull base and the medial aspect of the middle turbinate appeared ischemic. Neither blood cultures, nor cultures obtained from the right ethmoid and frontal sinuses showed fungal elements or growth. Subsequently, pathology from the right middle turbinate biopsy reported vasoinvasive fungal sinusitis consistent with Mucormycosis, at which point IV Amphotericin B and Posaconazole were initiated. Despite appropriate therapy, she did not improve clinically developing proptosis of the right eye with a repeat MRI brain showing edema and thickening of the medial rectus muscle, leptomeningeal enhancement in bilateral orbitofrontal regions and enhancement along the right optic nerve sheath. As she continued to decline clinically her immunosuppressive therapy was discontinued, despite her history of solid organ transplant, which resulted in complete resolution of her presenting symptoms prior to discharge 24 days after admission. She was restarted on low dose Tacrolimus approximately one month after her initial diagnosis and continues to be asymptomatic. In immunocompromised patients presenting with headaches or sinusitis, mucormycosis must be considered as part of the differential diagnoses. Endoscopic sinus examination must be sought as early as possible as the diagnosis of mucormycosis relies upon the identification of organisms in tissue by histopathology as the culture may yield no growth and imaging may be normal. A review of 208 patients with ROCM showed the most significant factors associated with death were delayed diagnosis, presence of hemiparesis/hemiplegia, bilateral sinus involvement, leukemia, renal disease, and treatment with deferoxamine. Overall mortality from ROCM ranges from 25 to 62 percent with the best prognosis in patients with infection confined to the sinuses. Therefore, it is imperative to diagnosis this disease as early as possible for the best prognosis.

COMMON SYMPTOMS FOR AN UNUSUAL DIAGNOSIS

Nidhi Saini DO, Verde Valley Medical Center, PGY-2

Case of 72 year old female with worsening exertional shortness of breath. Nidhi Saini, DO – OGME – 2, Verde Valley Medical Central – Cottonwood, AZ 72y/o F with PMH for CLL diagnosed in 2003 was treated with Rituximab from 2010 till April of this year. She presents to the ED with SOB and dyspnea that is worst with exertion for two-month duration. Patient denied any chest pain, or pressure, she denies lower extremity edema, orthopnea or PND. Patient also reports hypoxia prior to admission with oxygen saturation of 78% at home. Patient was placed on Levaquin for possible atypical pneumonia and prednisone was restarted at the time of her admission. Further history revealed recent history of pleurisy for which her oncologist started her on a prednisone tapering dose. Last dose of prednisone was day prior to admission. CXR showed diffuse bilateral infiltrates and CT of the chest was negative for
pulmonary embolism. Spirogram during her stay showed restrictive pattern. Bronchoscope was obtained for further evaluation. Given the patients immunocompromised status the possibility of CMV, pneumocystis jiroveci, and HSV pneumonia were considered. During the procedure biopsies were obtained and were sent for Legionella stain and culture along with culture for TB, fungal and GMS stain for pneumocystis jiroveci. GMS was consistent with PCP and patient was placed on Atovaquone, due to reported sulfa allergy.

Both typical and atypical pneumonia and its complications are one of the common causes of admission. In patients who are immunocompromised or who do not appear to improve with empiric therapy, it is important to look for other etiologies. Pneumocystis pneumonia is a life-threatening opportunistic infection in immunocompromised individuals. It was first noted in the 1980’s in AIDS patients. Now it is most commonly seen in patients with malignancy due to use of various immunosuppressive agents. Drug of choice for treatment is TMP-SMX. For patients with mild- moderate PCP who cannot tolerate TMP-SMX, Atovaquone is a good option. For patients with severe PCP who cannot tolerate TMP-SMX, pentamidine is as effective as TMP-SMX. Glucocorticoids are recommended for HIV-infected patients with moderate to severe PCP but there is no clear evidence of efficacy in treatment of PCP in HIV-negative patients. Prophylactic treatment is indicated also for patients who are receiving high dose glucocorticoids (>20mg for one month or greater) who have another causes of immunosuppression, such as immunosuppressive drugs or certain hematologic malignancies.

**BOWEL PERFORATION IN A PATIENT WITH ANCA-ASSOCIATED VASCULITIS**

**Christopher Savage MD, St. Joseph’s Hospital & Medical Center, PGY-2**

Betre Workie

Bowel Perforation in a Patient with ANCA-Associated Vasculitis

Christopher Savage, MD, MA, Betre Workie, MD St. Joseph’s Hospital and Medical Center, Creighton University – Phoenix Campus

Case Report: A 56 year old Caucasian male with a past medical history significant for vasculitis was admitted for worsening numbness and weakness of his hands and feet. The patient failed to improve on low dose steroids and was started on Solu-Medrol 1g/day. Two days after the initiation of this treatment he began to complain of diffuse stomach pain and nausea. That night he abruptly became tachycardic and hypotensive. A STAT KUB revealed significant free air and the patient was emergently taken to the OR where approximately 20 feet of grossly necrotic small bowel was removed. Resulting pathology showed frank necrosis as well as vasculitic changes. His vasculitis was found to be C-ANCA (PR3-ANCA) positive. We planned to start cyclophosphamide but a CT guided biopsy of a recently discovered upper lobe cavitary lesion revealed acid-fast bacilli. Empiric treatment for atypical micobacterial disease was begun, and after induced sputum returned negative for AFB we chose to proceed with pulsed IV cyclophosphamide 800mg/m2 with Mesna, and then a transition to 50 mg TID orally. The patient had multiple complications during his stay including atrial fibrillation, upper extremity DVT, hyponatremia, and severe deconditioning. The patient remains on prednisone 50 mg BID and oral cyclophosphamide.
Following a lengthy convalescence he was discharged to a rehabilitation facility and later to home with frequent outpatient follow up.

Discussion: The clinical picture of arthralgias, mononeuritis multiplex, leukocytoclastic vasculitis, cavitary lung nodule and positive C-ANCA points to the diagnosis Granulomatosis with PolyAngiitis (GPA), formerly known as Wegner’s although Microscopic PolyAngiitis (MPA) can not be completely ruled out. Approximately 90% of patients with GPA are ANCA-positive, of which 80% to 90% have PR3-ANCA (C-ANCA) and the remainder have MPO-ANCA (P-ANCA). In our literature review, we found very few reported cases of ANCA-associated vasculitis complicated by bowel ischemia, necrosis or perforation. Indeed intestinal complications are not only rare but also hard to recognize. In our case the impending bowel perforation was not recognized because the patient was on high-dose steroid which masked early peritoneal signs of bowel ischemia. Combination of cyclophosphamide and glucocorticoid is the standard treatment recommendation. In this case, cyclophosphamide treatment was delayed until serious mycobacterial infection was ruled out. This is a unique and interesting case because it has rare disease with rare complication and with significant diagnostic and treatment challenges.

Conclusion: Intestinal ischemia and bowel perforation are rare complications of ANCA-associated vasculitis. Patients who are already on high dose steroid treatment may not exhibit early peritoneal signs and high index of suspicion is necessary for timely diagnosis and intervention.

**KOUNIS SYNDROME: ST ELEVATION WITH A STING**

Dmitriy Scherbak DO, Banner Good Samaritan, PGY-2

Mohamad Lazkani MD, Akil Loli MD

Kounis Syndrome: ST Elevation That Stings!

Dmitriy Scherbak DO, Mohamad Lazkani MD, Akil Loli MD Banner Good Samaritan Medical Center, Department of Cardiology, Phoenix, AZ

Introduction: Kounis syndrome is a rare, non-thrombogenic cause of angina or myocardial infarction triggered by the release of inflammatory mediators following an allergic or anaphylactic reaction. This so called "allergic angina" is seen in the setting of anaphylactic reactions and is believed to be due to mast cell release causing coronary vasospasm. The treatment of such cases is often with epinephrine, which has also been described in the literature as another rare cause of coronary vasospasm.

Case Description: A 46 year old male suffered multiple bee stings while landscaping in his yard. He developed an anaphylactic reaction with subsequent loss of consciousness. He was promptly treated with IM epinephrine injection by paramedics at arrival and approximately 20 minutes later developed marked ST elevations seen on the ambulance run sheet. Upon arrival to the Emergency Department (ED) his ST elevations were redemonstrated in the inferior leads with reciprocal ST depressions in the anterior leads. His troponin peaked at 13 ng/mL and tryptase level was 15 ng/mL (normal <10 ng/mL). He developed two cardiac arrests in the ED, the first one leading to one round of CPR and 1mg of Atropine, and the second one leading to one round of CPR with administration of 1mg of epinephrine. He was then promptly sent for coronary catheterization which showed non-diseased coronary arteries
and a normal ejection fraction without evidence of vasospasm. After his left heart catheterization he was treated with an epinephrine drip for distributive shock. Interestingly his syndrome was not provoked when re-challenged with this therapy, suggestive of an allergic reaction rather than epinephrine as the etiology of his presumed vasospasm. He did well in ICU with supportive care including mechanical ventilation. His epinephrine drip and ventilator were eventually weaned off and he was discharged home in good health with a prescription for an Epipen.

Discussion: Kounis syndrome is a rarely described phenomenon. It has been associated with food and drug allergies, as well as with allergy to bee venom. This patient’s ST segment elevation and troponin elevation was likely Kounis syndrome and not epinephrine-induced vasospasm. However, he was treated with an epinephrine injection prior to any strips being run, and therefore epinephrine-associated vasospasm cannot be ruled out as a possible cause. Awareness that anaphylactic reactions can lead to Kounis syndrome can lead to prompt appropriate treatment in this life threatening condition.

SERUM SICKNESS AFTER FIRST DOSE OF RITUXIMAB

Jaspreet Singh DO, Sierra Vista Regional Health Center, PGY-2

Abdalla Fadda MD

Serum sickness is traditionally described as development of antibodies in a host against non host serum proteins leading to sickness due to the immune complex formation. The term has also been used for the triad of fever, arthralgia and rash that can form after administration of certain medications and even after some infectious diseases. 68 year old female with history of non-Hodgkin’s lymphoma who presented to the hospital with diffuse arthralgias, fever, rash on her anterior chest wall for one day and she was unable to talk secondary to pain in her TMJ joint bilaterally. She also complained of painful ulcers in her mouth for 5 days. She had received her first dose of Rituximab 2 weeks earlier for her non-Hodgkin’s lymphoma. She was worked up for infectious and auto-immune etiologies with monospot test, ESR, CRP, ANA, rheumatoid factor, C3, C4, CCP IgG levels and blood cultures. Patient was started on cefepime for any possible infectious source of her fever. Patient’s monospot test was negative, ESR and CRP were elevated at 92 and >9 respectively. On the second day of hospitalization blood cultures showed no growth but patient continued to have diffuse arthralgias, anterior chest wall rash and fever of 100.4 Fahrenheit. At that time patient was clinically diagnosed with serum sickness and IV benadryl and solumedrol were started. Third day of hospitalization blood cultures still showed no growth but patient’s condition was much improved, including arthralgias, rash started to regress, TMJ pain improved and she was able to talk and temperature dropped to 96.24 Fahrenheit. Patient’s antibiotics were discontinued secondary to low suspicion of infection. Fourth day of hospitalization patient had been afebrile for 24 hours, rash and arthralgias had diminished significantly. Patient’s ANA was negative, CCP IgG <16, C3: 81, C4 was undetectable. Patient was monitored for another 24 hours off antibiotics and discharged home on oral prednisone. This case shows the potential of developing serum sickness after just one dose of Rituximab. It is important to differentiate this diagnosis when evaluating patients through thorough history and physical exam. The reaction seen in this patient is rare but a predictable one. Recognizing this condition early in its process and instituting the correct treatment is crucial.
TOXIC EPIDERMAL NECROLYSIS

Bryce Swenson DO, Verde Valley Medical Center, PGY-2

Romi Coolidge DO, PGY-2

Toxic Epidermal Necrolysis (TEN) is an idiosyncratic reaction of the skin in most cases triggered by medication. The pathogenesis of this reaction is still not fully understood but is thought to be the result of an immunologic process. This disease is characterized by involvement of more than 30% of the skin surface. It causes full thickness epidermal involvement and results in sloughing of the skin. The management of this disease is complicated due to skin sloughing leading to a presentation similar to burn victims, with increased insensible losses and risk of infections. Diagnosis of this disease can be difficult due to an extensive differential diagnosis list and a varying spectrum of presenting complaints. Another difficult factor in making the diagnosis is the identification of an offending agent, as timeframe between drug ingestion and symptoms can vary. Management is complex and complicated by skin breakdown and possibility of bacterial infection that may require treatment with antibiotics that could further exacerbate the skin reaction. This presentation will discuss the complexities of this diagnosis by describing the presentation, diagnosis and management of a case of TEN. Here will be reported a case of TEN seen as a result of antibiotic therapy with ciprofloxacin. The patient presented with an erythematous rash covering a large portion of her body including the palms and soles of the feet and the mucous membranes of her mouth. Severe areas of rash were noted to desquamate within the first few days of treatment. After significant investigation the likely causative agent was identified and discontinued. The patient was placed on corticosteroids and all medications including antibiotics were discontinued. This decision was difficult to make as the patient continued to experience symptoms of the initial infection that prompted antibiotic use. Over a period of a week the patient’s rash improved and faded, and areas of desquamation healed. A biopsy specimen was taken with results suggestive of toxic epidermal necrolysis. This case was a very challenging case that required an extensive history of the patient’s recent medical history, with special attention to recent medications taken in the last 6 months. Included in the list of medications were over the counter and herbal medications. Family member’s went through the patient’s home and brought in any bottles of medications and supplements that could be found. After verifying with the patient which medications she had been taking we were able to identify ciprofloxacin as the offending agent.

A RARE REASON FOR ACUTE PANCYTOPENIA IN THE DESERT

Maja Udovcic MD, Mayo Clinic Arizona, PGY-2

Ryan Robetorye MD, Raoul Tibes MD

Introduction: Panctopenia yields a broad differential that necessitates a thorough history and physical for diagnosis. Although hematologic conditions are most common, it is important to consider infectious etiologies. The following case presents an uncommon cause of pancytopenia, diagnosed in an unlikely region of the United States. Case Description: A 41 year old female with no medical history presented with one week of fatigue, fevers, headache, lower extremity rash, and dark urine. One week before presentation she travelled to Sedona, AZ where she went hiking. Travel history also revealed a trip to
Connecticut one month prior, where she sustained bug bites from an unknown insect. In addition, her pet dogs had a recent tick infestation. On exam, she was afebrile and tachycardic. She had petechiae on her legs and patchy erythema on her back. Laboratory evaluation revealed new pancytopenia, elevated AST and LDH. Urinalysis showed proteinuria and urobilin. Blood film showed rare RBC inclusions of ring forms consistent with Babesia microti. Microbiology evaluation confirmed Babesiosis by PCR. Testing for Ehrlichia and Anaplasma was negative. She was treated for symptomatic Babesiosis with Atovaquone and Azithromycin for 10 days. Her pancytopenia resolved. It remained unclear where she contracted Babesiosis, with possibilities including Connecticut, Sedona, or from her pet dogs with ticks. Discussion: Babesiosis is a protozoan infection seen in the Northeast and Upper Midwest regions of the United States, making Arizona an unusual location to contract the infection. The vector of transmission is the deer tickIxodes scapularis. Although Babesiosis is a notifiable condition in 18 states, Arizona limits notification of tick borne diseases to Anaplasma and Lyme disease. Symptoms begin 1 to 6 weeks after tick bite, although many patients are not aware of a bite. Symptoms can range from mild flu-like illness to severe illness marked by end-organ damage, pancytopenia, DIC, and splenic infarct. Babesiosis is suspected after travel to endemic areas and exposure to tick bites with flu-like symptoms. Microscopy is used for diagnosis. PCR and serology are optional. Co-infection with Lyme disease or Anaplasmosis should be considered if a patient does not respond to treatment for Babesiosis, if they present with erythema chronicum migrans indicative of Lyme disease, or persistent leukopenia indicative of Anaplasmosis. Treatment includes Atovaquone and Azithromycin or Clindamycin and Quinine for 7 to 10 days. This patient responded well to initial treatment. Conclusion: This case highlights the importance of including a broad differential in the workup for pancytopenia and assuring a thorough history and physical. Although a primary hematologic etiology is most likely, this case demonstrates an infectious source especially unique to the southwest region of the United States.

A NEW FORMULA: ANTI-FREEZE AND PROPYLENE GLYCOL

Elizabeth Ulliman MD, UACOM at South Campus, PGY-2

Aswani Alavala

Introduction Anti-freeze ingestion is a medical emergency due to the toxicity caused by ethylene glycol, a common major component. Recently anti-freeze manufacturers have been changing ingredients from typical ethylene glycol to other alcohols such as propylene glycol. The high fatality and need for urgent recognition and treatment in toxic ingestions underscores the necessity of quality history taking and understanding of alcohol metabolism. Case Presentation: A 34 year old woman with history of bulimia nervosa and substance abuse presented with nausea and stated she had ingested half a gallon of anti-freeze and attempted to end her life with incisions on her wrists. The patient denied any co-ingestions or alcohol ingestions. Initial labs an elevated anion gap of eighteen, measured serum osmolality of 342, an osmolar gap of 56, lactate of 9.9, a significant rise in patient’s creatinine to 3.0, and an ABG showed pH of 7.27. Salicylate toxicity, diabetic ketoacidosis, ethanol toxicity, sepsis, and other causes of anion gap metabolic acidosis with osmolar gap were ruled out with initial lab work. Comprehensive toxicology panel was sent on initial labs, though results would take approximately 24-36 hours. Thus, it was decided to treat the patient as ethylene glycol toxicity, with fomepizole. During course of admission patient remained alert and oriented. With treatment, the patient produced urine
and all abnormal labs trended down. The gas chromatography resulted with a negative ethylene glycol level. A negative ethylene glycol level in the setting of anion gap metabolic acidosis and extremely elevated osmolar gap, the only fitting diagnosis is propylene glycol. The lack of IV benzodiazepine administration and negative urine toxicology for benzodiazepines was quite peculiar. The patient was then remembered the brand of anti-freeze that she ingested, which uses propylene glycol as primary ingredient instead of ethylene glycol. Discussion Alcohol ingestions are medical emergencies that can be fatal if not identified and treated appropriately. Ethylene glycol is renally cleared and, in low levels, can be cleared without causing any damage. Only when parent alcohols are oxidized to metabolites, that devastating end organ damage occurs. Alcohol dehydrogenase metabolizes ethylene glycol to glycolate, glyoxylate and oxalate. These metabolites target the kidney tubules and lead to reversible acute kidney injury. Fomepizole and IV ethanol block alcohol dehydrogenase, preventing the formation of toxic metabolites, allowing the ethylene glycol to be eliminated unchanged through the kidney. Propylene glycol toxicity is seen in patients receiving IV lorazepam or diazepam, propylene glycol is a solvent in the preparation. There is no antidote for propylene glycol toxicity. Treatment includes stopping the offending agent and hemodialysis, depending on the severity. Conclusion: With changing anti-freeze formulas, clinicians must consider alternate causes of toxicity, so that appropriate therapy can be provided to patients.

TINU: A UNIQUE CASE OF TUBULO-INTERSTITIAL NEPHRITIS WITH UVEITIS (TINU) SYNDROME PRESENTING WITH POLYURIA AND POLYDIPSIA

Courtney Walker DO, UACOM at University Campus, PGY-2

Mahmoud Kamel, Huthayfa Ateeli

Acute tubulo-interstitial nephritis with uveitis (TINU) is a rare syndrome. Approximately 200 cases have been reported worldwide since it was first described by Dorbin and Associates in 1975. We report a case of TINU syndrome that presented with polyuria and polydipsia. A 32 year old African-American female with history of Grave’s Disease presented to an outside hospital (OSH) with complaint of frequent urination and excessive thirst for 2 months. On initial presentation, patient described clear, large volume diuresis with nocturia, thirst, dry, cracked lips, blurry vision, and dry eyes. Urine chemistry at the OSH revealed low urine osmolality, mildly elevated serum osmolality and low urine sodium concerning for diabetes insipidus. A magnetic resonance image of the brain showed an absence of the posterior pituitary bright spot, which can be seen in patients with central diabetes insipidus, and was therefore discharged home on desmopressin therapy. Four weeks following the initial diagnosis - the patient continued to have significant polyuria and polydipsia despite desmopressin therapy. She was seen in nephrology clinic at our institution and was admitted to our hospital for further workup. Admission labs showed acute kidney injury with a creatinine of 2.0 and fractional excretion of sodium consistent with an intrinsic renal process. Urinalysis was negative for red blood cells, proteinuria, or casts. Mild hypokalemia with a non-anion gap metabolic acidosis and anemia of chronic disease were present. ESR and CRP were elevated. Creatinine kinase, ACTH, and cortisol levels were within normal limits. Autoimmune antibodies including antinuclear antibody, antineutrophilic cytoplasmic antibodies, rheumatoid factor, complement levels, angiotensin converting enzyme anti-Ro/SSA and anti-La/SSB were normal. Chest xray did not show any features suggestive of sarcoidosis. Water deprivation test
showed continued sodium and water diuresis. Ophthalmology was consulted for evaluation of dry eyes and blurry vision. Slit lamp exam revealed significant “granulomatosis panuveitis”. Patient’s kidney function continued to deteriorate and a renal biopsy was performed. Pathology was consistent with “active tubulo-interstitial nephritis with diffuse interstitial fibrosis and moderate arteriosclerosis”. No granulomas were present to suggest sarcoidosis. Given the clinical history, uveitis, and kidney biopsy findings- patient was diagnosed with TINU syndrome. Desmopressin was discontinued and she was discharged to home on high-dose steroids. Follow-up one month later showed some improvement in renal function and polyuria. Tubulointerstitial Nephritis and Uveitis syndrome is an underdiagnosed syndrome which can result in incomplete recovery and persistent chronic kidney disease despite treatment. Early recognition and a multidisciplinary approach involving ophthalmologists and nephrologists or pediatricians is crucial for early diagnosis and treatment.

CREUTZFELDT-JAKOB DISEASE PRESENTING AS ALIEN HAND SYNDROME

Jessica Weiss DO, Banner Good Samaritan, PGY-2

Lise Harper

Introduction Creutzfeldt-Jakob Disease (CJD) is a rare prion disease in which abnormal protease-resistant prion proteins deposit in cerebral tissue resulting in fatal spongiform encephalopathy. CJD usually has a subacute onset and is a very rapidly progressing disease with mental and motor decline leading to death within about three months of diagnosis. The symptoms of CJD are variable and are often initially misdiagnosed as other neurodegenerative disorders. Cognitive decline and myoclonus are the commonest manifestations. Alien hand syndrome (AHS) is an unusual phenomenon that has been described as a presenting feature in rare cases of CJD. Case Presentation A 71-year-old male presented to an outside hospital with a two-week history of ataxia, disequilibrium and difficulty using his left leg, resulting in several falls. He was diagnosed with Guillain-Barre syndrome on the basis of a minimally elevated spinal fluid protein. The patient was treated with IVIG and transferred to an acute rehabilitation facility. His symptoms worsened and he was transferred to Banner Good Samaritan Medical Center for further neurologic evaluation. On exam, vital signs were normal and he was alert and oriented, but had difficulty understanding questions and performing tasks. Exam was further notable for difficulty performing movements but there were also spontaneous, non-purposeful movements of the left upper extremity. Extensive testing, including evaluation for metabolic, infectious, autoimmune, malignant and paraneoplastic etiologies was unremarkable. Brain MRI was notable for subtle diffusion restriction involving the right frontoparietal and temporal cortex as well as the right caudate head and putamen, suspicious for CJD. This prompted further testing, including both CSF 14-3-3 protein and EEG, which were negative. However, CSF total tau protein was markedly elevated, further pointing toward CJD. The patient was discharged home on hospice. He exhibited a progressive decline in mental status prior to death less than one month later. Post-mortem brain analysis confirmed a diagnosis of CJD. Discussion Alien hand syndrome (AHS) is a “combination of involuntary hand movements with a sense of alienness or estrangement of the affected hand”. It has been described as a loss of voluntary control and loss of ownership of one’s hand without sensory loss. AHS has been reported in cases of stroke, subarachnoid hemorrhage, neoplasm, corticobasal degeneration, trauma, Alzheimer’s disease and CJD. Rapidly progressing neurologic symptoms with AHS and suggestive MRI findings, with or without
positive CSF 14-3-3 protein or suggestive EEG findings, is strongly suggestive of CJD. Thus physicians should be familiar with AHS as this should prompt clinical suspicion for CJD.

**ENTEROPATHY ASSOCIATED T-CELL LYMPHOMA**

Jessica Yan MD, UACOM at University Campus, PGY-2

Jonathan Schatz

Enteropathy associated T-cell lymphoma Jessica Yan, Resident, Department of Medicine, University of Arizona, Tucson, AZ. Jonathan Schatz, MD, University of Arizona Cancer Center, Department of Medicine, and Bio5 Institute. Enteropathy associated T-cell lymphoma (EATL) is a rare peripheral T-cell lymphoma originating from intraepithelial T lymphocytes of the intestines strongly associated with Celiac disease. The under recognition, infrequency, and nonspecific manifestation of this disorder creates a diagnostic challenge for physicians. A 55-year-old man of Iraqi descent presented with a 6 month history of intermittent abdominal pain, watery diarrhea, bloating, decreased appetite and unintentional weight loss of 10 pounds. Two days prior to admission, he had worsening abdominal distension, pain, constipation and obstipation. Pertinent past medical history was hypothyroidism. On admission to the hospital, computed tomography revealed diffuse severe small bowel wall thickening involving ileal loops in the right lower quadrant with markedly distented small bowel loops proximal to the areas of thickening, consistent with partial small bowel obstruction. To further evaluate the small bowel abnormality, MRI was performed and demonstrated multiple regions of focal thickening within the small bowel most significant focus in the distal ileum measuring 0.7cm causing a partial small bowel obstruction. Despite conservative management of his small bowel obstruction, he continued to have abdominal pain and was subsequently taken to the operating room. Diagnostic laparoscopy was initially attempted but his bowels were diffusely dilated, requiring exploratory laparotomy. Intraoperatively, he was found to have a firm distal ileal mass adherent to the abdominal wall with diffuse mesenteric lymphadenopathy. Segmental ileal resection was performed with side-to-side anastomosis and the diagnosis of EATL was ultimately confirmed on biopsy results. Pathology revealed severe villous blunting and markedly increased intra-epithelial CD3, CD8 positive T cells suggesting undiagnosed celiac disease. The patient received six cycles of cyclophosphamide, doxorubicin, vincristine, and prednisone and achieved complete remission. This case highlights a potential complication of celiac disease, the commonest food intolerance disorder in Western populations. Although the lymphoproliferative malignancy seen in this patient is rare, the poor outcomes of EATL with a 5-year mortality rate of 80% to 92% underscore the importance of diagnosis of celiac disease. Adherence to a gluten-free diet would minimize the risk of developing this highly aggressive lymphoma.

**A CONUNDRUM OF A CASE: HEMOPHAGOCYTIC LYMPHOHISTIOCYTOSIS IN AN ADULT PATIENT**

Qi Yu DO, UACOM at South Campus, PGY-2

Hani M Babiker, Myke Green, Ravitharan Krishnadasan

Introduction: Hemophagocytic Lymphohistiocytosis (HLH) was recognized sixty years ago by Farquhar and Claireaux. HLH is a rare and fatal hematologic disease that can lead to multi-organ failure. Its
incidence varies, from 1 in 100,000 children to 1 in 800,000 in adults. The disease stems from defective cytotoxic T cells causing widespread inappropriate inflammatory cytokine release, creating a chaotic cascade of “hyperinflammation,” proliferation of histiocytes, and an inability to control infections. The syndrome can result from genetic mutation of genes that regulate the cytotoxic effect of T cells (Primary HLH). Other etiologies include infection, rheumatologic disease, or malignancy (Secondary HLH). This rare but grave disease presents with fever, jaundice, confusion, pancytopenia, rash, hyperlipidemia, hepatosplenomegaly, and inexplicable hepatitis. Given the high fatality of the HLH, early recognition and treatment is imperative. Case Presentation: A 58 year-old female with a history of Systemic Lupus Erythematous presented with generalized fatigue for 4 months, intermittent fevers for 1 year, and unintentional weight loss (40lbs). Prior hospitalizations with extensive infectious disease, rheumatologic, and gastroenterological workup were unrevealing. On physical examination her vitals revealed HR of 90-100, BP of 110s/60-70s, and temperature of 101.6°F. She was pale, lethargic, and moderately confused. She had hepatosplenomegaly on physical examination. Labs revealed microcytic anemia, transaminitis, hyperferritinemia (36,912 ng/ml), hypertriglyceridemia, and Epstein Barr virus infection. Bone marrow biopsy revealed significant hemophagocytosis and increased histiocytes. She was diagnosed with HLH and was started on the HLH-94 protocol, which includes steroids, etoposide, and cyclosporine. Her symptoms completely resolved after 3 weeks of treatment. Thereafter, she underwent a match sibling-related allogenic stem cell transplant successfully. Conclusion: HLH is a syndrome that more commonly occurs in the pediatric population and rarely in adults. In normal individuals, an antigen triggers the activation of nature killer and cytotoxic T cells, leading to induction of apoptosis of infected cells by cytotoxic enzymes. However in HLH, defects in either the enzymes or the release of granules results in uncontrolled T cell and macrophage activation, and therefore a “cytokine storm”. This leads to the presenting symptoms as described above. Given the high mortality rate, timely diagnosis and prompt treatment are crucial. Large scale clinical trials for the treatment of HLH are lacking in adults, therefore the pediatric HLH-94 protocol has been adapted for the treatment of adults. Studies have shown that effective early therapy has reduced the mortality rate from 95% to 30-35%. Our case report highlights the emergence of this rare disease entity in adults, the importance of early recognition in patients presenting with such constellation of symptoms, and the success of pediatric chemotherapeutic regimens followed by allogeneic stem cell transplant in adults.

PGY-1 SUBMISSIONS

ACERULOPLASMINEMIA

Raed Al-Adham MD, St. Joseph's Hospital & Medical Center, PGY-1

Fadi Alrabadi, MD Michael Roberts, MD.

Introduction: Aceruloplasminemia is an autosomal recessive disorder primarily described in the Japanese population, with incidence of 1 / 2,000,000. There is no data regarding incidence and prevalence in Western European countries and the USA. It is characterized by progressive neurodegeneration of the retina and basal ganglia. It has been found to be associated with specific inherited mutations of the ceruloplasmin gene and complete absence of ceruloplasmin ferroxidase activity. Which leads to iron accumulation in brain, liver and pancreas. It typically presents in the fourth
or fifth decade with neurological symptoms, retinal degeneration, anemia and diabetes mellitus. Case description: A 40 year old Native American male patient presented with headache, right hemianopsia, anemia and uncontrolled diabetes. Work up for the hemianopsia including brain MRI showed diffuse iron accumulation in the basal ganglia, dentate nuclei, left temporal lobe and cerebellum. Further workup showed hemoglobin of 9.3 gm/dl, MCV of 84 fl, ferritin level of 530 ng/ml (normal level 12-300 ng/ml), transferritin saturation of 9.4%, Serum Iron :23 ug/dl (normal level 60-150 ug/dl ), ceruloplasmin level was less than 2 mg/dL and copper level was less than 10ug/dL. Patient was known to have diabetes mellitus type 2, for 2 years, he presented with HbA1c of 15.8% although he was on 20 units of insulin before breakfast, lunch and dinner. An MRI of the abdomen showed significant iron deposition in the liver and pancreas. A genetic workup came back with CP gene mutation. The patient responded to deferoxamine 500mg IV daily for 7 days, and was discharged on deferasirox 125 mg po twice daily. Because of the anemia, the role for phlebotomies is limited Discussion: Although aceruloplasminemia is a rare disease, this case illustrates the need to screen patients who present with anemia, uncontrolled diabetes and neurological symptoms for aceruloplasminemia. Recognition of this syndrome and early treatment can prevent further neurological deterioration.

**MEDICATION USE, PSA AND PSA VELOCITY**

**Amit Algotar MBBS, PhD, MPH, UACOM at University Campus, PGY-1**

Roxanna Behnejad, Steven P. Stratton

Background: Prostate specific antigen (PSA) and PSA velocity (rate of PSA change over time) are important biomarkers for diagnosis and prognosis of prostate cancer (PCa). Many of the men who are at high risk for PCa also have associated co-morbidities such as coronary artery disease, arthritis, hyperlipidemia and hypercholesterolemia for which they are taking long term aspirin, other non-steroidal anti-inflammatory drugs (NSAIDs) and/or statins. Hence it is important to determine the effect of these medications on PSA and PSA velocity (PSAV) especially in a population of men at high risk for PCa to determine if these medications interfered with the diagnosis or prognosis of PCa. Method: Using a population of 699 men enrolled in a Phase 3 chemoprevention trial conducted to investigate the effect of selenium supplementation on PCa incidence, we investigated the effect of aspirin, other NSAIDs and statins on PSA and PSAV using a longitudinal study design. During the course of this trial 73 men were diagnosed with PCa. Multiple linear regressions were used to investigate the association of medication use with PSA at baseline and mixed effects models were used to investigate the association of medication use with PSAV during the trial. Using a longitudinal study design allowed the measurement of PSA at multiple time points providing a stronger estimated of the subject’s PSAV and hence increasing the power of this study. Results: Results from this longitudinal study indicate that aspirin, other NSAIDs or statins did not demonstrate statistically significant association with PSA (p-values 0.79, 0.68, and 0.79 respectively) or PSAV (p-values 0.23, 0.43, and 0.84 respectively). These results did not change upon stratifying the analysis by PCa diagnosis or after adjusting the statistical model with variables such as age, race, body mass index and smoking. Conclusion: Although larger studies with more specific study designs are needed to investigate this relationship, results from this current study indicates no effect of above medications on PSA and PSAV in elderly men at high risk for PCa. These results indicated that
above medications may not interfere with the diagnosis and prognosis PCa and hence may be safely used in men at high risk for PCa.

**A MYSTERIOUS CASE OF HYPERNATREMIA**

*Saed Alnaimat MD, Pending, PGY-1*

Courtney Walker, DO

Hypernatremia is a common electrolyte disturbance in the clinical setting. Left untreated, it can lead to serious neurologic complications. This is a case of hypernatremia presumed initially due to multiple factors (diarrhea, fever, decreased oral intake, and glycosuria). However, further investigations unraveled something else. A 40 year-old Caucasian male patient with past medical history of Hypertension, poorly- controlled Diabetes Mellitus, Obesity, and Gastroesophageal reflux disease, presented to the Emergency Department with fever, left upper quadrant abdominal pain, and watery diarrhea of one day duration. The patient underwent Roux-en-y bypass surgery one week before, reports postoperative were essentially uncomplicated; he had been tolerating bariatric liquid diet at home. The patient reported that over a 2 month period prior to surgery, he had felt excessively thirsty and drank up to 8 L of water every day; however, this had been attributed to poor glycemic control (home blood sugar 300-400 mg/dl). On the present admission, labs showed a WBC count of 14,800 /UL, plasma glucose of 301 mg/dl, and serum sodium of 161 mEq/L. CT abdomen was suggestive of intra-abdominal infection that was treated with empiric IV Ciprofloxacin and Metronidazole, with subsequent improvement. The patient’s glyceria was controlled with hospital insulin protocol. It is known that gastrointestinal surgeries can induce dehydration secondary to restricted oral intake. Fever, diarrhea and glycosuria can aggravate dehydration and produce hypernatremia. However, free water replacement, based on free water deficit, did not completely correct the patient’s sodium level. Further workup revealed inappropriately low urine osmolality (137 mOsm/kg H2O) and low urine sodium (<10 mEq/L) in the face of elevated serum osmolality (339 mOsm/kg H2O), which raised the suspicion of Diabetes Insipidus as the cause of this patient’s hypernatremia. Therefore, water deprivation test was performed and the patient failed to concentrate his urine. ADH stimulation significantly raised the patient’s urine osmolality. MRI of the pituitary and hypothalamus was unremarkable. ANA, Anti-ds DNA, ANCA, and Anti-ADH auto-antibodies were not detected. Thus, the patient was diagnosed with Primary (Idiopathic) Central Diabetes Insipidus. He was discharged home with 10 mcg Intranasal Desmopressin nightly and encouraged to drink free water, with improvement of his sodium. This complex case highlights Central Diabetes Insipidus as a cause of hypernatremia, with a presentation that was unmasked after reduced fluid intake in a postoperative patient. The diagnosis was challenging since there were multiple confounding causes of hypernatremia that needed to be corrected to reach the precise diagnosis. Diabetes Insipidus symptoms can be bothersome to patients. If left untreated, Diabetes Insipidus can lead to dehydration from excessive free water loss, various electrolyte disturbances, and serious neurologic sequelae or brain damage.
IN SEARCH FOR THE HIDDEN BUG; A RARE CASE OF CLOSTRIDIUM DIFFICILE AND CYTOMEGALOVIRUS CO-INFECTION

Fahad Alobaidi MD, UACOM at University Campus, PGY-1
Cristian Dominguez MD, Vijay Chandiramani MD, Santhosh G John MD

The management of a patient with persistent Clostridium difficile diarrhea can be confounded by presence of co-infection with other pathogens. This can lead to a delay in diagnosis and treatment. We present a case of a 63 year-old Caucasian man with history of squamous cell carcinoma of the lower lip, status post resection and chemoradiation presenting with intractable diarrhea. Our patient presented with nausea, vomiting, foul-smelling watery diarrhea and intermittent crampy abdominal pain of 2 weeks duration. At the time of admission, the patient was febrile, tachycardic and hypotensive. Physical examination findings included vague diffuse abdominal tenderness and hyperactive bowel sounds. Initial blood workup showed severe leukocytosis and lactic acidosis. Stool studies were positive for white blood cells on gram stain, but negative for ova, parasites, Salmonella, Shigella and Campylobacter jejuni. Stool Clostridium difficile toxin by polymerase chain reaction (PCR) was positive for NAP1 strain. He was started on oral vancomycin solution 250 mg every 6 hours without any significant response even after 7 days of therapy. Subsequently a computerized tomography of abdomen and pelvis demonstrated findings of severe diffuse pancolitis. Oral vancomycin dose was increased to 500 mg every 6 hours and IV metronidazole was added. Despite being on this regimen for another 5 days, patient continued to have watery diarrhea and leukocytosis. Vancomycin enemas were given when he developed ileus during the course of the infection. Patient was subsequently started on Fidaxomicin. Repeat C. difficile toxin by PCR came back negative. In search for other etiologies, further work up including viral panels in blood and stool were done. His blood and fecal quantitative Cytomegalovirus (CMV) PCR came back positive with high titers. Lower gastrointestinal endoscopic biopsy could not be performed for concern of perforation. Hence, patient was started empirically on oral Valganciclovir. He had a dramatic response in 48-72 hours of treatment and had complete resolution of diarrhea in 5 days after the initiation of treatment. His leukocytosis and tachycardia also resolved. Patient was discharged to a skilled nursing facility on oral Valganciclovir and a tapering dose of oral Vancomycin for a total of 4 weeks. CMV infection can occur in a patient with C. difficile, either as a coexistent infection or secondary to disruption of the colonic mucosa caused by C. difficile. This case report highlights the importance of high index of suspicion as well as work up for other rare causes of diarrhea in patients with persistent diarrhea and who test positive for C difficile. Though histological diagnosis has high sensitivity and specificity, treatment should not be delayed in symptomatic patients with positive CMV PCR in blood and stool, as this could result in high fatality.

THE WAY TO A MAN’S HEART, THROUGH THE STOMACH

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Sairav Shah, MD, Joseph Charles, MD

Introduction: Uncovering the underlying etiology of syncope is often challenging. In fact, up to 20% of cases have no clear cause. Cardiogenic syncope is often the most worrisome and potentially fatal. Case:
An 82-year-old male with a past medical history of esophageal cancer treated by resection, chemotherapy, and radiation without known recurrence, hiatal hernia, and chronic loose stools presented with a syncopal episode. That morning he experienced a minute long loss of consciousness upon standing after passing a loose stool. Additional history included a recent 10-lb weight loss and multiple prior syncopal events related to positional changes and meals. Physical exam, laboratory investigation, electrocardiogram, and chest x-ray revealed no definitive pathology. With question of pulmonary embolus, a subsequent chest CT angiogram demonstrated a hiatal hernia without evidence of pulmonary emboli or other significant abnormality. Accordingly, the patient was admitted and placed on telemetry, which revealed intermittent PVCs that resolved with metoprolol. However, the pre-syncopal symptoms continued. Given symptom persistence, an echocardiogram was obtained, which demonstrated a mass, measuring 4.5 cm at greatest dimension, adjacent to the heart with left atrial compression. Subsequent evaluation with EGD revealed a phytobezoar located in the gastric pull-through above the diaphragm. The phytobezoar was removed endoscopically, and after extraction, the symptoms resolved. The patient was counseled on bezoar prevention and discharged with scheduled follow-up motility studies. Discussion: The differential diagnosis for syncope includes any process that impedes blood flow or nutrient delivery to the brainstem, including cardiogenic, neurologic, orthostatic, and reflex-mediated causes. In addition to a thorough history and physical, the recommended workup includes orthostatic blood pressure measurement and electrocardiogram. A wide-array of additional testing tailored to the suspected syncopal type is then often pursued. For cardiogenic causes, echocardiogram, rhythm monitoring, and stress testing are among higher-yield studies, but specific context may alter further investigation. Phytobezoars are not a known cause of syncope. They are composed of undigestable vegetable matter and are often identified incidentally. Treatment includes chemical dissolution or manual extraction. Preventive measures for reducing the recurrence include increasing fluid intake while avoiding difficult to digest foods, increasing mechanical breakdown with thorough mastication, and correcting any underlying motility disorder. Hiatal hernias infrequently cause cardiovascular complaints, such as postprandial chest pain and syncope. Interestingly, a phytobezoar within a hiatal hernia leading to cardiogenic syncope has not previously been reported in the literature. This case of cardiogenic syncope secondary to phytobezoar highlights a rare etiology of syncope which was confounded by multiple other potential causes on initial presentation: arrhythmogenic, neurogenic, and orthostatic types. It illustrates the importance of a methodical history and physical as well as the need for continual re-evaluation of the differential without anchoring bias to establish a correct diagnosis.

**PULMONARY EMBOLISM AS INITIAL PRESENTATION OF MEMBRANOUS NEPHROPATHY**

Ramasubramanian Baalachandran MBBS, UACOM at University Campus, PGY-1

Snehal Patel, Kirsten A Cooper

Pulmonary embolism as initial presentation of Membranous nephropathy Introduction Membranous nephropathy is a type of nephrotic syndrome characterized by hypoalbuminemia, heavy proteinuria and edema. Hypoalbuminemia is found to be the dominant independent risk factor for venous thromboembolism in membranous nephropathy. We describe a case of membranous nephropathy presenting as pulmonary embolism and hypoalbuminemia. Case Report A 48-year-old gentleman, a
truck driver, presented with acute onset chest pain and shortness of breath. Bilateral leg swelling was present for a month. On examination he was apyrexic, with pulse rate of 80, respiratory rate 18/min, blood pressure 125/88 and spo2 95% on room air. Air entry was decreased on right side with central position of trachea and dull percussion note. CT angiography revealed right pulmonary embolism and moderate sized right loculated pleural effusion. He was treated with heparin drip. Proteinuria and hypoalbuminemia (1.5 mg/dl) were detected during routine lab tests. Antithrombin III and factor V mutations were normal. Therapeutic and diagnostic thoracocentesis of right pleural effusion revealed an exudative effusion. Further investigation of proteinuria revealed, urine spot PCR was 10 and 24-hour urine protein and albumin were 33 g and 561 mg respectively. Renal function was normal (serum creatinine 0.9 mg/dl). A diagnosis of nephrotic syndrome was confirmed. Renal biopsy to investigate cause of proteinuria revealed diffusely thickened glomerular capillary loops, due to the presence of numerous subepithelial electron dense immune-type deposits consistent with membranous nephropathy. Secondary causes were ruled out as HIV, hepatitis panel and screening for SLE were negative. Patient denied use of drugs like NSAID’s, penicillamine and no evidence of malignancy could be found. Though lambda light chains and cryoglobulins were increased serum protein electrophoresis and urine protein electrophoresis were normal. Deep vein thrombosis and renal vein thrombosis was ruled out by ultrasound. Lisinopril, furosemide, metolazone, statins and low protein and low salt diet were started. He was discharged on lifelong anticoagulation as his disease predisposes to hypercoagulability. Discussion Hypoalbuminemia is associated with hepatic overproduction of fibrinogen and factors V and VII, urinary loss of inhibitors of coagulation (Antithrombin III, plasminogen), a 6-fold increase in plasminogen activator inhibitor, hyperaggregability of platelets – the end result being hypercoagulability. A review of eight studies evaluating thromboembolic complications in nephrotic syndrome found 81 (18%) of 458 patients with deep venous thromboembolism or pulmonary embolism. The renal venous thrombosis is the most frequently detected; it is asymptomatic in 90% of cases. Whether high risk patients with hypoalbuminemia (<2.8 g/dl) should receive prophylactic anticoagulation is still controversial and elaborate analysis of risks and benefits.

**Third Place – PGY-1 Poster**

**NOT JUST ANY GIST**

**Kareem Bannis MD, UACOM at South Campus, PGY-1**

Anju Nair

Introduction: With an incidence of 1-2/100,000 population, gastrointestinal stromal tumors (GIST) are extremely rare. The vast majority of these connective tissue tumors are benign; those measuring greater than 2 cm have malignant potential. In relation to the GI tract 90% originate in the stomach and duodenum with the remaining tumors located in the esophagus, colorectal and appendix. Extremely rare cases have been reported that involve the jejunum.

Case description: The patient is a 59 year old Hispanic male that originally presented with a history of worsening vague abdominal pain for several months. Examination findings: hypotension, tachycardia, normocytic anemia, abdominal tenderness and distension. Abdominal CT with IV contrast visualized a hemorrhagic mass. Hospital course was complicated by acute blood loss anemia and hypotensive shock.
Urgent exploratory laparotomy and retroperitoneal exploration revealed a large amount of blood inside his abdomen, and a large hemorrhagic bleeding mass was identified and dissected. The mass measured 10cm in the jejunum and also involved the sigmoid colon, mesentery and appendix. Small bowel anastomosis with end colostomy was performed. Pathology revealed a GIST tumor that was c-KIT exon-9 mutation, CD 34 positive with high mitotic index of 30/50 on high power field. After an uncomplicated post-operative course, the patient was discharged with oncology follow up, where Imatinib was started as adjunctive chemotherapy. Later in his disease course, patient suffered episodes of disease recurrence that were identified and successfully treated with chemotherapy, based on repeat imaging studies. Unfortunately, during a later hospitalization for deep venous thrombosis, new acute anemia was identified. At this time, he also complained of constipation without obstipation for several days and repeat CT scan at this time demonstrated extensive metastasis to the point where radiologist were unable to differentiate the abdominal contents. Surgical oncology reported a dismal prognosis, recommending alternative palliative therapies with possible nasogastric tube decompression with or without bypass as a last resort. Outcome: Hospice and comfort measures. Discussion: We present here a rare and unfortunate case of malignant GIST tumor that hastily developed into severe metastatic disease. After 2 years of remission after initial diagnosis, with an overall benign follow up, the tumor was reactivated and rapidly progressed within 1-2 months to go from several distinct foci to extensive metastasis. The mass effect and possible intraluminal metastasis caused massive distention, constipation and obstipation. Management strategy is unclear as no clear guidelines exist. Perhaps molecular analysis may further predict those tumors that possess a predilection for rapid metastasis. Conclusion: Patients with certain molecular characteristics have poor outcomes despite initial remission. Identification of high risk phenotypes (c-kit exon 9 mutation, CD 34 +) using molecular analysis may prove to have prognostic and therapeutic value.

**PROTEIN-LOSING HYPERTROPHIC GASTROPATHY: HELICOBACTER PYLORI INFECTION MIMICKING MÉNÉTRIER"S DISEASE**

Roberto Bernardo MD, UACOM at University Campus, PGY-1

Fernando J. Lopez, Martin Tagle

Protein-losing enteropathies (PLE) are characterized by an excessive loss of serum proteins into the gastrointestinal tract, resulting in hypoalbuminemia, edema, and, in some cases, anasarca. A variety of conditions characterized by mucosal folds enlargement in the stomach (hypertrophic gastropathy) may be associated with PLE; giant hypertrophic gastropathy (Ménétrier"s disease) is the most common of these conditions. Helicobacter pylori infection may present with hypertrophic gastropathy and PLE, mimicking the clinical and pathological findings of Ménétrier"s disease. A previously healthy 86 year-old Hispanic woman presented with progressive lower extremities edema of 1-month duration; there was no history of dyspnea, ortopnea or other symptoms. Physical exam revealed pitting edema up to the thighs bilaterally, but was otherwise unremarkable. Laboratory studies revealed a WBC count of 5.6 and a hemoglobin level of 10.7. Renal function was within normal limits. Liver transaminases, alkaline phosphatase, bilirubin, INR, and TSH were normal. The patient’s total protein and albumin were both low at 3.24 g/dL and 1.85 g/dL, respectively. Iron studies revealed a low transferrin at 156. Serum protein electrophoresis showed moderate hypogammaglobulinemia and severe hypoalbuminemia. A 24
hour urine study revealed 0.15 grams of protein in 24 hrs. A serum gastrin level was normal and anti-transglutaminase antibodies were negative. The echocardiogram showed preserved systolic function (EF 67%), no diastolic dysfunction, pericardial effusion or signs of pulmonary hypertension. An abdominal ultrasound was negative for signs of chronic liver disease or presence of ascites and a contrast CT scan of the abdomen revealed diffusely enlarged rugal folds of the gastric fundus and body; the antrum was relatively spared. An upper endoscopy (EGD) showed mild diffuse antral gastritis with severe enlargement of the gastric muscosal folds; at this point, the differential diagnosis included Ménétrier’s disease, Zollinger-Ellison syndrome, sarcoidosis, lymphoma, eosinophilic gastroenteritis and H. pylori gastritis. The biopsy studies revealed a diffuse inflammatory infiltrate, mainly composed of lymphocytes and plasmatic cells, foveolar hyperplasia and presence of H. pylori 2+. Enteral high protein nutrition and triple therapy with omeprazole 20 mg PO bid, amoxicillin 1g PO bid and clarithromycin 500 mg PO bid for 10 days were prescribed. Two months after treatment the edema had completely resolved; the serum albumin was 3.27 g/dL and the EGD showed resolution of the hypertrophic gastropathy. PLE should be considered in patients with hypoalbuminemia in whom other causes such as nephrotic syndrome and liver disease have been excluded. The most common nonerosive gastrointestinal condition that may be associated with PLE is Ménétrier’s disease. H. pylori infection can be associated with inflammation and enlargement of the gastric folds, mimicking the findings in Ménétrier’s disease. As illustrated by the case, eradication of H. pylori may lead to resolution of the clinical picture.

First Place – PGY-1 Posters

BETWEEN A ROCK AND A HARD PLACE- AN UNCOMMON CAUSE OF PEA CODE ARREST

Rishi Bhargava MD, UACOM at South Campus, PGY-1
Varun Takyar, Carmen Luraschi MD

Introduction Return of spontaneous circulation (ROSC) is the beginning of a critical time. Rapid recognition of the etiology of code arrest has been shown to improve outcomes in major studies and the advent of coronary intervention and therapeutic hypothermia has clearly improved cardiac and neurologic recovery. However, intracranial hemorrhage (ICH) is an overlooked cause of pulseless electrical activity as etiology of code arrest. Case 51 year old female with past medical history of stage 4 cervical cancer was found unresponsive 5 minutes after she was in her normal state of health. Emergency Medical Service (EMS) arrived and found her in Pulseless electrical activity (PEA) code arrest. Cardiopulmonary resuscitation (CPR) was initiated and after 10 minutes the patient regained ROSC. On arrival at our emergency department, the patient was hypothermic with a pulse at 102, respiratory rate at 16 and BP at 97/65, intubated and unresponsive, with a Glasgow coma scale of 4. Laboratory studies revealed a creatinine of 2.2 mg/dL and troponin of 0.19 ug/dL. Electrocardiogram (EKG) revealed non-specific ST-T changes in V3-V5. An urgent bedside transthoracic echocardiogram revealed LVEF of 20% and multiple wall motion abnormalities. A CT head was performed and showed a large posterior subarachnoid hemorrhage (SAH) and early cerebral edema. As the patient had a previous Stage 4 cervical cancer diagnosis and given her poor neurologic state with presence of early cerebral edema the patient was made comfort care and eventually passed on Day 2. Discussion ICH including SAH is a devastating cause of sudden cardiopulmonary arrest. It can mimic primary coronary events with serum
troponin elevation along with non-specific ST-T changes on EKG and specific wall motion abnormalities on initial echocardiogram. However, the treatment of primary coronary events will worsen ICH. In our literature review, we found 4 studies with a total of 4232 Out-of-hospital Code Arrests (OHCA), 133 (5.5%) being ICH-related code arrests. Of these cases, PEA/Asystole code arrest occurred in 97% of ICH-related cases. Currently, there are no guidelines that clearly describe role of emergent CT head in patients who have ROSC after OHCA. Recent studies have suggested that getting an initial CT head in ROSC PEA/Asystole cases can change outcomes. This case emphasizes the need to consider adding “Head bleed” to the ACLS’ “Hs and Ts” algorithm.

IDIOPATHIC COMPARTMENT SYNDROME

Prince Buzombo MD, UACOM at South Campus, PGY-1

Ali Raoof

Acute compartment syndrome (ACS) occurs when increased pressure within a compartment bounded by unyielding fascial membranes compromises the circulation and function of the tissues within that space. We present a case of a healthy 42-year-old Hispanic male with no significant past medical history who presented to the ED with acutely worsening right lower extremity sharp pain that radiated down his leg. His symptoms started a week prior to his presentation when he noted sudden onset right calf pain that quickly progressed to involve the lateral aspect of his right thigh. The pain was associated with fever of 103 F and nausea. The patient denied any preceding history of trauma, surgery, excessive exertion, limb compression, injection drug use, insect or animal bites. On inspection, patient was in extreme pain with noticeable difficulty in bearing weight on the affected extremity. His initial vital signs were Temperature 100.3, blood pressure 133/71, heart rate 100, respiratory rate 16. He had substantial pain on palpation of the right lateral thigh that worsened with passive stretch and felt tense with a firm "wood-like" feeling. There was no visible pallor. Also, sensation and motor strength were noted to be intact. His initial labs were significant for an elevated WBC count of 14.3(85%N), CPK - 1120, ESR – 33 and CRP of 134.50. His CT Right lower extremity was consistent with the exam findings and demonstrated swelling and edema involving the right vastus intermedius and right vastus lateralis muscles. With adjacent fluid along the entire cranial to caudal extent of both of these muscles concerning for compartment syndrome. Patient was taken to OR for an immediate fasciotomy. A large incision was made and as soon as the fascia was opened, the muscle immediately extruded, however, appeared viable throughout. The rest of the compartments were soft and without evidence of injury. The muscles were too edematous to allow primary closure. ACS most often develops soon after significant trauma, particularly involving long bone fractures of the lower leg or forearm. ACS may also occur following non-traumatic causes, such as ischemia-reperfusion injury, coagulopathy, certain animal bites, injection of recreational drugs, and prolonged limb compression. Idiopathic compartment syndrome, however, is a very rare disease entity with only a few case reports in published literature. Due to the numerous causes of lower leg pain and limited accuracy of physical exam findings, compartment syndrome can easily be overlooked if there is no obvious etiological factor in the history. This could delay appropriate and timely management leading to extremely high morbidity associated with this disease. Acute compartment syndrome is a surgical emergency which calls for prompt surgical fasciotomy if irreversible muscle necrosis is to be avoided.
A BLOODY CANCER: PROSTATE CANCER PRESENTING AS DIC

Jaspreet Chahal MD, UACOM at University Campus, PGY-1

Hani M. Babiker, MD.; Andrew Kovoor, MD.; Myke R. Green, Pharm.D.; Emad Hammode, MD.; Frederick R. Ahmann, MD

Introduction: Disseminated intravascular coagulation (DIC) is a rare and potentially lethal complication of solid tumors, often indicating advanced disease. Prostate cancer (PC) is associated with DIC in 13-30% of cases, however only 0.4-1.65% present with symptomatic bleeding diathesis. Patients with advanced prostate cancer most commonly present with bone pain or elevation in screening PSA. We present the first case of high-grade PC-associated DIC rapidly managed with gonadotropin releasing hormone (GnRH) receptor antagonist, degarelix.

Case: 61-year-old male with history of benign prostate hyperplasia presented with excessive bleeding from a leg wound, ecchymosis, left leg pain, and urinary retention for 2 weeks. Physical examination was significant for diffuse ecchymosis. Work-up revealed hemoglobin 8.2g/dL (14-16g/dL), platelets 72x103 (150-400x103/ul), fibrinogen 40mg/dL (130-350mg/dL), D-dimer >20µg/ml (<8804; 0.40µg/ml), PT 20.9s (11.5-15.5s), PTT 46.9s (25-36s), anti-thrombin activity 64% (81-121%), alpha2-antiplasmin activity <50% (88-120%), factor V activity 50% (72-139%), PSA 673ng/mL (<8804; 4ng/mL), and alkaline phosphatase (ALP) 1231IU/L (40-150IU/L). MRI revealed diffuse osteoblastic disease of the spine, sacrum, iliac bones, and ribs. MRI also revealed rare epidural deposits of metastatic disease in the spine. Biopsy was deferred given bleeding risk and evidence supporting diagnosis of PC. The patient received the rapid-onset GnRH receptor antagonist degarelix, 240mg subcutaneous injection. The DIC was treated supportively with a cumulative of 70 units cryoprecipitate, 8 units plasma, and 4 units packed red blood cells. A temporary foley was placed for urinary retention, determined to be secondary to mechanical obstruction and not spinal cord compression. Seven days following degarelix, clinical improvement was evident with normalization of coagulation parameters. PSA decreased to 125ng/mL, ALP to 600IU/L, and he regained full bladder function. The patient was discharged on monthly degarelix for management of his PC.

Discussion: DIC is a serious bleeding diathesis that may be the initial finding of undiagnosed advanced PC. Cancer-derived thromboplastic substances result in fibrinogen consumption, but excessive fibrinolysis demonstrated in the present case is rare. Fibrinolysis in patients with solid tumors is normally compensated by cancer cell-derived fibrinolytic inhibitors. In contrast, patients with advanced PC may rarely develop DIC with systemic bleeding due to excessive fibrinolysis resulting from over-production of plasminogen activators and depletion of fibrinolytic inhibitors. Degarelix, a rapid-onset once-monthly GnRH receptor antagonist, was chosen in this treatment-naive patient to rapidly control DIC. Our case is the first demonstrating the rare entity of DIC with epidural deposits in a PC patient rapidly treated with a single dose of degarelix. DIC is a clinicopathologic manifestation of PC and adroit management requires anti-tumor therapy and supportive measures to control bleeding. Although DIC forebodes a poor long-term prognosis in metastatic PC patients, the rapid response from degarelix is promising and exemplifies the drug as a critical agent in treatment of advanced PC complicated by DIC.
CEREBRAL SALT WASTING WITHOUT CEREBRAL PATHOLOGY

Parinita Dherange MBBS, UACOM at South Campus, PGY-1

Raul Medina, Mahmoud Kamel MD, Bijin Thajhudeen MD FASN, Saravanan Balamuthusamy MD FASN

Cerebral salt wasting WITHOUT cerebral pathology. Parinita Dherange MBBS, Raul Medina, Mahmoud Kamel MD, Bijin Thajhudeen MD FASN, Saravanan Balamuthusamy MD FASN.

INTRODUCTION: Hyponatremia seen in cerebral salt-wasting syndrome (CSW) is thought to be a disorder of salt and water homeostasis in patients with intracranial disease. It is characterized by hyponatremia and mild hypovolemia and is often misdiagnosed as SIADH. Recognition of this syndrome is important because the treatment differs significantly from other causes of euvoletic hyponatremia. CASE

PRESENTATION: A 78 year old female patient with history of essential hypertension and bipolar disorder was admitted to the ED for acute altered mental status, somnolence and confusion. Patient had two hospitalizations in the last two months due to hyponatremia. On examination she was afebrile, saturating 94% at room air, blood pressure 104/65 mmHg, HR 82 and RR 12-15/min and was euvoletic. Her systemic examination was unremarkable. Her significant lab workup included sodium of 114 with a baseline of 134 five months ago, potassium 4.1, bicarbonate 27, Cr 0.9, BUN 28, glucose 115, osmolality of 246 mOsm/kg and uric acid 2.8. CBC was unremarkable. Urine studies showed unremarkable analysis, urine sodium of 48 and osmolality of 480 mOsm/kg. Her CXR was unremarkable and Head CT showed no mass or lesions. A diagnosis of SIADH secondary to medications was made and patient was placed on fluid restriction and Lisinopril and Trazodone were discontinued which showed an initial improvement of sodium to 119 mMol/L; however two days later her clinical exam showed mild hypovolemia with moderate improvement in her mental status. Her serum uric acid and phosphorous were 2.4 mg/dL and 1.8 mg/dL respectively. Hypouricemia and Hyponatremia are seen in both SIADH and RSW. Fractional excretion of urate was 18%. Due to the mild hypovolemia, renal salt-wasting Syndrome (RSW) was considered in the differential diagnosis. She was started on normal saline 1 liter over 5 hours which improved her sodium by 6 meq in 24 hours to 128mMol/L and urine osmolality decreased to 320 mOsm/kg. Twenty four hours later her serum sodium was 133 mMol/L and urine osmolality was 310 mOsm/kg. Mild hypovolemia, low serum uric acid, elevated FeUrate in addition to improvement in serum sodium with no improvement in serum uric acid and FeUrate after isotonic saline substantiate the diagnosis of RSW and differentiates it from SIADH in our patient.

CONCLUSION: Hyponatremia in CSW/RSW is associated with mild hypovolemia and hypouricemia. Cerebral pathology is not a diagnostic requirement for this disorder. Fractional excretion of urate is a useful clinical tool to differentiate SIADH from RSW.

A CASE OF LEMIERRE’S SYNDROME AKA THE FORGOTTEN DISEASE

Nana Domfeh MD, St. Joseph's Hospital & Medical Center, PGY-1

Matt Stapleton MSIII, Sadia Moinuddin PGY3, Wesley R. Shealey MD
Lemierre’s syndrome also known as postanginal sepsis is a rapidly progressing and potentially life threatening disease seen in healthy adolescents and young adults, most commonly caused by Fusobacterium necrophorum. Fusobacterium necrophorum is a non-motile, sporulating gram negative anaerobe which is part of the normal flora of the pharynx, gastrointestinal tract and female genital tract. Infection usually begins with a sore throat, followed by fever, septicemia, thrombosis and metabolic abscess. We present this case this case of Lemierre’s syndrome to bring attention to the “forgotten disease” as with increasing antibiotic resistant organisms and decreasing awareness of the syndrome, this disease has reemerged.

ASE PRESENTATION: A 20 year old male presented to the emergency room with fever, chills and neck swelling for one week. Patient denied recent travel, sick contacts, drug abuse and recent dental work or tooth ache. On admission, he was febrile, tachycardic and had an elevated white count at 13000. On physical exam, he had cervical lymphadenopathy; however his lungs were clear to auscultation. Initial work up revealed negative Strep Group A and chest x-ray was negative for consolidations. Computed Tomography (CT) scan of the neck showed possible sialadenitis of the left submandibular gland; multiple enlarged, mildly dilated prominent adjacent nodes with no drainable fluid collection. CT scan of the neck also showed a patchy biapical pulmonary consolidations which prompted a CT scan of the chest. CT scan of the chest revealed diffuse ground glass nodular opacities as well as small left lower lobe cavitory lesions. Septic emboli could not be excluded from the scan. Patient was then started on piperacillin/tazobactam once blood cultures came back positive for Fusobacterium. Patient started to improve and was discharged home on two weeks of intravenous ampicillin/sulbactam and 4 weeks of oral amoxicillin clavulanate. DISCUSSION: This case emphasizes the importance of having a high clinical suspicion for Lemierre’s syndrome which is an uncommon but potentially lethal complication of otolaryngological infection. Pharyngitis (often viral) is the most common preceding otolaryngological infection but others such as peritonsillar abscess and otitis media have also been implicated. Infection usually spreads to the internal jugular vein resulting in thrombophlebitis with septicemia within one week of initial infection. Pulmonary involvement is most common in Lemierre’s syndrome. Lemierre’s syndrome is confirmed with a positive blood culture of Fusobacterium necrophorum as well as visualization of the internal jugular vein thrombosis by ultrasonography, contrast CT or MRI. Proper treatment should be immediately started with intravenous antibiotics to prevent further development of the disease. Having Lemierre’s syndrome on a physician’s differential diagnosis is important as Lemierre’s syndrome can progress quickly if not properly treated and may be fatal.

**WHAT HAPPENS IN VAGUS: A CASE OF POST PRANDIAL SYNCOPE**

Natalie Ertz MD, Mayo Clinic Arizona, PGY-1

Samuel Money, MD, Joseph Hoxworth, MD, Fadi Shamoun, MD

Introduction: Paragangliomas (PGL) are extremely rare, extra adrenal tumors originating from neuroectoderm, occurring anywhere from skull base to pelvic floor. These tumors migrate outside of the sympathetic chain and acquire glandular characteristics called paraganglia. Half of these occur in the head and neck especially in the highly vascularized carotid body or vagal nerve. We report a case of an extensive, unresectable paraganglioma with a possible hereditary component. Case: A 59 year old, previously healthy woman presented to an outside hospital with two weeks of post prandial syncope.
and labile blood pressure. However, she had endured a four month history of lightheadedness and intermittent neck pain prior to having her first syncopal episode. She recalled having a neck mass for a number of years. She also reported having a paternal family history of benign, asymptomatic, neck masses in three generations. At an outside facility these episodes were subsequently diagnosed as vasovagal bradycardia. Physical exam demonstrated bilateral palpable masses just under the angle of the mandible. Differential diagnosis at this point included congenital anomalies as an incidental finding on physical exam, not related to syncopal episodes. Magnetic Resonance imaging revealed multiple bilateral paragangliomas extending from the carotid bifurcations to the base of the skull. The tumors were engulfing the vagus nerve which made them in-operative. These were non functional by laboratory assessment of urine and plasma metanephrines and catecholamines. Genetic analysis revealed a Succinate Dehydrogenase mutation, an autosomal dominant mutation resulting in risk of paraganglioma tumors. Radiation therapy was selected as the best option available giving the local extension of the tumors. The patient underwent stereotactic radiation treatment of 30Gy over 5 days with a positive response on imaging and reduced frequency of syncopal events overall. She developed new hoarseness as a complication of radiation therapy. Discussion: PGL’s occurring in the head and neck are tumors of the parasympathetic division of the autonomic nervous system. Usually PGL tumors are usually benign in nature and are cured surgically if anatomic location is permitting of intervention. More recent literature suggests stereotactic radiation therapy should be first line therapy. Given the scarcity of cases it is likely best to consider each case on an individualized basis.

LOOKING OUTSIDE THE COLON IN MYH-ASSOCIATED POLYPOSIS

Ashley Garrett MD, Mayo Clinic Arizona, PGY-1

Kelly K. Curtis, MD

Introduction: MYH-associated polyposis is an autosomal recessive hereditary colon cancer syndrome, similar to Lynch syndrome and familial adenomatous polyposis (FAP) that involves germline mutations of the MYH gene which is involved in base excision repair. Desmoid tumors have been reported to affect 12-38 percent of patients with FAP, but only one published case reports a desmoid tumor in association with MYH-associated polyposis. We report a case of MYH-associated polyposis with adenocarcinoma of the colon and later development of a desmoid tumor.

Case: A 50-year-old female presented with newly diagnosed Stage IIA (T3, N0, M0) adenocarcinoma of the cecum found on a colonoscopy performed for workup of iron-deficiency anemia. She was also found to have multiple tubulovillous adenomas of the ascending, transverse, descending and sigmoid colon and a tubular adenoma of the rectum as well as multiple polyps in the stomach and duodenum. She underwent laparoscopic total colectomy with ileal rectosigmoid anastomosis. Pathology showed a moderately differentiated adenocarcinoma, grade 3/4. There was no lymphovascular invasion or perineural invasion, surgical margins were clear and there was no lymph node involvement. After the surgery, she opted to begin adjuvant chemotherapy with six cycles of 5-fluorouracil/leucovorin therapy. She then had genetic testing to evaluate for genetic colon cancer syndromes. She was found to have biallelic MYH mutations (Y165C), confirming a diagnosis of MYH-associated polyposis. No mutations of her APC genes were found. She continued to have biannual surveillance CTs and two years later was found to have a 2.9cm left upper quadrant mesenteric soft tissue mass. The mass was noted to have
hypermetabolism on PET CT. She then underwent exploratory laparoscopy with resection of the mass from the small bowel mesentery and retroperitoneum and small bowel resection. Pathology revealed a desmoid tumor negative for CD117, S-100, desmin, SMA, ALK, HMB-45, EMA with nuclear positivity for beta-catenin, consistent with desmoid tumor. She was placed on sulindac therapy post-operatively. Discussion: Extra-colonic manifestations of MYH-associated polyposis are diverse and include epitheliomas, congenital hypertrophy of the retinal pigmented epithelium and osteomas, with the most common being duodenal adenomatosis and gastric fundic gland polyps and adenomas. Desmoid tumors, while commonly seen in FAP, are rarely seen in this syndrome. This case reports the uncommon finding of a desmoid tumor in association with MYH-associated polyposis in a patient with no mutation of APC genes and no family history of malignancy, highlighting the diverse presentation of the syndrome and the importance of continued surveillance for extra-colonic manifestations.

LEFT FOOT ACTINOMYCETOMA: A CASE FROM ARIZONA

Laith Ghazala MD, UACOM at University Campus, PGY-1

Laith Ghazala1, MD, Danniel Zamora, MS-IV, Joseph Nelson, MS-III, Eileen Lorenz2, MD, Mihra Taljano, MD, Jennifer Hill, MD

Mycetoma is a chronic, granulomatous infection that can be either bacterial or fungal in origin. It tends to follow a long course of slowly growing mass along with the development of sinuses and discharge of granules containing the infected organism. It is a rare condition in the United States with only a few cases being described in the literature. This would be the first case originating in Arizona. A 53 year old, healthy, Native-American female, native to Arizona, presented with an ulcer and swelling over her left foot of 21 years duration. The ulcer initially appeared in 1991 as a result of a puncture injury where she stepped on a snake bone. The wound grew progressively and developed active yellow drainage. She sought medical attention twice in the past without complete resolution. She was diagnosed with actinomycetoma based on tissue culture in 1994. This time, she presented with a large ulcer extended over the dorsum of the foot from the metatarsals proximally with multiple yellowish discharges actively draining from the ulcer. She had intact Pedal and popliteal pulses. No lymph nodes appreciated. Sensation was intact throughout the lower limbs. Laboratory studies were unremarkable. X-ray demonstrated extensive erosive and destructive osseous changes. Lower limb MRI showed dot-in-circle signs characteristic of actinomycetoma. Tissue and bone biopsy were performed and histopathology report revealed partially devitalized bone with focus of filamentous Gram-positive bacilli bacteria, consistent with actinomycetes. Both bacterial and fungal cultures were negative. Therapy was initiated with Penicillin G 5 million units IV. Mycetoma is a chronic, subcutaneous infection that can be caused either by fungi or bacteria. In the case of fungus it is referred to as eumycetoma and in the case of bacteria it is called Actinomycetoma. Mycetoma is more common in Mexico, India, southeast Asia, Middle East and Sudan. It follows the mycetoma belt which encircles the entire world above and below the equator. Typically the organism is found in the form of granules in the soil. Infection is acquired through traumatic implantation of the organism into the cutaneous and subcutaneous tissue. Clinically, patients may experience a painless nodule in the initial area of the infection. The lesion usually becomes larger and forms the characteristic tumefaction. Eventually this mass may rupture and begin to form draining sinus tracts, which drain pus and granules that contain the causative agent. Diagnosis of
Mycetoma includes combination of culture, histology and radiology. Management of actinomycetoma has not changed dramatically over the last decade and includes the use trimethoprim-sulfamethoxazole for a prolonged period. In addition amikacin may be used in refractory cases.

**ZOONOTIC ENDOCARDITIS**

**Kirat Gill MD, St. Joseph’s Hospital & Medical Center, PGY-1**

Sadia Moinuddin MD PGY3, Nana Domfeh PGY1, Mirella Mircescu MD

Staphylococcus intermedia, identified in 1976, is a coagulase-positive gram-positive cocci occurring singly or grouped in pairs and clusters. It is a zoonotic organism that is part of the normal flora of dogs primarily, yet can also be found in cats and bird species. In addition, it is an invasive pathogen in these animals causing infections in many organs, but primarily the skin. Although it is pathogenic, rare cases of Staphylococcus intermedius infections have been reported in humans. Most cases involve close contacts with dogs however isolated cases have been reported in which patients denied contact with animals. Most importantly, Staphylococcus intermedius is often mistakenly diagnosed as Methicillin-Resistant Staphylococcus Aureus (MRSA) due to the close morphology of these two bacteria. To our knowledge, we present the only reported case of Staphylococcus intermedius bacteremia causing infective endocarditis with vegetations. An 81 year-old female with a past medical history significant for normal pressure hydrocephalus s/p VP shunt, diabetes mellitus type 2, hypertension, hyperlipidemia and bladder incontinence presented with a one day history of altered mental status and abdominal distention. Patient was found to have partial small bowel obstruction and was admitted to the hospital. While in the hospital, the patient was found to be septic secondary to a urinary tract infection from Klebsiella pneumoniae and Enterobacter Cloacae. On admission, blood cultures were drawn which were initially found to grow gram-positive cocci in clusters (GPCC) suggestive of MRSA. However, on hospital day four, the GPCC were in fact found to be Staphylococcus intermedius via detection with Peptide nucleic acid fluorescence in situ hybridization (PNA FISH). Subsequently, the patient underwent a transesophageal echocardiogram (TEE) for further evaluation of the newly diagnosed bacteremia. Interestingly, TEE revealed two 3mm fibrinous strands on the aortic valve suggestive of vegetations thought to have occurred secondary to the bacteremia. Repeat urine and blood cultures showed no growth. The patient was treated with long-term intravenous vancomycin therapy with gradual improvement of mentation. Of note, the patient denied recently being in contact with animals or having any at home. This case highlights the importance of being cognizant of the fact that not all GPCC are truly Staphylococcus aureus. Furthermore, it illustrates the utility of PNA FISH to differentiate between the various Staphylococcus species. Staphylococcus intermedius should be taken into consideration when the Staphylococcus genus has been isolated in culture, especially if the patient reports recent contact with animals.

**EMPHYEMA NECESSITATIS DUE TO METHICILLIN SENSITIVE STAPHYLOCOCCUS AUREUS, A CASE REPORT**

**Mohammed Hasoon MD, Maricopa Medical Center, PGY-1**

Layth Al-Jashaami MD, Ali Al-yacoobi MD, Syed Zaidi MD
Empyema Necessitatis Due to Methicillin Sensitive Staphylococcus aureus (MSSA), Case Report
Mohammed Hasoon MD, Layth Al-Jashaami MD, Ali Al-Yaqoobi MD, Syed Zaidi MD

Introduction: Empyema necessitatis is a rare complication of pleural space infections with contiguous spread outside of the pleural space to involve the soft tissues of the chest wall. It is commonly related to infections with Mycobacterium tuberculosis (TB), Actinomyces, Fusobacterium nucleatum, and Staphylococcus aureus.

Case: A 47-year-old male was diagnosed with left lower lobe pneumonia associated with a small left parapneumonic pleural effusion by his PCP one month prior to admission to our institution. He was treated as an outpatient with ceftriaxone 1 g single IM injection and then given a course of amoxicillin/clavulanic acid and azithromycin. He did not undergo a thoracentesis. He presented to the emergency department with painful swelling of his right hand and left posterior chest wall, beneath the left scapula. He recalled a fall onto his right hand with a flexed wrist in the interval since treatment for pneumonia. He reported weight loss and intermittent fevers for the previous month. He denied cough, shortness of breath, nausea, vomiting, loss of sensation in his upper extremities, history of TB/TB exposure. Upon admission, vital signs were normal. Physical exam showed a tense, tender, >10 cm left axillary mass and the scapula was displaced by this subcutaneous mass which was warm to the touch but without drainage. Decreased breath sounds in the left base, without wheezes, crackles or rhonchi. The right hand was swollen and had erythema with tenderness on the dorsum of the hand. No epitrochlear or axillary adenopathy was found. There were no lymphangitic streaks. Labs were remarkable for leukocytosis of 21.2 x 103/μL, hemoglobin of 9.2 g/dL, urine and blood cultures positive for MSSA. CT chest with contrast showed large left pleural effusion and a multiloculated rim-enhancing fluid collection in the chest wall measuring 15 x 13 x 7 cm. Lungs showed scattered nodules and an abscess in the left lower lobe. Echocardiogram did not show any evidence of endocarditis. Incision and drainage of the right hand abscess grew MSSA. The patient underwent drainage of both the subcutaneous abscess, with a pigtail catheter, and pleural space with a tube thoracostomy; cultures grew MSSA. He was treated with a 14 day course of cefazolin.

Conclusion: Primary care providers need to recognize complications of pneumonia. Parapneumonic pleural effusions add to the complexity and severity of community acquired pneumonia due to potential for complications. This case reminds us of the importance of diagnostic thoracentesis of every effusion that is encountered with pneumonia. Prompt recognition of parapneumonic effusion characteristics allows us to provide appropriate treatment based on current guidelines and helps prevent local and systemic complications.

Second Place – PGY-1 Poster
DISSEMINATED NOCARDIA CYRIACIGEORGICA INFECTION IN A LIVER TRANSPLANT PATIENT RECIPIENT
Allon Kahn MD, Mayo Clinic Arizona, PGY-1
Elizabeth J. Carey, M.D. and Holenarasipur R. Vikram, M.D.
Introduction: Liver transplantation has provided life-saving therapy for many patients with end-stage liver disease, with 1-year and 5-year survival rates of 85% and 68%, respectively. While advances in immunosuppression have decreased complications of organ rejection, increased susceptibility to infections poses a significant mortality risk. Infection leads to 25% of non-hepatic mortality in liver transplant patients and is the leading cause of death in the first 3 years post-transplant.

Case Description: A 37-year-old woman underwent deceased-donor liver transplantation for autoimmune hepatitis and primary sclerosing cholangitis overlap syndrome. Her post-transplant course was complicated by multiple episodes of acute cellular rejection requiring pulsed IV steroid therapy in addition to baseline immunosuppression with mycophenolic acid, tacrolimus, and prednisone. Despite this, she developed evidence of plasma cell-mediated autoimmune chronic rejection on liver biopsy and was treated with rituximab. Two months later, she developed a warm, tender right posterior thigh nodule that was evaluated by ultrasound, initially diagnosed as a hematoma due to a lack of vascularity and overlying redness. Three days later, she presented to the emergency department with fever, chills, tachycardia, and worsening pain and erythema of the nodule. The lesion was incised and drained and the patient was started on empiric piperaxillin/tazobactam and vancomycin. Gram stain and modified acid-fast stain revealed rare branching gram-variable, acid-fast organisms. Antimicrobial therapy was changed to TMP/SMX and imipenem. Brain MRI revealed no concerning lesions, however a chest CT revealed multiple cavitory nodules in the right lung. Bronchoalveolar lavage was negative for fungal elements. The patient’s fever and tachycardia resolved and she was discharged on hospital day three. Culture of the drained fluid subsequently grew out Nocardia cyriacigeorgica sensitive to both TMP/SMX and imipenem. She was treated with a 2-month course of IV imipenem and instructed to continue oral TMP/SMX 800/160 mg for one year. At most recent follow-up, patient is doing well and has no evidence of recurrent infection.

Discussion: Nocardiosis is an opportunistic infection seen most frequently in the first year following liver transplantation. Nocardia cyriacigeorgica is a newly identified species previously known as a subset of Nocardia asteroides with a particular antimicrobial susceptibility pattern. A high index of suspicion for Nocardia is important, particularly in disseminated disease, as estimated mortality rates range from 14-50%. Rapid identification of Nocardia by gram and acid-fast staining is critical, as most species are not susceptible to common empiric antimicrobial therapies, such as piperaxillin/tazobactam and vancomycin, as demonstrated in this case. Antimicrobial selection requires careful consideration, as greater than 40 percent of the common isolates N. farcinica and N. abscessus are resistant to TMP/SMX and imipenem, respectively.

SOMETHING FOUL IN THE BOWEL

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Natasha Sharda & Bujji Ainapurapu

INTRODUCTION: Superior mesentery artery syndrome (SMAS) is a clinical disorder of vascular compression affecting the third part of the duodenum. The superior mesentery artery transverses at an angular path past the duodenum supported by normal fat and lymphatics. In SMAS this angle is
narrowed which can be a rare cause of small bowel obstruction. Here we discuss an unusual case of severe duodenal obstruction caused by SMAS.

CASE PRESENTATION: The patient is a 46-year-old woman with a complicated past medical history of severe Rheumatoid arthritis, with intra peritoneal abscess s/p draining from chronic immunosuppression, Diverticulitis s/p partial bowel resection and anastomosis, who presented with altered mental status and accelerated hypertension. During a prolonged hospital course incidental finding on KUB reviled severely dilated loops of bowel. Upon questioning, patient had a history of recent weight loss of 60lbs in the last year and current complaints of abdominal pain, hiccups, intense burping and vomiting. She had no bowel movements for several days and had not been passing gas. Given the patients past surgical history there was concern for small bowel obstruction and CT was ordered to rule out small bowel obstruction. CT scan revealed marked gastric distention. Nasogastric tube was placed for gastric decompression with low intermittent suction, until endoscopy could be preformed. During endoscopy scope was passed through the pylorus into the first part of the duodenum without resistance. Imaging yielded a 25mm nodular ulcer with irregular raised borders on the greater curvature of the stomach. As endoscopy did not explain the gastric distension MRI was performed and showed significant pinching of the transverse duodenum between the aorta and SMA with dilatation of the duodenum proximal to this area without any evidence of masses or lymph nodes. Given the patients recent weight loss it was proposed that her symptoms was the result of superior mesentery artery syndrome. Patient was treated conservatively and she slowly recovered and tolerated diet.

DISCUSSION: Superior mesenteric artery syndrome is a rare but well recognized cause of duodenal obstruction. Through our review we have found approximately 400 cases described within the literature. This condition can be treated conservatively with gastric decompression, nutritional support, and special swallowing techniques. Surgical interventions to bypass the obstruction such as duodenojejunostomy or gastrojejunostomy or techniques to alter the aorto-mesenteric angle have been employed with success when conservative measures fail. SMAS may easily be confused with gastric outlet or proximal small bowel obstruction, however a high clinical suspicion is crucial for diagnosis and radiographic imaging with CT or MRI can provide confirmation.

ALTERED MENTAL STATUS IN HIV-INFECT PATIENT: OPPORTUNISTIC INFECTION OR ANTIRETROVIRAL THERAPY

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Altered mental status in an HIV infected patient comprises a very broad differential. In developing countries altered mental status often results from opportunistic infections of central nervous system due to unavailability of antiretroviral therapy. However, in developed countries, rarely an opportunistic infection is isolated as a cause of neurologic disease. Though antiretroviral therapy (ART) has dramatically decreased the incidence of opportunistic infections, ART itself is held responsible for patients’ altered mental status as it manifests as Immune Reconstitution Inflammatory Syndrome (IRIS). IRIS is a clinical condition constituting an exaggerated inflammatory response in a HIV patient upon
commencing antiretroviral therapy. It typically manifests as a paradoxical worsening of a treated opportunistic infections or unmasking of a subclinical infection, after starting antiretroviral therapy. Non-infectious IRIS has also been described in the literature and our patient is an example of such a case. 38 year old female with recent diagnosis of HIV/AIDS (CD4:160) with PCP pneumonia on antiretroviral therapy (Stribild: combination of elvitegravir, tenofovir, emtricitabine and cobicistat) for almost two months and Bactrim prophylaxis that presented with an episode of sudden onset of confusion. On admission patient was alert and oriented; however, per her husband, patient was unresponsive at home for almost 20 minutes, without any clinical manifestations of seizure-like activity. Review of systems was significant for new onset headaches for one week, fevers, one episode of abnormal eye movement with facial twitching. Patient was admitted to medicine floor with a working diagnosis of subclinical seizures. Over the next four days patient developed febrile seizures without supportive EEG findings and underwent extensive negative work up, including several lumbar punctures, for infectious etiology. Patient was managed symptomatically with anti-seizure medications (phenobarbital, levetiracetam, phenytoin and volpraic acid) without any resolution. Lastly, high dose dexamethasone was started empirically to treat immune reconstitution inflammatory syndrome and patient started to improve clinically. Infection negative IRIS is a fatal condition that presents as a subacute generalized encephalopathy with alteration in mental status and seizures, and if untreated results in coma and death. In these patients the T-cells are assumed to be targeting viral reservoirs in the brain or the brain tissue itself with a possible autoimmune etiology. Extensive analysis does not yield infectious cause and these patients respond well to corticosteroids.

**TRANSCATHETER PATENT FORAMEN OVALE CLOSURE VERSUS MEDICAL THERAPY FOR CRYPTOGENIC STROKE: A META-ANALYSIS OF RANDOMIZED CLINICAL TRIALS**

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Irbaz Riaz, Abhijeet Dhoble, Ahmad Mizyed, Paul Hsu, Muhammad Husnain, Kapildeo Lotun, Kwan S Lee

Introduction: It is shown that 30-40% of strokes are cryptogenic in nature. Many observational studies have demonstrated an association between cryptogenic stroke and patent foramen ovale (PFO) with a prevalence of around 25-30%. The optimal treatment strategy for this condition is still unclear. The purpose of this study was to analyze and aggregate data examining the safety and efficacy of transcatheter device closure versus standard medical therapy in patients with patent foramen ovale (PFO) and cryptogenic stroke.

Methods: A meta-analysis from the search of published data identified 3 randomized clinical trials for inclusion. The primary outcome was a composite end-point of death, stroke and transient-ischemic attack (TIA). Pre-defined subgroup analysis was performed with respect to baseline characteristics including age, sex, atrial septal aneurysm and shunt size. Data was synthesized using a random effects model and results presented as hazard ratios (HRs) with 95% confidence intervals (CIs).

Results: A cohort of 2,303 patients with a history of cryptogenic stroke and PFO were randomized to device closure (n=1150) and medical therapy (n=1153). Mean follow-up was 2.5 years. Transcatheter closure was not superior to medical therapy in the secondary prevention of stroke or TIA in intention-to-treat analysis (HR: 0.66, 95% CI: 0.43 to 1.01; p = 0.056). However, the results were statistically
significant using per-protocol analysis (HR: 0.64, 95% CI: 0.41 to 0.98; p = 0.043). Males had significant benefit with device closure (HR: 0.48, 95% CI: 0.24 to 0.96; p = 0.038).

Conclusions: This meta-analysis found that device closure of PFO was found to be superior to standard medical therapy in secondary prevention of cryptogenic stroke using per-protocol analysis. However, the difference was not statistically significant when intention-to-treat analysis was performed.

**VITAMIN K DEFICIENCY SECONDARY TO MALNUTRITION FROM AGGRESSIVE CHEMOTHERAPY**

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Malignancies will induce a hypercoagulable state and may require aggressive chemotherapy resulting in an inability to obtain essential nutrients. If a patient develops a DVT, they will require prolonged anticoagulation. Vitamin K Deficiency may arise due to the inability to ingest food. This deficiency is relatively rare and will result in excessive bleeding states. A 57 year old female patient presented to the SVRHC Emergency Room with an initial complaint of shortness of breath and was incidentally found to have a supratherapeutic INR that was not measurable. The patient had previously been on coumadin due to a past medical history of Factor V Leiden mutation, ovarian cancer, and Stage III sigmoid adenocarcinoma. She had previously been receiving aggressive chemotherapy to treat her Stage III sigmoid adenocarcinoma and was not able to tolerate adequate PO nutrition as she had been experiencing weight loss of 40+ pounds over the last several months. The patient was admitted to the hospital and was determined to have taken an excess amount of coumadin resulting in her present condition. Coumadin was immediately stopped, Vitamin K and FFP was transfused, and after 3 days of monitoring the patient was subsequently discharged to follow up with her PCP while holding coumadin until that visit. One week following the initial ER visit, the patient sent back to the ER after PCP found the patient’s INR had been increasing regardless of holding coumadin. The patient’s INR was again not measurable and prior to reversal with FFP and vitamin K, a DCP (Des-Gamma-Carboxy Prothrombin) was ordered and sent for interpretation. She was stabilized and during the course of her stay in the hospital, it was determined that the patient had a vitamin K deficiency secondary to malnutrition. During the patient’s stay in the hospital, she was started on adjunct TPN and PO diet, and was stabilized. This case illustrates the potential for DCP to be used in patients who are hypercoagulable secondary to malignancies who present with supratherapeutic INR. Although coumadin is likely the cause of a high INR, patients who are receiving aggressive chemotherapy or have in the past may also suffer from severe vitamin deficiency. Recognition of this possibility may allow for better management of hypercoagulable states.

**BASAL CELL CARCINOMA AND IT”S EXTENT OF EROSION**

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Basal cell carcinoma (BCC) accounts for 80% of all skin cancers, and most commonly presents as a papule or nodule with telangiectasia, plus no associated systemic symptoms. The risk of metastasis is estimated to be between 0.05 to 0.1 percent [1,2]. Patients diagnosed with BCC have approximately 20-40% chances of recurrence within one to five years respectively [3]. The prognosis for most patients
with BCC is excellent, if treated early. These lesions are typically slow-growing, but when left untreated can ulcerate and cause considerable morbidity by local destruction of skin, cartilage and bone. The patient was a 63 year old Spanish speaking male patient, who worked in the outdoor fields his entire life, and had not seen a primary care physician in 18 years. He was brought to the Emergency Department in an altered mental state, hypotensive, tachycardic, and tachypneic. On physical examination, a 15 cm x 15 cm large full thickness necrotic ulcer with foul smelling odor was seen on the left side of his face. As per his daughter, it started as a skin tag in front of the left ear in 1995, which gradually transformed into an invasive ulcer that on imaging and physical exam showed erosion into the mandible, adjacent soft tissues, exposure of his carotid artery and internal jugular vein, and temporal bone. There was also temporal/occipital brain involvement, which caused his right eye to protrude out of the orbital cavity with complete loss of vision for the past few years. He was initially resuscitated in the ICU for septic shock and severe anemia (Hg 4), and subsequently underwent a biopsy that showed poorly differentiated basal cell carcinoma. Several consultations were placed including plastic surgery, neuro-surgery, ENT, and wound care for evaluation of his extensive bone and intracranial involvement. Unfortunately, due to the extensive invasion of the tumor he was not considered a surgical candidate. Hospice options were discussed and recommended to the patient and family. Basal cell carcinoma (BCC) is the most common skin malignancy with a benign and non erosive presentation. Apart from presenting with a pearly pink or flesh colored papule, patients with BCC generally have no other complaints referable to the tumor. Due to the increased reoccurrence rate of BCC after initial diagnosis and treatment, dermatologists recommend regular follow up every 6 months in the first year post treatment, and then annually from thereon. Our case illustrates the destructive potential of basal cell carcinoma and the extent of damage it can cause when left unattended, by eroding into the surrounding structures, which depending on the location, can be quite detrimental. With appropriate follow up, complications such as these can be avoided, and patients can have an improved morbidity and mortality.

**NOT A SIMPLE ABSCESS**

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Muna Omar, Naser Mahmoud, Ahmad Al-Khashman

Introduction: Infections are the leading cause of morbidity and mortality in kidney transplant patients; the increasing number of the immunocompromised patient has not only augmented infections by specific pathogen but also with opportunistic infections such as nocardia. Case presentation: We describe a 49 year old female patient with history of Adult Polycystic Kidney Disease (APKD) that was treated with kidney transplantation and on chronic immunosuppressive therapy. She presented with worsening shortness of breath, dry cough and right sided pleuretic chest pain of 10 days duration, associated with right upper abdominal painful skin lump. Physical exam: Temperature: 39.2 C, pulse: 103, RR: 20, BP: 171/87, O2 SAT: 98 on room air. Chest exam showed decrease air entry bilaterally on the bases with coarse crackles on the right base. A 3 cm fluctuant tender localized skin induration on right upper abdominal wall was identified. Labs and Imaging: WBC:6.6 N:81, HB:8.8, plt:224, creat:2.5, Na: 135, k:5.5, HCO3:14, CA :9.1, ALT:60, AST:11, Lactate:1.2 , CMV by PCR Neg, Coccidioidomycosis serology Neg, Blood culture Negative, Pus culture from the abdominal abscess showed Nocardia species.
CXR showed multifocal pneumonia, CT of the chest showed bilateral multifocal pneumonia but No abscesses is seen. Renal Ultrasound was unchanged from prior with no acute changes. Course in hospital: The Patient was admitted as a case of multifocal pneumonia and started on intravenous Meropenem and Vancomycin. Skin abscess was drained in the ED and the pus sent for cultures. Wound culture came back 2 days later showing Nocardia. Sputum culture was not sent because the patient was unable to produce sputum. Patient was continued on iv meropenem and stared on double strength Bactrim twice daily with gradual improvement in symptoms with a working diagnosis of Disseminated Nocardiosis. Discussion: Nocardiosis is a localized or disseminated infection caused by gram positive rod shaped bacteria, that is catalase positive, partially acid fast beaded branching filament. The infection most commonly affects the lungs, skin, and CNS. It principally occurs in individuals with cellular immunodeficiency and should be suspected in those individual when presented with respiratory, cutaneous or neurological symptoms. The lungs are the most common site of infection and can cause disseminated infection if treatment is not adequate initially. Trimethoprim/sulfamethaxazole is the treatment of choice, and minocycline is an alternative to Trimethoprim/sulfamethaxazole, also imipenem, amikacin and linezolid can be used in severe respiratory infection. Treatment is usually for 6 months in immunocompent patients and for 1 year in immunocompromised patients. Proper wound care is also critical for those with skin infection.

REFRACTORY HYPERCALCEMIA IN A CASE OF DIFFUSE LARGE B CELL LYMPHOMA

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Primary lymphoma of bone, usually Diffuse Large B Cell Lymphoma, accounts for less than 2% of all lymphomas, and rarely presents as refractory hypercalcemia. A 71 year-old male presented to the University of Arizona Medical Center Emergency Department with a 3-month history of fatigue, constipation and 50 lb weight loss. Patient also complained of a 4-6 month history of right knee pain, which had acutely worsened during the last 2-3 weeks. Previous MRI of left femur showed idiopathic fracture, with no soft tissue mass surrounding the joint. Interventional Radiology guided bone biopsy of the right femur had inconclusive results of necrotic cells, suspicious for malignancy. Laboratory workup upon presentation revealed a Ca2+ level of 18.1 mg/dL. Malignancy workup for hypercalcemia was initiated. Computed tomography of chest, abdomen, and pelvis, showed a small nodule in the right posterior lung base. Bone scan showed a single blastic lesion in the right femur. Lactate dehydrogenase was within normal limits, and a 1,25 hydroxyvitamin D was elevated. PTH and PTHrP were low/low normal. SPEP and UPEP were positive for free kappa light chains in the urine, with no immunologic M spike or monoclonal predominance. A bone marrow biopsy showed no plasma cell predominance. Second IR guided bone biopsy was only positive for necrotic cells suspicious for malignancy. Orthopedics was consulted for an open bone biopsy. Prior to biopsy a repeat MRI of the leg was done which showed a large soft tissue mass with invasion into the distal femur. Open bone biopsy with soft tissue biopsy confirmed primary lymphoma of the bone, diffuse large B cell subtype. Hypercalcemia was controlled with intermittent pamidronate and oral prednisone and he was scheduled for R-CHOP therapy as an outpatient. Primary lymphoma of bone is a rare lymphoma, that may present in various forms, making it an increasing challenge to diagnose. Patients presenting with hypercalcemia, refractory
to treatment, along with localizing symptoms of bone pain must be investigated aggressively. Furthermore, in patients with pathological fractures the need for open surgical bone biopsy should be considered as a crucial step in diagnosis.

**DRESS SYNDROME - A LIFE THREATENING CONDITION**

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Juxiang Huang, DO

DRESS Syndrome – A life-threatening Condition  Archana Nair MD; Juxiang (Jennifer) Huang, DO; Bujji Ainapurapu, MD. University of Arizona College of Medicine at South Campus  DRESS syndrome (Drug reaction with eosinophilia and systemic symptoms) is a life threatening idiosyncratic hypersensitivity reaction featuring a constellation of symptoms including fever, rash, lymphadenopathy, hematological abnormalities like eosinophilia, atypical lymphocytosis and internal organ involvement. This case describes a 38 year-old male with DRESS syndrome secondary to Vancomycin with resolution of symptoms secondary to prompt diagnosis and high dose steroid use. A 38 year old male, with PMH of diabetes mellitus type 2 presented with fever of 102.70 F. He had MRSA pneumonia and bacteremia 3 weeks ago and was initially treated with Vancomycin. He was discharged home to continue a total of 4 weeks of IV Vancomycin. 17 days after his first Vancomycin dose, he came back to the emergency department with fever. He had been receiving his antibiotic as scheduled and also had therapeutic range of Vancomycin trough levels. Blood cultures and urine cultures were obtained which were all negative. Fever, macular rash and leukopenia while on treatment, were contributed to Vancomycin-related drug fever. He was then switched to Daptomycin. However, patient continued to have fever at home and represented back to the emergency department in two days with fever and significant non-pitting edema of the entire body especially the face and periorbital region. There was also a patchy confluent non-blanchable, non-exfoliating macular rash all over his body with sparing of mucous membranes. Axillary and inguinal lymphadenopathy were also palpable. Labs showed a WBC count of 7600/µL with remarkable eosinophilia of 1600/µL. His liver function tests were also elevated and CT chest demonstrated subcarinal and hilar lymphadenopathy. Dermatology was consulted and skin biopsy performed which revealed no evidence of immune deposits for IgA, M, G, C3, C4 or fibrinogen ruling out a close differential diagnosis, Steven Johnson syndrome. The patient was diagnosed to have DRESS syndrome and was transferred to the intensive care unit. Patient received IV fluid hydration, high dose steroid and all antibiotics were stopped. His fever has subsided on day 3 of Prednisone treatment. His body edema and rash improved. In our patient, diagnosing DRESS syndrome was a challenge as there where many confounders that could explain his symptoms and progress of disease. Differential diagnosis were persistent sepsis, lymphoma, atypical infections and other hypersensitivities like Stevens-Johnson syndrome. DRESS has a significant mortality rate of 10%. Fever with a macular rash following treatment with a drug typically within 2-6 weeks, eosinophilia in the serum and associated organ involvement should have prompt differential of DRESS syndrome. Early recognition of the syndrome and cessation of the offending agent could have mortality benefit.
ANOMALOUS CORONARY ARTERY ARISING FROM THE OPPOSITE SINUS: AN INSIDIOUS CLINICAL ENTITY

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Introduction: Congenital coronary artery anomalies describe a broad set of anatomical abnormalities. A subset of this category is when a coronary artery arises from the opposite coronary sinus, which can cause angina, syncope, and predispose to sudden cardiac death – usually during intense exercise. It has been found that the specific course of the anomalous coronary artery can help predict risk of sudden death. Making an early diagnosis is crucial, as surgery is often indicated to decrease this risk. Yet, congenital coronary artery anomalies can masquerade as other more common clinical entities in otherwise healthy young persons; thus, clinical suspicion must be high.

Case: The patient is a 22-year-old male who presented to the outpatient cardiology department with chest pain and dyspnea upon exercise since childhood. These symptoms had been attributed to asthma for most of his life. However, the patient did not improve with standard asthma treatment. About one year ago the patient was running on a mountain and suddenly developed nausea, vomiting, and subsequent chest pain. At this point his primary care provider referred the patient to a cardiologist. On exam, vital signs were normal. The patient was tall, slim, well developed and in no acute distress. Cardiovascular and pulmonary exams were normal. Electrocardiogram revealed a subtle rSr pattern in the anterior leads consistent with intraventricular conduction delay within the right ventricle. Transthoracic echocardiogram revealed a left ventricular ejection fraction of 60% with no wall motion abnormalities. An exercise nuclear stress test did not reveal any myocardial ischemia. A computed tomography angiogram revealed that the ostium of the right coronary artery (RCA) arose from the left coronary cusp. The RCA had a partially intramural course within the aortic root. The patient was placed on metoprolol and referred to cardiothoracic surgery for evaluation and repair.

Conclusion: This case illustrates how anomalous coronary arteries arising from the opposite coronary sinus of Valsalva can masquerade as more common clinical conditions. Notably, the literature reveals that a stress test is often negative despite a symptomatic anomaly, as in this case. Coronary arteries arising from the opposite coronary can cause chest pain, syncope, and sudden death, though most are asymptomatic. This abnormality is quite common; precisely how common remains uncertain. One large prospective study reported an incidence of 1.07%. More controversial is what proportion of these anomalies predisposes to sudden death and should therefore be surgically repaired. One risk factor, which is present in this case, is an intramural course of the anomalous artery. Further investigation into how to risk stratify these patients is necessary for more rational treatment in the future.

HYPERCALCEMIA AND HYPOKALEMIA ASSOCIATED WITH DISSEMINATED COCCIDIOIDOMYCOSIS AND HIGH DOSE FLUCONAZOLE THERAPY

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Irbaz Bin Riaz (MD), Jawad Bilal (MD)
Although infectious diseases are not often considered in the differential diagnosis of hypercalcemia, hypercalcemia is a well known complication of infectious and non-infectious granulomatous diseases. We report a case of hypercalcemia and hypokalemia in a patient with disseminated coccidioidomycosis on fluconazole therapy. Case Description: A 22 year old African American male presented with fatigue and constipation. He had previously been diagnosed with disseminated coccidioidomycosis in May of 2013. At that time he had presented with fever and back pain. He was found to have a L2 vertebral body and epidural abscess and a right psoas abscess. Cocci titers were positive at 1:256. The patient was started on amphotericin B and underwent abscess drainage. His condition improved and he was discharged on fluconazole 600mg daily. During a follow-up visit in infectious disease clinic, he was found to have calcium of 15.5 and potassium of 2.1. He was admitted for further evaluation. He was found to have suppressed levels of PTH, normal levels of 1,25-dihydroxy vitamin D, and negative PTHrP. Morning cortisol, aldosterone, and renin levels were normal. He was treated with potassium replacement, hydration, subcutaneous calcitonin, and pamidronate. His calcium level normalized on hospital day 5, but he continued to require potassium replacement. He was discharged on oral potassium chloride and fluconazole, which was increased to 800mg daily. Discussion: Disseminated coccidioidomycosis is an uncommon but reported cause of hypercalcemia. In sarcoidosis, there is strong evidence that hypercalcemia is associated with high serum levels of calcitriol. Activated pluripotent macrophages in granulomatous tissue convert circulating 25-hydroxy Vitamin D to 1,25-dihydroxy Vitamin D. Most reported cases of coccidioidomycosis have normal calcitriol levels, as was seen in our patient. Only one case of disseminated coccidioidomycosis and hypercalcemia along with an elevated PTHrP has been reported. Another theory of hypercalcemia in coccidioidomycosis relates to elevated cytokines with bone resorptive potential in patients with active disease. However, apart from sarcoidosis, the pathogenesis of hypercalcemia in other granulomatous diseases has not been studied in depth. If it is mild, infection-related hypercalcemia usually responds to treatment of the underlying infection. Acute management of hypercalcemia due to coccidioidomycosis includes the infusion of normal saline plus administration of furosemide. Our patient had a more robust response with pamidronate compared to normal saline. It is suggested that in difficult to treat infections, treatment of hypercalcemia with corticosteroids should be avoided, though it has not been studied directly.

Hypokalemia in our patient is presumed to be a side effect of high dose anti-fungal therapy as no other cause could be ascertained. He will follow up with infectious disease clinic for continued management.

AN INTRIGUING CASE OF TWO CARDIOMYOPATHIES

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Steven Lester M.D.

Introduction. Left ventricular noncompaction (LVNC) and hypertrophic cardiomyopathy (HCM) are both primary genetic cardiomyopathies which may share similar mutations in genes encoding for sarcomeric proteins. LVNC is characterized phenotypically by hypertrabeculations of the myocardial wall with deep intertrabecular recesses resulting in a bilayered myocardium consisting of a compacted and noncompacted layer while HCM is characterized by unexplained thickening or hypertrophy of the left and sometimes right ventricle wall. Within family members sharing the same gene mutation, the phenotypic expression of HCM (primarily the apical variant) in one member and LVNC in another has
been recognized and reported. This case however, illustrates the evolution of the phenotypic expression of HCM to LVNC in a single individual over a span of three years not previously reported.

Case. A 47 year old man with HCM diagnosed at the age of 29 presented to establish medical care in 2006. He was asymptomatic at that time. Echocardiography revealed HCM with a LV septal wall thickness of 25mm and no resting or provable left ventricular outflow tract obstruction. His left ventricular ejection fraction was 46%. Genetic testing for HCM revealed a mutation in the Myosin Binding Protein C (MYBPC3) gene. The c.772G>A nucleotide change with amino acid change p.Glu258Lys as a result of a splice site or missense mutation was reported as causative of HCM. The patient’s mother shares the same mutation and has too been diagnosed with HCM. Three years later, he started to experience shortness of breath on exertion. A repeat echocardiogram revealed that morphology of the left ventricle had changed to now that of LVNC. There was concordance for all three available diagnostic criteria for LVNC with the ratio of the thickness of the compacted layer (C) to noncompacted layer (NC) greater than 2:1 measured at end-systole (Jenni et al.), ratio of compacted to sum of compacted and noncompacted thickness at end diastole less than 0.5 (Chin et al.) and four or more trabeculations in the LV wall visible in a single plane (Stollberger and Finsterer).

Discussion. This case questions our current understanding of LVNC as an arrest of myocardial development, highlights how the features of LVNC and HCM may overlap and the variability of phenotypic expression.

**DOUBLE, DOUBLE TOIL AND TROUBLE - AN UNUSUAL CASE OF DYSPHAGIA**

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Varun Takyar MD

Double Double Toil and Trouble, An Unusual Case of Dysphagia

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Introduction: Dysphagia in elderly patient is an alarming symptom warranting immediate evaluation. Esophageal etiologies of dysphagia include intrinsic or extrinsic mechanical lesions, motility dysfunction or functional disorders. Here we describe a rare cause of esophageal dysphagia due to epiphrenic diverticula.

Case: 79 year-old female with a PMH of GERD, atrial fibrillation, hypothyroidism and HTN presents to our hospital with dysphagia, epigastric abdominal pain, nausea and vomiting. Epigastric pain is described as 10/10 sharp, non-radiating, which began shortly after swallowing her medications, and was relieved by vomiting. ROS reveals difficulty tolerating solids with weight loss of ~10 lbs. in the last 2 months. She was previously placed on a PPI without alleviation of dysphagia and pain. Upon examination, vital signs were stable and she presented with a soft non-distended abdomen with epigastric tenderness. Laboratory studies obtained CBC, CMP, PT/INR, lactate, U/A, all being within normal limits. Barium esophagram revealed obstruction of distal 3rd of esophagus with two partially constricting thick rings without pouching in between. EGD showed tortuosity of middle to distal third of esophagus with abnormal middle esophageal motility and spasticity of esophagus body. Two medium-
sized non-bleeding diverticuli were present at lower 3rd of esophagus ~ 30cm from the incisors. High Resolution Manometry was performed as an outpatient and revealed LES pressure 14.8mmHg, LES length of 4.0cm, esophageal length 25.9cm, abdominal length 1.5 cm and relaxation 78%. 50% esophageal ineffective motility with contraction amplitude 89.2mmHg. Interpretation of findings revealed diffuse esophageal spasm with dysmotility. She returned to clinic with continued epigastric discomfort and dysphagia. Given her advanced age, she was advised to take pureed diet in slow fashion and to consider diverticulectomy myotomy using robotics.

Conclusion: Epiphrenic diverticula are rare as they account to <10% of all esophageal diverticula. Epiphrenic diverticula usually present with dysphagia, odynophagia, chest pain or regurgitation. The size of the diverticula impact the severity of the symptoms and in many, are debilitating with progressive worsening dysphagia and poor overall quality of life as seen with our case. High Resolution Manometric evaluation is a necessary component of the workup as it usually reveals an underlying esophageal motility disorder. Diverticulectomy combined with selective myotomy has provided resolution of dysphagia and abdominal pain. Medical management such as CCB or PPI has been tried in the past however found ineffective in alleviation of symptoms.

MAKING BIPHASIC WAVES

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Aaron Fernandes MD

Introduction: Wellen’s Syndrome, known as Left Anterior Descending (LAD) coronary T-wave syndrome, is a pattern of electrocardiographic T-wave changes in unstable angina pectoris patients. It”s associated with a critical stenosis of the LAD artery. Sometimes, ECG may be the only indication of an impending extensive anterior wall myocardial infarction in an asymptomatic patient. This case illustrates the importance of early recognition and intervention in a patient presenting with the less common variant of this potentially lethal syndrome.

Case Description: A 73-year-old female with a past medical history of hypertension, CHF and an MI with stent placement presented with severe, sharp, sub-sternal chest pain that radiated up to her left shoulder and neck. Symptoms were relieved with administration of three sublingual Nitroglycerins. While in the emergency room an EKG and Troponin were within normal limits. Patient was diagnosed with Unstable Angina. A repeat EKG (Figure 1) was performed which showed biphasic T-waves in leads V2 and V3 without pathologic Q waves or ST segment elevation. Patient underwent urgent coronary angiography, which showed a new 60% focal, heavily calcified proximal to mid LAD stenosis. Successful PCI of proximal to mid LAD with placement of a drug-eluting stent was performed and the patient was started on dual antiplatelet therapy. The following day the patient remained clinically stable and chest pain free.

Discussion: The simplified criteria for Wellen"s syndrome are: • History of chest pain • Little or no cardiac enzyme elevation • No pathologic precordial Q waves or ST-segment elevation • No loss of precordial R waves • Characteristic ECG findings The characteristic ECG changes of this syndrome occur in the T-waves and occur in 2 forms. The more common (76%) of the 2 forms is Type II, which presents
with deep inversion of T-waves in precordial leads V1-V4. The less common (24%) of the 2 forms and potentially more lethal is Type I, which consists of biphasic T waves in leads V2-V3, as seen with our patient. The sensitivity, specificity, and positive predictive value of T-wave inversion for significant LAD stenosis is 69%, 89%, and 86%, respectively. In conclusion, when evaluating and risk stratifying patients with Unstable Angina, the ECG is of great value for recognizing a subset of patients with a proximal LAD lesion having a poor prognosis. The ECG abnormalities of Wellen’s syndrome occur during a pain-free interval when other evidence of ischemia or angina may be absent. The ECG may be a physician’s only clue to identifying patients that are at high risk for potentially fatal anterior wall myocardial infarctions. It is imperative that Wellen’s Syndrome be considered in the physician’s differential so early cardiac catheterization and potentially life-saving therapy can be performed.

EFFUSIVE-CONstrictive PERicarditis: An UNCommon MANIFESTATION OF Graft-VERSUS-Host DISEase?

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Dr. Christopher Appleton, Dr. Evan Kransdorf

Introduction: Effusive-constrictive pericarditis is a late sequela of pericarditis that can lead to thickening, scarring, and contraction of the pericardium and ultimately low cardiac output diastolic heart failure.

Case description: A 64 year old gentleman with a history of myelodysplastic syndrome who had previously undergone a matched, unrelated stem cell transplant, developed acute graft-versus-host disease and presented seven months after transplant with pleuritic chest pain and an ECG with diffuse ST segment elevation. An echocardiogram showed a small posterior pericardial effusion, pericardial thickening, and a LV ejection fraction of 50%. The patient was diagnosed with acute pericarditis and was treated with prednisone. A week later, he complained of fatigue and lightheadedness and was found to be in atrial fibrillation with a rapid ventricular response of 148 beats per minute. After spontaneous conversion his metoprolol dose was increased, colchicine was initiated, and he was discharged. Three months later he was re-hospitalized with rapid atrial fibrillation and was treated with intravenous amiodarone. A repeat echocardiogram showed a moderate pericardial effusion with an early tamponade physiology. Pericardiocentesis led to the removal of 300 mL of serosanguinous fluid, described as anterior epicardial fibrinous exudate, with no evidence of confirmed infection and no malignant cells identified. A repeat echocardiogram post-procedure revealed a diffuse rind over the left and right ventricles, cardiac adhesions, and continued evidence of increased intracardiac pressures.

Discussion: Graft-versus-host disease is a common sequela of allogeneic stem cell transplantation in which T-cells derived from the donor’s stem cells recognize the recipient’s tissues as foreign. This leads to activation and tissue destruction that most commonly occurs in the skin and gastrointestinal tract. Pericarditis, pericardial effusion and effusive-constrictive pericarditis are rare manifestations of the disease, which should be treated aggressively to prevent supraventricular arrhythmias and the development of constrictive pericarditis, which requires surgical intervention in these immunocompromised patients. Physicians from multiple specialties should have a high index of suspicion for cardiac involvement in graft-versus-host disease, as timely recognition has an impact on long-term outcomes.
NEUROSARCOIDOSIS: A DIAGNOSTIC CHALLENGE IN AN OTHERWISE HEALTHY PATIENT

Tijana Skrepnik MD, THMEP, PGY-1
William Lamear MD, David Teeple MD, Robert Aaronson MD

Purpose: To demonstrate a case of neurosarcoidosis.

Case Description: A previously healthy 42 yo male firefighter in Southern Arizona developed asymptomatic subcutaneous nodules on his arm and knee. One month later, he presented to a local hospital with fever to 105F, intermittent occipital headaches, night sweats, generalized weakness and neck pain. After a negative workup including MRI/MRA brain and MRI spine, he was discharged home afebrile and on no new medications. Two weeks later, he presented to a local ED with a new left facial droop and tongue numbness and was felt to have Bells palsy. He was treated with prednisone and acyclovir. Due to increasing weakness, lethargy and persistent fevers and facial droop, he was admitted to a local hospital two weeks later. A CT heard, MRI brain and C-spine with contrast were negative. An LP was abnormal, with 0 RBC, 33 WBC (97% lymphocytes) and protein 88. Coccidiomycosis, viral and fungal CSF analyses were negative, as was an ACE level (2.5). His facial droop improved and he was discharged home with a diagnosis of viral meningitis and Bell’s palsy. He then developed right-sided facial paralysis, and was admitted to a third institution, where repeat brain MRI and LP were performed, with similar profile but increased total protein (164); cocci serology was still notably negative. His brain MRI demonstrated enhancement of bilateral CN VII. Seronegative coccidioidomycosis meningitis was strongly suspected, and he was started on fluconazole. He subsequently developed bilateral tinnitus, hearing loss, and gait instability. His subcutaneous nodules persisted and biopsies revealed non-caseating granulomatous inflammation. Soon thereafter, the patient developed hypercalcemia, acute, and diffuse lymphadenopathy and presented to a fourth institution. Sarcoidosis was suspected, but an alternative diagnosis of lymphoma was entertained. An ACE level was found to be elevated at 160 and review of prior CT and chest x-ray studies revealed mediastinal and bilateral hilar adenopathy with splenomegaly. These findings, along with his subcutaneous non-caseating granulomata, recent cranial nerve deficits, fever and neck pain led to his diagnosis of neurosarcoidosis. He received high-dose steroids, with improvement in his constitutional and neurologic symptoms, hypercalcemia and renal failure, and he was sent home on 100mg prednisone daily for 2-3 months and Bactrim prophylaxis while on steroids.

Summary of Results: MRI Brain with enhancement of bilateral CN VII; subcutaneous nodule biopsy showing granulomas, inflammation; ACE level 160, negative coccidioides serology and CSF culture; LP with elevated protein, 97% lymphocytes, 0 RBCs, CT and Chest X-ray with mediastinal and hilar adenopathy. Conclusions: 42yo otherwise healthy male firefighter presented with multiple cranial nerve deficits, fever, headache, receiving extensive workup, and found to have neurosarcoidosis after fourth hospital presentation.

POST MYOCARDIAL INFARCTION VENTRICULAR SEPTAL DEFECT – THING OF THE PAST?

Michael Sultan MD, Banner Good Samaritan, PGY-1
Tomas Rivera
Ventricular sepal defects have been thought to be complications of the past given the current era of percutaneous intervention for acute myocardial infarctions. A 56 year old man with a history of hypertension and hyperlipidemia presented to the emergency room with complaints of shortness of breath and chest pain. Initial EKG showed ST segment elevation along the percordial leads with an incomplete left bundle and right axis deviation with inferior Q waves. The STEMI phone was activated and the patient was taken for emergent percutaneous intervention. A coronary angiogram was performed that showed proximal LAD occlusion. Patient then underwent a successful bare-metal stent to the proximal LAD with restoration of TIMI 3 flow. The next day the patient had a bedside ECHO while in the ICU that showed a new onset VSD in the setting of symptomatic systolic heart failure. Patient was evaluated for emergent surgical repair for his VSD and ultimately taken to the operating room. The patient ultimately had acquired post MI systolic heart failure with an ejection fraction of 20%. This case brings up an important reminder of the complications of myocardial infarctions. VSD secondary to myocardial infarctions are in fact present in the ERA of PCI. The importance of echocardiography in post myocardial infarctions became apparent in this patient.

**PREVALENCE OF POSITIVE TUBERCULIN SKIN TEST AND BOOSTER IN A POPULATION WITH EARLY RHEUMATOID ARTHRITIS**

Carlos Tafich MD, UACOM at University Campus, PGY-1

Background/Purpose: Chronic inflammation and the use of DMARDs increase the risk of reactivation of latent tuberculosis (TB) in RA. Although other tests are available, tuberculin skin test (TST) is still used to screen for latent TB prior to the use of DMARDs. While TB prevalence is high in our community, there are currently no reports on TST reactivity in patients with early RA. In addition, there is no evidence on the usefulness of a TST Booster in the detection of latent TB in patients with early RA. The purpose of our study was to evaluate the response to the TST and Booster in patients with early RA.

Methods: A cross-sectional analytical study was conducted. Patients who fulfilled the ARA-1987 classification criteria with less than 1 year from the beginning of symptoms were included. Five units of PPD CT-68 were administered with the Mantoux technique. Induration >8 mm was considered positive. Patients with negative TST were scheduled within 15 days for a Booster dose. History was documented along with ESR and DAS 28. We reviewed clinical records and settled a database for analysis using the statistical package SPSS v. 20.

Results: Fifty two patients, 47 female (90.6%) and 5 male patients (9.4%), with an average age of 43.88 (17-64) were included. The mean DAS 28 score at baseline was 5.55 (1.94-8.22). Twenty three patients were taking prednisone at the time of the TST with an average dose of 8.89 mg/day. In our study 50% of patients had a positive TST response. In the initial application 18/52 patients (34.6%) had a positive TST. Of 34 patients with a negative TST, 31 received a Booster dose resulting in 8 positive reactions (25.8%). Patients with a positive TST were sent for specialist evaluation. None of them developed active TB infection. The number of patients with a positive TST was higher in the group without prednisone (41.4%) than in the group taking prednisone (26.1%) but without any statistical significance \(p = 0.25, OR = 0.5, CI 95% 0.15-1.64\). The number of patients with a positive TST was higher in the group without DMARDs (39.3%) than in the group taking DMARDs (29.2%) also without statistical significance \(p = 0.44, \)
OR= 0.63, CI 95% 0.19-2.03]. There were no differences in age, DAS 28 and prednisone dose between positive and negative TST groups.

Conclusion: The prevalence of positive TST in patients with early RA in this study was 50%. The prevalence of positive TST Booster was 25.8%. A Booster dose yielded 16% more positive reactions. It is not possible to determine whether the use of prednisone or DMARDs is associated with a negative TST in our study.

First Place – Patient Safety/Quality Improvement Poster

IMPROVING COMPLIANCE WITH SURVIVING SEPSIS CAMPAIGN RESUSCITATION BUNDLES AT UA - SOUTH CAMPUS

Anjali Takyar DO, UACOM at South Campus, PGY-1

Gordon Carr MD, David Ingram OMS-IV

Introduction: More than 1.5 million cases of sepsis are diagnosed each year, with a mortality rate of approximately 30%. The Surviving Sepsis Campaign (SSC) has developed evidence-based guidelines that recommend 3-hr and 6-hr resuscitation bundles. Multiple reports have shown that compliance with such bundles is associated with improved mortality. The Severe Sepsis Campaign’s 3-hour and 6-hour Bundles (SSB) have now been proposed as quality measures. We have initiated a quality improvement (QI) project to optimize our processes of care for sepsis.

Methods: As the first phase of an ongoing sepsis QI project, we performed a retrospective analysis of patients presenting to the University of Arizona south campus emergency department between July 2012 and September 2013 with ICD-9 coding for sepsis, severe sepsis or septic shock. Three-hour SSB and 6-hour SSB compliance were assessed. The 3-hour bundle included serum lactate measurement, drawing blood cultures, administration of antibiotics, and adequate fluid resuscitation. The 6-hour bundle included vasopressor initiation, obtaining central venous access, adequate CVP and SVO2 measurements and a repeat lactate measurement.

Results: The baseline data presented here is part of an ongoing Sepsis task force Quality improvement project. A total of 123 patient charts were reviewed, and 69 patients met severe sepsis criteria. Of these, 40 patients were admitted to the ICU and 29 patients were admitted to the medical floor. The majority of patients had signs and symptoms suggestive of pneumonia; 50% of ICU patients and 44.8% of floor patients. For patients admitted to the ICU, total compliance with the entire 3-hr SSB was 12.1%. For specific elements, compliance was 70% for lactate measurement, 67.5% for blood cultures, 42.5% for antibiotic administration, and 33.3% for fluid resuscitation. Compliance with the entire 6-hr SSB was 0%; for the elements, compliance was 95% for vasopressor use, 74.2% for central line placement, 6.45% for CVP &#8805; 8, 12.9% for Svo2 &#8805; 70, and 34.5% for lactate re-measurement. Overall hospital mortality was 40% in the ICU group. For patients admitted to the medical floor, compliance with the entire 3-hr SSB was 0%, specifically 72.4% for lactate measurement, 51.7% for blood cultures, 17.2% for antibiotic administration, and 21% for fluid resuscitation. Compliance with the entire 6-hr SSB was 0%, specifically 6-hr SSB compliance was 77.8% for vasopressor use, 35.7% for central line placement,
14.3% for CVP &≤8805; 8, 0% for SvO2 &≤8805; 70%, and 36.8% for lactate re-measurement. Overall hospital mortality was 31% in the inpatient group.

Conclusion: Our baseline QI data demonstrates suboptimal compliance with all aspects of the SSC 3 and 6 hr bundles particularly with timely fluid resuscitation and antibiotic administration. The next step in our QI project will focus on improving these two measures as well as overall bundle compliance.

THE LESSER OF TWO EVILS: DRUG INDUCED RENAL DYSFUNCTION

Vanessa Tang MD, Mayo Clinic Arizona, PGY-1

Daniel L. Roberts, MD, Dept of Med., and Maxwell L. Smith, MD, Dept. of Lab and Path

Introduction: Acute interstitial nephritis (AIN) is caused by an insult to the renal interstitium resulting in reduced creatinine clearance. Eventually AIN may lead to cardiorespiratory complications. The differential diagnosis for AIN is broad and requires the performance of a thorough history. Case: A 70 year-old Caucasian man with rheumatoid arthritis presented with two weeks of decreased appetite, nausea, fatigue, and a 20-pound weight gain. On exam, he had anasarca with 3+ lower extremity pitting edema. Investigative studies showed an elevated sedimentation rate of 74 and a serum creatinine of 3.5 (0.9-1.0 at baseline). Urinalysis revealed 3+ protein and 24-hour urine collection revealed 5 grams of protein. No urine eosinophils were identified. He had recently started on naproxen, a non-steroidal anti-inflammatory drug (NSAID) for rheumatoid arthritis and omeprazole, a proton pump inhibitor (PPI) for gastrointestinal prophylaxis. Initially, his use of omeprazole was not reported since he had started and stopped the drug on his own. With obvious renal injury and proteinuria, he was suspected to have AIN with nephrotic syndrome. His naproxen and omeprazole were not restarted on admission. However, his creatinine and potassium continued to rise, necessitating dialysis for symptomatic uremia. Renal biopsy revealed patchy interstitial nephritis with marked tubular injury consistent with allergic interstitial nephritis. He was advised to avoid NSAIDs and PPI for life and discharged home with outpatient dialysis. One month after his admission he regained sufficient kidney function to no longer require dialysis.

Discussion: Although systemic diseases can cause AIN, drug therapy is often the root cause. NSAIDs unlike other causative medications, often cause a nephrotic picture, with edema being a common symptom. This patient had an autoimmune disease and history of two known causal medications (naproxen and omeprazole), but the presence of proteinuria with his uremia, this is consistent with NSAID-induced AIN and nephrotic syndrome. In addition to reinforcing this unique presentation of a common disease, this case is a reminder of the importance of taking a complete and detailed medication history.

A BUGGING PAIN

Maria Tumanik DO, UACOM at South Campus, PGY-1

Parinita Dherange, MD; Patel Krunal, MD; Eugene Trowers, MD
Schistosomiasis is caused by an infection with parasitic blood flukes called schistosomes, mostly prevalent in endemic areas. Disease can have significant mortality and morbidity and due to rarity in clinical practice in the US often goes undiagnosed and untreated for a long time causing significant effect on patient’s daily quality of life and well being. A 28 year old Burundian male presented to clinic to re establish care recently. He complained of intermittent dull diffuse abdominal pain which improved after meals for many months. The pain was alleviated with bowel movements and Vicodin. There was no history of diarrhea or hematochezia this time. Per the records, in 2009, patient had a history of similar intermittent abdominal pain and hematochezia which self resolved. Physical examination was significant for mild lower abdominal tenderness. Initial labs remained normal. However, Hp lori serology was indeterminate which was treated with antibiotics. The abdominal pain continued despite that and he was treated for presumed diagnosed of IBS with dicyclomine. In the interim, patient was referred to the Gastroenterology team for further evaluation. Owing to his past history of hematochezia and recurrent abdominal pain, colonoscopy was performed. Colonoscopy was negative for signs of IBD/hemorrhoids, but significant for mild patchy erythematous mucosa in sigmoid colon. Cold forcep biopsy revealed small bowel mucosa without histopathologic abnormalities, well-formed villi without significant inflammation, but colonic mucosa with demonstrated intestinal schistosomiasis with associated mucosal eosinophils. A diagnosis of colonic schistosomiasis was made and Praziquantel was started. Over the next few weeks, his abdominal pain improved significantly. This case illustrates the negative effect of lingering and undiagnosed schistosomiasis and its impact on our patient’s life. Schistosomiasis often represents a significant diagnostic challenge with asymptomatic patients or with vague symptoms such as fatigue, but significant morbidity with long-term infection often resulting in serious complications including periportal fibrosis, esophageal varices, cor pulmonale, colonic strictures and even colon cancer. Screening includes serology and microscopy that should be performed on every patient with minimal suspicion of schistosomal exposure, such as immigrant refugees or travelers to endemic areas. Clues to diagnosis include presence of eosinophilia, anemia from chronic blood loss, and thrombocytopenia, elevation in alkaline phosphatase and GGT, stool and urine microscopy positive for ova. Our patient did not have any of these suspicious findings, which led to years of undiagnosed condition. Definitive diagnosis often takes a long time due to low sensitivity of the above less-invasive screening tests and long time to invasive procedures such as biopsy; therefore, patients should be treated with Praziquantel empirically to avoid incidence of complications. Why do we think this case is important? Diagnosis can be delayed. With delay cases of carcinoma have been reported. Early diagnosis and treatment is essential.

**A SNEAKY MALIGNANCY: AN UNUSUAL PRESENTATION OF SMALL CELL LUNG CANCER AND IT’S COMPLICATIONS**

Brian Wojcick MD MPH, Mayo Clinic Arizona, PGY-1
Ilko Ivanov MD, Thomas Lidner MD

Introduction: The diagnosis of dermatomyositis in adulthood can be a harrowing sign of malignancy. Age appropriate screening is recommended with limited additional testing for those at high risk for malignancy. This report will discuss dermatomyositis as a primary presentation and superior vena cava syndrome as a complication of small cell carcinoma.
Case Report: A 67 year old female with past medical history significant for COPD and heavy tobacco abuse presented to the emergency room with diffuse rash and polymyalgias for three weeks duration. She was initially treated for eczema but her rash rapidly extended at which point she sought evaluation in our emergency room. She complained of increasing proximal muscle weakness and pain. She reported having difficulty picking up a can of soda to drink, and raising her arms over her head. She was no longer able to ambulate independently and required a wheelchair. On exam the patient had a tender, erythematous, flaky, ulcerating rash with extension to the shoulders, back, extremities, and scalp. Gottron’s papules were present on her PIP joints bilaterally. Patient had diminished proximal strength and deep tendon reflexes in all four extremities. Patient had elevated CK of 2809, which improved with prednisone to 399. Electromyography findings revealed chronic myopathy with underlying necrosis. Based on these findings she was diagnosed with dermatomyositis. Given her tobacco history a CT chest was performed. A lobulated soft tissue mass within the right upper lobe near the hilum, with enlarged adenopathy was found. She underwent bronchoscopy with endobronchial ultrasound guided biopsy which confirmed small cell carcinoma. Within 10 days of initial diagnosis she returned to the emergency department with acute worsening dysphagia for both solids and liquids with postprandial coughing and choking. Lambert-Eaton syndrome was ruled out via paraneoplastic panel. Her dysphagia was attributed to dermatomyositis. A PEG tube was placed to maintain nutrition. On the day of her PEG placement she complained of acute shortness of breath. A CT scan was performed which showed increasing size of her mediastinal mass at 5.0 x 3.5 x 5.6 cm from 3.3 x 2.7 cm three weeks prior. There was severe mass effect on her superior vena cava, narrowing it to a thin slit. She was diagnosed with superior vena cava syndrome. Patient had acute respiratory decompensation requiring transfer to ICU and intubation. She passed secondary to multifactorial acute respiratory decompensation.

Discussion: Dermatomyositis may result in unusual primary presenting symptoms of small cell carcinoma. In this case the patient’s concomitant dermatomyositis, small cell carcinoma, and superior vena cava syndrome were complicated by chemotherapeutic effects and co-morbidities. The complication of dermatomyositis is uncommon in the hematology/oncology population with 13% of patients with lung cancers developing dermatomyositis.

**Third Place – Research Poster**

**MECHANISM OF DUAL MTORC1/MTORC2 INHIBITOR-INDUCED APOPTOSIS IN HUMAN LYMPHOID MALIGNANCIES**

Seong seok Yun MD, UACOM at University Campus, PGY-1

Nicole D. Vincelette, Katherine L. Knorr, Andrea E. Wahner Hendrickson, Scott H. Kaufmann

Seong Seok Yun, MD1,2, Nicole D. Vincelette2, Katherine L. Knorr2, Andrea E. Wahner Hendrickson, MD3, Paula Schneider4, Kevin L. Peterson, PhD4, Karen S. Flatten4, and Scott H. Kaufmann, MD, PhD2,4 1, Department of Internal Medicine, University of Arizona Medical Center, Tucson, AZ 2Department of Molecular Pharmacology and Experimental Therapeutics, Mayo Clinic, Rochester, MN 3, Division of Medical Oncology, Mayo Clinic, Rochester, MN 4Division of Oncology Research, Mayo Clinic, Rochester, MN
The serine/threonine kinase mammalian target of rapamycin (mTOR) plays a central role in regulation of cell growth, proliferation, metabolism and apoptosis. Dysregulation of the pathway involving phosphoinositol-3 kinase (PI3K) and mTOR is frequently observed in lymphoid malignancies. Extensive studies, including phase II trials, have demonstrated that mTORC1-selective rapamycin derivatives are a viable treatment option in various hematologic malignancies, although the efficacy is relatively limited due to incomplete suppression of mTORC1 and hyperactivation of mTORC2 upon mTORC1 inhibition. These limitations fostered the generation of new dual mTOR inhibitors. The purpose of the current study is to identify the mechanistic basis for the pro-apoptotic effects of dual mTOR inhibitors in malignant human lymphoid cells in vitro. Tetrazolium dye reduction (MTS) assays indicated diminished viable cell mass following mTOR dual inhibitor treatment of a variety of lymphoid cell lines, including Jeko (mantle cell lymphoma), Jurkat (T cell ALL) and Nalm6 (pre-B cell ALL). Propidium iodide staining followed by flow cytometry demonstrated that mTOR dual inhibitors induced apoptosis, as indicated by the presence of subdiploid cells and staining with annexin V to detect phosphatidylserine exposure. Immunoblotting revealed effective dephosphorylation of phospho-Thr37,46-4EBP1 (a rapamycin resistant mTORC1 substrate) and phospho-Ser473-Akt (an mTORC2 substrate) with mTOR dual inhibitors. Additional immunoblotting revealed upregulation of the proapoptotic Bcl-2 family members Bim and Puma after 48 hours of treatment with mTOR dual inhibitors but not with rapamycin. Small hairpin RNA mediated knockdown of mRNAs encoding Raptor and Rictor, critical components of the mTORC1 and mTORC2 complexes, respectively, phenocopied the effect of mTOR dual inhibitors by inducing upregulation of Bim and Puma as well as apoptosis. Further quantitative PCR analysis also indicated a 4-fold increase of Bim mRNA and a 10-fold increase of Puma mRNA with dual inhibitors. Similar changes were also observed in Bim promoter and Puma 3'UTR luciferase reporter assays, suggesting that Bim is upregulated by activation of its promoter, whereas Puma is upregulated by stabilization of mRNA. Subsequent analysis of Bim promoter truncation constructs localized the major mTOR regulated response element to a 12-base pair region, and inhibition of mTORC1 revealed elf4E/elf4G-dependent regulation of Puma mRNA. Collectively, these results identify a critical pathway for mTOR dual inhibitor induced cytotoxicity, and also suggest that mTORC1/mTORC2 dual inhibition is a potentially effective treatment strategy for a wide variety of human lymphoid malignancies.

**RARE EXTRANODAL LARYNGEAL NK/T CELL LYMPHOMA**

**Bianca Zangeneh MD, UACOM at University Campus, PGY-1**

Snehal Patel

Extranodal natural killer (NK) cell lymphomas are a rare group of aggressive neoplasms. Most cases reported involve the nasal area; a primary finding in the larynx is extremely rare with only 8 cases currently reported in literature. We report a case of a NK/T cell lymphoma originating in the larynx after an initial negative biopsy. A 79-year old Thai female presented with 30 lb weight loss, drenching night sweats and worsening odynophagia for 3 months. Patient had initially been evaluated at another institution and had a laryngo-endoscopy that showed edematous oropharyngeal mucosa with biopsy of epiglottis consistent with epiglottic squamous mucosa with ulceration and arytenoid cartilage showing highly atypical lymphoid infiltrates suspicious for lymphoproliferative disorder. Immunohistochemistry was diffusely positive for Vimentin as was CD45 and CD10 however negative for cytokeratin 5/6, CK20,
CEA, S100, p63, CK7, AE1/3, and CD3. Due to the uncertainty of her previous biopsy results, a CT neck was performed which showed edema of both aryepiglottic folds with linear mucosal enhancement and narrowing but no significant lymphadenopathy. Patient underwent another laryngoscopy with biopsy, which revealed a 1cm mass around the right arytenoid with rest of the laryngeal surface appearing inflamed. Biopsy of the right arytenoid revealed extranodal NK T-cell lymphoma on pathology with expression of CD3, CD56, Granzyme B, TIA-1. In addition, FISH Epstein-Barr virus (EBV) encoded mRNA was positive. Further imaging did not reveal any brain or abdominal metastasis however CT chest displayed left apical opacity suggestive of scarring. Bone marrow biopsy did not reveal any abnormal findings. LDH was normal and beta-2 microglobulin was elevated. PET scan revealed diffuse thickening of supraglottic and glottis larynx, FDG uptake with hypermetabolic lymph nodes at right neck and at the left lung hilar lymph node consistent with inflammatory changes. Patient is currently staged IIB NK T-cell lymphoma and planned to have radiation and chemotherapy with cisplatin. Extranodal non-Hodgkin lymphomas account for less than 10-35% of all cases reported, moreover, less than 1% of these are primary in the larynx. NK/T cell lymphoma is most common in Asia and South America. The pathogenesis of the disease is poorly understood but malignant cells usually arise with a NK cell phenotype. Diagnosis is often difficult and is based on immunotyping and presence of EBV. Most studies looking at prognosis and treatment are based on nasal NK/T cell lymphoma and are retrospective studies taking place outside of the US. Treatment is based on staging and remains controversial as to do radiation alone or combined with chemotherapy. Studies do show chemotherapy alone is associated with worse outcomes. Prognosis remains poor for this extranasal aggressive lymphoma and is associated with better outcomes if diagnosed and treated in the early stages.

MEDICAL STUDENT SUBMISSIONS

A MYSTERIOUS CASE OF THROMBOCYTOPENIA: ACQUIRED AMEGAKARYOCYTIC THROMBOCYTOPENIA

Gabrielle Brown MS, UofA College of Medicine - Tucson, MS-IV

Hani Babiker, MD; Ravi Krishnadasan, MD

Introduction: Acquired amegakaryocytic thrombocytopenia (AAT) is a rare disorder that is characterized by severe thrombocytopenia and diminished or absent megakaryopoiesis in the setting of otherwise normal bone marrow.1 AAT is associated with other hematologic and rheumatologic conditions, including myelodysplasia, aplastic anemia, acute myeloid leukemia, and systemic lupus erythematosus.2,3 The exact mechanism has not been elucidated, however the presence of antithrombopoietin IgG antibodies in patients with AAT suggests a dysregulated humoral immunity.4 Dysfunction of cell-mediated immunity has also been postulated, in which monoclonal T-lymphocytes obtained from a patient with AAT were found to inhibit megakaryocyte lineage in vitro, but not other cell lineages.5

Case Description: A 40-year-old woman with a past medical history of migraine headaches presented to her primary care physician for a routine physical accompanied by vague complaints of fatigue. Further questioning revealed a long-standing history of easy bruising and frequent nosebleeds. A routine
complete blood count revealed a platelet count of $12 \times 10^9/L$ [150-425 $10^9/L$], after which she was referred to a local hematologist. She was initially diagnosed with idiopathic thrombocytopenia purpura and treated with prednisone, with no improvement in platelet count. A bone marrow biopsy revealed hypercellular marrow with trilineage hematopoiesis, and mildly decreased megakaryocytes. Due to the persistent thrombocytopenia, her treatment regimen was modified to include weekly platelet infusions. An appropriate elevation in platelets would be observed after each transfusion with a subsequent decline over the following days. At this time, the patient was referred to our hematology service for further evaluation and management. On physical examination, the patient had several discrete petechiae and ecchymoses on the extremities, and no rash, hepatosplenomegaly, or lymphadenopathy were present. A complete blood count revealed thrombocytopenia with a count of $25 \times 10^9/L$ [150-425 $10^9/L$]. Repeat bone marrow biopsy revealed thrombocytopenia with absent megakaryocytes and otherwise normocellular elements with no evidence of dysplasia, which was consistent with acquired amegakaryocytic thrombocytopenia (AAT). The patient was admitted to the bone marrow transplant unit for a four day course of anti-thymocyte globulin (ATG) at $3.5mg/kg/day$ followed by an extended outpatient course of cyclosporine (CsA) lasting at least 6 months. The patient required one platelet transfusion shortly after discharge, and nearly 2 months after ATG/CsA treatment the platelet levels increased to $92 \times 10^9/L$.

Discussion: Standard treatment guidelines have not been established for acquired amegakaryocytic thrombocytopenia, and few case reports have been published regarding the management of this disorder. Herein, we report a rare case of AAT successfully managed with ATG and CsA. In addition to the ATG and CsA regimen, several treatment approaches have been utilized in patients with AAT with variable degrees of success, including Rituximab2, Azathioprine3, and bone marrow transplant6.

**UNILATERAL COLOR CHANGE IN THE LOWER EXTREMITY OF AN ELDERLY DIABETIC MALE: EMERGENCY OR CHRONICITY?**

Daniel Butler BS, UofA College of Medicine - Tucson, MS-IV

Mary Teeple MD, Douglas Rappaport MD, Tiffany Link MD PhD, Taranee Paravar MD

70 year old Caucasian male with a history of non-small cell lung cancer status post chemotherapy in 1999, diabetes mellitus complicated with chronic osteomyelitis status post right lower extremity amputation in 2008 and left lower extremity amputation in July 2013 presented to dermatology clinic in August 2013 with concerns regarding recent color changes of the distal residual left lower extremity. The patient reported that his distal left leg felt “cooler” with diminished touch sensation. He received a below the knee amputation two weeks prior due to a 5 year history of chronic osteomyelitis. The patient denied recent fever, chills, nausea, or vomiting. The patient’s past medical history also included peripheral vascular disease, hypertension, and a deep vein thrombosis. Physical exam of the patient revealed an elderly man in no apparent distress resting calmly in his wheelchair. Inspection of the left lower extremity revealed a well-healed below knee amputation with no evidence of wound infection or dehiscence. There was blue-grayish discoloration circumferential around the distal residual leg. The color change stretched from the end of the limb extending three inches proximally. The area of discoloration had minimal temperature change as compared to uninvolved skin. There was no edema and it was not tender to palpation. Touch and vibratory sensation were intact, and left knee had regular
range of motion and strength. The remainder of the physical exam was within normal limits except for bluish discoloration of the sclera bilaterally as well as of the teeth. With concern for ischemia, the emergency department was called for a STAT consult to assess perfusion of the left extremity. An ultrasound of the popliteal artery revealed a bounding pulse without evidence of compromised flow. Next, the team found prior notes which revealed a history of scattered patches of discoloration prior to amputation. The team asked about remote medication use, and he recalled taking Minocycline for a substantial period of time years back. A thorough review of his records revealed he was started on Minocycline in November 2008 for his chronic osteomyelitis and continued until October 2009. After discussing with patient, he recalled the minocycline pigmentation and deferred biopsy. Minocycline-induced skin pigmentation has an estimated incidence between 3-15% of those who use the medication for prolonged periods, usually at doses between 100-200mg daily for over 3 months. Pigmentation of the nails, bones, and thyroid has also been noted in the literature. This case highlights the expansive differential for skin discoloration which can include both acute and chronic processes. Clinicians should be aware of discoloration side effects of medications, which include antimalarials, chemotherapeutics, heavy metals, antiretrovirals, and certain psychotropic agents.

FISHING FOR SNAILS: SCHISTOSOMIASIS PRESENTING AS MASSIVE SPLENOMEGALY

David Ingram, Midwestern AZCOM, MS-IV
Anjali Takyar DO, Shivani Ruben MD, Eugene Trowers MD

Introduction Massive splenomegaly is rarely encountered. Causes of splenomegaly include portal hypertension secondary to cirrhosis and heart failure, and infiltrative diseases including infections and hematologic malignancies. In this report, we describe a rare cause of portal hypertension resulting in massive splenomegaly.

Case Report A 34-year-old man originally from Uganda presented to the clinic with worsening left-sided abdominal pain and early satiety for the past 2 years. Past medical history was significant for documented esophageal varices requiring banding. Review of systems was unremarkable except for intermittent nausea and occasional melenic stools. Physical exam revealed an afebrile, thin man with palpable splenomegaly. Initial labs showed bilirubin of 0.7 mg/dL, albumin of 3.8 mg/dL, AST of 64 IU/dL, ALT 247 of IU/dL, alkaline phosphatase of 256 IU/dL and INR of 1.5. Hematologic labs revealed WBC of 3.9 K/dL with 30% Eosinophilis, Hb of 12.6 g/dL and platelets of 107 K/dL. Abdominal CT revealed a portal vein measuring 21.6 mm and an enlarged spleen measuring 22.1 cm in span, displacing a decompressed stomach and the left kidney. An extensive workup was negative for Hepatitis B and C, Quantiferon gold, RPR, HIV, ova and parasites in stool, and filaria. It was significantly positive for Schistosoma Antibody IgG. Upon further questioning, it was discovered that the patient worked as a fisherman near the Enyau River in Uganda prior to the onset of his symptoms. The patient was started on Praziquantel. Because he continued to have persistent abdominal pain, he ultimately underwent an elective splenectomy. Post-splenectomy, the patient is being seen in our clinic for surveillance upper endoscopies and hepatology follow-up.

Conclusion Schistosomiasis is a common cause of noncirrhotic portal hypertension worldwide, but is rarely encountered in the United States. S. japonicum and S. mansoni are the two main species known
to cause liver disease. S. japonicum is predominantly in Asia while S. mansoni is endemic in Egypt, Africa, Middle East, and South America. Infection occurs when worms enter the skin, reach the mesenteric veins and produce thousands of eggs. These eggs can then enter the portal vein and become trapped in portal venules, eliciting an immune response and inflammation leading to fibrosis and portal hypertension. Patients with chronic schistosomiasis commonly present with features of portal hypertension including variceal bleeding, hepatomegaly and splenomegaly with hypersplenism, defined as splenomegaly plus a deficiency in one or more of the peripheral cell lines: hemoglobin, leukocyte count, or platelet count. Praziquantel can be used to treat acute schistosomiasis. In chronic schistosomiasis, the worms no longer lay any eggs; therefore anthelmintic treatment may not be necessary. The sequelae of portal hypertension are managed with techniques including nonselective beta-blockers, blood replacement, endoscopic varietal banding, TIPS, and splenectomy.

HEART OF LYMPHOMA: PRIMARY MEDIASTINAL LARGE B-CELL LYMPHOMA WITH ENDOMYOCARDIAL INVOLVEMENT

Elisa Rogowitz BS, UofA College of Medicine - Tucson, MS-IV

Varun Bhalla, MD.; Hani M Babiker, MD, Ravitharan Krishnadasan, MD, Clint Jokerst, MD, Thomas P Miller, MD, Michael Bookman, MD

Primary mediastinal B-cell lymphoma (PMBCL) is an uncommon aggressive subset of diffuse large B-cell lymphomas. Although PMBCL frequently spreads locally from the thymus into the pleura or pericardium, it very rarely invades directly through the heart. Herein, we report a case of a young Mexican female diagnosed with PMBCL with clear infiltration of lymphoma through the cardiac wall and into the right atrium and tricuspid valve leading to tricuspid regurgitation. This was demonstrated by cardiac MRI and trans-thoracic echocardiogram. In addition, cardiac MRI and CT scan of the chest revealed the large mediastinal mass completely surrounding and eroding into the superior vena cava (SVC) wall causing a collar of stokes. The cardiac and SVC infiltration created a significant therapeutic challenge as lymphomas are very responsive to chemotherapy and treatment could potentially lead to vascular wall rupture and hemorrhage. Despite the lack of conclusive data on chemotherapy-induced hemodynamic compromise in such scenarios, her progressive severe SVC syndrome and respiratory distress necessitated urgent intervention. In addition to the unique presentation of this rare lymphoma, our case report highlights the safety of R-CHOP treatment.

PSEUDO-DKA SECONDARY TO HYPERTRIGLYCERIDEMIA

Muhammad Omar Salim , Midwestern AZCOM, MS-IV

K. Choudry MD; Q. Mashood, MD; M. Shahlapour, MD; M.H. Horani, MD

Case Study: A 44 year old female presented with a 24 hour history of abdominal pain radiating to the back, lethargy, nausea, vomiting, and a fever of 103.7. Past medical history is significant for lipodystrophic diabetes mellitus, hypertriglyceridemia, previous episode of pancreatitis, psoriatic arthritis. The patient does not smoke, drink, or use any non-prescription drugs. The patient was noncompliant in taking fenofibrate, methotrexate, and levemir. The physical exam was significant for abdominal distension in the epigastric region. Vital signs showed a blood pressure of 143/74, pulse 101.
Respirations 16, pulse ox of 99, and a temperature of 38.1. Laboratory findings revealed Hgb of 15.5, platelet count of 246, and WBC of 11.3. Sodium was 131, chloride 97, potassium 3.6, bicarbonate 18, BUN/Cr 11/.73, glucose level of 321, and anion gap of 25. Lipase was 1,228 U/L and amylase was 90 U/L. Lipid panel showed cholesterol at 632 mg/dl, triglycerides at 4,502 mg/dl, HDL 25 mg/dl, and LDL 33mg/dl. UA showed a specific gravity of 1.035 with 3+ ketones, and 4+ glucose. The patient received fluids, insulin, and IV antibiotics. The day after, serum glucose was 167 mg/dL, lipase decreased to 769, sodium was down to 127, triglycerides were still 4,500 mg/dl, and the bicarbonate dropped to 3. The ABG, however, showed a bicarbonate of 19.5. The endocrinology consult considered a likely familial dyslipidemia and recommended a nephrology consult for plasmapheresis for triglyceridemia-induced pancreatitis and continuing IV insulin treatment. Nephrology prescribed acute plasmapheresis and concluded hyponatremia and low bicarbonate were due to dilution.

Discussion: Insulin deficiency in DKA enables the increased release of free fatty acids, which increases the formation of VLDL. Less VLDL is removed from the plasma due to reduced activity of lipoprotein lipase, which results in hypertriglyceridemia. This can decrease electrolyte measurements and bicarbonate via the dilutional effect of high amounts of serum lipids. This causes inaccuracy in the BMP, so the ABG was necessary. In severe hypertriglyceridemia, high plasma chylomicrons are hydrolyzed in the pancreatic capillaries and result in the release of free fatty acids (FFA). These FFAs cause free radical damage to the capillaries. In the majority of cases, hypertriglyceridemia can be controlled by insulin without using lipid lowering agents. In severe hypertriglyceridemia, however, plasmapheresis should be considered to avoid complications. If DKA is treated with fluids and insulin, yet no improvement is present, then a lipid panel should be considered to rule out hypertriglyceridemia-induced pancreatitis and pseudo-DKA.

http://www.uptodate.com/contents/hypertriglyceridemia-induced-acute-pancreatitis

EVALUATION OF DIASTOLIC FUNCTION IN RATS WITH CHRONIC HEART FAILURE

Pablo Sanchez BS, UofA College of Medicine - Tucson, MS-IV

Jordan Lancaster MS, Elizabeth Juneman MD, Steven Goldman MD, Joseph Bahl PhD

Background: Chronic Heart Failure (CHF) is a condition causing vast morbidity and mortality and the leading cause of hospital readmissions in the US. It may result from systolic or diastolic dysfunction, which many times coexist. Much research is available delineating the pathophysiology and therapeutics for systolic dysfunction; conversely there is a paucity of data for diastolic dysfunction. Most clinical studies use improvement in left ventricular (LV) systolic function as an endpoint. However, since diastolic dysfunction post-myocardial infarction, independent of systolic function, carries an important prognosis for patients and our available armamentarium for clinically treating diastolic dysfunction is quite limited, the importance of constructing a model cannot be overstated. Future delineation of the effectiveness of therapy at preventing diastolic function deterioration or improving diastolic function is important. Conventionally, evaluating changes in diastolic function with invasive hemodynamic measurements is cumbersome to obtain in patients. Echocardiographic evaluations through blood and
tissue Doppler evaluation have provided a dependable, non-invasive method of assessing diastolic function.

Purpose: To evaluate the capability of echocardiography to define changes in diastolic function in a rat coronary artery ligation model of CHF. Methods: Adult male Sprague-Dawley rats were randomized to undergo thoracotomy and left coronary artery ligation or thoracotomy alone. Echocardiography was performed with a dedicated rodent echocardiography system (Vevo2100) at 3 and 6 weeks post-intervention. Mitral valve inflow patterns, M-Mode for LV functional analysis, and Tissue Doppler for quantification of myocardial tissue movement in diastole (anterior LV wall) were used to assess function. Hemodynamic pressure measurements were performed at 6 weeks post-ligation (PL) with Millar solid state micromanometer catheters.

Results: At 3 weeks post-ligation, echocardiography reveals systolic and diastolic functional changes: Sham Ejection Fraction (EF) = 76.1 ± 4.3 (n=4) compared to 43.86 ± 23.3 (n=10) in CHF rats (p<0.05); Sham anterior wall $E'$ = 30.4 ± 2.3 (n=3) vs 24.4 ± 5.2 mm/s (n=8) in CHF (p<0.05). At 6 weeks Sham $E'$ = 37.1 ± 12.5 (n=2) vs 22.0 ± 1.2 mm/s (n=9) in HF group (p<0.05) and Sham E/E' = 12.9 (n=1) vs 30.0 ± 3.1 (n=9) in CHF rats (p<0.05). Hemodynamics: Sham left ventricular end-diastolic pressure (LV EDP) = 19 ± 3 (n=3) vs 7 ± 2 mmHg (n=8) in CHF rats and Sham time constant of isovolumic relaxation (Tau) = 16.6 ± 3.6 (n=3) vs 30.9 ± 3.2 msec (n=8) in CHF rats.

Conclusions: The data show that we can use echocardiography to measure changes in diastolic function in this rat model of CHF, which will allow us to access the effects of new experimental treatments in CHF.

**A TAKE OF TWO CANCERS: THE CO-EXISTENCE OF ENDEMIC BURKITT’S LYMPHOMA AND OSTEOSARCOMA IN AN HIV PATIENT**

**Shana Semmens , UofA College of Medicine - Tucson, MS-IV**

Anju Nair, MD, Josephine Taverna, MD, Soham Puvvada, MD

Introduction: Burkitt’s lymphoma (BL) is a highly aggressive B cell neoplasm characterized by three distinct clinical forms: endemic, sporadic, and immunodeficiency-associated BL1,2,3. Endemic BL classically presents as a jaw or facial bone tumor affecting children from Equatorial Africa and can spread to extranodal sites (i.e. mesentery, ovary, testis, kidney, breast, meninges); however, involvement of the peripheral lymph nodes, mediastinum, and spleen are uncommonly seen. Sporadic BL presents with an abdominal mass or lymphadenopathy with CNS and bone marrow involvement occurring in 15-30% of cases. Immunodeficiency-related BL is related to the underlying immunodeficiency (eg, AIDS, congenital immunodeficiency, acquired immunodeficiency due to hematopoietic or solid organ transplantation) and often involves lymph nodes, bone marrow, and CNS. Case Presentation: We present a fascinating case of a 38-year-old Hispanic transgendered female with HIV (CD4 count 31) who presented with constitutional B symptoms, HIV encephalopathy and an enlarging left mandibular mass. Excisional biopsy of this mass confirmed a high grade Burkitt’s Lymphoma with plasmacytoid differentiation. The cells expressed CD20, CD79a, CD10, BCL6, had a high Ki-67 proliferative index of 95%, and expressed EBV. Routine karyotype as well as FISH analysis demonstrated rearrangement of the MYC gene at chromosome 8q24 region and
IGH/MYC fusion. Bone marrow and CSF analysis were normal on initial presentation. Additionally, an MRI of the brain revealed multiple lesions along the calvarium and a right superior skull biopsy confirmed an osteosarcoma. She was initiated on highly active antiretroviral therapy and subsequently treated with three cycles of modified CODOX-M (Cyclophosphamide, Vincristine, Doxorubicin, and Methotrexate) with complete regression of her left mandibular mass. Her medical course was complicated by prolonged pancytopenia despite growth factor stimulation and a repeat bone marrow biopsy revealed a hypocellular marrow which was likely related to her chemotherapy and underlying HIV.

Discussion: Based on our review of the literature, osteosarcoma of the calvarium and endemic BL have never been reported in a single individual. Osteosarcoma of the skull is a rare phenomenon and risk factors include a history of Paget’s disease, previous irradiation, and retinoblastoma. Our patient did not have any known risk factors for developing osteosarcoma. Her case was also unusual in that she presented with an endemic form of BL rather than immunodeficiency-related BL, which we would have expected given her HIV status. The severe immunosuppression resulting from her chemotherapy and underlying HIV presented us with a therapeutic challenge since patient could not tolerate further doses of chemotherapy. We discuss the treatment challenges and chemotherapeutic management of our severely immunocompromised patient with two rare and potentially life-threatening malignancies.

**OMINOUS TRUTH UNDERLYING A SWOLLEN ELBOW**

**Amber Bellafiore , UofA College of Medicine - Tucson, MS-III**

Kristina Voss, Dr. Santhosh G John

Delay in the diagnoses of slow growing tumors due to its atypical presentation is not uncommon in the medical literature. This is often exaggerated in the absence of systemic symptoms. Synovial sarcomas are rare tumors of pluripotent mesenchymal cells, named somewhat inappropriately for their propensity to arise adjacent to joints, instead of their histopathology. Patients with synovial sarcoma usually present with a palpable soft tissue mass or swelling, seen most in adolescents and males. An association with trauma has also occasionally been cited in the literature, although most cases are likely coincidental. We present a case of a 55 year old woman admitted with progressively worsening right elbow pain and swelling. Our patient presented to the ED with right elbow pain status post patient-reported trauma to the elbow one year previously. For seven months, the elbow swelled progressively and became increasingly painful. At the onset of her symptoms, initial imaging findings were suggestive of a small nonspecific soft tissue mass. After five months passed without symptomatic resolution, the patient presented to emergency for workup. Physical exam was significant for a firm mass on the dorsal aspect on the radial side without overlying skin changes. MRI revealed 9x9x8.3 cm heterogenous mass and CT showed multiple pulmonary masses. Further assessment via biopsy revealed a CD 56 positive, vimentin positive, CD 99 positive BCL-2 positive tumor consistent with synovial sarcoma, confirmed by FISH analysis of SYT gene rearrangement on chromosome 18q11. CT guided biopsy of the pulmonary masses confirmed metastatic sarcoma. Further review by the oncologist finalized the diagnosis as a stage 4 grade 2/3 synovial sarcoma with pulmonary metastasis. Due to the advanced stage of disease and wide dissemination, the patient was not a surgical candidate. The patient was discharged with a plan for outpatient care for chemotherapy. This case illustrates prolonged tumor diagnosis attributed to confounding features and demonstrates the necessity for a high index of clinical suspicion. Primarily, synovial sarcomas are often not considered given their rarity. The occurrence of elbow
swelling secondary to musculoskeletal trauma is without question significantly higher and therefore the most likely. Furthermore, as epidemiologic studies suggest that synovial sarcomas occur most often in adolescent males, with rare involvement of the upper extremities, suspicion of synovial sarcomas in our patient is low. The most important features associated with synovial sarcoma that may lead to a mistaken diagnosis of a benign indolent process are slow growth (average time to diagnosis, 2-4 years) and small size (<5 cm at initial presentation). This case highlights the need for physicians to remain suspicious of rarer conditions as the delay in diagnosis may result in advanced disease and thus worse prognosis.

**Second Place – Medical Student Poster**

**A CASE OF THROMBOSED PERSISTENT SCIATIC ARTERY ANEURYSM RESULTING IN AMPUTATION**

Hersh Goel MD, UofA College of Medicine - Phoenix, MS-III

Christopher Goettl, MD

Arterial supply to the lower leg is initially provided by the sciatic artery during early embryologic development. Later, the superficial femoral artery arises as the ultimate conduit to the lower leg, and the sciatic artery involutes. However, in 0.025-0.04% of adults, the sciatic artery persists as an extension of the internal iliac artery, resulting in either incomplete (25%) or complete (75%) aberrant arterial supply to the lower leg and foot. Although most remain asymptomatic, presentation of known cases are most often due to aneurysm or complications arising from aneurysm thrombosis, with approximately 8% of patients eventually requiring limb amputation. In this case, an 83 year old ambulatory female with prior history of hypertension, hypercholesterolemia, type 2 diabetes and chronic right lower extremity claudication presented with a cold right leg after sustaining a ground-level fall. She described her pain as more severe than her typical chronic claudication pain, constant in nature and localized to her right buttock, posterior thigh and calf. Following patient transfer from an outside facility, CT angiography was performed to assess for suspected aortoiliac dissection, but instead demonstrated a thrombosed right persistent sciatic artery with proximal aneurysm near the ischial tuberosity and arterial thrombosis down to the level of the knee. Heparin drip was initiated and subsequent IR thrombectomy was attempted. Regrettably, all interventions failed to restore patency of the persistent sciatic artery due to the extensive nature of the vascular occlusion. Ultimately, above-the-knee amputation was performed. This case demonstrates the rare yet classic presentation of persistent sciatic artery with complications arising from aneurysm thrombosis. In this instance the delay in initial therapy due to the presence of acute on chronic limb ischemia, combined with the patient’s underlying advanced peripheral arterial disease, contributed to the unsuccessful outcome of medical and surgical therapy. In the rare but clinically significant instance of persistent sciatic artery thrombosis, it is important that physicians across the acute care spectrum are aware of this anatomic variant, as prompt diagnosis and vascular intervention are crucial for maximizing of limb salvage.

**METHAMPHETAMINE USE WITH SUBSEQUENT THYROID STORM, PTU AGRANULOCYTOSIS AND THYROIDECTOMY: A CASE REPORT**

Deanna Menapace , Creighton COM, MS-III
Thyroid storm is a rare, yet potentially lethal condition involving collapse of the hypothalamic–pituitary–thyroid feedback loop. Thyroid storm carries a mortality rate of 20-30% therefore it is imperative to identify and treat this condition promptly. 47-year-old Hispanic female presented to the ED complaining of palpitations, shortness of breath and intractable emesis for twenty-four hours that began immediately after using methamphetamine. Past medical history was significant for untreated hyperthyroidism. Physical exam was notable for a prominent, palpable thyroid and distended neck veins. TSH<0.004 uU/mL, Total T3=3.49 ng/dL and Free T4=4.47 ng/dL. With a Burch-Wartofsky Score of 35, the patient was medically managed for thyroid storm with propylthiouracil (PTU), SSKI, Propranolol and IV Hydrocortisone. On day 6, an idiosyncratic drug reaction to PTU resulted in agranulocytosis. PTU was withheld and a total thyroidectomy performed on day 8. On day 10, the patient was discharged on levothyroxine. Patients with under-treated hyperthyroidism may be predisposed to catecholamine-induced thyrotoxicosis due to catecholamine hypersensitivity. Therefore, with known methamphetamine use, methamphetamine toxicity and a methamphetamine-induced thyroid storm should be included in the differential diagnosis in a patient presenting with hyperadrenergic symptoms. If anti-thyroid medication fails, a total-thyroidectomy utilizing ligation of the vascular supply as the initial step prevents extrusion of thyroid hormone into the blood stream during thyroidectomy and prevents the possibility of future thyroid storm.

**RECURRENT MASSIVE PLEURAL EFFUSION. CARE PROVIDERS MISUNDERSTANDING OF AN INTERESTING PATHOLOGY. ATTRACTIVE DIAGNOSTIC APPROACH. A CASE REPORT**

**Meenal Misal , UofA College of Medicine - Tucson, MS-III**

2) Huthayfa Ateeli PGY-3, 3) Justin Lee PGY-1 4) Irbaz Riaz PGY-2

**INTRODUCTION** Thoracentesis is a simple bedside procedure that permits pleural fluid to be rapidly sampled, visualized, microscopically examined, and quantified. A systematic approach to analysis of the fluid in conjunction with the clinical presentation should allow the clinician to diagnose the cause of an effusion in ~75% of patients at the first encounter (1). Here we present a case of hepatic hydrothorax with an interesting pathology diagnosed by nuclear imaging.

**CASE PRESENTATION & DISCUSSION** A 63 year old female with a past medical history of alcoholic liver cirrhosis, esophageal varices, and diabetes mellitus type 2 presented to an outside hospital with progressive shortness of breath and was found to have a large right pleural effusion. The patient underwent multiple diagnostic and therapeutic thoracentesis in different hospitals that revealed transudative effusion according to light’s criteria with no evidence of malignancy. None of the healthcare providers were convinced that her effusion was purely hepatic in origin due to the absence of clinically significant ascites and the rapid reaccumulation of unilateral pleural effusion within days after each thoracentesis. The patient was transferred to our university hospital for evaluation. At time of admission the patient had recurrent massive right thoracic effusion. Abdominal ultrasound showed minimal free intrabdominal fluid. She had signs of third spacing on her lower extremities. Our impression from the history and records was hepatic hydrothorax despite minimal ascitic fluid. While the exact mechanism involved in the development of hepatic hydrothorax is incompletely understood, it probably results from the passage of ascites from the peritoneal to the pleural cavity through small diaphragmatic defects. These are typically < 1 cm (and may be
microscopic) and are generally located in the tendinous portion of the diaphragm (2-6). The negative intrathoracic pressure generated during inspiration favors the passage of fluid from the intra-abdominal to the pleural space. Thus, many patients have clinically undetectable or mild ascites. Our patient underwent intraperitoneal injection of Technetium 99mTc albumin aggregated (99mTc-MAA). After less than one hour most of the tracer migrated into the right hemithorax consistent with hepatic hydrothorax.

CONCLUSION Health care providers need to know the diagnostic approach to this interesting entity in order to avoid unnecessary repeated thoracentesis and its potential complications. Once the diagnosis is made treatment will target volume status management with salt and water restriction, diuretics, and other validated options for portal hypertension.

**POLYSOMES: POLYMERIZED LIPIDIC VESICLES FOR SELECTIVE INTRACELLULAR DELIVERY OF MACROMOLECULES**

Jimmy Pham MHS, MA, Midwestern AZCOM, MS-III

Jarvis Walters, Bill Brownlow, and Tamer Elbayoumi

TITLE Polysomes: Polymerized Lipidic Vesicles for Selective Intracellular Delivery of Macromolecules Jimmy Pham*, Jarvis Walters*, Bill Brownlow**, and Tamer Elbayoumi** *Arizona College of Osteopathic Medicine, Midwestern University, Glendale, AZ **College of Pharmacy, Midwestern University, Glendale, AZ

ABSTRACT One principal aim of chemotherapy is specific eradication of cancer cells, mostly through the induction of apoptosis. Genistein, the major soy isoflavone, has been shown to induce apoptosis and arrest cancer cell growth, both in-vitro and in-vivo. Specifically, genistein has been recently reported to induce apoptosis mediated by activation of caspase-9, and -3 and was associated with a decrease in mitochondrial trans-membrane potential and cytosolic release of cytochrome c, in different solid and hematological cancer cells. Owing to the lipophilic nature of genistein, it was an ideal candidate for loading into liposomal, (Lip), nanoemulsion (NE) and micellar (Mic) nanocarriers (NCs). The nano-encapsulation of genistein would afford not only improved delivery of the drug into cancer tissues due to improved solubilization but it also offers enhanced means of intracellular translocation of this partially ionic compound, within interstitial and intracellular pH milieu. Based on different recognized QSAR model-driven criteria for mitochondrial selectivity, genistein, as free drug molecule would be a highly likely mitochondriotropic candidate compound. Hence, lipidic-membrane incorporation of mitochondria-specific ligand would be quite feasible. Therefore, our current work investigated the mitochondria-specific pro-apoptotic activity of genistein-targeted lipid nano-emulsified particles (mean size of 20 & 160 nm for Mic and NE, respectively). Specifically, NCs, with high membrane-incorporation of genistein molecules, in order to facilitate specific delivery to mitochondria of model cancer cells. The mitochondria-specific accumulation of FITC-labeled gensitein NCs, was confirmed microscopically, in different cancer cells. Such targeted delivery strategy would accelerate and enhance the mitochondria destabilization effect of genistein, as well as further induce more caspasedependent apoptotic cancer cell death. The enhanced mitochondrial-specificity and pro-apoptotic activity of gensitein-NCs can serve as a promising drug delivery platform for lipophilic anticancer chemotherapeutic drugs, to augment their anti-neoplastic effectiveness. Tamer Elbayoumi was supported by Midwestern University Intramural funds.

**IS THE BLOCK ABOVE OR BELOW THE AV NODE**
Catherine Phan MD, UofA College of Medicine - Tucson, MS-III

Is the Block Above Or Below the AV Node?  Catherine Phan, M.D., Associate PGYIII, University of Arizona, Tucson, AZ

Introduction: When initial analysis of a rhythm strip shows second degree block corresponding to 2:1 the next question in mind should be: Is this a type-I or type-II second degree block. Rhythms that display non-conducting p-waves on every other beat make it impossible to specify type of second-degree block. Location of block cannot be discerned in second-degree 2:1 AVB. Second-degree type I block indicate conduction problems within the AV node itself whereas type II correlates to conduction problems in the His-Purkinje bundle. Maneuvers utilized to identify block location albeit that definitive location is defined via electrophysiologic. Provocation of vagal tone in slowing AV nodal conduction thereby allowing His-Purkinje tissue more time to recover. Vagal maneuvers improve conduction if the block location was infranodal however worsen AV nodal blocks. On the flip side, exercise and B1 adrenergic pharmacologics increase AV nodal conduction and sinus rate improve AV nodal blocks and worsen infranodal blocks.

Case Study: A 72-year-old male with history of CVA, CAD s/p 3V-CABG, DM, Htn and paroxysmal atrial fibrillation presented to emergency department after 3 weeks of shortness of breath, worsened with exertion and dizziness. Preliminary blood work revealed acute kidney injury (AKI) with Cr 3.6, hyperkalemia associated with metabolic acidemia and bradycardia. His presenting blood pressure was 78/46 mm Hg with atrial fibrillation and corresponding heart rate 48 beats per minute (bpm). His electrocardiogram was negative for the salient cardiac reflections of hyperkalemia or heart block: absent peaked T-waves and no QRS widening. In the ED, the patient was given nebulized albuterol, kaxelate, lactulose, regular insulin with D50W and placed on sodium bicarbonate drip before he was admitted to ICU with cardiac monitoring. The patient’s subsequent blood work revealed appropriate response to hyperkalemic treatment. His AKI resolved with intravenous fluids along with short course of rocephin for UTI and renal ultrasound was negative. Telemetry indicated second-degree atrioventricular (AV) block (AVB) with 2:1 conduction. However, whether the block was a type-I or type-II second-degree heart block was uncertain. Heart rate ranged between 40’s to low 60’s however patient had no lightheadedness, syncope or weakness. Patient was instructed ambulate which surfaced a second-degree type 1 (Wenekebach) heart block. Cardiologist confirmed the assessment and permanent pacemaker placement was not indicated. Diagnostic workup of symptomatic bradycardia should always involve simple maneuvers; i.e Valsalva, carotid massage, exercise, B1 adrenergic provocation; if the patient does not have any contraindications to aid in differentiating type of block since bradycardic arrhythmia treatment depends on type of block. Appropriate management of bradycardia varies with type of AVB.

NECROTIZING FASCITIS; "NEVER UNDERESTIMATE A SMALL SORE ON YOUR FOOT": A CLINICAL CASE REPORT

Alexander Sabre, Universidad Autonoma de Guadalajara, MS-III

Patricia White MD, Carlos G Robles, Laurie Farricielli MD

Necrotizing fasciitis (NF), is a form of soft tissue infection characterized clinically by fulminant tissue destruction of the poor blood supplied muscle fascia and overlying subcutaneous fat, sepsis, organ failure,
and often high mortality. These infections are composed of two distinct bacterial etiologies; Type I polymicrobial infection origin or type II Group A Streptococcus (GAS) based.[1] Many virulence factors are present in GAS that makes infection spread to deep fascia planes through venous and lymphatic channels. [5] Stevens et al described the changing potential of GAS from rheumatic fever to toxic shock-like syndrome concluding with the importance of “scarlatina toxin”. [4] Renamed the M antigen, this filamentous protein acts as a super antigen in vivo as well as possessing anti-phagocytic qualities; cases of streptococcal toxic-shock syndrome with this factor are associated with 50% increased incidence of NF [3] In regards to epidemiology, there is an incidence of 3.5 cases of GAS infections per 100,000 individuals in United States, with NF infections making up approximately 6 percent of these cases. [2] Significant mortality is associated with NF, 21% with type I and 14-34% with type II. [1] Necrotized soft tissue infections are associated with predisposed conditions such as NSAID drug use, obesity, recent surgery, immunosuppression, traumatic wounds and diabetes. [3] Important manifestations of NF include fever, soft tissue involvement with pain out of portion with visual findings, rapid progression to swelling, brawny edema, dark red induration, bullae, and ending with presentation of necrotic skin. Laboratory work-up includes elevated WBCs and creatine kinase levels with decreased BUN and sodium [1]. Blood cultures are positive in only 60% of cases with GAS. [2] Imaging studies include MRI, CT, doppler, and X-ray of soft tissues in order to delineate infection progression. Following clinical suspicion of NF, it is imperative for clinicians to understand current treatments based upon literature. This condition is considered a surgical emergency and a greater than 24 hour delay in surgery was a factor for increased mortality. [1] Diagnosis of NF can only be done with explorative surgery of affected areas and treatment consist of early aggressive surgical debridement of tissue necrosis along with empirical antibiotic therapy in order to control the spread of infection. In this presentation, we discuss a case of type II necrotizing fasciitis in a 39 year-old female patient troubled with many predisposing factors, who presented to the emergency department with cellulitis of her right foot and lower leg that rapidly developed into necrotizing fasciitis. This case is of particular interest due to a long history of difficulties following surgical treatments and will also include information regarding the importance of early detection and prompt treatments in order to avoid the catastrophic consequences with necrotizing fasciitis.

**BREWING UP TROUBLE**

**Muhammad Osman Salim, Midwestern AZCOM, MS-III**

C. Anneski, MD; M. M. Salim, MD; M. Vasiq, MD; Y. Shareef, DO; M. H. Horani, MD

Introduction:

Foodborne botulism is reported 25 times annually in the U.S. Potential lethality combined with public health outbreak merits close clinical study.

Case Study:

A 24 year old Native American male prisoner along with seven other inmates in Arizona presented with dizziness, ptosis, dysphagia, dysarthria, weakness, descending paralysis, nausea, and vomiting. They claimed to have drunk “hooch”, prisonmade alcohol, two days ago that was concocted from potatoes and fruits inside a garbage bag. They were suspected of botulism poisoning. All eight patients were placed in the ICU for monitoring, supportive care, and all received antitoxin by the CDC. Seven were intubated due to
respiratory failure and many placed on NG tubes alongside propofol sedation. Comprehensive function was intact, but movement was limited to gestures.

Labs: Vital signs WNL, WBC was 8.4 c/mL, Hgb 16.4 g/dL, alkphos 143 IU/L, sodium 133 mEq/L, potassium 3.7 mEq/L, chloride 96 mEq/L, BUN 10 mg/dL, creatinine 1.08 mg/dL, glucose 99 mg/dL, alcohol level <3. AST/ALT <40 IU/L, TBili .16 mg/dL, and CXR negative.

Seven of the patients' serum tested positive for C. botulinum Type A toxin, a more common and potent strain. Many patients developed polymicrobial pneumonia, suggesting aspiration versus ventilator associated. Also, around day five post-antitoxin, a serum sickness reaction, which may occur, was suspected as several patients were febrile without new infections being isolated with a normal WBC. Ileus was another concern since the spores are ingested, contributing to paralysis. GIT transit was encouraged with pharmacologically induced peristalsis.

Discussion:

Clostridium botulinum is a gram positive, spore-forming, obligate anaerobic bacilli that produces preformed toxins that cleave synaptobrevin, which mediates fusion of NT containing vesicles, thereby inhibiting acetylcholine release, causing botulism. In adults, the disease is typically caused by toxin ingestion cultivated in closed containers, whereas in infants, it is frequently caused by spores found in honey or infantile GIT colonization. Symptoms due to muscarinic inhibition include symmetric descending flaccid paralysis, diplopia, dysarthria, dysphagia, xerostomia, nausea, vomiting, diarrhea, and abdominal pain. Severity of illness can range from mild/self-limiting to rapidly fatal, especially if respiratory muscles and/or the diaphragm are affected. No vaccine or natural immunity is known.

Definitive diagnosis is made by toxin presence in stool or serum. If suspicion is high, the CDC should be contacted for investigation, such that emergency antitoxin may be shipped and promptly administered. This would not reverse paralysis, but arrest disease progression. Recovery follows the regeneration of neuromuscular connections.

Conclusion:

Because clinicians are the first to treat patients with this rare disease, they must be proficient in rapidly assessing and treating foodborne botulism. Procuring the antitoxin early to avoid lethality is a must alongside involving the CDC so that investigations may counteract a public health outbreak.

References:

http://emergency.cdc.gov/agent/botulism/clinicians/index.asp

http://wwwnc.cdc.gov/eid/article/15/1/08-1024_article.htm

**AN UNCOMMON CAUSE OF DIARRHEA IN POST CARDIAC TRANSPLANT PATIENT**

**Kristina Voss , UofA College of Medicine - Tucson, MS-III**

Amber Bellafiore, Santhosh G John MD
AN UNCOMMON CAUSE OF DIARRHEA IN POST CARDIAC TRANSPLANT PATIENT  Kristina Voss, Amber Bellafiore, Santhosh G John MD University of Arizona College of Medicine

Blastocystis is an unusual enteric unicellular protozoan parasite found in human and animal intestines. Its clinical relevance is still subject to discussion with numerous conflicting reports on its ability to cause disease. Accumulating epidemiological, in vivo, and in vitro data strongly suggest that Blastocystis is a pathogen. Given the increasing prevalence of organ transplantation in the current era, physicians need to consider this pathogen in the differential of acute diarrheal disease. We present a case of a 69 year old man with history of cardiac transplantation, admitted with acute diarrhea. Our patient presented to the ED with fever, chills, rigors, nausea, vomiting, crampy abdominal pain and profuse watery diarrhea of 4 days duration. His past medical history is significant for cardiac transplant 2 years ago and is currently being maintained on adequate immunosuppressive therapy. On admission, patient had a temperature of 103°F, tachycardia with stable blood pressure and O2 sats. Physical examination findings were unrevealing other than for vague diffuse abdominal tenderness. In light of his complex past medical history, extensive workup for infectious causes was done including Chest x-ray, CT of chest, abdomen and pelvis. Bronchoscopy with BAL was negative for any pulmonary pathology. Serological workups for infectious and inflammatory process were also negative. Urinalysis is negative for Legionella antigen assay, but positive for a few colonies of MDR E. coli urinary tract infection. Blood cultures were negative and there was no evidence of pyelonephritis on imaging studies as well. Given his immunocompromised state, patient was started on IV Ertapenem with rapid improvement in his urinary symptoms. However the patient continued to have persistent watery diarrhea. Stool culture was negative for Shigella, Salmonella, C. Diff, and Campylobacter. The stool sample was, however, positive for Blastocystis hominis. He was started on Metronidazole orally. Patient had rapid improvement in his diarrheal symptoms in 2-3 days of treatment and was discharged home to complete therapy for a total of 7 days. This case illustrates the potential for Blastocystis hominis, a controversial pathogenic or commensal parasite, to demonstrate opportunistic qualities in an immunocompromised host. Identification of Blastocystis hominis in this patients stool may represent an aberrant subspecies capable of opportunistic and/or primary infection; however, subspecies classification was not determined on this specimen. The debate regarding the pathogenicity of Blastocystis hominis remains inconclusive; however, this case demonstrates that Blastocystis hominis infection should be given weight in the overall clinical picture. Metronidazole is currently considered the first line agent for the treatment of this disease.

**First Place – Medical Student Poster**

**CAMPAIGN AGAINST TEXTING AND DRIVING**

Sandeep Bains , UofA College of Medicine - Tucson, MS-II

Peter Rhee MD, Viraj Pandit MD, Daniel Judkins RN, and Bellal Joseph MD

**INTRODUCTION:** Distracted driving is a growing public safety problem which is estimated to cause over 387,000 critical injuries and over 3,300 fatalities annually. Distracted driving has been primarily considered as a problem among the young drivers however; in recent years there has been an increasing incidence of distracted driving among adults. The primary aim was to identify the incidence of distracted driving among health care providers. Our secondary aim was to create awareness and prevention strategies against distracted driving. We hypothesized that distracted driving is prevalent among health care providers.
METHODS: We performed a prospective interventional study of all the staff members at our hospital. The trial involved three phases: Phase one was 1 week pre-intervention observation outside employee parking garage. Phase two was 1 week intervention phase carried out in hospital cafeteria, banners at garage exit, and survey questionnaire via email. Phase three was 1 week post-intervention observation. Observations were carried out at three time intervals: 6.30-8.30am, 4.40-5.30pm, and 6.30-7.30pm. We defined distracted driving as texting or talking on cell phones. Hospital employees were identified with: badges and scrubs, employees exiting through employee gate, and parking pass on the car. Our primary outcome measure was incidence of distracted driving pre and post intervention. Univariate analysis was performed to compare incidence of distracted driving pre and post intervention. RESULTS: A total of 10,859 observations (Pre: 6,639, Post: 4,220) and 520 survey respondents were collected. The mean age of respondents was 44±27.5 years and 88% were female. 35.5% respondents admitted to texting while driving while 4.5% respondents were involved in an accident due to texting and driving. 77% respondents felt more informed after the survey and 91% respondents supported a state legislation against texting and driving. There was a significant reduction in distracted driving pre and post intervention in each of the time interval of observation (6.30-8.30am: 9% vs. 4%, p=0.01; 4.30-5.30pm: 19% vs. 14.6%, p= 0.02; and 6.30-7.30pm: 29% vs. 24.5%, p=0.04). On sub-analysis, there was a significant reduction in talking (10.1% vs. 4.8%, p= 0.001) and texting (5.2% vs. 2.3%, p= 0.01) while driving post intervention. CONCLUSION: Distracted driving is prevalent among healthcare professionals. We recorded greater than 50% reduction in the incidence of distracted driving during the post-intervention phase. Implementation of a national education campaign against distracted driving is warranted.

**INFLUENCE OF THE SOY ISOFLAVONE GENISTEIN ON JEJUNAL FUNCTION IN OB/OB MICE: POTENTIAL CLINICAL BENEFIT?**

Scott Cochran, Midwestern AZCOM, MS-II

L. Leung, D. Larsen, R. Jelenick, J. Willey, T. Broderick and L. Al-Nakash

The leptin-deficient ob/ob mouse is obese and diabetic. The goal of this study was to characterize the effect of the naturally occurring phytoestrogen, genistein, administered via diet (600 mg/kg diet, 600G) for 4-weeks on jejunum structure, function and physical characteristics in this clinically relevant mouse model of diabetes. Female and male ob/ob mice were aged 12-13 weeks. Comparisons were made to groups fed standard rodent chow. Genistein diet, 600G, significantly reduced body weight by 6g in females, but was without effect in males. We measured transepithelial short circuit current (Isc, a measure of chloride secretion), across freshly isolated segments of jejunum. Basal Isc was significantly increased with 600G diet, by 2-fold in males and 1.6-fold in females. Total expression of the cystic fibrosis transmembrane conductance regulator, CFTR, the major route for chloride exit across jejunum epithelia, was decreased by 70% in females fed 600G (no effect in males), suggesting that a change in CFTR protein (normalized to GAPDH) was not responsible for the 600G-mediated increase in Isc in females. In addition, 600G increased crypt depth 13% in males, but decreased crypt depth 25% in females; this may account in part for the increased basal Isc in males, and may also explain the loss of CFTR protein with 600G in females. Interestingly, 600G increased wall thickness 10% in males (no effect in females). We predict that genistein may be of benefit in improving gastrointestinal transit via increased smooth muscle contractility in ob/ob males. The jejunum is the major site for absorption, thus we determined the effect of 600G on expression...
of key transporters (normalized to GAPDH); SGLT-1 expression, responsible for glucose and galactose absorption, was unaffected by 600G in both males and females, GLUT5 expression (responsible for fructose absorption) was increased 2-fold in females (no effect in males), and GLUT2 expression (responsible for monosaccharide transport across the basolateral membrane) was decreased 50% with 600G in males (no effect in females). These data suggested that the 600G-mediated weight loss in females is not associated with concomitant loss of monosaccharide transporters. These data support our hypothesis that soy products may have sex-dependent clinical benefits to those with diabetes and obesity.

**A CROSS-SECTIONAL STUDY OF DIETARY BEHAVIORS AND PERCEIVED BARRIERS TO HEALTHY EATING IN AT-RISK PATIENTS FOR TYPE 2 DIABETES MELLITUS AT THE FRANK BRYANT HEALTH CENTER (SAN ANTONIO, TEXAS)**

Mary Hoang, Midwestern AZCOM, MS-II

Introduction Type 2 diabetes mellitus (T2DM) is a major public health concern in San Antonio, Texas, affecting 14% of its population. It is well-understood that the development of T2DM is influenced by both genetic factors and lifestyle choices, the latter of which include healthy eating. Several studies have suggested that potential barriers to healthy eating include accessibility, cost, difficulty of food preparation, and social environment. This study is to investigate the dietary behaviors of at-risk patients for T2DM and to assess, thereafter, the potential barriers to healthy eating that increase the risk of development of T2DM.

Methods Forty-four adult patients were recruited by convenience sampling from the primary care clinic waiting area at the Frank Bryant Health Center in San Antonio, Texas. Subjects were stratified into two groups: at-risk for diabetes or non at-risk for diabetes, based on whether they had a history of gestational diabetes and/or a family history of diabetes. Descriptive statistics and percentage comparisons are used.

Results Of the 44 participants, 33 were at-risk for diabetes and 11 were non at-risk for diabetes. 84% of subjects were overweight or obese. For the at-risk group, cost was the greatest perceived barrier to healthy eating. For both groups, social environment presents a potentially hidden barrier to healthy eating. Both groups show a lack of diabetes education and proper nutritional understanding. Conclusion Clinicians should emphasize weight loss in overweight and obese patients and provide diabetes education and proper nutrition education to patients at-risk for T2DM. Friends and family members should be included in implementing dietary changes. Health clinics can work with local diabetes prevention programs to promote healthy lifestyle habits in the community.

**SEX-DEPENDENT DIFFERENCES IN OB/OB MALE AND FEMALE MICE: CLINICAL RELEVANCE?**

Ryan Jelinek, Midwestern AZCOM, MS-II

L. Leung, D. Larsen, S. Cochrane, J. Willey, T. Broderick and L. Al-Nakkash

The ob/ob mouse lacks functional leptin and presents with obesity, and hyperglycemia. The goal of this study was to characterize sex-dependent differences in jejunal structure and function in this clinically relevant mouse model of diabetes. Comparisons were made to lean counterparts. Mice were 12-13 weeks of age. Body weights of female (51.9±1.3 g, n=9) and male (50.9±2.0 g, n=10) ob/ob mice were comparable. Basal transepithelial short circuit current (Isc, a measure of chloride secretion), across freshly isolated segments of jejunum was significantly increased 1.5-fold in female ob/ob mice compared to males.
Expression of the cystic fibrosis conductance transmembrane regulatory protein, CFTR (normalized to GAPDH), the major route for chloride exit across jejunum was significantly increased 3.5-fold in ob/ob females compared to males. Crypt depth (the site of chloride secretion) was comparable for both sexes. We determined the expression profile of several key transporters associated with monosaccharide absorption within jejunum (all were normalized to GAPDH). GLUT5 (responsible for fructose absorption) was significantly reduced 60% in ob/ob females compared to males. This could be related in part to the significant 20% decrease in villi length measured in the ob/ob females. Interestingly, GLUT2 (responsible for monosaccharide transport across the basolateral membrane) and SGLT1 (responsible for glucose and galactose absorption) remained similar for both ob/ob females and males. These data suggest that the increased basal jejunal lsc in ob/ob female mice appears to be directly associated with an increase in CFTR expression, without structural change in crypts. Whether this correlates to an increase in gastrointestinal transit time in females compared to males, remains to be seen. The increase in fructose absorption in males, a consequence of the increased GLUT5 expression, may be a major contributor towards the ob/ob male phenotype, and thus may be a target for therapeutic manipulation in males. Our data supports the idea that sex-dependent treatment options would likely be optimal.

12 WEEKS TO LIVE? CHARACTERISTICS OF LONG TERM SURVIVORS (> 12 MONTHS) TREATED ON ONCOLOGY PHASE I STUDIES. THE VGPCC/TGEN EXPERIENCE

Gagan Preet Kaur, UofA College of Medicine - Tucson, MS-II

Mark Slater, Glenn Weiss, Gayle Jameson, Cathy Mast, Erica White, Molly Downhour, Daniel D Von Hoff

Introduction: Phase I clinical trials are restricted to patients with advanced incurable cancers without any treatment options. The life expectancy for these patients is 3-5 months and few survive > 12 months. Eligible patients need to have an estimated life expectancy of > 12 weeks. Phase I studies were once thought of as the last resort for advanced cancer patients, however with molecular target selection of agents in individual patients, we are increasingly seeing long term survivors. Methods: The medical records of patients entered onto phase I clinical trials in 2008 and 2009 were reviewed. IRB approval was obtained for review of patient characteristics.

Results: We identified 30 patients (approximately 8% of patients seen in 2008-2009) with > 12 months survival from study entry. The characteristics of these 30 are described. The median age is 63.4 years (range 54-84).

Performance status: ECOG 0 in 60% and ECOG 1 in 40%. 66% were male. Tumor types were: prostate 17%, pancreas 13%, basal cell 10%, breast 10% and lung 7%. The remainder (43%) included liposarcoma, small bowel, ovarian, uterine, cervical, appendiceal, gastric, colon, neuroendocrine and adrenal tumors. The median survival of this group (n=30) is 27.9 months from start of the first phase I trial.

Tumor involvement: 13% locally advanced, 70% had one organ (usually liver), 13% had 2 organs involved and 3% had 3 organs involved. The majority (90%) were treated with a first in human molecular targeted agent. The agents were TKI (20%), PI3K (15%) and a variety of other molecular inhibitors (cMET, H1F1-#945; Anti-myostatin, Hedgehog, W, HSP-90ee-1, CHK2, CDK etc).
Conclusions: A subset of patients (about 9%) treated with molecular targeted agents have prolonged (>12 months) survival, which is encouraging. A good performance status and <1 organ involvement correlates with survival. In the near future, all patients will have a complete genomic profile to select individualized therapy. In our program, molecular profiling with whole genome sequencing, CGH etc is already being introduced to appropriately select biomarkers for therapy.

**PARAGANGLIOMA TYPE 1**

Khalid Salim, Midwestern AZCOM, MS-II

K. Salim MS; M.O. Salim MS; A. Choure MD; M. Shahlapour MD; M. H. Horani MD

Case presentation: A 32 year old morbidly obese female with a history of hypertension and hypothyroidism presented to the ER after a sporadic syncopal episode at home. She complained of a four month history of intermittent episodes of blurry vision, headache, dizziness, and neck pain. She went to the ER after an episode three months ago and was found to be hypotensive, which was attributed to her medication. After discontinuing them, she continued to have episodes. Her family history is significant for neck masses in her father, sister, and paternal grandfather, as well as hypertension and diabetes. There was no family or personal history of MEN syndrome, thyroid cancer, skin lesions, pancreatic tumors, nor pheochromocytomas.

The physical exam was significant for tenderness to palpation on the left neck. Vital signs showed borderline hypotension with systolic in 90-100s, otherwise normal. Hgb/Hct was 12.6/38.3, platelets were 244, and WBC was 7.6. Sodium was 137, potassium was 3.7, chloride was 105, bicarbonate was 23, and BUN/Cr was 12/66.

A head CT was negative. A neck CTA showed heterogenously enhancing mass lesions in the carotid sheath bilaterally between the internal and external carotid arteries suggestive of paragangliomas. A neck MRI confirmed 2.5 x 1.7 cm masses bilaterally. Soft tissue U/S of head/neck/thyroid revealed diffuse heterogenous enlargement consistent with diffuse multinodular thyroid goiter and nodules. Genetic testing revealed one amino acid mutation at Proline-81-Leucine in the SDHD gene, consistent with paraganglioma-pheochromocytoma syndrome type 1.

An electrophysiology consult placed a dual-chamber pacemaker, alleviating syncopal episodes. An endocrinology consult ruled out a secretory paraganglioma and pheochromocytoma by checking catecholamines, metanephrines, dopamine, and chromogranin A. Calcitonin and CEA markers returned normal, ruling out thyroid cancer. The patient followed up with neurosurgery, who recommended bilateral excision.

Background/discussion: Hereditary paraganglioma is an autosomal dominant disorder involving mutations involved in at least 4 genes SDHD, SDHAF2, SDHC, and SDHB, which correspond to paraganglioma type 1, 2, 3, and 4 respectively. These genes modify the enzyme succinate dehydrogenase which, if dysfunctional, leads to succinate accumulation causing abnormal, yet benign cell growth. This can manifest into parasympathetic nerve cell bunches found on head, neck, trunk, or sympathetic bundles on the adrenals as a pheochromocytoma. The patient had bilateral nonsecretory parasympathetic neuroendocrine masses on the carotid bodies, causing symptomatic bradycardia secondary to vagal symptoms related to paranglioma.
There is accurate diagnosis. Excision is the definitive treatment for secondary symptoms.

References:


http://www.cancer.gov/cancertopics/pdq/treatment/pheochromocytoma/HealthProfessional

**IMPROVING EARLY SCREENING AND DIAGNOSIS OF AUTISM IN UNDERSERVED POPULATIONS IN ARIZONA THROUGH THE ARIZONA LEADERSHIP EDUCATION IN NEURODEVELOPMENTAL AND RELATED DISABILITIES (AZLEND) PROGRAM**

Alexandra Tsontakis BA, UofA College of Medicine - Tucson, MS-II

Purpose for study: The University of Arizona Leadership Education in Neurodevelopmental and Related Disabilities (AZLEND) program is an interdisciplinary graduate training program that trains professionals who care for individuals with disabilities; they come from a variety of disciplines and are trained to be leaders in their fields. Fellows participate in a yearlong research project designed to improve early identification of and referral for children with developmental disabilities and autism in rural and border counties to increase early detection of disabilities in rural populations and to thereby minimize healthcare disparities in these areas.

Methods: The project improves compliance with the American Academy of Pediatrics’ developmental surveillance and screening guidelines in early childhood and collects pre- and post-screening and referral patterns to detect rates of change. The research project requires fellows to: interview key players in rural clinical practices; develop a surveillance and screening process flow integrating formal screening tools and surveillance into each practice; provide training on developmental screening tools and the new clinic operation processes to physicians and staff; provide follow-up technical assistance; and collect screening and referral data through medical record abstraction. Four sites were recruited to participate in the 2012-2013 study, one in each of the following Arizona counties: Yuma County, Coconino County, Cochise County, and Santa Cruz County.

Results: Sunset Community Health Center in Yuma County completed the program and implemented the PEDS screening tool in all three of their locations successfully. There were 190 eligible well-child visits (9-, 18-, or 24-month) representing 187 children (54% male and 87% Hispanic) from the Sunset Community Health Center sites. In addition, 86% of the families were either on public assistance or had no insurance. There was an evenly distributed number of 9-, 18-, and 24-month visits during the study from September 2012 through March 2013. Findings from Sunset Community Health Centers showed that formal developmental screening rates increased significantly from 0% pre-intervention to 85% post-intervention (Chi-square 135.7, p=.000).

Conclusion: The success at Sunset Community Health Centers provides evidence that a developmental screening program can be successfully incorporated into rural community health practices with AZLEND fellows acting as primary contacts and providing technical assistance and training. Key lessons learned from the experience indicate that the success of implementation and continued adherence to the new policy is dependent on a local physician advocate within the healthcare practice to ensure protocol adoption by reiterating the need communicated by AZLEND fellows. The project will be continued with an attempt to...
include more practices from different parts of the state that deal with underserved populations. Presenting Author: Alexandra Tsontakis, BA, The University of Arizona College of Medicine

LIBMAN SACKS ENDOCARDITIS

Jawad Hussain , UofA College of Medicine - Phoenix, MS-I

K. Salim MS; T. Jamal, H. Salim; P. Patel MD; M. H. Horani, MD

Introduction: Cardiac involvement is common among patients with systemic lupus erythematosus. Cardiac disease in SLE can be divided as follows: Pericardial Disease, valvular disease, myocardial dysfunction, and coronary arteritis.

Case Study: A 62 year old female was admitted with one episode of syncope and fatigue. Her past medical history included pericardial effusion s/p pericardial window 6 months ago, pancytopenia with negative bone marrow biopsy, diabetes, and hypertension. While in the hospital, the patient developed fever. CT of the abdomen showed a splenic infarct. A detailed infectious workup followed with negative bacterial etiology. Transesophageal echocardiogram was performed and revealed a 0.5 cm vegetation attached to the aortic valve with mild regurgitation. Broad spectrum antibiotics were started for possible culture negative endocarditis. Her CBC showed WBC of 1.1, Hgb 8.9 g/dl, platelet 75,000. Pt was found ANA positive, and double strand DNA was highly positive 8,334 IU/mL. SSA and SSB were negative; complement was low and antiphospholipid antibody was elevated. Cardiolipin IGM was 82 MPL (0-12 nl), and Beta-2 Glycoprotein-1 Abx >150 (0-20 normal). The diagnosis of SLE was made with positive antiphospholipid antibody with cardiac involvement including the pericardium and valvular disease in the form of non infectious vegetation (Libman-Sacks endocarditis). The patient was started on steroid and antibiotics were also continued with eventual improvement of her symptoms. Discussion and conclusion: Pericarditis is the most commonly recognized cardiac problem in SLE. It may occur as the initial manifestation of SLE, or at any point of the disease process. Pericardial tamponade is rare, but may occur at any time in the course of SLE. Treatment is usually with NSAIDs or steroids for patients who do not tolerate or respond to NSAID. Drainage of the fluid considered if clinically indicated. Valvular pathology in SLE is common. Cases may present as noninfectious vegetations (Libman-Sacks endocarditis). Valvular thickening is the most common echocardiographic finding followed by vegetation and valvular insufficiency. Valvular lesions in SLE may occur at any time, and their presence does not correlate with disease activity. Vegetations are usually near the edge of the valve and consist of accumulations of immune complexes, hematoxyline bodies, fibrin, and platelet thrombi. Steroid or cytotoxic therapy does not appear to have an effect upon valvular lesions. Valvular surgery may be indicated if the patient developed severe valve regurgitation, or rarely stenosis.

Third Place – Medical Student Poster

OPTIMIZATION OF PORCINE HEART DECELLULARIZATION

Tiffany Son , UofA College of Medicine - Tucson, MS-I

Ning Qu, Anthony Louis, Courtney Hemphill, Alice Ferng, Brigid Smith, Katherine Stavoe, Kitsie Penic
The traditional treatment of Stage IV cardiac disease is limited by available donor organs. As heart disease mortality grows worldwide, the disparity between the large patient population and the small heart donor pool is an increasing problem that makes cardiac tissue regeneration an anticipated solution. A method for organogenesis is to use a decellularized heart as a biological scaffold which bypasses the complications of organ donation and allows for clinical cardiac transplantation. This study developed a decellularization protocol for porcine heart and compared experiment durations of 5 hours and 10 hours in order to optimize this technique. The system was pressure regulated and monitored to simulate physiological conditions with gradual flow rates ($\leq$2200ml/min) and pressures ($\leq$400 mmHg) to decellularize porcine hearts ($n=9$). Decellularization involved perfusion with 1-3% triton and 1-2% sodium dodecyl sulfate and a porcine heart was perfused with only ddH2O for comparison. Native and decellularized hearts were histologically analyzed. After a decellularization process of 5 hours or 10 hours, the non-native hearts appeared grossly translucent, indicating loss of cellular components. Histological data indicated removal of nuclear material with varying ranges of structural architecture in the decellularization procedures. The 5 hour reaction depicts the optimization of this procedure with the cardiomyocytes retaining the most structural integrity and minimal nuclear material compared to the other procedures. The aim of monitoring the decellularization protocol resulted in a shorter cardiac decellularization method in order to provide a viable scaffold for stem cell re-seeding.