Florida Chapter
45th Annual Scientific Meeting
Cuc T. Mai, MD FACP - Scientific Program Chair

✧ Resident & Medical Student ✧
Poster Competition

✧ Doctor’s Dilemma© Competition ✧

October 4 - 6, 2013
Sirata Beach Resort & Conference Center
St Pete Beach, Florida
Table of Contents

CME Information .................................................................................................. 2
Faculty .................................................................................................................... 3
Guest Speakers & Resident Presenters ................................................................. 4
Special Program Notes ........................................................................................... 5
Scientific Program Agenda .................................................................................... 6
Doctor’s Dilemma Participants .............................................................................. 8
2013 Formal Poster Judging Presentations - Resident ......................................... 9
2013 Formal Poster Judging Presentations - Medical Student .............................. 46
2013 Poster Presentations – Display Only............................................................. 67

Governors

Michael A. Zimmer, MD, FACP
Mailing Address: 509 Jackson Street N
St Petersburg, FL 33705
Telephone: 727-820-7800
Fax: 727-820-7801
E-mail: drzimmer@michaelazimmer.md

Michelle L. Rossi, MD, FACP
Mailing Address: 1600 SW Archer Road, # 4102
Gainesville, FL 32610
Telephone: 352-273-7761
Fax: 352-271-4560
E-mail: mrossimd@hotmail.com
Florida Chapter Annual Scientific Meeting  
*October 4 - 6, 2013*

### Learning Objectives

At the conclusion of this activity, the participant will be able to:

- Apply updated knowledge of internal medicine to clinical practice;
- Improve clinical practice by incorporating major new developments in subspecialty medicine; and
- Understand recent advances in internal medicine.
- Satisfy requirements of state-mandated educational courses on HIV and Prevention of Medical Errors

### CME

Accreditation and Designation Statement

This activity has been planned and implemented in accordance with the Essential Areas and policies of the Accreditation Council for Continuing Medical Education through the joint sponsorship of the Florida Medical Association and the Florida Chapter American College of Physicians. The Florida Medical Association is accredited by the ACCME to provide continuing medical education for physicians.

The Florida Medical Association designates this live activity for a maximum of eighteen (18.0) *AMA PRA Category 1 Credits™*. Physicians should claim only the credit commensurate with the extent of their participation in the activity.

The planners of the activity disclosed no financial relationships relevant to the content to be presented.

The speakers listed on page four and five disclosed no financial relationships relevant to the content to be presented with the exception of the following:

Charurut Somboonwit, MD FACP - Speakers Bureau, Gilead Sciences, Consultant - Boehringer-Ingelheim

### Resident & Medical Student Activities and Events

Clinical vignettes prepared by Residents will be presented at the meeting as well as posters prepared by Residents and Medical Students. The Resident oral presentations winner is invited to represent the Chapter at the 2014 national competition (2014 Internal Medicine) and will receive up to $1,000 funding from the Chapter. The winners of the poster competition will receive a plaque. Winners of the Doctors’ Dilemma™ will represent the Chapter at the 2014 Internal Medicine Doctors’ Dilemma™ competition and each resident may receive up to $1000 funding.
2013 Annual Meeting Faculty

Brooke T. Baldwin, MD
Ankush K. Bansal, MD FACP
Carmel J. Barrau, MD FACP

Charles W. Brock, MD
Michelle L. Estevez, MD
Robert D. Geck, MD

Jason Goldman, MD FACP
Michael L Howell, MD FACP
Lara W. Katzin, MD

Owen Linder, MD FACP
Cuc T. Mai, MD FACP
Naresh H. Pathak, MD FACP

Michelle L. Rossi, MD FACP
John F. Rubin, MD FACP
John T. Sinnot, MD FACP
2013 Annual Meeting Faculty

Charurut Somboonwit, MD FACP
Brice T. Taylor, MD
Stephanie P. Taylor, MD MS
Howard S. Tuch, MD MS
Ana P. Velez, MD FACP
Michael A. Zimmer, MD FACP

Guest Speakers

Charles Cutler, MD FACP
Chair, Board of Regents, ACP
Christopher L. Nuland, Esq
FL Chapter ACP Counsel
Clifford G. Rapp, LHRM
VP, Risk Management, The Doctors Co

Resident Presenters

Vertilio M. Cornielle, MD
Mount Sinai Medical Center
Emmanuel S. Coronel, MD
University of Miami/Jackson Memorial
Morganna Freeman-Keller, MD
University of South Florida
Oral A. Waldo, MD
Mayo Clinic Florida
Friday, October 4, 2013

Highlights in HIV Medicine for the Internist
Meet the requirements set forth in Florida Statute 456.033, and Rules 64B8-13.005 & 64B15-13.001 requiring an HIV/AIDS course for the first license renewal.

Saturday, October 5, 2013

2nd Annual Humanism in Medicine Lecture Series:
Art of Medicine: Diagnosing the Canvas
John T. Sinnott, MD, FACP

Residents and Medical Students Workshop:
"Paging Dr. Twitter! Pitfalls in Online Medical Professionalism"
- Discuss the impact of electronic communication and social networking on physician-patient interaction
- Using examples of potential confidentiality breaches, encourage audience discussion on ethical and professional issues in each case
- Learn guidelines for future use, including safeguards for online social behavior and respect of patient privacy

Prevention of Medical Errors
Meet the requirements set forth in Florida Statute 456.013 for biennial license renewal

Living Well Practicing Medicine
- Understanding Economics of Managed Care and the flow of funding from CMS to you.
- Profitability of Capitation Contracts for Primary Care Providers
- Transforming "Pay for Performance" into "Profiting with Performance"

Formal Poster Competition for Residents and Medical Students
Awards Reception

Sunday, October 6, 2013

Fellowship (FACP) Mentoring Breakfast (pre-registration required)
This is a mentoring session designed for those practicing physicians who would like more information on advancement to Fellowship (FACP)

HIPAA Compliance Made Easy
How to comply with the new HIPPA Privacy, Security Rules effective 9/23/2013

Tales from the Trenches
What residents and medical students need to know about selecting a practice, including practice models and lifestyle choices.

Doctors' Dilemma Competition - Finals
Florida Hospital Orlando
University of Florida
University of Miami/ Jackson Memorial Hospital
Scientific Program and Special Events

Florida Chapter ACP 2013
Scientific Meeting Program

Updates to Strengthen the Internist’s Core

Friday, October 04, 2013 Majestic Palm Ballroom

7:00 a.m. - 8:00 a.m. Registration & Breakfast

7:55 a.m. Governors’ Welcome
Michelle L. Rossi, MD FACP, Governor, ACP Florida Chapter
Cuc T. Mai, MD FACP, Scientific Program Chair

8:00 a.m. Highlights in HIV Medicine for Internists*** (Mandatory for New Licensees)
Charurut Somboonwit, MD FACP

9:00 a.m. Updates in Sleep Medicine for the Internist
Robert D. Geck, MD

10:00 a.m. Networking Break with Exhibitors

10:30 a.m. Migraine Headaches: What Internists Need to Diagnose & Treat
Charles W Brock, MD

11:30 a.m. Dermatology for the Internal Medicine Physician
Brooke T. Baldwin, MD

12:25 p.m. Adjourn

12:30 p.m. Luncheon Program

1:45 p.m. Palliative Care in the Age of Health Care Reform
Howard Tuch, MD MS

2:45 p.m. Twenty Skin Infections that an Internist Should Know
Ana P. Velez, MD FACP

3:45 p.m. Networking Break with Exhibitors

4:15 p.m. What is the Real Risk of Radiation Exposure from Medical Imaging?
Stephanie P. Taylor, MD MS

5:15 p.m. What’s New in Community Acquired Pneumonia & Health Care Associated Pneumonia
Brice T. Taylor, MD

6:15 p.m. Adjourn
<table>
<thead>
<tr>
<th>Time</th>
<th>Event</th>
</tr>
</thead>
<tbody>
<tr>
<td>7:30 a.m.</td>
<td>Registration &amp; Breakfast</td>
</tr>
<tr>
<td>8:00 a.m.</td>
<td>Scientific Session</td>
</tr>
<tr>
<td>8:25 a.m.</td>
<td>Geriatric Assessment for the Internist</td>
</tr>
<tr>
<td>8:30 a.m.</td>
<td>Michelle L. Estevez, MD FACP, Scientific Program Chair, Presiding</td>
</tr>
<tr>
<td>9:00 a.m.</td>
<td>Peripheral Neuropathy for the Internist</td>
</tr>
<tr>
<td>9:30 a.m.</td>
<td>Lara W. Katzin, MD</td>
</tr>
<tr>
<td>10:00 a.m.</td>
<td>Networking Break with Exhibitors</td>
</tr>
<tr>
<td>11:00 a.m.</td>
<td>2nd Annual Humanism in Medicine Lecture Series: Art &amp; Medicine: Diagnosing the Canvas</td>
</tr>
<tr>
<td>11:00 a.m.</td>
<td>John T. Sinnott, MD FACP</td>
</tr>
<tr>
<td>12:00 p.m.</td>
<td>Town Hall Meeting: ACP College Highlights</td>
</tr>
<tr>
<td>12:00 p.m.</td>
<td>Charles Cutler, MD FACP, Chair, American College of Physicians Board of Regents</td>
</tr>
<tr>
<td>12:30 p.m.</td>
<td>Governor’s Luncheon</td>
</tr>
<tr>
<td>12:30 p.m.</td>
<td>The Doctor is &quot;IN&quot; but not &quot;ALL THERE&quot; - Balancing Life at Work and at Home</td>
</tr>
<tr>
<td>1:00 p.m.</td>
<td>Naresh H. Pathak, MD FACP</td>
</tr>
<tr>
<td>1:30 p.m.</td>
<td>A Dose of 'Alcohol' Saved Her Life</td>
</tr>
<tr>
<td>1:30 p.m.</td>
<td>Oral A. Waldo, MD (Mayo Clinic Florida)</td>
</tr>
<tr>
<td>1:30 p.m.</td>
<td>Residents Final Competition</td>
</tr>
<tr>
<td>2:15 p.m.</td>
<td>Networking Break</td>
</tr>
<tr>
<td>2:30 p.m.</td>
<td>Paging Dr. Twitter! Tips and Pitfalls in Online Medical Professionalism</td>
</tr>
<tr>
<td>2:30 p.m.</td>
<td>Morganna Freeman-Keller, DO</td>
</tr>
<tr>
<td>2:30 p.m.</td>
<td>Immediately following: The Council of Resident/Fellow Members and Council of Medical Students will meet (all are welcome)</td>
</tr>
<tr>
<td>2:00 p.m.</td>
<td>Prevention of Medical Errors *** (State Mandated Course)</td>
</tr>
<tr>
<td>2:00 p.m.</td>
<td>Clifford G. Rapp, LHRM &amp; Christopher L. Nuland, Esq</td>
</tr>
<tr>
<td>2:00 p.m.</td>
<td></td>
</tr>
<tr>
<td>4:00 p.m.</td>
<td>Living Well Practicing Medicine ***</td>
</tr>
<tr>
<td>4:00 p.m.</td>
<td>Owen Linder, MD FACP</td>
</tr>
<tr>
<td>4:00 p.m.</td>
<td>Moderator: Michael A. Zimmer, MD FACP</td>
</tr>
<tr>
<td>4:00 p.m.</td>
<td>Panelist: Carmel J. Barrau, MD FACP and Naresh H. Pathak, MD FACP</td>
</tr>
<tr>
<td>5:45 p.m.</td>
<td>Adjourn</td>
</tr>
<tr>
<td>6:00 p.m.</td>
<td>Formal Poster Competition</td>
</tr>
<tr>
<td>7:45 p.m.</td>
<td>Reception and Awards Presentation</td>
</tr>
</tbody>
</table>
Sunday, October 6, 2013  Majestic Palm Ballroom

7:45 a.m.  Registration & Breakfast

8:40 a.m.  Scientific Session
Cuc T. Mai, MD FACP, Scientific Program Chair, Presiding

8:45 a.m.  HIPAA Compliance Made Easy *** *(New Rules Effective Date 9/23/2013)*
Christopher Nuland, Esq

9:45 am  Tales from the Trenches ***
Moderator: Michelle L. Rossi, MD FACP
Panelists: Ankush K. Bansal, MD FACP, Jason M. Goldman, MD FACP,
Michael L. Howell, MD MBA FACP, John F. Rubin, MD FACP

10:30 a.m.  Networking Break with Exhibitors

11:00 a.m.  Doctor's Dilemma™
Florida Hospital Orlando
University of Florida
University of Miami / Jackson Memorial Hospital

12:00 p.m.  Announcements and Adjournment

---

**Doctor’s Dilemma Finalist Teams**

**Florida Hospital Orlando**
Yasmin Abaza, MD
Aditya Chada, MD
Divyanshu Malhotra, MD

**University of Florida**
John Brandt, MD
Islam Elgendy, MD
Vikas Khullar, MD

**University of Miami/Jackson Memorial Hospital**
Rhanderson Nascimento, MD
Maroun Sfeir, MD
Samantha Taghva, MD
2013
Florida Chapter ACP Resident
Formal Poster Judging Presentations
Resident Posters

Broward Health Medical Center Nova Southeastern University
- ALK Negative Anaplastic Large Cell Lymphoma Limited to Bone
  Aaron Heath, DO
- Metaplastic Breast Cancer: A Rare Diagnosis
  Heather Katz, DO
- Rare Infection of Implantable Cardioverter-Defibrillator Lead with Candida Albicans
  Nina Rivera, DO PhD

Cleveland Clinic Florida
- Sarcoid Reaction After Treatment for Diffuse Large B Cell Lymphoma
  Digantkumar Paghdal, MD

Florida Hospital
- Characterizing the Rate of Clostridium Difficile Infection by Different Clinical Practice Models
  Saira Ajmal, MD
- Hands on fire: An unusual presentation of Hypertriglyceridemia.
  Aditya Chada, MD
- Physical Examination: The dying art in modern medicine.
  Divyanshu Malhotra, MD

Florida State University
- Exhausted From Cancer Treatment
  Vikas Patel, MD
- An Unusual Presentation of AIDS
  Vinay Wayal, MD

Largo Medical Center
- Ventriculitis as a Result of Esophageal Perforation: A Case Report and Literature Review
  Tammy Ferro, DO

Mayo Clinic Florida
- Spontaneous Cryptococcal Peritonitis as a Primary Presentation of Disseminated Cryptococcosis
  Alphonso Lopez, MD
- Cardioembolic Stroke as the Initial Presentation of Cardiac Amyloidosis
  Jose Melendez-Rosado, MD
- A Case of Seasonal Allergies, Quadriplegia, and Cardiomyopathy
  David Snipelisky, MD

Mount Sinai Medical Center
- FRAX as a Tool for Osteoporosis Screening in the Outpatient Primary Care Setting
  Hara Rosen Berger, MD
- Milk Alkali Syndrome in Retrospect
  Li Li, MD
- Malassezia Furfur Fungemia: An Underestimated Reality
  Debjit Saha, MD

Northside Hospital
- Tracheal and Right Main Bronchial Stenosis Leading to Almost Complete Right Airway Collapse
  Kelly Kynaston, MD
- Enterococcus Faecalis Meningitis
  Ricarda White, MD
Orlando Health

- Post-traumatic Carotid Cavernous Fistula that Presented as Focal Neurological Deficits with Near Complete Symptoms Resolution after Therapeutic Coil Embolization
  Ekaterina Klevtsova, MD
- Never Say No! Case of Whipple’s Endocarditis with Initial Negative Small Bowel Biopsy
  Tania Lalani, MD
- Quality Improvement – A Humbling Experience Triggering Change in Resident Education
  Caroline Nguyen-Min, MD

University of Florida College of Medicine

- Shoulder Pain And Then Some: An Unusual Presentation Of Sickle Cell Disease
  Ayed Ayed, MD
- Demographics, Clinical Features, Treatments, and Outcomes of Infective Endocarditis Patients with Peripheral Embolization: A Single Tertiary Center Experience Over 15 years
  Islam Elgendy, MD
- Hughes-Stovin syndrome – A Rare Variant of Behçet’s Disease
  Roja Pondicherry-Harish, MD

University of Florida College of Medicine - Jacksonville

- Breast Cancer & Racial Disparity between Caucasian and African American Females: Part-1 (B.R.C.A-1)
  Khurram Tariq, MD
- Anomalous Origin of the Right Coronary Artery from the Left Sinus of Valsalva in a Young Female Presenting with Chest Pain
  Trevanne Matthews Hew, MD
- Unusual Cause of Pneumonia in Near Drowning Victim
  Jean Touchan, MD

University of Miami Jackson Memorial Hospital

- Recurrent angioedema in a patient with normal levels of C4, C1 inhibitor and lupus nephritis
  Bernice Acevedo, MD
- A Severe Case of Cocaine Induced Leukocytoclastic Vasculitis
  Zeina Hannoush, MD
- Paget-Schroetter Syndrome: An Unexpected Addition to a Circus Acrobat’s New Act
  Archana Ramireddy, MD

University of Miami Regional Campus

- Extramammary Paget Diseases of the Scrotum as an Important Clue to Diagnose Invasive Prostate Cancer
  Joao Braghiroli, MD
- A Case Report of IgG-4 Related Disease
  Aaron Garza, MD
- A Startling Starfield Pattern
  Megha Rao, MD

University of South Florida

- IgA Nephropathy in a Cirrhotic Patient Successfully Treated with Plasmapheresis
  Andrew Kuykendall, MD
Peripheral T cell lymphomas (PTCL) are an uncommon heterogeneous group of aggressive neoplasms within the non-Hodgkin lymphomas (NHL). They constitute less than 15 percent of all NHL in adults. ALK negative anaplastic large cell lymphoma (ALCL) is a rare subtype of PTCL accounting for only about 6% of cases. Most patients with PTCL present with generalized lymphadenopathy with or without extranodal involvement. Extranodal disease limited to bone with no lymph node involvement is exceedingly rare. We present a case of ALK negative ACLC with diffuse metastatic disease throughout the thoracolumbar spine with no lymphadenopathy or other organ involvement.

A 42 year old previously healthy African American female presented complaining of 2 months of worsening lower back pain. She had no significant past medical, surgical, or family history and was a lifelong non-smoker. Physical examination revealed no lymphadenopathy, masses or organomegaly. In the emergency room a CT scan of the abdomen and pelvis showed sclerotic metastatic disease throughout the spine and pelvis. A subsequent CT scan of the chest and lumbar spine showed diffuse sclerotic osseous metastasis throughout thoracic and lumbar spine. A thoracolumbar MRI revealed multilevel metastatic disease with a pathologic fracture at T7 for which the patient subsequently underwent kyphoplasty. Thoracic vertebral body biopsy revealed a poorly differentiated malignant neoplasm of unknown origin, with findings suggestive of a high-grade lymphoma. Previous laboratory workup revealed elevated tumor markers including CA 27-29, CA 15-3 and CA-125. The patient underwent a full workup to search for a primary malignancy. Initially this was thought to be a very unusual presentation for a hematologic malignancy given the lack of lymphadenopathy, and metastatic breast cancer was highest on the differential. A PET-CT scan revealed multiple areas of abnormalities and bony metastatic disease with no adenopathy. A repeat bone biopsy revealed a CD 20 negative ALK negative anaplastic large cell lymphoma. The patient was started on CHOP (Cyclophosphamide, Hydroxydaunomycin, Oncovin, Prednisone) and after 3 cycles a repeat PET-CT scan showed improvement in lytic and sclerotic lesions in the thoracolumbar spine. Additionally the patient’s lower back pain had resolved and tumor markers were trending down.

ALK negative ALCL is a rare subtype of PTCL. The prognosis of PTCL remains poor in comparison to B cell NHL due to lower response rates to standard combination chemotherapy. ALK negative ALCL tend to have a poorer prognosis compared with ALK positive ACLC. ALCL is a lymphoma of T cell or null cell type that typically presents with painless lymphadenopathy with or without systemic symptoms. This case represents a rare, unusual and aggressive case of ALK negative ALCL with the only site of disease being the bone with no lymphadenopathy or organomegaly.
In the United States, breast cancer is the most commonly diagnosed cancer and the second most common cause of cancer death in women. Metaplastic breast carcinoma, a neoplasm comprised of both epithelial and mesenchymal elements, is an extremely rare type of breast cancer representing less than 1 percent of all breast cancers. This paper serves to highlight a rare case of metaplastic breast carcinoma presenting as an axillary mass in a 45-year-old woman with a history of well-differentiated ER/PR positive, HER2 negative breast cancer.

A 45-year-old female with a past medical history of well differentiated ER/PR positive, HER2 negative right breast cancer status post right segmental mastectomy with sentinel node lymphadenectomy, tamoxifen therapy and radiation to the right breast presented with a new palpable mass in the right axilla. The patient denies any chest wall or breast tenderness, skin changes of the breast, axillary tenderness, fevers, chills, fatigue or weight loss. The patient admits to close follow up with her oncologist and surgeon as well as continuous breast exams every three months and annual mammograms for past three years since her initial breast cancer diagnosis.

Physical exam revealed stable vital signs, and a benign exam except for a palpable 2 cm right axillary mass. Laboratory data was unremarkable. Magnetic resonance imaging confirmed a 3.3 cm low right axially mass that invaded the pectoralis muscle.

An ultrasound-guided biopsy was performed and pathology revealed a malignant spindle cell tumor. The patient underwent bilateral nipple sparing mastectomies with immediate reconstruction. Final pathology revealed a normal left breast and a 3.5 x 15 x1.5 cm high grade, triple negative metaplastic spindle cell tumor in the axillary tail of the right breast. The margins were clean and seven lymph nodes were negative.

PET CT showed abnormal uptake in the right axillary region. Adjuvant chemotherapy using Doxorubicin and Cyclophosphamide was initiated after a baseline echo revealed an ejection fraction of 56%.

This case serves to illustrate a rare case of metaplastic breast carcinoma treated with an adjuvant chemotherapy regimen of Doxorubicin and Cyclophosphamide. There are no specific treatment guidelines for metaplastic breast cancer because of its low incidence and variable pathology. Studies on the molecular pathology of mataplastic carcinoma demonstrate EGFR gene amplification that could lead to targeted treatment. However, further research is needed regarding signaling pathways to gain a better understanding about these tumors.
Implanted cardiac devices, specifically pacemakers and implantable cardioverter-defibrillators (ICDs), have a relatively low rate of becoming infected, from 0.8 to 5.7 percent. Fungal infections of such devices represent an atypical phenomenon. They are typically associated with a high mortality. Both medical and surgical therapies are recommended for a successful outcome.

A 60-year-old female with past medical history significant for sarcoidosis, diabetes mellitus and chronic kidney disease presented with subjective fevers, chills, sweats, and productive cough for 1-2 weeks, followed by 3-4 days of atypical, pleuritic chest pain. Two years and two months prior to symptom onset, she underwent implantation of a single-chamber ICD due to the patient’s severe cardiac history including congestive heart failure with reduced ejection fraction less than 30% with class III symptoms, and episodes of non-sustained ventricular tachycardia.

Blood cultures were obtained immediately upon admission. Initially, a transthoracic echocardiogram (TTE) was performed, which found an improved ejection fraction of greater than 55% along with a highly mobile 2.09-cm by 4.49-cm mass associated with the ICD wire. By the completion of the TTE, blood cultures were found to be positive for Candida albicans. In light of the vegetation size, the patient’s septic state, the multiple comorbidities, and high-risk nature of the infectious agent, open heart surgery for direct extraction of the lead and vegetation was selected.

The patient underwent sternotomy with cardiopulmonary bypass and intraventricular intracardiac exploration for removal of the infected ICD and removal of the ICD generator with revision of the ICD generator pocket along an intraoperative TEE. The vegetation was attached to the right ventricle without interference with the tricuspid valve. It measured 4-cm by 2-cm by 2-cm and a fibrous capsule was found to extend up and over the ICD lead measuring 4-cm by 3-cm by 1-cm. Pathology and culture confirmed Candida albicans. She was treated with a combination of micafungin and fluconazole for 16 days.

In this case report, we describe the rare infection of an ICD lead with Candida albicans, in the form of a fungal ball. This is one of less than twenty reported cases of Candida device-related endocarditis. The majority occurs in relation to pacemaker wires, rather than ICD leads, as presented here. This is the first Candida device-related endocarditis reported in a female. This is also one of the largest vegetation that has been reported, measuring 4-cm at its greatest length. As Candida device-related endocarditis is so rare, and as fatality occurs in half, clinical management can only be derived from sporadic case reports. Therefore, the course of this patient’s disease and care will be a useful adjunct to the current literature for determining treatment and prognosis in similar cases.
SARCOID REACTION AFTER TREATMENT FOR DIFFUSE LARGE B CELL LYMPHOMA

Digantkumar Paghdal, MD; Faria Nasim, MD; Franck Rahaghi, MD.
Cleveland Clinic Florida

Introduction:
Sarcoid reaction is the occurrence of localized epitheloid granuloma with out signs of systemic sarcoidosis. Sarcoidosis may develop with malignancy. Similarly, sarcoid reaction is also known to be associated with various malignancies especially lymphomas.

Case description:
We present the case of a 58 year old gentleman who developed sarcoid reaction after successful treatment of diffuse large B cell lymphoma with chemotherapy. The patient was diagnosed with diffuse large B cell lymphoma (DLBCL) by excisional biopsy of a cervical lymph node. He had presented with a right neck mass. He underwent six cycles of conventional chemotherapy regimen CHOP- R (Cyclophosphamide, Doxorubicin, Vincristine, Prednisone and Rituximab) over four months. Complete radiological remission was noted on PET scan by oncology. Eight months later, surveillance PET scan came back positive for new perihilar and periportal lymph node enlargement suspicious for relapse of lymphoma. Laparoscopic biopsy of periportal lymph nodes was performed. Pathological review of biopsy specimen showed non-caseating granulomatosis with no evidence of lymphoma. Cultures including fungal cultures came back negative. Patient was asymptomatic. He had no systemic manifestations of sarcoidosis such as skin, eye, or lung lesions. His serum γ-globulin, calcium and ACE level were within normal limits. Diagnosis of sarcoid reaction after treatment of DLBCL was made. Patient is doing well and has been malignancy free for three years.

Discussion:
Differential diagnosis of localized lymphadenopathy after treatment of non Hodgkin’s Lymphoma should include recurrence of malignancy, concurrent sarcoidosis, fungal infection or sarcoid reaction. In the past, a sarcoid reaction has been reported in 7.3% of cases of non-Hodgkin lymphomas. DLBCL is the most common type of Non Hodgkin lymphoma in adults. We report the first case report of sarcoid reaction after treatment of DLBCL. Definitive diagnosis of post treatment lymphadenopathy should be made by tissue biopsy in order to choose correct clinical approach and to avoid unnecessary chemotherapy.
CHARACTERIZING THE RATE OF CLOSTRIDIUM DIFFICILE INFECTION BY DIFFERENT CLINICAL PRACTICE MODELS

Saira Ajmal, MD; Vincent Hsu, MD; John Wong, RN; Victor Herrera, MD
Florida Hospital Orlando

Introduction: Clostridium difficile infection (CDI) is a growing concern in this country, causing an estimated 20,000 deaths annually. CDI is one of the most dangerous healthcare associated infections (HAI), even replacing Methicillin Resistant Staphylococcus Aureus (MRSA) as the number one HAI. There is substantial interest in understanding factors associated with the occurrence and prevention of CDI; the association with practice model has been of particular interest. Varying data has been reported. A study in Quebec showed that academic institutions were associated with less CDI than non-academic hospitals. This was contrasted by a longitudinal-study in USA that showed the opposite. Other studies have reported that academic status has no significant impact on CDI. At our hospital we had the unique opportunity to study this relationship, as both teaching and non-teaching practices run side-by-side with the same patient population, staff and resources. By reducing confounders, if differences are identified, investigating these could help develop recommendations to reduce the incidence of CDI. Method: A retrospective study was performed using data compiled by Florida Hospital Infection Prevention from January 2011 through July 2012. Cases of Healthcare-Associated CDI on internal medicine services were identified, and classified by practice model. 6 academic physicians with 5380 total admissions, and 71 private physicians with 75196 total admissions were identified. The rate of CDI for the groups was compared with an unpaired 2-sample t-test and chi-squared test. Results: Data for CDI rates was compared using a chi-squared test, with our outcomes being number of CDI cases and number of admissions without CDI. Statistical analysis of this data (24 cases/5380 admissions for academic and 259 cases/75196 admissions for private) revealed a p value of 0.27, indicating no statistically significant difference. We subsequently compared the mean number of CDI cases amongst the 2 groups (4 academic vs. 3.65 private) using an unpaired two-tail t-test. The 2-tailed p-value was 0.84, confirming no statistically significant difference. Conclusion: The results revealed that at our hospital there is no statistically significant difference in the rate of CDI amongst the 2 common practice models; academic and private. There were several important issues raised during analysis; the most evident being the difference in the sample sizes. Given the lack of consensus, further studies are required to characterize this relationship and avoid the alarming trend of this HAI. Design must address reducing confounders, and involving a sample-size with sufficient power to make appropriate conclusions.
HANDS ON FIRE: AN UNUSUAL PRESENTATION OF HYPERTRIGLYCERIDEMIA

Aditya Chada, MD; Joshua Trabin MD ; Divyanshu Malhotra MD
Florida Hospital

The hypertriglyceridemia syndromes typically present clinically with acute pancreatitis, eruptive xanthomas, lipemia retinalis and hepatosplenomegaly. However, our case describes an unusual presentation of hypertriglyceridemia with postprandial erythromelalgia, or redness and burning sensation of the hands.

A 42 y/o Caucasian male presented with complains of redness and burning sensation of the hands. He described the sensation as “hands catching on fire” within a few minutes after having a heavy meal. The sensation persisted for a few hours and disappeared spontaneously. His medical history included diabetes mellitus, obesity, two episodes of spontaneous acute pancreatitis and three episodes of uric acid renal colic. On physical examination there was obvious erythema of both hands that extended proximally to wrists. His BMP and CBC were normal but his fasting lipid panel revealed high level of triglycerides(TG) of 3787, high density lipoproteins (HDL) of 29, low density lipoproteins (LDL) of <5 and very low density lipoproteins (VLDL) of 277. His symptoms resolved on administration of intravenous fluids. He was started on Fibrates and Omega fatty acids and his symptoms improved there after.

Hypertriglyceridemia is biochemical diagnosis based on fasting plasma triglyceridemia levels. Fredrickson classified hypertriglyceridemias into six types based on the quantitative and qualitative differences in the levels of lipoproteins. Human genome studies have identified 32 single nucleotide polymorphisms that raise plasma TG levels. The level of triglyceridemia correlates with the sum of these alleles together which is called Genetic Risk Score. Secondary risk factors like obesity, metabolic syndrome, pregnancy, Systemic lupus erythematosis (SLE) can cause genetically susceptible individuals to present with clinical symptoms. This is a case of Type V hyperlipoproteinemia characterized by very high levels of chylomicrons, triglyceride and VLDL with low levels of LDL and HDL. TG levels are often higher than 1000 mg/dL. Clinically it can manifest as multiple episodes of acute pancreatitis, hyperuricemia with urolithiasis, eruptive xanthomas, and rarely as erythromelalgia due to hyper viscosity as in our patient. For the last few decades hypertriglyceridemia has not been considered to be a risk factor for cardiovascular disease (CVD) mainly because chylomicrons carrying tryglycerides are too large to enter endothelial cells. However, recent studies have shown that hypertriglyceridemia may be a risk factor for CVD by causing oxidative stress, inflammation and endothelial damage. The standard treatments of the hypertriglyceridemia syndromes are Fibrate medications and high-dose omega fatty acids. Statins alone are generally ineffective in reducing the TG levels. In acute life threatening conditions, plasmapheresis can also be considered.

This case illustrates that classic erythromelalagia, which is usually assosciated with myeloproloferative disorders, may be indicative of extreme hypertriglyceridemia. Physicians should be alert to this possibility when symptoms of erythromelalagia appear. This problem may become frequent given the rapid development of obesity and metabolic syndrome in our society.
For centuries, doctors diagnosed illness using their own senses, by poking, looking, listening. From these observations, a skilled doctor can make amazingly accurate inferences about what ails the patient. One recent study in the Journal of the American Medical Association examined stethoscope skills of various kinds among 453 practicing physicians and 88 medical students. Whatever their age or experience, the doctors correctly recognized only 20 percent of heart problems. We present a case highlighting the importance of physical examination. A 42 year male was brought to the emergency department after involving in a motor vehicle accident. Patient was hit from the side while driving his car. No external visible injuries were reported. In the ED patient was Alert but not oriented to time, place or person. He complained of neck pain worse on movement. On physical examination he had no external injuries, lungs and heart sounds were unremarkable for any abnormal findings. Central nervous system examination was remarkable for retrograde and antegrade amnesia, poor attention, poor calculations. Motor exam revealed 5/5 power on the right but 2/5 power on the left upper and lower limbs. His sensory system was abnormal with loss of pain and temperature on the right and vibration on the left. MRI revealed a herniated disc impinging on to the left side of Spinal cord. A diagnosis of Brown-Sequard was made. The Brown-Séquard syndrome is characterized by ipsilateral loss of motor function, vibration, and proprioception below the level of the lesion with contralateral loss of pain and temperature sensation beginning approximately two levels below the lesion. This pattern of deficits occurs because the spinothalamic tract travels on the ipsilateral side of the cord before crossing over to the contralateral side. Our patient had similar presentation but there was something strange in the pattern of sensory loss. There was not only loss of pain and temperature on the opposite side of lesion but also on the same side below the lumbar region. This prompted us to repeat an MRI of Lumbar Spine which revealed a herniated disc protruding on to the opposite side but to a lesser extent compared to the one above. Neurosurgery was immediately consulted who repaired both the defects. Physical examination is a primitive ritual which is losing its charm in modern day medicine. The patient in the bed has just become an icon to the patient in the computer. A thorough physical examination not only saves millions of dollars but also increases HCAPS scores as patient is often pleased by thorough examination. It is time to move medical rounds from closed rooms in front of 2-dimensional pictures to around the beds of real life 3-dimensional patient.
Chemotherapy with bleomycin is the standard of care for testicular cancer but may lead to significant lung toxicity with rapid deterioration. A 22 year old Caucasian male college student with a history of testicular cancer status post right orchiectomy and chemotherapy, marijuana use and cocaine inhalation, developed acute respiratory distress that woke him up from sleep. Six weeks earlier, he had less severe symptoms and a chest CT demonstrated patchy areas of ground-glass opacities superimposed on areas of consolidation especially at the bases. There were no pleural effusions, pericardial effusions, pneumothoraces or pneumomediastinum seen and corticosteroid treatment was initiated for suspected bleomycin-induced pneumonitis. In a subsequent chest CT, 3 weeks later, the patchy areas of consolidation were improved but there was now prominent pneumomediastinum, small pneumothoraces bilaterally, and extensive chest wall and neck soft tissue emphysema. He had visited a doctor at his student health clinic and a pulmonologist before admission for worsening dyspnea but declined hospitalization. In the emergency department, the patient was in acute respiratory failure with a respiratory rate of 48 breaths per minute and oxygen saturation of 38%. A chest x-ray revealed 20% right pneumothorax, pneumomediastinum, and subcutaneous emphysema. A right-sided chest tube was placed and breathing support was initiated with high-flow oxygen with 50L/min by nasal cannula that was titrated to 86-91% oxygen saturation to avoid worsening of bleomycin-induced lung injury. The patient later had worsening dyspnea with oxygen desaturation and had to be intubated with vent settings of FiO2 65%, PEEP 4cm H2O, Respiratory rate 21 breaths per minute, and Tidal volume 480mL. Nitric oxide support was initiated in an effort to lower the FiO2. Left lower lobe pneumonia was suspected and bronchoscopy with bronchoalveolar lavage did not reveal pneumocystis jirovecii infection. His condition continued to decline and he went into acute respiratory distress syndrome with a PaO2/FiO2 ratio of 91. He was later transferred to a facility capable of providing extra corporeal membrane oxygenation (ECMO) but he expired before ECMO was initiated. This case illustrates the severity of bleomycin-induced lung toxicity, particularly in the presence of pneumothorax and pneumomediastinum, and how quickly a patient’s respiratory status can decline even in the presence of adequate supportive treatment. The incidence of pneumothorax and pneumomediastinum in the presence of bleomycin-induced lung disease is very rare and only 4 such cases have been reported in the literature. Awareness should be increased so that early treatment can be initiated.
AN UNUSUAL PRESENTATION OF AIDS

Vinay Wayal, MD; Gregory Todd MD; Raymond Shashaty, MD; Vikas Pate, MD
Florida State University

HIV-infected patients with a CD4 count < 50 cells/microL are at risk for cryptococcal meningitis, which is uniformly fatal if untreated. Predictors of poor outcome include high cerebrospinal fluid cryptococcal antigen levels (titer > 1:1024), low body weight, and poor mental status on presentation. Management of these patients includes potentially toxic antifungals with careful monitoring for complications related to invasive fungal infection and inflammatory syndromes secondary to immune recovery. A 44-year-old male with no significant past medical history was admitted for new onset seizure and altered mental status. The patient’s girlfriend stated he had severe headaches for one week and was recently treated for sinusitis. Upon further questioning, she finally remembered that he was having chills and recalled his temperature was 102 degrees Fahrenheit two days earlier. He was started on empiric antibiotics, dexamethasone and seizure prophylaxis. An LP was significant for an opening pressure of 550mm/h20, protein 23.9, glucose 48, no WBCs, and culture growing yeast. The cryptococcal antigen was positive with titers > 1:1024, blood cultures positive for yeast, and a leukocytosis of 14.5. He was started on amphotericin and flucytosine. His HIV test was positive with a CD4 count of 50 cells/microL and started on prophylaxis both for Pneumocystis carinii pneumonia and Mycobacterium avium-intracellulare complex. Serial lumbar punctures relieved the intracranial pressure and decreased the opening pressure below 15, significantly relieving the patient’s headaches. Finally, after several weeks of treatment, the third set of blood cultures were negative for yeast but thrombocytopenia and acute kidney injury forced discontinuation of flucytosine, which was replaced with fluconazole and significantly improved the platelet count. The patient later told us that he had unprotected sex with 30-40 female partners over the past 5 years. The patient clinically improved throughout his hospital stay and was discharged home with fluconazole, HAART therapy, Pneumocystis carinii pneumonia and Mycobacterium avium-intracellulare complex prophylaxis. Upon follow-up he was in good spirits and grateful. This was a rare presentation of newly diagnosed AIDS in a community setting. A thorough history from family members and or significant others with detailed questioning can be the key to saving a patient’s life. Treating cryptococcal meningitis with complications of fungemia and a low CD4 count with abnormally high opening pressures can be very challenging. The three phases of treatment: induction, consolidation, and maintenance must be initiated and patients must be monitored closely for potentially harmful side effects. Prophylaxis for opportunistic infections must be started. Starting HAART therapy requires close monitoring for immune reconstitution inflammatory syndrome especially with a CD4 count < 50 cells/microL and an underlying infection.
VENTRICULITIS AS A RESULT OF ESOPHAGEAL PERFORATION: A CASE REPORT AND LITERATURE REVIEW

Tammy Ferro, DO
Largo Medical Center

INTRODUCTION:
Instrumentation injury of the esophagus alone account for 57-90% of all causes of esophageal injuries. Those resulting from dilation of malignant strictures are the most common cause of all reported cases among adult patients. Mortality as a result of esophageal perforation after dilation can be as high as 50%. Central nervous system (CNS) infection as a result of esophageal perforation is rare. There have been five adult cases of esophageal perforations resulting in CNS complications in the literature since the 1960’s.

CASE PRESENTATION:
I present a 72 year old female patient with history of coronary artery bypass grafting, essential hypertension, breast cancer with chest radiation and gastroesophageal reflux, who played tennis weekly. She underwent weekly esophageal dilations for a malignant stricture as a result of radiation for breast cancer treatment. The patient developed symptoms of pneumonia 2 weeks subsequent to this and was treated as so. Her final dilation 1 week after her pneumonia subsided demonstrated 2 mucosal defects proximal to the stricture. A CT-barium swallow revealed contrast in the trachea and mediastinum. Four days into her hospitalization she developed fevers, seizures and became unresponsive requiring mechanical ventilation. Multiple imaging modalities revealed vertebral osteomyelitis with compression fracture, ventriculitis, intraventricular abscess and hydrocephalus. Ultimately, she required craniotomies, ventriculostomies, a hemilaminectomy and ventricular drains. Her hospitalization course was complicated with nosocomial infections, persistent comatose state, pulmonary effusions and the need for tracheostomy for persistent mechanical ventilation needs. Her mental status declined to a baseline that was described as depressed with severe cognitive impairment. She required inpatient rehabilitation center for several months during which time her condition improved to where she was able to complete tasks of daily living with only moderate assistance. The patient was released home from rehabilitation 6 months after the inciting esophageal dilation.

DISCUSSION:
This case report brings forth a very rare case of CNS infection in a patient who underwent esophageal dilation for a malignant stricture. The recommended evaluation following a plain roentgenogram is an esophogram utilizing barium as it offers superior density and reveals small perforations better than gastrograffin but at the risk of necrotizing pneumonitis. Dilating a stricture antegrade with a bougienage seems to be the high-risk mechanism for which the perforation can result in. The venous drainage underlying the intimate anatomy the CNS and the esophagus shares is the main conduit for spread of the infection. Options for surgical repair of esophageal perforations include minimally invasive thoracoscopy, open thoracotomy that divert, directly repair or patch the damaged area. More recent literature in the last 10 year support successful conservative nonsurgical observation techniques including, frequent imaging, alternative methods of enteric feeding, and antimicrobial administration. Overall, iatrogenic perforations portend better prognosis than those that occur spontaneously.
SPONTANEOUS CRYPTOCOCCAL PERITONITIS AS A PRIMARY PRESENTATION OF DISSEMINATED CRYPTOCOCCOSIS

Alfonso S Lopez, MD; David Snipelisky, MD; Dooshanveer Nuckchady, MD; Ricardo Pagan, MD; Lisa Brumbl, MD
Mayo Clinic Florida

Introduction:
Cryptococcal peritonitis is an exceedingly rare entity with very high morbidity and mortality. This case describes an immunocompetent cirrhotic patient with cryptococcal peritonitis as a first manifestation of infection.

Case:
A 72-year-old male with past medical history of end stage liver disease secondary to non-alcoholic steatohepatitis undergoing weekly therapeutic paracentesis and end stage renal disease on hemodialysis was admitted to our service directly from the clinic after a culture showed cryptococcal species in his last ascitic fluid analysis.

On examination, the patient was afebrile and in no acute distress. He admitted to have increased lethargy over the past several days and mild abdominal pain. Pertinent laboratory analysis included a complete blood count and metabolic panel within normal limits. The patient’s model for end-stage liver disease score was 20. He was initially started on liposomal amphotericin B, with addition of oral flucytosine. Further laboratory evaluation found a serum cryptococcal antigen measured by indirect enzyme immunoassay of 1:256. Cerebrospinal fluid analysis found a protein level of 45 mg/dL, 15 erythrocytes/μL, and 18 leukocytes/μL. Human immunodeficiency virus testing was negative. Further conversation with the patient revealed that he had been exposed to dust from construction work in his neighborhood within the preceding weeks. Continued treatment with amphotericin B and flucytosine resulted in no deterioration in his condition and the patient will continue with consolidation therapy as an outpatient.

Conclusion:
In patients with systemic symptoms and recurrent ascitic fluid reaccumulation, cryptococcal peritonitis, although rare, should be considered. With an excessive mortality rate, our case illustrates that early diagnosis and treatment can result in resolution of the infectious process.
CARDIOEMBOLIC STROKE AS THE INITIAL PRESENTATION OF CARDIAC AMYLOIDOSIS

Jose Melendez-Rosado, MD; David Snipelisky, MD; Joseph Blackshear, MD; Juan Leoni, MD
Mayo Clinic Florida

Introduction:
Amyloid cardiomyopathy is a disease of misfolded proteins that infiltrate different tissues causing organ dysfunction. The most common type, AL amyloidosis, affects 6 to 10 cases per million people. Cardiac involvement can include atrial mechanical standstill, but to our knowledge no case has been reported with cardiac amyloidosis presenting as a cardiogenic embolic stroke in a healthy young person.

Case Presentation:
A 48 year old African-American personal trainer with no significant past medical history presented to an outside emergency department with slurred speech, left sided weakness, and left facial palsy. Head CT scan demonstrate right middle cerebral artery hyperdensity consistent with an acute stroke. The patient was transferred to our institution for further evaluation. At arrival, NIH stroke score was 13 and Glasgow coma scale was 15. The patient denied any chest pain, palpitation, fever or chills prior to his symptoms but did admit to shortness of breath and productive cough post exertion for two months. Endovascular right middle cerebral artery mechanical thrombectomy retrieved thrombus, and with subsequent intravascular tPA administration progressive improvement of neurological symptoms was noted. Troponin elevation to 0.64 ng/mL was found, along with a brain natriuretic peptide level of 643 pg/mL (normal for age .38 pg/ml) and sinus rhythm EKG with low voltage QRS in the limb leads. Trans-esophageal echocardiography showed an ejection fraction of 25%, global left ventricular hypokinesis along with concentric left ventricular hypertrophy, left atrial appendage spontaneous echo contrast and thrombosis in the left atrial appendage with severely diminished flow velocities despite the presence of sinus rhythm. Cardiac MRI was performed for the suspicion of cardiac amyloid and found abnormal delayed enhancement in the myocardium, consistent with an infiltrative process. Further diagnostic studies included, serum protein electrophoresis showing an M spike and kappa/lambda free light chain ratio was consistent with a monoclonal gammopathy. Bone marrow biopsy demonstrated atypical plasmacytosis with 30 to 40% of marrow involvement of plasma cells and focal amyloidosis. Fat aspirate was positive for congo red stain and typing was consistent with AL (lambda light chain) type cardiac amyloidosis. No hypercalcemia, renal failure, pathological fractures or bone lytic lesions were detected. The patient was discharge on appropriate heart failure medications, anticoagulation and is on AL amyloid chemotherapy.

Conclusion:
This case illustrates a pathologic consequence of stasis due to atrial mechanical standstill. Cardiac amyloidosis, although rare, should be in the differential diagnosis of any young patient with no history of cardiovascular disease or arrhythmias that presents with an acute stroke.
Decongestant ephedrine products are widely used. Common side effects include palpitations, nervousness, and headache. More severe adverse reactions include cardiomyopathy and vasospasm. We report a case of an otherwise healthy 38-year old woman who presented to our institution with acute onset quadriplegia and heart failure. She had an unremarkable chest radiograph on admission, but developed marked pulmonary edema and bilateral effusions the following day. Echocardiogram showed an ejection fraction of the left ventricle (LVEF) of 18% and no obvious intrinsic spinal pathology or foramen narrowing on spinal imaging. Laboratory screening was positive for metamphetamines in the urine and the patient admitted to using multiple ephedrine-containing products for allergy symptom relief over the past several weeks. She was ultimately diagnosed with an acute catecholamine-induced cardiomyopathy and spinal artery vasospasm due to excessive use of decongestants. Her symptoms resolved completely with supportive care and appropriate heart failure management. Repeat echocardiogram two weeks later showed improvement of the LVEF to 33%. Our case illustrates an unusual combination of side effects of ephedrine products presenting as acute quadriplegia and severe cardiomyopathy.
FRAX AS A TOOL FOR OSTEOPOROSIS SCREENING IN THE OUTPATIENT PRIMARY CARE SETTING

Hara Berger, MD; Violet Lagari-Libhaber, DO; Mathew Farbman, DO; Elizabeth Kury, MD
Mount Sinai Medical Center

Introduction:
Osteoporosis related fractures cause significant morbidity and mortality. Recently, FRAX (fracture risk assessment tool) was created to calculate the 10-year probability of hip fractures and major osteoporotic-related fractures in patients with osteopenia (T-score of -1 to < -2.5). The objective of this quality assurance study is to identify patients at high risk for fracture using FRAX with or without a screening dual x-ray absorptiometry (DXA) in an outpatient primary care clinic.

Methods:
This study is a retrospective chart review using our electronic medical records of primary care visits from January- December 2012 that included patients from the outpatient primary care clinic at Mount Sinai Medical Center, Miami Beach, FL. Descriptive statistics were used to characterize the study population and their clinical risk factors. For each patient, the FRAX score was calculated with DXA included (if available) and also without DXA.

Results:
A total of 132 patients were included (106 women, ages 65-90, and 24 men, ages 70-90). Seven patients were excluded for age > 90. Characteristics of the study population include: average age 75 yr + 6.8, weight 155 lb. + 37.0, height 70.8 in + 8.9, vitamin D levels 31.4 mg/dl +17.7 (79 patients), calcium levels 9.05 mg/dl +.48 (126 patients). There were 28 Caucasian, 85 Hispanic, and 14 African Americans patients. 48% (52/109) were on calcium supplementation. 10% of patients had previous fracture history, and 7.7% had parental fracture history. 31% (4/13) of patients with a previous fracture history were on bisphosphonate treatment. FRAX major osteoporotic fracture mean risk was 10.6 +7.1%, FRAX hip fracture risk was 3.9+5.5%, FRAX major osteoporotic risk with BMD excluded was 10.8+6.4%, and FRAX hip fracture risk with BMD excluded was 4.4+4.9%. 57% (4/7) of patients who had osteopenia based on DXA T-scores and had high FRAX scores (major risk of fracture ≥ 20% or hip fracture ≥ 3% in the next ten years) were not on anti-osteoporosis therapy. 41.3% (19/46) of patients who did not have screening DXAs were identified as having high FRAX scores.

Conclusion:
The population of this outpatient clinic is characteristic of the South Florida area. This study identified a significant number of patients with osteopenia by DXA and high FRAX who were not receiving treatment with an anti-osteoporosis therapy. When FRAX was used alone (in patients without DXA), a number of patients were identified who should be considered for anti-osteoporosis therapies. In our outpatient clinic, this study increased awareness of the need for increased screening with tools like FRAX and DXA. Our goal is to ultimately reduce fracture risk in high-risk patients through the initiation of appropriate anti-osteoporosis therapies.
MILK ALKALI SYNDROME IN RETROSPECT

Li Li, MD
Mount Sinai Medical Center

Milk alkali syndrome is a fairly uncommon cause of hypercalcemia, characterized by hypercalcemia, renal insufficiency, and metabolic alkalosis. Diagnosis is largely based on a patient history of excessive calcium and absorbable alkali intake.

An 80 year old white female presents to the Emergency Department (ED) with progressive generalized weakness for 3 days duration and altered mental status (AMS). Her husband reported that she suffered from a 2 year history of short term memory impairment aggravated by a significant decline in cognitive function over previous several days. Also notes over past 3 days, the patient had become more inactive, progressing from difficulty rising from a chair, to difficulty standing without support. She has history significant for osteoporosis treated with daily calcium and vitamin D supplements as well as hypertension treated with amlodipine. The both denied use of any other medications, including over the counter medications, or herbal remedies. Physical exam was notable for impaired short term memory, orientation to person, place, but not time, and 4/5 strength throughout. Initial laboratory results showed significant hypercalcemia with serum calcium of 15.4 mg/dl as well renal insufficiency. Further workup revealed normal intact parathyroid hormone (iPTH), parathyroid hormone related protein (PTHrp) and Vitamin D levels. Serum and urine immunofixations were normal. Image study did not find any mass lesion in the lung and brain. Patient’s hypercalcemia, renal insufficiency, and altered mental status resolved after vigorous intravenous hydration and treatment with calcitonin. Upon further questioning, and as her mental status improved, she gave a history of ingesting large quantities of Tums for dyspepsia, likely exacerbated by short-term memory impairment.

This case illustrates the potential etiology of severe hypercalcemia due to excessive ingestion of Tums. The importance and difficulty of elucidating an adequate history in patients with AMS warrants a high index of suspicion of milk alkali syndrome when a patient presents with severe hypercalcemia.
Malassezia species are lipophilic yeasts that require medium-chain fatty acids to grow and are part of the normal skin flora. These organisms most commonly cause a superficial mycosis known as Pytiriasis versicolor and seldom, they cause disseminated disease. With recent development of lipid-based total parenteral nutrition (TPN), there have been an increasing number of cases of Malassezia furfur fungemia, unraveling a new and interesting spectrum of nutrition-related and catheter-related infections. A 61-year old man was referred from his Gastroenterologist’s office for chronic intermittent fever. He had a history of chronic intestinal pseudo-obstruction secondary to gastrointestinal dysmotility. He underwent multiple abdominal surgeries due to small bowel obstructions (SBO), eventually requiring a percutaneous endoscopic gastrostomy (PEG) tube placement. However, due to chronic SBO, his PEG tube was unused, requiring lipid-based TPN, through a left subclavian port, over the previous 14 months. During the first of his multiple admissions, he was found to have Staphylococcus epidermidis bacteremia, which was treated accordingly and his PEG tube and port were replaced. During subsequent admissions, his blood and urine cultures were consistently negative, requiring no further treatment. Upon his most recent admission, febrile, tachycardic and borderline hypotensive. There were no signs of infection at the PEG tube or port sites and physical exam was unremarkable. Urine and two sets of blood cultures were ordered along with complete blood and metabolic panels. Laboratory studies showed leukopenia and thrombocytopenia. Trans-Esophageal echocardiogram was negative for valvular vegetations. Peripheral blood smear showed intracellular and extracellular budding yeasts and the final blood cultures with lipid supplementation grew Malassezia species. The patient was treated with Amphotericin B for two weeks, became afebrile on his second day of antifungal treatment and his complete blood count improved. After a port replacement, he was discharged in good condition. Blood-borne infections are usually caused by skin flora and are often related to intravenous catheters; which are sites of colonization and/or the port of entry. Lipid-based parenteral nutrition could support the growth of certain lipophilic fungi, like Malassezia furfur. Unfortunately, standard culture plates, used to identify common blood-borne pathogens, are not supplemented with enough amounts of lipids in order to grow these fungi, which suggest that this condition may be currently underestimated and even misdiagnosed. This case illustrates the importance of recognizing the risk factors for this disease and the use of pursuing lipid-based cultures when this etiology is suspected, as well as cultures from the nutritional solutions, which would be critical to prevent intravascular infections from this unusual organism.
TRACHEAL AND RIGHT MAIN BRONCHIAL STENOSIS LEADING TO ALMOST COMPLETE RIGHT AIRWAY COLLAPSE

Kelly Kynaston, MD; Mohamed Ali, MD FCCP
Northside Hospital

Case Description:
A 65-year-old Bosnian female, recently diagnosed with narrowing of the right main bronchus via CT scan at her outpatient pulmonology office, presented with difficulty breathing and stridor. The patient was a lifetime never smoker, though she was exposed to secondhand smoke from her husband; she carried a diagnosis of COPD and had undergone several months of steroid therapy as high as 60 mg of prednisone per day. Despite this, the patient was having increasing dyspnea with only minor activities. She underwent bronchoscopy: white cotton like material was seen infiltrating the trachea, right main bronchus, and left main bronchus. This was worse, however, on the right side leading to almost complete right airway collapse. Biopsies were taken during the bronchoscopy and were sent to pathology ruling out malignancy. The patient was empirically started on caspofungin to cover probable candida, less likely aspergillus. Maxipime was added to cover the possibility of septic pulmonary emboli until a 2D echocardiogram ruled out endocardial vegetations. During her hospital course, the patient did develop hypercarbic respiratory failure, requiring intubation. Weaning was difficult and the patient underwent tracheostomy. On day eleven of her hospital stay, preliminary cultures came back positive for Mycobacterium tuberculosis. She was started on quadruple therapy after which clinical improvement was noted, though stridor continued. The patient eventually underwent cryotherapy to the narrowed bronchus.

Discussion:
Endobronchial Tuberculosis is a rare form of lower lung field tuberculosis. Because the symptoms are nonspecific, it is often misdiagnosed as bronchial asthma or lung cancer. It is defined as Mycobacterium tuberculosis infection of the tracheobronchial tree. Seven different forms have been identified, whose pathophysiology will be reviewed. Variations range from absence of parenchymal disease (20% of cases) to severe bronchostenosis causing obstructive atelectasis and respiratory failure. Because of the occult nature of the former and the morbid nature of the later, high index of suspicion is required in the diagnosis. With timely diagnosis and initiation of early treatment, lesions can heal without complications. Bronchial stenosis, however, occurs in 60-95% of untreated cases. Once they have progressed to this stage the pathology is irreversible. Moreover, Mycobacterium tuberculosis replicates albeit at low numbers in the stenotic lesion, so early identification and prompt eradication is of paramount public health importance.
ENTEROCOCCUS FAECALIS MENINGITIS

Ricarda White, MD; Nicholas Kotch, DO.; Anthony Welch, DO; Ann Threated, DO
Northside Hospital

Background:
Enterococcus meningitis is a rare cause of meningitis in adults. Although accounting for 0.3 to 0.4 % of cases of bacterial meningitis, it is associated with a high mortality.

Case presentation:
A 78 year old Cuban male presented to the hospital with a one week history of headaches. He was noted by family members to complain of intermittent dizziness, have an unsteady gait, along with confusion described as difficulty recognizing family members or locations. The patient had a history of CAD with 4 vessel CABG, severely dilated thoracic aorta, severe aortic regurgitation requiring porcine AVR and aortic root replacement with a Dacron graft eight years prior. He developed staphylococcus epidermidis endocarditis requiring re-do bioprosthetic AVR and aortic root/arch replacement with a 22mm homograft. He also had paroxysmal atrial flutter with sick sinus syndrome requiring PPM. There was no history of trauma, recent illness, or recent surgical procedure. In the emergency department, he was febrile with a temperature of 103.3F and found to have a leukocytosis of 13.81 (10e3/μL). On physical exam he exhibited meningeal signs and was disoriented to person, place, and time. He underwent lumbar puncture in the emergency department after undergoing a negative computed tomography brain scan with cerebrospinal fluid studies showing WBCs of 4916/μL, glucose 17 mg/dL, protein 271.3 mg/dL. Gram staining showed gram positive cocci with resulting CSF and blood cultures showing enterococcus faecalis. He was empirically started on IV vancomycin, ceftriazone, and ampicillin and admitted to the Intensive Care unit. Intravenous dexamethasone was also given until cultures returned. He was later switched to daptomycin, gentamicin, and ampicillin/sulbactam over concerns for endocarditis. A transesophageal echocardiogram was performed showing normal valve function with no evidence of vegetations. Computed tomography scans of his abdomen and pelvis were negative for abscesses and urinalysis was negative for urinary source. An Indium-111 labeled WBC body scan was negative for focal abscess. Subsequent blood cultures remained negative. His hospital course was significant for hypoxic respiratory failure due to congestive heart failure exacerbation improved with diuresis and subsequently extubated. His mental status continued to improve back to his baseline and he was ultimately discharged home to complete a six week course of intravenous vancomycin to cover meningitis as well as possible endocarditis.

Conclusions:
Enterococcus faecalis, though rare, can be a cause of meningitis.
POST-TRAUMATIC CAROTID CAVERNOSA FISTULA THAT PRESENTED AS FOCAL NEUROLOGICAL DEFICITS WITH NEAR COMPLETE SYMPTOMS RESOLUTION AFTER THERAPEUTIC COIL EMBOLIZATION

Ekaterina Klevtsova, MD
Orlando Health

Introduction:
Carotid cavernous fistula (CCF) occurs between the carotid artery and the cavernous sinus and is usually due to the rupture of the internal carotid artery within the cavernous sinus more frequently after a fracture of the cranial fundus by trauma. CCF is a rare but serious condition; patients usually experience progressive ocular complications and may even develop intracerebral hemorrhage. Progressive neurologic deterioration can occur resulting in permanent damage if the condition is not diagnosed and treated promptly.

Case:
A 46-year-old Caucasian male with the history of a motor vehicle crash six months prior presented with complaints of acute onset of expressive aphasia and focal neurological deficits for three days. On presentation, the patient was noted to have a facial droop, tongue deviation, and moderate proptosis and chemosis of the left eye with no visual changes. Magnetic resonance imaging of the brain showed an arterio-venous fistula arising from the anterolateral pre-cavernous left internal carotid artery directly communicating with the adjacent cortical veins along the medial aspect of the left temporal lobe resulting in venous congestion in the left frontal opercular region, the left insula, and subinsular tracks. The patient underwent an intracranial coil embolization of left carotid cavernous fistula. Post-embolization left internal carotid artery cerebral angiogram was performed showing a complete absence of previously seen retrograde filling of enlarged left cortical vein and absence of retrograde filling of pial venous structures on the cortical surface. No complications were encountered and the patient had near complete resolution of neurologic deficits and left eye proptosis.

Discussion:
The estimated incidence of traumatic brain injury (TBI) in United States is 1.5 million per year. Approximately 0.2-0.3% of all traumatic brain injuries are complicated by carotid cavernous fistula (CCF). The classic symptom triad of CCF includes exophthalmos, conjunctival injection, and an audible orbital bruit. However, signs and symptoms may range from isolated ocular manifestations to life-threatening brain edema and intracranial hemorrhage. This case is notable for acute onset of symptoms consistent with a middle cerebral artery distribution stroke followed by near complete resolution of symptoms and physical findings after the endovascular procedure when brain edema rapidly decreased due to reversal of blood flow through cortical veins. In conclusion, the diagnosis of CCF should be kept in mind while evaluating a patient with focal neurological deficits since it is a treatable condition in settings of timely diagnosis.
NEVER SAY NO! CASE OF WHIPPLE’S ENDOCARDITIS WITH INITIAL NEGATIVE SMALL BOWEL BIOPSY

Tania Lalani, MD; Carmelo Licitra, MD; Mario Madruga, MD
Orlando Health

Introduction:
Whipple’s disease previously known as “intestinal lipodystrophy” is a chronic infectious multisystem disease predominately affecting males in their 40-60’s. The disease is caused by *Tropheryma Whipplei*, a PAS positive bacillus, which can be identified via PCR but is difficult to culture. Although fewer than 1000 cases have been reported worldwide, most individuals present with symptoms involving the GI tract and have PAS positive histiocytes present in their small bowel biopsy. Culture negative endocarditis, a rare manifestation of the disease, has been identified in fewer than 100 cases in literature.

Case Report:
This is a case of a 57 year old Caucasian male with history of inflammatory arthritis who presented with 60lb weight loss over the past year. He had an EGD, colonoscopy, gastric/small bowel biopsy, Gliadin and Endomyseal IgA six months earlier which were normal. He was ruled out for other age-appropriate malignancies with normal PSA, normal SPEP, and CT chest/abdomen/pelvis. During this admission, his hemoglobin was 7.0 g/dl with MCV of 107, and platelet of 24x10³/ul. His bone marrow biopsy with flow cytometry found 2% kappa light chains of undetermined significance. On subsequent days, he also developed a small subarachnoid hemorrhage as well as bilateral ischemic infarcts. Questioning an embolic event with cardiac origin, a TTE was performed with findings of Mitral and Aortic valve vegetations. Three sets of blood cultures were negative. Serologies for culture negative endocarditis were all negative except positive Whipple PCR. The patient subsequently underwent a second biopsy of the small bowel with findings of PAS positive foamy histiocytes corresponding with Whipple’s disease. He was started on IV Ceftriaxone for 4weeks followed by TMP/SMX PO for one year. On follow up visit, the patient was regaining his weight and his hemoglobin and platelet levels were improving.

Discussion:
Although there have been case reports of Whipple’s disease affecting almost every organ, most patients present with GI symptoms, arthralgia and weight loss. The case described above is a rare presentation of Whipple’s disease as the patient presented with endocarditis and weight loss without any GI symptoms. His initial small bowel biopsy was also normal.
Gubler described four patients with Whipple’s endocarditis involving Aortic and Mitral valves without overt GI symptoms. All patients had positive Whipple PCR of their valvular vegetation with negative small bowel biopsy and symptoms improved with antibiotics. Our patient’s endocarditis was complicated by embolic brain infarcts. Whipple’s disease has also been associated with ITP and anemia, both of which were present in our patient. His positive Whipple PCR and findings of PAS positive histiocytes on subsequent small bowel biopsy confirmed the diagnosis of Whipple’s disease. Multiple studies have reported rapid recovery with appropriate antibiotics; however, the disease can be fatal if left untreated.
QUALITY IMPROVEMENT — A HUMBLING EXPERIENCE TRIGGERING CHANGE IN RESIDENT EDUCATION

Caroline Nguyen-Min, MD; Malisa Agard, MD; Bridgette Provost, MD, MPH; Kwabena Ayesu, MD
Orlando Health

Introduction:
Quality Improvement has recently gained significant importance after the Institute of Medicine’s landmark report “To Err Is Human” in 1999, which showed an unperceivable number of patient deaths due to medical errors. In light of this, quality improvement (QI) has now become part of the ACGME core competencies for residents. However, many organizations lack a formal QI training curriculum. With this in mind, Orlando Regional Medical Center (ORMC) has taken steps in formulating a standardized QI training curriculum for residents.

Methods:
A literature survey was done to identify available questionnaires for assessing QI knowledge; however, none were found. Questions that were deemed relevant to the basic knowledge of QI were then selected to create a baseline pre-test questionnaire. The pre-test consisted of questions regarding previous experiences; ability to perform QI projects; as well as relevant basic QI knowledge. The pre-test questionnaires were administered to the residents in IM, EM, Ob/Gyn, Surgery, Orthopedics, Pediatrics and Pathology. Residents were then given access to IHI QI training modules and were instructed to complete four particular modules. Residents were then reassessed. Post-test questionnaires consisted predominately of questions from IHI modules and several from the pre-test questionnaire. Pre and post-test responses were then evaluated.

Results:
On the pre-test, if the resident stated that he/she has knowledge of QI, but scored less than 75%, they were considered to have a discrepancy in their perceived and actual knowledge. IM, EM and orthopedics had the highest percentage of perceived knowledge (42%, 37% and 33% respectively). Pediatrics had the lowest perceived knowledge of 11% followed by Pathology 13%. The actual knowledge or percent of residents who stated they knew QI and passed the pre-test was 8% for IM, 7% for Orthopedics, and 4% for EM, Surgery and Pediatrics; whereas Pathology and Ob/Gyn had 0% of resident with actual QI knowledge. On the post-test, if the resident scored greater than 75%, then he/she was considered to have passed the QI basic training. The departments with the highest passing rate were Ob/Gyn and Pathology at 100% followed by Orthopedics at 88.9% and Pediatrics 78.9%. IM, Surgery and EM were 64.3%, 64.7% and 52.6% respectively.

Conclusion:
QI is now becoming the forefront of all residents' training. Here at ORMC we took the initiative to start a preliminary QI curriculum and to determine its effects on resident knowledge. Pre and post-tests were our method of defining efficacy. We concluded that residents from all departments have limited actual QI knowledge and that there was a significant discrepancy between perceived to actual knowledge of QI. Furthermore, the post training results revealed that residents from all departments have benefitted from the training. With these results, we hope to establish a mainstream curriculum here at ORMC.
SHOULDER PAIN AND THEN SOME: AN UNUSUAL PRESENTATION OF SICKLE CELL DISEASE

Ayed Ayed, MD; Bradley Fletcher, MD, PhD
University of Florida

Introduction:
Sickle cell disease (SCD) can present with various clinical manifestations with vaso-occlusive crises being at the forefront. These painful episodes arise from a complex interplay between sickled erythrocytes, endothelium, leukocytes, and inflammatory mediators leading to vaso-occlusion of the microcirculation and resultant ischemia and pain. Vaso-occlusive crises tend to have recognizable patterns of pain often involving multiple sites and commonly affecting the back, lower extremities, trunk, and abdomen. Unusual pain patterns on presentation may obscure the diagnosis of a crisis and delay treatment, potentially leading to serious complications.

Case Description:
An 18-year-old male with known history of SCD (HbS 94%, HbF 2%, HbA2 3.9%) presented with a 2-day history of severe right shoulder pain described as dull and cramping in nature. He noted that it would radiate to the right upper arm and worsen with movement. On further questioning, he reported engaging in strenuous exercise and bench-pressing prior to onset of symptoms but denied any history of trauma or injury. He also reported that his presentation is different from his typical pain crises. Physical examination was pertinent for swelling and tenderness over the posterior aspect of the right shoulder without limitation in range of motion. The physical examination was otherwise unremarkable. Laboratory data showed a hemoglobin of 11.0 g/dL, a hematocrit of 33.4%, a white blood cell count of 13.4 thousand/mm3 with a normal differential, and a platelet count of 478 thousand/mm3. Total creatine kinase (CK) was elevated at 4034 U/L (30 – 170 U/L) and lactate dehydrogenase (LDH) was elevated as well at 322 U/L (135 – 225 U/L). A 3-view plain radiograph of the right shoulder was negative for bony disease. Given patient’s acute and severe symptoms and significant laboratory findings, an MRI of the right shoulder was done. The latter showed swelling and increased signal intensity on T2-weighted imaging of the right supraspinatus muscle consistent with myositis possibly related to a sickle crisis. Hematology was subsequently consulted and recommended treatment of myositis as a vaso-occlusive crisis. Patient received appropriate treatment including hydration, analgesia, and physical therapy. Over the next three days, his pain and swelling markedly improved. His CBC remained relatively stable and his CK and LDH trended downward appropriately (CK of 1105 U/L on discharge). He was released from hospital with primary care and physical therapy follow-up.

Discussion:
This case demonstrates the importance of recognizing unusual presentations and pain patterns of SCD. Myositis is a relatively rare form of vaso-occlusive crisis but can lead to deleterious consequences if not properly identified and treated, including myonecrosis, myofibrosis, and contractures. Elevated CK and LDH levels, along with typical MRI findings, can aid in making the diagnosis. Early medical treatment and physical therapy are key to preventing long-term complications.
DEMOGRAPHICS, CLINICAL FEATURES, TREATMENTS, AND OUTCOMES OF INFECTIVE ENDOCARDITIS PATIENTS WITH PERIPHERAL EMBOLIZATION: A SINGLE TERTIARY CENTER EXPERIENCE OVER 15 YEARS

Islam Elgendy, MD; Ashkan Karimi, MD; John Petersen, MD; Sarah Capplman, MS4; Charles Klodell, MD
University of Florida-College of Medicine

Objectives:
We aimed to study the demographics, clinical features, microbiological profiles, and outcomes of infective endocarditis patients who developed clinical or radiographic evidence of peripheral embolization as part of their presentation.

Background:
Peripheral embolization is a prevalent and potentially devastating manifestation of infective endocarditis; however, few prior studies have investigated the clinical characteristics and the effect of this complication on treatment and outcome of infective endocarditis.

Methods:
Between 1998 and February 2013, 316 patients were treated for infective endocarditis at our institution. 228 patients who had an echocardiogram at our institution, which confirmed the presence of at least one vegetation, were included in this study. Clinical data were gathered by review of the electronic and paper medical records and data was analyzed with the SPSS statistical software v.17 (SPSS Inc., Chicago, IL). The P value of

Results:
Among 228 study subjects, 43% (97) had evidence of peripheral embolization. 78% (73) of peripheral embolizations were symptomatic and 22% (21) were asymptomatic. Embolization occurred most commonly to the brain 38% (37), lung 31% (30), and spleen 28% (27). Among the entire cohort staphylococcus aureus was the most commonly isolated organism in 39% (89), followed by streptococcus 17% (38), and enterococcus 15% (35). Nine percent of cases were culture negative. Univariate analysis showed that younger age (48 vs. 53 y/o; p=0.02), intravenous drug abuse (36% vs. 12%; p < 0.0001), co-infection with hepatitis C (12% vs. 3%; p=.007), and streptococcal infection (23% vs. 12%; p=0.038) were associated with higher rate of peripheral embolization. Also patients with peripheral embolization had higher white blood cell count on admission (14.3 vs. 12.2 kcell/µL; p=0.047). Diabetes was associated with lower rate of peripheral embolization (16% vs. 29%; p=.015). Multivariate logistic regression analysis showed that intravenous drug abuse was the only independent predictor of peripheral embolization (adjusted odds ratio of 3.1; confidence interval of 1.3-6.9; p=0.006). Among the entire cohort 71% (161) of patients were treated surgically, 20% (46) were treated medically, and in 9% (21) surgery was indicated but not performed due to comorbidities or patient’s preference. There was no difference between treatment strategies among those with or without peripheral embolization. Among the entire cohort hospital mortality rate was 14.5% (33/228). Peripheral embolization was associated with higher hospital mortality (23% vs. 8%; p= 0.001).

Conclusion:
Among all tested demographic, clinical, microbiological, and serologic variables; intravenous drug abuse was the only independent predictor of peripheral embolization in patients with infective endocarditis. Peripheral embolization was associated with higher hospital mortality. We plan to determine if echocardiographic characteristics of vegetations and associated valve disease provide additional prediction of the risk of embolization.
HUGHES-STOVIN SYNDROME – A RARE VARIANT OF BEHÇET’S DISEASE

Roja Pondicherry-Harish, MD; Roland Staud, MD; Hassan Alnuaimat, MD
University of Florida

Introduction:
Hughes-Stovin syndrome (HSS) is a rare clinical disorder that is characterized by venous thrombosis and multiple pulmonary artery aneurysms. Exact etiology and pathogenesis of HSS remains unknown, but it is considered to be a variant form of Behçet's disease (BD). Specifically pulmonary involvement is often indistinguishable between the two entities. Patients with HSS usually present with cough, dyspnea, fever, chest pain, and hemoptysis. Aneurysmal rupture leading to massive hemoptysis is the leading cause of death. Therefore, early diagnosis and timely intervention is crucial in improving the prognosis.

Case Description:
31 year old Caucasian male, without significant medical history, was admitted for one month history of headache, fever, and dyspnea. Workup showed extensive thrombosis involving the dural venous sinus, right atrium, bilateral subclavian and jugular veins, right common femoral vein, and multiple bilateral pulmonary artery emboli. He was started on argatroban for possible HIT and transferred to our hospital for further evaluation. Extensive work-up for hypercoagulable state, connective tissue disorder, and infectious disease was negative. He was empirically treated with 4 weeks of antibiotics for suppurative thrombophlebitis without improvement. CTA Chest showed multiple bilateral pulmonary artery aneurysms containing thrombus. Thorough history and physical examination did not suggest evidence of Behçet's disease. However, because of the coexistence of multiple pulmonary artery aneurysms and venous thromboembolism, Hughes-Stovin syndrome was suspected. He was treated with pulse dose IV methylprednisolone and IV cyclophosphamide. Hospital course improved dramatically with resolution of his respiratory distress. He was discharged on prednisone, enoxaparin, and monthly infusion of cyclophosphamide. On follow up one month later, he reported increased exercise tolerance and did not have fever, cough, chest pain, or hemoptysis. CTA Chest three months after discharge showed near-complete resolution of the multiple pulmonary artery aneurysms, as well as significant improvement in thromboembolic disease involving the pulmonary arteries.

Conclusion:
Hughes-Stovin syndrome is a very rare clinical disorder with less than 40 published cases described. Due to lack of controlled trials, there are no standard treatment guidelines for management of HSS. However, as Behçet's disease and HSS share certain clinical manifestations, the management of HSS can be tailored along the lines of BD. Medical management of HSS includes the use of steroids and cytotoxic agents, particularly cyclophosphamide. Antibiotics have no proven role in HSS, while anticoagulation may be considered in cases of intracardiac thrombi or massive pulmonary embolism. Surgical management is reserved for cases of massive hemoptysis. This case increases awareness of HSS and its management. We hope it will aid clinicians make prompt diagnosis and institute appropriate treatment early in the course of this rare disease. Since aneurysmal rupture is the leading cause of mortality, early diagnosis and treatment is crucial in improving the prognosis of patients with Hughes-Stovin syndrome.
BREAST CANCER & RACIAL DISPARITY BETWEEN CAUCASIAN AND AFRICAN AMERICAN FEMALES: PART-1 (B.R.C.A-1)

Khurram Tariq, MD; Naeem Latif, MD; Robert Zaiden, MD; Nick Jasani, MD; Fauzia Rana, MD
University of Florida Jacksonville

Background:
Breast cancer is a commonly diagnosed malignancy and the second leading cause of cancer related death among American women today. The overall lifetime risk of developing breast cancer is 10% for African American women and 14% for Caucasian women. Despite the lower incidence of breast cancer among African American women they are more likely to die from the disease each year compared to their Caucasian counterparts. Socioeconomics has historically played a significant role in this racial disparity, however, our study design predates universal health care measures under the Patient Protection and Affordable Care Act and we are yet to establish if the implementation of these measures will actually lead to improved access to health care, enhanced breast cancer awareness and a heightened patient attitude towards breast cancer screenings.

Methods:
We present a retrospect cohort study of the tumor registry data from electronic medical records of patients diagnosed with breast cancer at University of Florida Health Jacksonville from the year 2000 to 2005.

Results:
A total of 907 patients were diagnosed with breast cancer at University of Florida Health between 2000 and 2005. A total of 445 patients with invasive breast cancer had complete medical information available in their medical records and were selected for this review. Advanced stage breast cancer was defined as stage IIB or higher and was present in 43% of African American females and in about 28% of Caucasian females with a p-value of 0.0007. Aggressive breast cancer was defined as triple negative breast cancer and was present in 12% of African American and 7% of Caucasian patients with a p-value of 0.074. Estrogen/Progesterone receptor positive breast cancer was present in 60% of African American women and 72% of Caucasian patients with a p-value of 0.006. Her2/neu was over-expressed in 30% of African American women and 21% of Caucasian women with a p-value of 0.075. At the time of diagnosis about 30% of Caucasian and 31% of African American women were pre-menopausal, while 76% of Caucasian compared to the 65% of the African American females had health insurance at the time of diagnosis.

Conclusions:
Our data suggests that African American females present with a more advanced stage and aggressive subtype of breast cancer than Caucasian females, and are less likely to have health insurance. However, we are yet to establish if universal health care insurance can lead to an improved health care access, better breast cancer awareness and an enhanced attitude towards breast cancer screenings. All of these can ultimately lead to an earlier diagnosis and better outcomes. We aim to revisit this study with a follow up research study, BRCA-2, a few years after the implementation of universal healthcare reforms.
ANOMALOUS ORIGIN OF THE RIGHT CORONARY ARTERY FROM THE LEFT SINUS OF VALSALVA IN A YOUNG FEMALE PRESENTING WITH CHEST PAIN

Trevanne Matthews Hew, MD; Amit Babbar, MD; Jason Hew, MD
University of Florida Jacksonville

The origin of the right coronary artery (RCA) from the left sinus of valsalva is a rare congenital anomaly affecting less than 1% of the general population. The clinical presentation is highly variable from the asymptomatic individual to sudden cardiac death. In one study an anomalous origin of the coronaries was identified in 13% of cases of sudden cardiac death in patients younger than 35 years old. Among symptomatic patients most will develop chest pain during exercise or a stressful event. We report on a case of a 30 year old obese female with no prior cardiac history who presented to our hospital with severe retro-sternal chest pain. Her initial Electrocardiogram demonstrated non-specific T wave changes and she had negative cardiac enzymes. She subsequently underwent an exercise stress test which had to be aborted prematurely because of severe angina and non-sustained ventricular tachycardia. A diagnostic left heart catheterization was then undertaken which revealed patent coronary vessels with an abnormal takeoff of the RCA from the left coronary cusp. This was confirmed on Coronary Computer Tomography angiography (CCTA) which further delineated the course of the vessel which traversed between the aorta and right ventricular outflow tract (RVOT) and was identified as the dominant artery supplying the posterior descending artery (PDA). Cardiothoracic surgery was consulted and this patient was taken to the operating room where she had successful decompression of the RCA by translocation of the main pulmonary artery. This case highlights an unusual cause of angina in a young female patient who had a low pretest probability for cardiac disease. It also demonstrates the usefulness of non-invasive techniques such as CCTA in illustrating coronary anatomy. Although the exact cause of myocardial ischemia and death in these patient is not known several mechanisms have been proposed. In this case compression of the RCA via the aorta and the RVOT was deemed the culprit and was managed by translocation of the main pulmonary artery.
Introduction:
With the advance of microbiology & widespread use of antibiotic, there has been an increased recognition of ubiquitous bacteria causing disease in humans; one of these bacteria is Shewanella algae, we hereby reports shewanella algae pneumonia caused by near drowning in salt water. Case presentation: 61 year old man with past history of hypertension, hyperlipidemia & coronary artery disease who was admitted to the intensive care unit after a near drowning episode while he was walking at Jacksonville beach, bystanders started CPR & he returned to spontaneous circulation after two to three minutes, when paramedic arrived, his Glasgow coma scale was only 3 & he was intubated for airway protection. He was admitted to the critical care unit, given elevated cardiac troponin & EKG changes he had coronary angiogram which showed an occluded OM1, percutaneous intervention was attempted unsuccessfully due to calcification, while he was intubated in the intensive care unit, he developed ARDS, leukocytosis, hypotension & low grade fever, he was started on pressors & had bronchoscopy with bronchoalveolar lavage which showed Shewanella Algae ( sensitive to cefepime, ciprofloxacin, gentamicin, levofloxacin & Piperacillin/tazobactam) & E coli ( sensitive to Piperacillin/tazobactam as well), he was started on Piperacillin/tazobactam & his leukocytosis, fever & vital signs improved, he was extubated after 4 days & was transferred to general medicine service for rehab & completion of intravenous antibiotics course.

Discussion:
Shewanella is a gram negative rod that was first described by Derby & Hammer in 1931 after being isolated from tainted butter; they first named it Achromobacter putrefaciens, & then successively called Pseudomonas putrefaciens, Alteromonas putrefaciens, and finally Shewanella spp., of which 30 species have now been identified, it has been recently recognized as a potential pathogen in humans, although this is still controversial. A recent literature review by Vingier et al identified 239 cases of Shewanella infections in human reported since 1973, the most common clinical scenario is skin & soft tissue infection & bacteremia, followed by gastrointestinal tract infection(mainly biliary tract) then ear infection, pulmonary infection and/or colonization has been reported, but less frequently. Although Shewanella was proposed as pathogenic to humans since the 1970s, most case reports in the literature were published after 2000, due to the advance in microbiologic technic & increased awareness, there has been one report of outbreak in single general surgical ward of Seoul University Hospital between June of 2003 and January of 2004. Most cases of human infections involve contact with marine environment especially salt water, One study that look at the susceptibility of 67 ear isolates of Shewanella algae found all strains to be uniformly susceptible to ciprofloxacin, gentamicin, imipenem, tetracycline, aztreonam, and cefotaxime while resistant to sulfonamides, trimethoprim, and polymyxin B.
RECURRENT ANGIOEDEMA IN A PATIENT WITH NORMAL LEVELS OF C4, C1 INHIBITOR AND LUPUS NEPHRITIS

Bernice Acevedo, MD; Camilo Gomez, MD; Juan C Duque, MD; Jair Munoz Mendoza, MD
University of Miami-Jackson Memorial Hospital

49 y/o Haitian female, presented to the ER with worsening hypertension and recurrent angioedema. Her PCP tried multiple anti-hypertensive medications including angiotensin-converting enzyme inhibitors, diuretics, calcium channel blockers and beta-blockers but apparently she developed angioedema with each one of them. She had two prior episodes of angioedema when she was a teenager thought to be secondary to penicillin. She had intermittent diffuse joint pain and intentionally weight loss. Her past medical history was significant for hypertension and a benign breast mass. Her family history was negative for angioedema. Physical examination was remarkable for blood pressure of 226/123 mmHg, pulse of 100 beats/min. She was not in respiratory distress, her lips and tongue were swollen but had no hives in the skin. Her breath sounds were decreased at the bases, and she had mild bilateral lower extremity edema. The rest of the exam was unremarkable. Laboratory analysis showed a serum creatinine of 0.96 mg/dL, albumin 2.5 g/dL, C3 43 mg/dL, C4 12mg/dL, rheumatoid factor 100 mg/dl, CH50 < 13 U/mL, C1 Inhibitor functional 100%. A Kidney biopsy showed Lupus nephritis class IV + V, with 39% fibrocellular crescents. She was started on danazol, nifedipine, steroids and mycophenolate mofetil and had good response. Two months after discharge, her blood pressure was controlled, she had no more episodes of angioedema, her urine sediment was inactive, her proteinuria resolved and her creatinine remained normal.

Discussion:

Hereditary angioedema (HAE) has been described in 2% of patients with lupus erythematosus. Classically there are two types of HAE; type I (80%) is characterized by low C1 inhibitor and low C4 levels while patients with type II (15 - 20%) have normal or high C1 inhibitor and low C4 levels. Recently a new group was described, the so-called type III, which is characterized by normal levels of C4, C1 inhibitor like our patient. To the best of our knowledge this would be the first case reporting association between hereditary angioedema type III in a patient with lupus nephritis.
A SEVERE CASE OF COCAINE INDUCED LEUKOCYTOCLASTIC VASCULITIS

Zeina Hannoush, MD; Rafael Arciniegas, MD
University of Miami – Jackson Memorial Hospital

In 2009 the prevalence of cocaine use in the United States was estimated at 2.8% in the population aged 15 to 64 years. Seventy percent of illicit cocaine consumed in this country is contaminated with levamisole. Most commonly used as a veterinary antihelmintic agent, levamisole is a known immunomodulatory agent that has been associated with cutaneous vasculitis and agranulocytosis in several case reports. We describe the case of a 55 year old man with documented cocaine use who presented with a 5 day history of a severe maculopapular and painful purpuric rash with hemorrhagic bullae on the buttocks, groin, lower and upper extremities, associated with fever and acute kidney injury. Labs were significant for urinalysis without active sediment, negative C-ANCA and P-ANCA, ANA 1:160, and negative Hepatitis C and HIV serology. His renal function rapidly improved with intravenous fluids. On the second day of hospitalization the patient developed acute respiratory distress with new bilateral ground glass opacities found on chest computed tomography, consistent with hypersensitivity pneumonitis. Normal transthoracic echocardiogram ruled out cardiogenic pulmonary edema. Acute pulmonary hemorrhage was ruled out by bronchoscopy. Patient’s respiratory status improved with supportive therapy. Skin biopsy showed leukocystoclastic vasculitis. According to a systematic review there are only 32 case reports of levamisole induced vasculitis from 1964 to 2011. Even though the clinical course, urinalysis and bronchoscopy findings were not consistent with involvement from the vasculitic process, this case represents a severe presentation of cocaine induced vasculitis with hypersensitivity pneumonitis not previously reported in the literature. Given the high incidence of levamisole contaminated cocaine consumption and the potential life threatening complications associated with its use, it is extremely important to increase patient and physician awareness of this condition to ensure proper diagnosis and treatment.
PAGET-SCHROETTER SYNDROME: AN UNEXPECTED ADDITION TO A CIRCUS ACROBAT’S NEW ACT

Archana Ramireddy, MD; Gabriel Hernandez, MD; Amit Badiye, MD; Jose Pinero, MD
University of Miami Jackson Memorial Hospital

Introduction:
Paget-Schroetter Syndrome or effort thrombosis refers to axillary-subclavian vein thrombosis (ASVT) that is associated with strenuous and repetitive activity of the upper extremities as well as anatomical abnormalities at the thoracic outlet (i.e. Cervical rib)

Case Presentation:
26 year-old male circus acrobat with no medical history presented with 2-week history of pain, swelling, and erythema of his left upper extremity. He had recently joined the circus 3 months prior, and his routine was on the “German wheel”, a strenuous activity involving mainly the upper body, he otherwise denied any trauma to the left upper extremity.

Upon arrival, ultrasound evaluation of the left upper extremity showed a complete deep venous thrombosis (DVT) involving the left subclavian and extending into the axillary vein. Pulmonary embolism was ruled-out with CT angiography. Subsequent left upper extremity venogram demonstrated an acute partial thrombus within the left axillary, subclavian, and brachial-cephalic veins with partial clot in the superior vena cava. Given the patient's age and acute DVT, continues thrombolysis with tPA infusion was pursued via peripheral catheter for a total of 16 hours. Repeated venogram showed marked improvement in the left subclavian vein, but abducted arm images demonstrated almost complete occlusion of the subclavian vein and appearance of collateral circulation, consistent with thoracic outlet obstruction. The patient was then taken to the operating room for decompression and removal of an unexpected extra cervical rib and first rib. Patient was discharged home with oral anticoagulation to complete 3 months for provoked DVT.

Discussion:
Paget-Schroetter Syndrome or effort thrombosis is an uncommon condition that accounts for 30–40% of spontaneous ASVT and for 10–20% of upper extremity DVT. Although pathogenesis includes repetitive and strenuous activity of the extremity such as wrestling or gymnastics, anatomical abnormalities involving the thoracic outlet has been described.

In our patient, repetitive use of his upper extremities in addition to a cervical rib (present in 1% of the general population), contributed the development of a thoracic outlet obstruction followed by effort thrombosis, culminating in Paget-Schroetter Syndrome. Early diagnosis is crucial given the risk for pulmonary embolism as well as recurrence of DVT if outlet obstruction is not treated.
EXTRAMAMMARY PAGET DISEASES OF THE SCROTUM AS AN IMPORTANT CLUE TO DIAGNOSE INVASIVE PROSTATE CANCER

Joao Braghiroli, MD; Naiara Fraga Braghiroli, MD; Samer Fahmy, MD
University of Miami Regional Campus

Introduction:
Extramammary Paget disease (EMPD) is a rare cutaneous malignancy, representing 6.5% of all cutaneous Paget’s disease. It affects predominantly women between 50 to 80 years of age, with a peak age of 65. The vulva is the most commonly affected site (65%), with the perianal skin (20%) and male genitalia (14%) affected in fewer cases.

Case Report:
62 year old Caucasian gentleman with extensive history of smoking and previously treated actinic keratosis and resected squamous cell carcinoma of the lip, who presented to outpatient clinic with a erythematous and itchy skin lesion at the right inguinal region extending into the scrotum for over eight months. The patient reported that it was previously diagnosed as intertrigo, but failed to improve with topical antifungal. Skin exam showed an extensive erythematous plaque involving the right side of the scrotum and perineum. Biopsy of the lesion revealed intraepidermal Paget’s cell, which is a large, round, clear staining cell with a hypochromatic nucleus. Further investigation for underlying malignancy reveled presence of Barret’s esophagus and elevated PSA of 34ng/ml. Ultrasound guided prostate biopsy showed prostate adenocarcinoma, Gleason 7 (3+4). He was treated with extensive surgical resection of the skin lesion followed by local and prostatic radiation therapy.

Discussion:
The pathogenesis of EMPD has been debated, but most cases are thought to arise as a primary intraepidermal neoplasm of glandular origin. A minority seems to represent intraepithelial spread of an underlying dermal adnexal malignancy or an underlying regional neoplasm (genitourinary or gastrointestinal adenocarcinoma). Therefore, EMPD favors the apocrine gland rich areas of the body, especially the vulva, scrotum, and perianal areas, but it can be found anywhere on the skin or mucosa. In contrast to mammary Paget’s disease, in which there is nearly always an underlying adenocarcinoma, EMPD often has no associated neoplasm. For EMPD on the male genital skin the most likely sites of other tumors are colorectal and the prostate. In those cases the most common site of skin involvement are scrotum, penis and perianal area. On physical examination penoscrotal Paget’s disease manifests as an erythematos, eczematoid and well demarcated lesion. The diagnosis is often delayed because the common presenting symptoms of scrotum eczema and pruritus are relatively nonspecific and consequently it is initially dismissed or attributed to benign disease. Usually the prognosis for patients with EMPD is fairly good. Although the minority of the cases of EMPD is associated with internal malignancy literature emphasize the importance of a thorough examination for other tumors, considering the life threatening potential associated with these malignant conditions. All patients must be followed periodically by their physicians, with a minimum follow-up time of at least 5 years.
A CASE REPORT OF IGG-4 RELATED DISEASE

Aaron Garza, MD; Jasmin Martinez, MD; Kumar Suresh; MD, FACP, FACR
University of Miami Regional Campus

Introduction:
IgG-4 related disease is a newly described inflammatory and fibrotic condition characterized by protuberant lesions, lymphoplasmacytic infiltrate that contains IgG-4 positive plasma cells, a typical pattern of fibrosis and elevated serum IgG4 concentrations. Most of the patients are men and older than 50 years which is in contrast to other autoimmune diseases. Genetic susceptibility, autoimmunity and infectious agents have been proposed initiating mechanisms. Due to its histological similarities among affected organs it has been compared to sarcoidosis.

Case:
56 year old female with SLE, lupus nephritis stage V on hydroxychloroquine/ACEI and autoimmune hepatitis treated with ursodiol presented with abdominal discomfort, anorexia, weight loss of 20 pounds for 3 months. On physical exam, patient was afebril and hemodynamically stable. She had no oral ulcers, malar or discoid rash. She had left axillary adenopathy. Her cardiovascular and lung exam showed no abnormalities. On abdominal exam, there was midepigastric tenderness upon palpation without organomegalies, rebound or guarding. No mass was palpated. Her musculoskeletal exam showed no abnormalities. A CT abdomen was performed which showed a 3 cm mass in the junction of the distal body and tail of the pancreas. A PET scan showed a hypermetabolic area in the pancreas consistent with malignant tumor with splenic, mesenteric and bilateral axillary, iliac and inguinal regions as well. Subsequently, she had a biopsy of the pancreatic mass and the left axillary node that showed lymphoplasmacytic infiltration with no malignant cells. Later, she underwent bone marrow biopsy which was negative for lymphoma. Serum IgG4 levels were normal. IgG/IgG4 immunohistochemical staining showed rare plasma cells

Discussion:
The disease is often identified incidentally, though two findings are rare tumor-like lesions and allergic diseases.Histological analysis of the involved tissue remains the keystone for diagnosis and elevated concentrations of IgG4 in tissue and serum are helpful in diagnosing but neither one is a specific diagnostic marker. About 30% of patients have normal serum IgG4 concentrations, despite classic histopathological and immunohistochemical findings. Glucocorticoids are the first line of therapy and as seen in our case, a remarkable response to glucocorticoids is characteristic. Azathioprine, mycophenolate mofetil and MTX are used as steroid-sparing agents or remission-maintenance. For patients with recurrent or refractory disease, rituximab can be used. The best indicator of treatment responsiveness is the degree of fibrosis within the involved organs.
Fat embolism is a clinical entity that includes neurological, pulmonary and cutaneous manifestations. It can pose a diagnostic dilemma to clinicians in the absence of this classical triad.

Case:
25 year old male with a history of seizure disorder, non-compliant with antiepileptic medications, and bradycardia with pacer implantation, presented after a traumatic fall while escaping from a beehive attack. On admission, a displaced proximal left femur shaft fracture was diagnosed and an open reduction and internal fixation (ORIF) of left femur was planned. The next morning, the patient experienced a witnessed seizure and, hence, was loaded and maintained on Dilantin. ORIF was performed after he was medically stabilized without surgical complications. The patient was noted to be lethargic post operatively and neurology was consulted to evaluate the alteration in mental status. The physical exam was notable for obtunded mentation, equal pupillary response to light, positive doll’s eye phenomenon, minimal response to pain in the lower extremities with no spontaneous movement, 1+ reflexes bilaterally and mute Babinski’s sign bilaterally. A stat CT Brain without contrast did not display any acute intracranial bleed or any other abnormalities. An MRI was initially thought was unattainable - considering the presence of a pacer - however after extensive consultation with the manufacturer and the radiology, it was identified as MRI-compatible. A stat MRI Brain with/without contrast displayed numerous small, variable-in-size, diffusion-weighted, FLAIR and T2-weighted hyperintensities consistent with a “Starfield” pattern. TEE failed to reveal PFO or vegetations. A diagnosis of cerebral fat embolism was made based on a high index of clinical suspicion, along with the aforementioned radiological findings, despite the absence of pulmonary or cutaneous manifestations (i.e. respiratory distress and petechiae, respectively). Supportive treatment, including physical and occupational therapy, was provided. The patient displayed steady improvement with time and was discharged close to 2 weeks following initial presentation with a nearly full neurological recovery. The presence of a “Starfield Pattern” on MRI conjures a finite list of differential diagnoses. This finding in the setting of a recent long-bone fracture and the gradual development of either altered mentation, respiratory distress or petechiae over the ensuing 24-48 hours post-trauma should create a high index of suspicion for the fat embolism syndrome (FES). Importantly, not all three organ systems necessarily need to be affected in order to make this diagnosis. It is a critical diagnosis to consider early in the disease process – because it can obviate the need for further diagnostic testing or active interventions that may be costly and do more harm than good. Additionally, as FES is usually associated with an excellent prognosis, it can enable activation of the hospital multidisciplinary team early in this disease process and thereby help ensure a full recovery.
IGA NEPHROPATHY IN A CIRRHOTIC PATIENT SUCCESSFULLY TREATED WITH PLASMAPHERESIS

Andrew Kuykendall, MD; Karan Vyras, MD; Stephanie P. Taylor, MD
University of South Florida

Introduction:
The association between hepatic cirrhosis and secondary IgA nephropathy has been well described. While the deposition of IgA in the glomerulus in the setting of cirrhosis is very common, symptomatic disease secondary to IgA nephropathy is more rare. Medical therapy is predominantly focused on managing portal hypertension, however in rare cases of rapidly progressive glomerulonephritis secondary to IgA nephropathy, immune modulating therapies have been employed. We present a case of rapidly progressive glomerulonephritis due to IgA nephropathy in a young cirrhotic patient treated successfully with plasmapheresis.

Case Presentation:
A 32 year old Hispanic male with a history of alcoholic cirrhosis presented to the emergency department with acute onset of bilateral lower extremity edema and hematuria. On exam, the patient was hypertensive, confused, with a distended, nontender abdomen and asterixis. Labwork revealed hyperammonemia, hypoalbuminemia, non-anion gap metabolic acidosis and acute renal failure. Urine evaluation revealed gross hematuria and nephrotic range proteinuria without urine eosinophils or evidence of infection. A fluid challenge did not result in improvement in renal function. Urine electrolytes were not consistent with hepatorenal syndrome. Serologies for autoimmune, connective tissue, and HIV disease were negative. A renal biopsy was performed showing diffuse IgA and C3 deposition with crescent formation, without fibrosis. A diagnosis of rapidly progressive glomerulonephritis secondary to IgA nepropathy was made. Aggressive therapy targeted toward lowering portal pressure was initiated with propranol and diuresis, however there was no improvement in renal function. High-dose steroids did not result in improvement. Finally, plasmapheresis was initiated. The patient’s creatinine normalized, with decreased proteinuria and resolution of hematuria over the course of 10 plasmapheresis treatments. Labs obtained two months after discharge revealed a stable creatinine.

Discussion:
The association between IgA nephropathy and cirrhosis has been well described, with a proposed mechanism of reduced IgA clearance by damaged Kupffer cells. In most cases, this is merely an incidental finding with little clinical relevance. This case describes a cirrhotic patient with acute renal failure found to be due to rapidly progressive glomerulonephritis secondary to IgA nephropathy. This is the first case to our knowledge in which plasmapheresis was used successfully to treat IgA nephropathy associated with cirrhosis.
2013
Florida Chapter ACP
Medical Student
Formal Poster Judging
Presentations
Medical Student Posters

Florida State University College of Medicine
- Telaprevir Associated DRESS Syndrome Complicated by Staphylococcus Endocarditis.
  Sheldon Brown - MS IV

Lake Erie College of Osteopathic Medicine
- Unusual Levels of Intact Parathyroid Hormone Levels in a Patient with Total Parathyroidectomy with Autotransplantation: A Case Report
  Sunny Kar – OMS IV

Nova Southeastern University College of Osteopathic Medicine
- The Bloody Aortic Stenosis: A Case of Heyde Syndrome
  Milla Kviatkovsky - MS IV
- A Comparative Analysis of Immunological Response of Brucella sp in a Chronically Infected Patient
  Erice Turse - MS III

University of Central Florida College of Medicine
- An Unusual Etiology for a Large Anterior Chest Wall Mass
  Cathleen Courtney - MS III
- The Curse of the Nine-Banded Armadillo
  Galal Elsayed - MS II

University of Florida College of Medicine
- Out of the Frying Pan and into the Fire: Rare Malignant Transformation
  Elissa Finkler - MS IV
- A Rare Cause of Recurrent Altered Mental Status
  Mayur Mody – MS IV

University of Miami Miller School of Medicine
- Extrapulmonary Small Cell Carcinoma Presenting as a Seemingly Infected Neck Mass
  Brandon Hendricksen – MS III
- TyG Index as a Surrogate Marker of Insulin Resistance: A Comparison With HOMA-IR in Subjects of the Hepatitis C Antiviral Long-Term Treatment Against Cirrhosis (HALT-C)
  Leah Katta – MS IV
- A Case of Confusing Classification for an Atypical Intradermal Smooth Muscle Neoplasm
  Jennifer Schwenk – MS III
- A Novel Model for Regulatory T Cell Differentiation in Ulcerative Colitis: PPAR-gamma Activation via TLR4-Mediated Stimulation
  Guarav Singh – MS II

University of South Florida Morsani School of Medicine
- Why are My Counts so Low?
  Michael Belsky – MS IV
- Out Of Thin Air: A Case Report Of Pneumotasis Intestinalis Secondary To Lactulose Treatment
  Clay Evans – MS IV
- Why the Long Face?
  Norberto Mancera – MS III
- Peripartum Cardiomyopathy; An Uncommon Diagnosis Presenting Uncommonly
  Anand Parekh – MS IV
- Lung and Subcutaneous Abscess in the Clinical Presentation of a Papillary Fibroelastoma (PFE)
  Tara Saco – MS IV
- A Piercing Blow to the Heart: One Patients Shocking Case of Twiddler Syndrome
  Gregory Sinner – MS IV
- Straightening Out SMA Syndrome
  Mohamad Zetir – MS IV
TELAPREVIR ASSOCIATED DRESS SYNDROME COMPLICATED BY STAPHYLOCOCCUS ENDOCARDITIS

Sheldon Brown, MS IV; Kulvir Nandra, MS IV, Mia Klein MS IV
Florida State University College of Medicine

A 59 year old male, JP, was diagnosed with hepatitis C and began treatment with Telaprevir, peginterferon, and Ribivirin. 8 weeks into treatment, he developed a severe total body skin rash that would not remit. Dermatopathologist biopsy confirmed DRESS syndrome and he his treatment was stopped at week 10 and steroids were started. 4 weeks after developing the initial rash, he was admitted to the hospital with complaints of malaise and intermittent chest pain. He was found to have atrial fibrillation with decompensated rapid ventricular response requiring cardioversion. He also had a left arm wound related to his now resolved rash. CT scan revealed pericardial effusion which enlarged on serial echocardiograms and a pericardial window was done. Blood cultures, pericardial fluid, urine cultures, and wound culture were positive for Methicillin-sensitive Staph aureus. He was initially started on IV Vancomycin and Cefepime for the Staph bacteremia, and then switched to IV Ancef for 6 weeks via a PICC line. Hospital course was complicated by right axillary vein DVT which required Coumadin and acute renal failure which improved. He was discharged home after being hospitalized for 2 weeks, and was readmitted several days later due to palpitations. Patient was then found to have atrial flutter with decompensated rapid ventricular response requiring Amiodarone. Repeat CT scan revealed recurrent pericardial effusion and a left chest tube was placed. A 2nd pericardial window was done which resolved the pericardial effusion, and the chest tube was removed. Patient was discharged 4 days later afebrile with negative blood cultures. He was kept on IV Ancef for 7 weeks, and after repeat echocardiogram revealed no pericardial effusion, the PICC line was removed. Despite having stopped treatment for Hepatitis C, the patient remains disease free at 6 month follow up. This is a case of DRESS syndrome complicated by Staph bacteremia and endocarditis. While the incidence of DRESS syndrome with Telaprevir has been documented [1], this is the first case complicated with Staphylococcus bacteremia and infectious endocarditis. Though DRESS syndrome indeed requires the exclusion of infection or sepsis as a cause, this case is unique in that bacteremia developed after DRESS syndrome was diagnosed and steroid treatment was started. The arm wound and steroids may have contributed to the development of infection in this patient.
UNUSUAL LEVELS OF INTACT PARATHYROID HORMONE LEVELS IN A PATIENT WITH TOTAL PARATHYROIDECTOMY WITH AUTOTRANSPLANTATION: A CASE REPORT

Sunny Kar, OMS IV; Sneha Rathod, MS IV; Pran M. Kar, MD FACP
Lake Erie College of Osteopathic Medicine

Introduction:
Secondary hyperparathyroidism (SHPT) is a common complication of end stage renal disease (ESRD). Total parathyroidectomy with auto transplantation (TPTX+AT) is a treatment option for patients not refractory to medical management alone. Post-transplant patients often clinically stabilize despite continuously elevated intact parathyroid hormone levels (i-PTH). The recurrence rate of high i-PTH following TPTX+AT has been reported in 10-12% of cases (median follow up of 36 months). Our patient developed SHPT with variable i-PTH ranging from 24 to 1389 ng/L after TPTX+AT. This case report serves to remind readers that such findings are not uncommon.

Case Description:
The patient is a 73-year-old obese male status post cadaveric renal transplant 19 years ago, on chronic immunosuppressive therapy. He had TPTX+AT 15 years ago. He presented to the renal clinic for a routine follow up. His home medications included albuterol, allopurinol, aspirin, cyclobenzaprine, cyclosporine, isosorbide, lisinopril, metoprolol, omeprazole, pravastatin, prednisone, and tramadol. Physical examination revealed only trace edema on both lower extremities and was otherwise unremarkable. Significant laboratory data included fluctuating i-PTH values with no appreciable changes in calcium, vitamin D or renal function over the course of the past three years.

Discussion:
Inconsistent i-PTH levels in TPTX+AT patients are a routine finding. Serial labs can show a very broad range of values from high normal to a twentyfold elevation. Not only are such levels expected, but they are used as an initial measure for stratifying TPTX+AT viability. Persistently elevated i-PTH levels are observed regardless of auto transplant location. These findings may be secondary to transplant proximity to blood draw site, intrinsic transplant activity or retained parathyroid tissue. In routine healthcare screenings, recurrent elevations in lab values can serve as a nidus for subsequent retesting, monitoring and management. Such laboratory findings are isolated phenomenon with no appreciable clinical sequelae. Due to intrapatient variability in lab values, one time levels should be used with caution to guide interventions in TPTX+AT patients.
THE BLOODY AORTIC STENOSIS: A CASE OF HEYDE SYNDROME

Milla Kviatkovsky, MS IV; A. Alekseyenko; J. Peguero
Nova Southeastern University

Introduction:
In 1958, Heyde published 10 cases describing the association of aortic stenosis (AS) and iron deficiency anemia due to arteriovenous malformations (AVMs) of the gastrointestinal tract. To date this association is still controversial. However, the postulated link between AS and anemia is secondary to an acquired type IIA von Willebrand’s Disease (vWD) via breakdown of high molecular weight multimers of von Willebrand Factor (HvWF) across the stenotic valve. The anemia is generally secondary to the development of GI bleeds from angiodysplasia. Research suggests an Odds Ratio of 4.5 for the association of angiodysplasia with AS while case reports demonstrate cessation of GI bleeding via AVR.

Case Report:
We present a case of a 74 year-old male who presented with chest pain and shortness of breath and found to be anemic (hemoglobin/ hemocrit (H/H): 9.8/28.6). Patient had a history of frequent admissions for angina exacerbated by Hb <10 which consistently improved via transfusion. His GI bleeds were treated via cauterization multiple times with suspected diagnoses ranging from hematopoietic disorder, myelodysplastic disorder to anemia secondary to renal failure. Further review of the patient’s chart revealed that he received his first transfusion in June 2007 during initial presentation with the aforementioned symptoms. A TEE from 2007 suggested only mild aortic valve sclerosis. During this hospital stay, he received blood transfusions with normalization of his H/H and temporary resolution of symptoms. Considering his history of AS, a transesophageal echocardiography (TEE) was performed which demonstrated an aortic valve area (AVA) of 0.8 cm2. Six years and 56 transfusions later, the newly diagnosed severe AS finally raised suspicion for the diagnosis of Heyde Syndrome. Additional laboratory findings revealed a prolonged collagen-epinephrine and collagen-ADP time suggestive for diagnosis of acquired vWD. This patient was subsequently treated via AVR with resolution of bleeding and now stable H/H.

Discussion:
Research shows that the mean trans-valvular pressure gradient is correlated with the degree of loss of HvWF multimers and studies indicate that aortic valve replacement (AVR) has reversed the aforementioned laboratory abnormalities by first postoperative day. Current indications for AVR include those patients with severe AS (defined by AVA <1.0 cm2) who are symptomatic or those with severe stenosis who fulfill various other criteria- none of which include patients suffering from an associated bleeding disorder. As evidenced in our patient, a bleeding disorder secondary to stenosis may present even with mild valvular disease. Because valve replacement is indicated as curative in this population, severity of stenosis and/ or gradient should not be sole criteria for patient selection. We recommend further review of symptoms and clinical parameters to establish separate criteria for AVR in this population with efforts to raise early clinical suspicion and improve outcomes.
A COMPARATIVE ANALYSIS OF IMMUNOLOGICAL RESPONSE OF BRUCELLA SP IN A CHRONICALLY INFECTED PATIENT

Erica P Turse, MS IV; Ernesto Martinez Duarte, MD; Paula Dilanchian, DO; Dipnarine Maharaj, MD FACP; Nancy Klimas, MD
Nova Southeastern University College of Osteopathic Medicine

Introduction:
Brucellosis is the most common bacterial zoonotic infection presenting as an acute febrile illness, which may also persist as a chronic infection. Myalgic Encephalomyelitis/Chronic Fatigue Syndrome (ME/CFS), a complication of chronic infection, is characterized by fatigue and associated with increased immune activation and inflammatory cytokine levels, decreased NK cytotoxicity, and alterations in lymphocyte function. We analyzed the immunological markers of a 28-year-old man diagnosed with Brucellosis and ME/CFS.

Methods:
A literature search for immunological markers identified in adult Brucellosis cases and ME/CFS cases published in the English language was conducted using PubMed, Embase, and Scopus databases, without limitations regarding date, journal, author or type of article. MeSH terms used were: Brucella, Brucellosis, immune, immunology, immunity, myalgic encephalitis, and chronic fatigue syndrome. We compared immune markers found in the literature to those of a 28-year-old male with serology analysis, symptoms and exposure consistent with Brucellosis and ME/CFS.

Results:
Similarities were seen in NK activity, TNF-α, IL-1b, IL-2, IL-6, IL-17, IL-23, and CD4-count between our patient and historical ME/CFS serology obtained from the literature. CD4+/CD8+, CD54 (ICAM), TGF-β1, IgA, and IgE were not measured.

Conclusion:
Recent literature states that Brucellosis can lead to symptoms and secondary diagnosis of ME/CFS. In our patient diagnosed with Brucellosis and ME/CFS multiple immunological markers were found to have similar values. Theories regarding chronic infections inducing ME/CFS in the literature warrant further research, including subgrouping strategies, and utilization of Brucellosis diagnostics in cases with historic risk factors.
A 63 year-old man with human immunodeficiency virus (HIV) infection presented with an anterior chest wall mass for 2 weeks. There were no fevers or chills, but he reported fatigue and intermittent discomfort at the site. On exam, the patient had normal vital signs. A visible, hard mass, approximately 10 cm in diameter, was centered the mid-clavicular line, and there was no associated erythema or skin discoloration. There were also non-distinct, enlarged cervical nodes bilaterally. The remainder of the exam was unremarkable. The patient was receiving combination antiretroviral therapy (cART), and his CD4 count was 367 cells/μL (31%) with an HIV viral load that was below the limits of detection. CT of the chest revealed an extraparenchymal lobulated enhancing mass in the right upper thorax with internal vascularity. The mass appeared to originate from the chest wall with displacement of the right upper lobe posteriorly and the right pectoralis major muscle anteriorly. Left sided cervical and supraclavicular lymphadenopathy was also noted. However, there was no significant hilar or mediastinal lymphadenopathy. A whole body PET/CT demonstrated FDG-avid bulky bilateral neck lymphadenopathy. There was also avid uptake in the soft tissue mass in the right anterior chest wall. A biopsy of the mass revealed spindle cells associated with inflammatory cells and fibrin material. The histopathology was inconclusive due to scant material; however, it suggested an inflammatory lesion or soft tissue neoplasm. A subsequent excisional biopsy of a lymph node in the neck showed numerous Reed Sternberg cells with prominent collagen bands consistent with Hodgkin lymphoma of the nodular sclerosis subtype. The patient was referred to an oncologist for a full staging evaluation and therapy. HL characteristically originates at a specific site within the lymphatic system, typically a lymph node. It then progresses to adjacent nodes via the lymphatic system before disseminating to distant sites and organs. Chest wall involvement occurs in about 6.4% cases of HL, most commonly with infiltration of parasternal soft tissues by direct extension from anterior mediastinal nodes. A mass located beneath the pectoral muscle without contiguous mediastinal or axillary involvement, as occurred in this case, is unusual. HL is not an AIDS-defining malignancy, but population-based studies have demonstrated an increased incidence of the disease in the setting of HIV infection. Furthermore, the incidence of HIV-associated HL appears to be increasing since the advent of cART. The prognosis of HIV-associated HL in patients treated with cART and standard HL therapy is comparable to that observed in non-HIV infected patients. With the changing epidemiology of HIV-associated HL and its favorable prognosis, it is important for clinicians to be aware of atypical presentations and diligently pursue the evaluation necessary for diagnosis.
A 41 year old avid hunter and fisherman presented with a generalized skin rash of several months duration. He was otherwise healthy and reported no fevers, chills, joint symptoms, or neurological complaints. On physical exam, he was afebrile with normal vital signs. There was a skin eruption characterized by over forty erythematous, non-ulcerated symmetrical patches across the buttocks, flanks, arms and legs. The remainder of the exam was normal. He was on no medications. He lived in Florida and had not traveled outside of the United States and Canada in the past decade. He first noted the rash 4 months earlier, at which time there were only 3 lesions on the lower flank. A skin lesion was biopsied and sent for histopathology and culture.

Histopathology revealed the presence of acid-fast organisms within cutaneous nerve twigs, a pathognomonic finding for Mycobacterium leprae. The biopsy cultures yielded no growth, which is also characteristic for this organism, since it cannot be cultivated on artificial laboratory medium. Confirmatory testing was performed at the National Hansen Disease Center in Carville, LA, and the final diagnosis was active borderline lepromatous leprosy. Upon further questioning, the patient reported dysesthesia and hypoesthesia of the lesions manifest by pruritus. He also admitted to multiple attempts to snare an armadillo one year before his initial presentation. This was his only apparent risk factor for infection. Treatment was started with rifampin, dapsone, and minocycline for a projected course of 24 months. After 4 weeks of treatment, sensation was restored to his upper extremities, and the skin lesions were receding. Follow up was scheduled on a monthly basis.

Leprosy or Hansen’s disease is an ancient condition characterized by hypopigmented patches with central clearing and erythematous borders that progress to become plaques with hypoesthesia. There are approximately 150 new cases reported in the United States each year, with 85 active cases of lepromatous leprosy. In the southern United States, the nine-banded armadillo (Dasypus novemcinctus) is a large natural reservoir for M. leprae. Several case reports have suggested that armadillos may be a source of M. leprae for clinical cases, and contact with armadillos has been shown to be a significant risk factor in several case-control studies. Most patients with leprosy are not infectious, since the organism remains intracellular. However, person-to-person spread is possible from patients with lepromatous leprosy, since M. leprae may be excreted from the nasal mucosa and skin. Early diagnosis and treatment gives a good prognosis and is an effective means to prevent complications of disfigurement, social stigma, and disability. Since it is exceedingly rare, leprosy can be a significant diagnostic challenge. Clinicians should consider leprosy in the differential diagnosis when confronted with chronic skin lesions.
Case Presentation:
A 52 year old nulligravida female presented to her primary care physician with lower extremity edema. Ten years prior to presentation, she had undergone a hysterectomy and unilateral salpingo-oophorectomy for dysmenorrhea that had failed conservative management. Pathology confirmed benign disease. Intraoperative consultation with general surgery was conducted for evaluation of a 12 x 12 centimeter (cm) mass within the liver capsule. A biopsy was not performed given concern for a vascular growth such as a hemangioma. CT scan revealed a 12 x 12 cm cystic lesion with at least one loculation and irregularly thickened cystic walls (Figure 1). Follow-up ultrasound, CT with venous pooling and MRI were recommended but the patient was unable to follow up for financial reasons. Her interval health history was uneventful until she presented 10 years later with abdominal distention and bilateral leg swelling. She had no pertinent additional past medical or surgical history. Laboratory studies were remarkable for direct bilirubin of 4.5 mg/dL, AST 356 U/L, and ALT 130 U/L. CT scan on admission revealed a large complex cystic mass compressing the inferior vena cava and innumerable liver metastases. Paracentesis revealed serum to albumin ascites gradient of 10.0. Fluid cytology was negative. MRI with Eovist and fine needle aspiration confirmed metastatic biliary cystadenocarcinoma (Figures 2,3). After consultation with Hepatology and Medical Oncology, the patient opted for hospice care.

Discussion:
Hepatobiliary cystadenomas have an overall incidence of about one in 20,000-100,000 and account for approximately 5% of cystic lesions of the liver. Cystadenocarcinomas are much more rare (incidence of one in 10 million). Cystadenomas can be misdiagnosed as simple cysts by imaging. Radiographic findings of septations, irregular borders, calcifications, loculations, and septal enhancement are hallmarks of cystadenomas. Cystadenocarcinomas have similar radiographic features and have internal septation with nodularity. Cystadenomas are thought to be premalignant as the age of presentation for cystadenomas is generally at least a decade earlier than cystadenocarcinomas, cystadenomas not completely resected can recur as cystadenoma or cystadenocarcinoma, resected cystadenocarcinomas have been found to contain benign epithelium, and a case of serial biopsies of a benign cystadenoma demonstrated papillary proliferation followed by malignant transformation. This evolution of disease likely explains the difficulty in reliably differentiating the two by imaging alone. Since radiographic diagnosis of cystadenomas and cystadenocarcinomas can be challenging, clinicians should be familiar with these conditions and maintain a high level of suspicion in patients who present with suggestive radiographic findings. Because of the potential for malignant transformation, complete surgical resection with negative margins is indicated in all biliary cystadenomas.
A RARE CAUSE OF RECURRENT ALTERED MENTAL STATUS

Mayur Mody, MS IV; Bahaaeldeen Ismail, MD; Maryam Sattari, MD MS
University of Florida College of Medicine

Case Presentation:
A 63 year-old Caucasian female was transferred to our tertiary center for further work-up of recurrent hyperammonemia and altered mental status (AMS). Past medical history is significant for seizures, hypertension, stereotactic radiosurgery in 2002 for acoustic schwannoma, and remote ureterosigmoidostomy for congenital bladder exstrophy. Her first episode of AMS and hyperammonemia occurred 2 years prior to presentation and improved transiently with lactulose and rifaximin. On transfer, patient was unable to follow commands and had incomprehensible speech. Labs revealed normal hepatic panel and renal function, ammonia level of 148 mc mol/L (normal < 51), bicarbonate of 10 mmol/L (normal=22-30), and chloride of 119 mmol/L (normal=98-107). Head CT was normal and MRI showed small vessel disease. EEG suggested metabolic encephalopathy, but no seizure activity. No structural hepatic abnormalities were detected by abdominal CT and ultrasound. CT IVP revealed a non-obstructing branching right kidney stone, but no ureteral or sigmoid colon obstruction. In addition to bicarbonate, lactulose, and rifaximin, patient was placed on Zosyn for empiric coverage of urea-splitting organisms. A rectal tube was placed to enhance urine drainage from sigmoid colon and patient was placed on low-protein, high-calorie tube feeding. Her mental status started to improve. At the time of discharge, she was alert and oriented with improved acid-base status and normal ammonia level. She was discharged on lactulose, rifaximin, and low protein diet, with outpatient follow-up with urology and gastroenterology. Urology will consider possible invasive urinary reconstruction if patient continues to have recurrent symptoms despite appropriate prophylactic regimen. Discussion:

We attribute our patient’s recurrent AMS to non-hepatic hyperammonemic encephalopathy due to ureterosigmoidostomy, as she did not have evidence of liver disease and urea cycle disorders seemed less likely. Ureterosigmoidostomy is associated with increased incidence of metabolic complications, including normal anion gap hyperchloremic metabolic acidosis and hyperammonemic encephalopathy, both of which may occur in the setting of normal renal and hepatic function. Possible mechanisms for hyperammonemia include increased production of ammonia in the colon from bacterial ureolysis and absorption of ammonia within the sigmoid colon, which can exceed hepatic excretory capacity.

Clinical presentation resembles hepatic encephalopathy. Patients present with AMS, somnolence, and asterixis, without other clinical stigmata of liver disease. Workup should include evaluation of liver structure and function to rule out concomitant liver disease. Lactulose and non-absorbable antibiotics such as rifaximin not only reduce ammonia levels in acute treatment, but can also be used, in addition to dietary modifications (i.e. low-protein diet), to prevent recurrences.

While most cases of hyperammonemic encephalopathy are due to liver disease, clinicians should be aware of unusual etiologies of hyperammonemic encephalopathy to prevent delay in diagnosis and treatment in patients without liver disease presenting with encephalopathy.
EXTRAPULMONARY SMALL CELL CARCINOMA PRESENTING AS A SEEMINGLY INFECTED NECK MASS

Brandon Hendriksen, MS III; Martin Aldana, MD; Aaron Garza, MD
University of Miami Miller School of Medicine

In the United States it has been estimated that Extrapulmonary Small Cell Carcinomas (EPSCC) account for between 0.1-0.4% of all cancers diagnosed annually. According to WHO classification, EPSCC is a type of grade 3 poorly differentiated neuroendocrine tumor. These cancers are highly aggressive, resistant to treatment, and prone to rapid dissemination. The relative rarity of these tumors has led to a dearth of evidence based literature describing the clinical presentation and the treatment of EPSCC. A 59 year old male with no past medical history presented to the ED complaining of an 8 week history of a painful right sided neck mass. The mass was first noticed with a sharp pain that the patient associated with a supposed insect bite. Over the course of 8 weeks, the lesion increased in size and caused increasing pain and shoulder weakness. The patient denied any history of smoking or illegal drug use. On admission vital signs were stable. Physical exam showed that the mass was hard, non-moveable, non-draining and erythematous located at the base of the right ear stretching along the anterior superior border of the sternocleidomastoid. Extensive lymphadenopathy extended from the lesion to the edge of the right clavicle. The mass measured 8 cm by 5 cm and was dark reddish-purple in coloration. Several puncture wounds were present where the patient had tried to "pop" the mass with a needle. CT imaging of the neck revealed a location of 2 cm from the right carotid artery and infiltration of both local lymph nodes and the parotid gland. Blood work returned within normal limits. Infectious etiology was suspected and labs were drawn for Bartonella, HIV, CMV, EBV, and Toxoplasmosis all of which returned negative. A biopsy was performed which revealed a monotonous population of small blue cells with a high proliferation index. Negative TTF and CK20 markers ruled out Merkel Cell involvement; neuroendocrine and KI-67 markers, as well as nuclear molding on cytology confirmed the diagnosis of small cell carcinoma. CT imaging of the head, thorax and abdomen were ordered to search for metastatic disease and revealed no further masses or lesions. The patient was started on treatment with Cisplatin and Etoposide matching current literature which suggests that prognosis and management of ESPCC is similar to that of small cell cancer of the lungs. This case illustrates the clinical course of a relatively rare form of small cell carcinoma and helps to demonstrate the importance of maintaining a broad differential when approaching any clinical situation. In the future, as more cases are presented through literature, more clinicians should begin to add EPSCC to a broad differential for any unidentified mass.
TYG INDEX AS A SURROGATE MARKER OF INSULIN RESISTANCE: A COMPARISON WITH HOMA-IR IN SUBJECTS OF THE HEPATITIS C ANTIVIRAL LONG-TERM TREATMENT AGAINST CIRRHOSIS (HALT-C) TRIAL

Leah Katta, MS IV; Jonathan Nahas, MS IV; Aymin Delgado-Borrego, MD MPH; Raymond T. Chung, MD
University of Miami Miller School of Medicine

Background and Aims:
A strong association between hepatitis C virus (HCV) infection and insulin resistance (IR) has been established. Insulin resistance is associated with increased liver steatosis, worsened fibrosis, and portal hypertension. IR also diminishes sustained virological response. Thus, it is important to assess IR in the care of the patient chronically infected with HCV. Existing measures of IR are impractical and costly or require laboratory studies that are not routinely measured. The triglycerides and glucose (TyG) index has recently been proposed as a measure of IR that is inexpensive and may be widely applicable. Thus, we performed an ancillary investigation within the lead-in phase of the Hepatitis C Antiviral Long-Term Treatment against Cirrhosis (HALT-C) Trial to evaluate whether TyG index may serve as a surrogate measure of IR. Our aim was to evaluate the correlation between TyG and homeostasis model assessment (HOMA-IR), a measure validated against the gold standard for assessment of IR.

Methods:
Eligible patients had failed prior therapy with interferon (IFN) with or without ribavirin and had histological evidence of advanced hepatic fibrosis. A total of 1,105 patients were included in a cross-sectional analysis. TyG index was calculated as Ln(fasting triglycerides (mg/dL) × fasting glucose (mg/dL))/2. HOMA-IR was calculated using serum insulin and fasting glucose levels. The primary outcome measure was the correlation between TyG index and HOMA-IR. Calculation of the Pearson’s r correlation coefficient was used to determine the association between Ln(HOMA-IR) and TyG index. A log transformation of HOMA-IR was used to improve normality of this variable. Correlations between TyG index and HOMA-IR with clinical characteristics, metabolic indices, and viral and histologic parameters were obtained.

Results:
There was a strong correlation between TyG index and Ln (HOMA-IR), with r = 0.627 (p
A CASE OF CONFUSING CLASSIFICATION FOR AN ATYPICAL INTRADERMAL SMOOTH MUSCLE NEOPLASM

Jennifer Schwenk, MS III; Carly Rivet, MS III; Andrew Shapiro, MD
University of Miami Regional Campus

Cutaneous smooth muscle tumors, or leiomyomas, are rare, benign tumors. The current subtypes of cutaneous leiomyomas are based on the skin tissue of origin, with cutaneous pilar leiomyomas arising from the arrector pili muscle. An atypical pilar leiomyoma differs from a simple cutaneous pilar leiomyoma in that it has features of nuclear pleomorphism, cytologic atypia, and minimal mitotic activity. It is important to distinguish the atypical leiomyoma (also referred to as an atypical intradermal smooth muscle neoplasm) from a leiomyosarcoma, which has potential for metastasis.

Patient E.G. is a 35-year-old white man who presented to his dermatologist with a 15-year history of a reddish-brown skin lesion on his left leg directly below the knee. The patient reported that the lesion had been stable in size and appearance during this time. Four months ago, the patient observed that the lesion began to “twitch by itself” while he was sitting and had become larger and redder in color. The lesion had also become painful and sensitive to touch. He reported no heat or cold sensitivity. The patient’s medical history is significant for asthma and allergic rhinitis. He also has a history of sunburns and tanning bed use. To the best of his knowledge, no family members have a similar dermatologic condition.

On physical exam, the lesion appeared as a smooth, firm, well circumscribed, reddish-brown nodule that measured 1.1 x 0.5 cm. The clinician performed a punch biopsy, which was sent to pathology for analysis. The skin biopsy returned with the diagnosis of atypical pilar smooth muscle tumor with involved margins. Another dermatopathologist was consulted and described the tumor as “something less than leiomyosarcoma.” Wide local excision of the lesion was recommended. This procedure was performed by a surgeon two months later with 0.5-cm margins and excision of full-thickness skin. The re-excision returned with margins free of tumor.

There is confusion in the literature regarding proper classification of atypical pilar tumors such as the one in our case report. The same pathology may be described as atypical intradermal smooth muscle neoplasms, atypical or symplastic pilar leiomyomas, and cutaneous leiomyosarcomas. Recent studies have supported their classification as atypical intradermal smooth muscle neoplasms and moving away from the term leiomyosarcoma, which brings the stigma of a more malignant disease process. These intradermal lesions display some mitotic activity and cellular atypia, but remain confined to the dermis and have an excellent prognosis, unlike the leiomyosarcomas which can be subcutaneous or deeper and have some metastatic potential. After excision of an atypical lesion, patients should be followed closely for any recurrences. However, labeling a patient with leiomyosarcoma could influence treatment and management decisions to go beyond what is necessary for what is truly a benign process.
A NOVEL MODEL FOR REGULATORY T CELL DIFFERENTIATION IN ULCERATIVE COLITIS: PPAR-GAMMA ACTIVATION VIA TLR4-MEDIATED STIMULATION

Gaurav Singh, MS II; Jose R Ruiz, BA; Masayuki Fukata, MD PhD
University of Miami Miller School of Medicine

Purpose:
The peroxisome proliferator-activated receptor gamma (PPARgamma) is abundantly expressed in intestinal epithelial cells (IECs), and suppresses inflammatory signaling. It is known that TLR4, a bacterial recognition protein, mediates crosstalk between IECs and macrophages. We aimed to determine whether this crosstalk regulates differentiation and expansion of naïve T cells into mucosal regulatory T cells (Treg) via epithelial PPARgamma activation.

Methods:
An in-vitro model of intestinal mucosa was established by co-culturing mouse peritoneal macrophages and human IECs in transwells. Lipopolysaccharide (LPS) was applied to transwell macrophages, and TLR4-expressing antigen presenting cells (APCs) were utilized from the SW480-APC cell line. Treg cells were generated in vitro by adding isolated mouse splenic naïve T cells to macrophages on the bottom wells of the mucosa model. The production of TNF-alpha (inflammatory cytokine) and IL-10 (anti-inflammatory cytokine) was measured in macrophages and IECs. PPARgamma and Treg generation were also measured by luciferase activity. Two in-vivo colitis models were established: the T cell transfer model, and DSS colitis model (in which dextran sodium sulfate was used to artificially induce colitis). The experimental groups of these models received daily treatment with PPARgamma inhibitor, whereas the control group did not. Cells were stained with FoxP3, a Treg cell marker, and counted under fluorescent microscopy.

Results:
Blocking PPARgamma decreased FoxP3+ Tregs in both the DSS and transfer colitis models. This reduction of FoxP3+ Tregs by PPARgamma inhibition was not observed in TLR4 knockout mice during DSS colitis—indicating that TLR4 is a key regulator of intestinal Treg cells. LPS stimulation of IEC-macrophage co-culture resulted in PPARganna activation in IECs, and generated FoxP3+ Tregs from naïve T cells within 3 days. This PPARgamma activation was inhibited when IECs were co-cultured with TLR4-deficient macrophages or macrophages incubated with COX2 inhibitor, indicating TLR4-induced macrophage activity activates PPARgamma. Furthermore, LPS stimulation induced IL-10 production from IECs in a PPARgamma activation-dependent manner. IL-10 production by IECs, in turn, resulted in TNF-alpha production by macrophages. Conclusions: Results uncover a novel immunological model of TLR4-mediated immunomodulation: during intestinal inflammation, infiltrated macrophages activate IEC PPARgamma via TLR4 signaling, supporting IL-10 expression and Treg proliferation while inhibiting destructive inflammation. While a colitis mouse model has limited generalizability to the human colon, steps in the aforementioned cascade may serve as novel colitis drug targets.
A 53 year old African American male with a past medical history significant for HIV disease presented to the emergency department with a two week history of progressive odynophagia/dysphagia, mild intermittent dry cough, subjective fevers, and unintentional 40lb weight loss over six months. He denied chills, night sweats, chest pain, gastrointestinal symptoms, or CNS symptoms. There was no history of recent travel or sick contacts. The patient reported that he had stopped taking his HAART therapy three months prior due to significant nausea and vomiting. He had continued PCP prophylaxis until he ran out of sulfamethoxazole several weeks prior to presentation. On exam, patient was febrile at 100.4°F, with no evidence of oral thrush, hepatosplenomegaly, lymphadenopathy, or rash. Other than cachexia, the remainder of his physical exam was normal. Labs revealed a recent CD4 count of 51 cells/mm3 and viral load >200,000 copies/mL.

Following a negative CMV PCR, empiric therapy with valcylovir and fluconazole was started for esophagitis, with resolution of odynophagia/dysphagia within 72 hours. Likewise, a treatment dose of sulfamethoxazole and azithromycin for possible Pneumocystis jiroveci and atypical pneumonia was also initiated. The cough resolved within 48 hours.

Laboratory data on admission revealed pancytopenia, which necessitated several transfusions throughout the hospital course. Per the chart, patient had a history of mild thrombocytopenia and anemia. However, 8 months prior, CBC was normal. An extensive anemia work-up revealed an elevated lactate dehydrogenase, decreased reticular hemoglobin equivalent, and significantly elevated ferritin at > 10,000 ng/mL. Immature reticulocyte fraction and immature platelet fraction were within normal limits, despite significant anemia and thrombocytopenia, indicating a hypoproliferative marrow. A bone marrow biopsy identified Histoplasma capsulatum within a poorly-formed granuloma.

Histoplasma capsulatum is a soil-based dimorphic fungus endemic to the Mississippi and Ohio River valleys. Progressive disseminated histoplasmosis (PDH) is the most common manifestation in AIDS patients. The macrophages of HIV-infected patients exhibit a decreased ability to bind yeast, and the decreased CD4 count results in a weakened CD4-cytokine profile, required to activate macrophages. As such, most clinical cases of PDH in HIV occur with a CD4 count

The most common signs and symptoms of PDH include fever, chills, weight loss, anemia, thrombocytopenia, leukopenia, hepatosplenomegaly, and meningitis. Rapid diagnosis can be made with visualization of organism in tissue, or detection of antigen in urine, which is present in 90% of PDH cases. Elevated serum LDH and ferritin >10,000 ng/mL, in the AIDS patient, is suggestive of PDH.
OUT OF THIN AIR: A CASE REPORT OF P NEUMOTASIS INTESTINALIS SECONDARY TO LACTULOSE TREATMENT

Clay Evans, MS IV; Daniel Poetter, MD
University of South Florida Morsani College of Medicine

Introduction:
Pneumotasis intestinalis (PI) is characterized by the presence of gas within the bowel wall, usually visualized on radiographic imaging. PI can range in severity from an incidental finding to a fatal complication. Lactulose is a semisynthetic disaccharide used commonly to treat constipation and occasionally as treatment for hepatic encephalopathy. The disaccharidases in the GI tract are unable to degrade lactulose. This results in less than 3% of ingested lactulose being absorbed; the majority is excreted. The side effects of lactulose are usually mild including excessive diarrhea, nausea, vomiting, bloating, and cramps. There are very few case reports that observed PI secondary to lactulose use. The case below is another example of this rare side effect. Case: A 45 year-old man with a past history significant for a laparoscopic gastric bypass three years ago and portal vein thrombosis presented with a one week history of AMS and “bizarre” behavior consisting of incoherent speech, unsteady gait with multiple falls, and occasional urinary incontinence. The father had noted a gradual decline in the patient’s cognitive function and malnutrition for approximately six months before admission. Lactulose was recently prescribed by an outside hospital, but the patient denied any current medications. At admission, the patient was oriented only to name with concentration deficits. Asterixis was noted on exam and a serum ammonia level was 225.5 mcg/dL. The patient was started on lactulose 20g/30mL solution BID and rifaximin 550mg BID. Lactulose was increased to 30g/45mL solution every three hours on HD2. The patient’s mental status significantly improved by HD3 to his baseline, A&Ox3. A distended abdomen with tympany led to an abdominal x-ray suspicious for PSBO. A follow-up CT scan was performed which revealed transverse and proximal descending colonic intestinal pneumatosis not seen on previous CT’s. The patient denied any pain or tenderness to palpation. General surgery and gastroenterology were consulted for evaluation and possible intervention. A flexible sigmoidoscopy was advanced 85cm into the descending and transverse colon; results revealed normal intestinal mucosa with no visible abnormalities. Given the patient’s clinical picture and lack of visible infarction, surgery was not indicated. The patient was placed on bowel rest and his abdominal exam returned to baseline by HD6. At time of discharge, the patient was tolerating a normal diet without issue.

Discussion:
It was concluded, based on the patient’s underwhelming clinical picture and lack of laboratory or visualized evidence, that the etiology of his PI was secondary to the aggressive lactulose treatment. Past studies have theorized that increased gas build-up by GI flora bacteria following lactulose administration should be considered as a possible cause of PI. It is important to appreciate the clinical context of pneumatosis intestinalis when evaluating and treating patients with this radiographic finding.
A 41yo African American male presented to the James A. Haley Veteran’s Hospital to establish care. He complained of right hip and lower back pain exacerbated by his new job in the hospital’s laundry room. Family history was noncontributory as the patient was adopted. PMH was significant for obstructive sleep apnea, pes plantus and a surgically repaired right inguinal hernia. Vital signs were unremarkable except for his height 76.5 inches and weight 179.2 lbs. On physical exam the patient presented with an impressionably large stature (6’4.5”) BMI 21, pectus excavatum, thin composition, and narrow face. Cardiovascular exam revealed a regular rate/rhythm with an apparent midsystolic mitral valve click. Inspection of his extremities showed minimal arch, wide arm span, swan-like neck, and possible arachnodactyly. Further examination revealed an S-curvature of the spine; passive and active ROM of the hip was normal.

Laboratory data including a CBC, CMP and UA were unrevealing. X-rays of the lumbar spine and right hip were remarkable for scoliosis. Opthamological exam did not show dislocated lens, although echocardiogram showed late systolic prolapse of the mitral valve suggestive of Marfan Syndrome.

Marfan syndrome is a disease caused by a genetic defect in the fibrillin-1 gene which plays an important role in the body's connective tissue, and can have clinical manifestations in physical development and cardiovascular health. It is an autosomal dominant disorder with a near-complete penetrance but variable expression. Fibrillin-1 plays an important role in skeletal, cardiovascular, and ocular findings. While approximately half of the patients diagnosed with Marfan syndrome have a positive family history, as many as 30% are due to de novo mutations. Clinical diagnosis is based on the Ghent nosology, which takes into account major and minor clinical manifestations. Variables in the diagnostic criteria include positive family history, enlarged aortic diameter, ectopia lentis, FBN1 mutation, mitral valve prolapse, and systemic structural features.

This case demonstrates the importance of doing a thorough physical exam and bearing in mind that what may seem to be separate and unrelated presentations may in fact be characteristic presentations of rare genetic disorders. If properly identified early on these disorders can be managed properly, thereby enhancing the patient’s lifespan and quality of life. Our patient’s complaints are relatively minor, yet common in Marfan patient. In Marfan’s, ligament relaxation occurs mainly where there is constant pressure. This shows up in the foot leading to pes planus, as in our patient. It may also occur in the spine leading to scoliosis, or kyphosis.

It has been well documented that the physical exam skills of a clinician deteriorates as one progresses in their medical career. This case demonstrates the art of physical exam and findings that can help elucidate the diagnosis.
PERIPARTUM CARDIOMYOPATHY; AN UNCOMMON DIAGNOSIS PRESENTING UNCOMMONLY

Anand Parekh, MS IV
University of South Florida Morsani College of Medicine

Introduction:
Peripartum cardiomyopathy is a rare form of dilated cardiomyopathy often leading to heart failure. By definition, it presents in the last month of pregnancy or within 5 months of delivery, in the absence of other causes of heart failure. Development of heart failure in these young women leads to a significant increase in mortality, requiring both close observation and strict adherence to medication for a considerable amount of time. The age and condition of these patients allows for better compensation than those with other causes of heart failure. This can lead to a delayed onset of symptoms, which may surpass the time interval provided and unintentionally decrease clinical suspicion.

Case Report:
A 29 year old Caucasian woman with a past medical history significant for asthma arrived via EMS for shortness of breath in early July. Progressive shortness of breath and nonproductive cough began one day prior. The patient reported difficulty breathing when going to sleep the previous night. She was given albuterol breathing treatment by EMS with only a slight relief of symptoms. She reported chills and sweating, but no fevers. When discussing her past medical history, the patient reported that she has had asthma all of her life, usually due to environmental exposures. Her past surgical history included two cesarean sections, the most recent being in early January at nine months gestation due to abruptio placentae. Social history elicited a nine year smoking history of one pack per week. Pertinent positives on physical exam included decreased breath sounds at the bases bilaterally, with mild end expiratory wheezing. In order to further evaluate the patient, a chest x-ray was done in the ER, which showed evidence of possible pulmonary vascular congestion. A CT-angiogram of the chest was also done to rule out a pulmonary embolism. While it was negative for thromboembolism, the CT-angiogram found diffuse bilateral interlobular septal thickening with patchy areas of ground glass airspace disease and bilateral pleural effusions. A trans-thoracic echocardiogram followed, which found a dilated left atrium and ventricle with an estimated ejection fraction of 20-25%, as well as global hypokinesis.

Conclusion:
The diagnostic criteria for peripartum cardiomyopathy include four criteria, one of them being the development of cardiac failure in the last month of pregnancy or within five months of delivery. Adhering to a rigid time interval, as well as the influence of compounding comorbidities can sway a clinician’s suspicion away from the diagnosis. This case may serve as an example to re-evaluate the diagnostic criteria in relation to time, and a reminder that younger patients may compensate better in the setting of heart failure.
LUNG AND SUBCUTANEOUS ABSCESS IN THE CLINICAL PRESENTATION OF A PAPILLARY FIBROELASTOMA (PFE)

Tara Saco, MS IV; Zachary Gales, MD; Chakrapol Sriaroon, MD
University of South Florida Morsani College of Medicine

Introduction:
Papillary fibroelastomas (PFEs) are the most common benign valvular tumors and the second most common cardiac tumor after myxomas. (3) While most patients are asymptomatic, complications include thromboembolic events, MI and valvular dysfunction. It is thought that PFEs lead to thromboembolic events either by causing turbulent flow, or fragmentation and embolization of the tumor’s fronds. We report a case of a patient presenting with a subcutaneous abscess extending from the lung. Work up revealed a pulmonic valve mass later diagnosed as a PFE. This is the first case of a PFE leading to lung infarction and subsequent lung abscess with extension to the subcutaneous area.

Case presentation:
A 66-year-old male with no significant medical history presented to the ER with a right sternoclavicular mass. The patient had presented to the ER ten days prior with similar symptoms, and was given NSAIDs for musculoskeletal pain. Since then, the mass grew from the size of a nickel to the size of a tennis ball. Chest x-ray revealed an air-fluid level in the right upper lung. Chest CT revealed a lung abscess extending to the chest wall and right clavicle. Blood cultures and cultures from CT-guided drainage of the abscess revealed streptococcus pneumoniae, for which the patient received intravenous antibiotics. A 2D-ECHO revealed a 2x1 cm mobile pulmonic valve mass, presumed to be infectious endocarditis. Surgery was consulted for removal.

A mobile, gelatinous, encapsulated, friable mass with multiple fronds on anterior pulmonic valve leaflet was removed. The histological findings were found to be consistent with a PFE without signs of infection. A six-week course of IV antibiotics lead to complete resolution of the lung and subcutaneous abscess.

Discussion:
We believe this patient’s PFE eventually lead to asymptomatic embolic pulmonary infarction. Subsequent lung abscess directly penetrated the chest wall, forming a subcutaneous abscess. Although embolization from the right heart is rare, pulmonary embolism and infarction can occur (1, 2). Although the streptococcus pneumoniae bacteremia had already cleared at the time of PFE removal, it is unlikely that septic embolization was the initial cause.

Conclusion:
We present an interesting clinical presentation providing evidence that right-sided PFEs could lead to serious embolic complications despite low incidence when compared to the left side. Antibiotic treatment alone is adequate for streptococcus pneumoniae lung and subcutaneous abscess. Tumor excision would provide a definitive diagnosis and eliminate recurrent embolic complications.
A Piercing Blow to the Heart: One Patient's Shocking Case of Twiddler Syndrome

Gregory Sinner, MS IV; Elizabeth Melzer, MD
University of South Florida Morsani College of Medicine

Introduction:
Twiddler syndrome is a rare complication of intracardiac device placement described as the dislodgement or retraction of leads resulting in device malfunction. With a reported incidence of 0.1-3%, the syndrome is thought to be caused by some form of conscious or unconscious manipulation of the device by the patient. We present a unique case of Twiddler syndrome with serious and potentially fatal consequences.

Case:
A 68-year-old man with paroxysmal ventricular tachycardia, diabetes mellitus, and hypertension presented with the subjective description of a “bubbling” sensation deep inside his heart two days after undergoing implantation of a biventricular cardiac defibrillator for non-ischemic cardiomyopathy. Although interrogation of the device at discharge showed the defibrillator was functioning appropriately, the patient now endorsed palpitations and a “new tick” along the left side of his chest. Repeat device interrogation showed ventricular lead R wave amplitude and lead impedance of 11.4mV and 623 ohms, respectively, and an RV threshold that failed to capture at 5V, suggesting lead displacement. Two days later the patient developed sharp, stabbing, rhythmic pains along his anterior chest wall. A bedside two-dimensional echocardiogram revealed evidence of right ventricular lead perforation and a small pericardial effusion without signs of tamponade. CT of the chest and thorax confirmed lead perforation of the right ventricle, and the close proximity of the ventricular lead tip with the inner aspect of the left anterior hemithorax was felt to be responsible for inappropriate pacing of the patient’s chest wall.

The next day, with cardiothoracic surgery present, the patient underwent right ventricular lead repositioning in the electrophysiology lab. A post-procedure echocardiogram showed no evidence of worsening effusion or tamponade. The patient received ibuprofen given the increased risk for pericarditis, and he was discharged without further complications.

Discussion:
Twiddler syndrome, first described by Bayliss et al. in 1968, is a complication of electrical pacemaker or cardioverter-defibrillator insertion typically occurring in the elderly, obese individuals, females, or children. Common findings include lead retraction, lead recoiling (similar to ‘reeling in’ a fishing line), or lead displacement resulting in failure to pace, contraction of the diaphragm, or rhythmic twitching of the upper extremity. Preventative maneuvers include active fixation of the leads into the myocardium and careful attention to avoid a disproportionally large pocket relative to the device size. To our knowledge, this case demonstrates an unusual occurrence involving lead perforation with subsequent pacing of the anterior chest wall that has not been described in the literature. Fortunately, the patient presented with only a small pericardial effusion and did not experience cardiac tamponade.
Superior mesenteric artery (SMA) syndrome is an uncommon cause of bowel obstruction, causing nausea and vomiting after feeding. This syndrome is often difficult to diagnose, and can be chronic or acute following significant weight loss or spinal corrective surgery.

A 20 year old female patient S/P spinal corrective surgery for scoliosis 7 weeks prior; presents with a 3 week history of nausea, vomiting, diarrhea, diffuse abdominal pain, fatigue, and a 20 pound weight loss. Symptoms occur approximately twenty minutes after eating. Patient presented 10 days prior to an outside hospital with similar symptoms. She was diagnosed with withdrawal from opiate medications given to her after the surgery.

On physical examination, patient’s vital signs were stable. Her Height was 160cm and weight was 36.29Kg, BMI 14.2. She was found to have diffuse abdominal tenderness in all quadrants, with no rebound or guarding. Bowel sounds were active. Abdominal CT showed marked distention of the stomach. An EGD indicated obliteration of the lumen of the third part of the duodenum with a pulsating mass consistent with SMA syndrome. G-J tube was placed and patient was educated on the importance of gaining weight to alleviate the obstruction.

SMA syndrome is caused by proximal intestinal obstruction and compression of the third part of the duodenum by the SMA due to fat pad loss between the SMA and aorta. Risk factors for SMA syndrome include significant weight loss brought on by medical or psychological conditions. Spinal corrective surgery for scoliosis is another well documented cause of SMA syndrome in the younger population. This procedure causes lengthening of the spine, which displaces the SMA origin reducing the distance between the aorta and the SMA. Patients with a low BMI prior to surgery are at a greater risk for developing the syndrome. Symptoms of SMA syndrome usually appear within a few days to a week following corrective spinal corrective surgery. This case was unusual secondary to the long length of time between surgery and presentation.

Given the patient’s clinical course, and CT scan findings, her presentation is consistent with a chronic SMA induced duodenal obstruction which was exacerbated by the spinal corrective surgery and subsequent weight loss.
2013
Florida Chapter ACP
Display Only
Poster Presentations
Poster Presentations – Display Only

Broward Health Medical Center Nova Southeastern University
- Diffuse Large B Cell Lymphoma Presenting as Primary Lymphoma Of Bone
  Julietta Militello, DO

Cleveland Clinic Florida
- Use Of The Adult Treatment Panel Iii (Atp-iii) Risk Score As A Predictor For Major Adverse Cardiac Events (Mace) In Patients Undergoing Single Photon Emission Computed Tomography (Spect)
  Marlow Hernandez, DO

Florida Hospital Orlando
- Aortic Dissection: A Rare Extraintestinal Finding in Crohn’s Disease
  Yasmin Abaza, MD
- Not Just a DVT - An intriguing Case of May Thurner Syndrome (MTS)
  Rumaisa Hameed, MD
- "Am I Having a Stroke?"
  Debby Sentana, MD

Mayo Clinic Florida
- Vibrio Parahaemolyticus Induced Necrotizing Fasciitis: An Atypical Organism Causing an Unusual Presentation
  Asim Ahmad, MD

University of Florida Jacksonville
- Secondary Cutaneous Diffuse Large B Cell Lymphoma Initially Described as a T Cell Histiocyte Rich B Cell Lymphoma
  Jason Hew, MD

University of Miami Jackson Memorial Hospital
- Not All Troponins Are the Same
  Cesia Gallegos, MD
- A Puzzling Case of Fever and Transaminitis
  Stacy Lieberman, MD

University of South Florida
- Altered Mental Status is not Always a Drug Overdose. Pancreatic Encephalopathy: A Case Report
  Shivam Kalan, MD
- I'm Not Crazy! A Case of Acute Intermittent Porphyria Diagnosed as Somataform Disorder
  Amit Patel, MD
- A Common Complaint with an Uncommon Cause
  Brijesh Patel, MD
- A Deceptive Presentation of Emphysematous Cholecystitis
  Alberto Sabucedo, MD

University of South Florida Morsani College of Medicine
- A Unique Case of Lyme Carditis
  Michael Schnaus, MS IV
DIFFUSE LARGE B CELL LYMPHOMA PRESENTING AS PRIMARY LYMPHOMA OF BONE

Julietta Militello, DO; Heather Katz, DO; Aaron B. Heath, DO; Natasha Bray, DO; Archana Maini, MD
Broward Health Medical Center

Diffuse large B cell lymphomas (DLBCL) are the most common lymphoid neoplasms in adults and the most common histologic subtype of non-Hodgkin lymphoma (NHL). They account for approximately 25 percent of NHL diagnosed annually. Extranodal disease is common and occurs in up to 40 percent of cases, however extranodal disease limited to bone with no lymph node involvement is rare. NHL originating in bone is referred to as a primary lymphoma of bone (PBL). PLB accounts for less than 3 percent of primary bone tumors and roughly 3 to 5 percent of extranodal non-Hodgkin lymphomas. We present a case of DLBCL presenting as diffuse metastatic disease throughout the thoracolumbar spine with no lymphadenopathy or other organ involvement.

A 63 year-old Hispanic female presented to our outpatient Oncology clinic after multiple previous hospitalization and extensive workup for pancytopenia. Her only complaint was lower back pain extending to her bilateral hips. Past medical history was significant for Crohn’s disease diagnosed in 2008 and treated with Infliximab and Sulfasalazine. Review of systems was significant for night sweats, fatigue and a 15 pound weight loss all over the past 3 months. Previous workup included a bone marrow biopsy which showed a B cell lymphoproliferative disorder that was suggestive of B-cell lymphoma but no definitive diagnosis was made. Physical exam was essentially normal with no lymphadenopathy, masses or organomegaly appreciated. Laboratory workup revealed pancytopenia with significantly elevated beta 2 microglobulin and LDH. A PET-CT scan was performed to look for a primary neoplastic site due to the patients previous biopsy results suggesting a B cell lymphoma. The PET-CT did not show any adenopathy or organomegaly, but did reveal multifocal areas of intense bone marrow uptake throughout the spine, ribs, sternum and pelvis suggesting metastatic bone disease. A subsequent thoracolumbar MRI was performed revealing abnormal enhancement throughout the lumbar vertebra and sacrum, consistent with diffuse metastatic disease. The patient underwent a CT-guided biopsy of the right pelvic bone. Histopathologic review demonstrated a diffuse large B cell lymphoma stage 4B. The patient was treated with R-CHOP (Rituximab, Cyclophosphamide, Hydroxydaunomycin, Oncovin, Prednisone) however at a 20% reduced dose secondary to severe pancytopenia.

Primary lymphoma of bone (PLB) is an exceedingly rare disease that accounts for less than 2 percent of all lymphomas in adults. The majority of cases of PLB are pathologically diagnosed as diffuse large B cell lymphoma. Most patients with PLB will present with bone pain not relieved by rest, however PLB lacks a specific presentation and thus can make diagnosis difficult and delay treatment. Once diagnosed, treatment is consistent with other NHLs, with the additional challenge of preventing or stabilizing a pathological fracture.
USE OF THE ADULT TREATMENT PANEL III (ATP-III) RISK SCORE AS A PREDICTOR FOR MAJOR ADVERSE CARDIAC EVENTS (MACE) IN PATIENTS UNDERGOING SINGLE PHOTON EMISSION COMPUTED TOMOGRAPHY (SPECT)

Marlow Hernandez, DO; Craig Asher, MD; Kaelin DeMuth, BS; Jaime Rodriguez, MD; Michael Shen, MD
Cleveland Clinic Florida

Background:
SPECT Appropriateness Criteria (AC) were created to improve proper referrals and therefore reduce imaging costs. However, AC has not been implemented in a large scale due to its subspecialty and subjective nature. ATP III score, on the other hand, has been validated and widely used as a 10-year predictor of MI or cardiac death. The objective of this research is to assess the utility of ATP-III risk score in the risk stratification of cardiac patients who underwent SPECT.

Methods:
Data was collected from consecutive outpatients and inpatients (who underwent SPECT) at Cleveland Clinic Florida from April 2008 to March 2009. ATP-III score, AC appropriateness level and SPECT imaging results were analyzed. Patients were retrospectively followed for Major Adverse Cardiac Events (MACE), defined as CABG, PCI, spontaneous MI or cardiac death) for 1 year after SPECT.

Results:
Of 645 patients who underwent SPECT, 36 patients (5.6%) suffered MACE. Based on AC, 322 (50%) pts were appropriate, 258 (40%) inappropriate and 65 (10%) uncertain. ROC identified ATP III > 10% as the most predictive value for MACE (AUC 0.641, p < 0.05). Patients with ATP III > 10% (n=238, 37%) were more likely to have a positive SPECT (OR 2.30, p < 0.05) and MACE (OR 2.27, p < 0.05). Although a positive SPECT was superior to ATP III > 10 in prediction of MACE (14.9% v. 9.1%, p < 0.05), a positive SPECT in the ATP < 10 population did not statistically predict MACE (5.98% vs. 3.70%, p = 0.27). If ATP III < 10 were used to exclude patients, inappropriate SPECT referrals would be reduced by 30%.

Conclusion:
In the low risk population, ATP III < 10 had a MACE equivalent to a positive SPECT study. Since ATP III score is a simple, objective, and well-accepted predictor of risk, it may serve as an adjunct to AC criteria for appropriate referral to SPECT. The use of ATP-III as gatekeeper for SPECT may be especially useful in the primary care setting. Further research on ATP score for guiding imaging referral is warranted.
AORTIC DISSECTION: A RARE EXTRAINTESTINAL FINDING IN CROHN’S DISEASE

Yasmin Abaza, MD; Daniel Tambunan, MD
Florida Hospital Orlando

Introduction:
Aortitis is a rare extraintestinal manifestation of Crohn’s disease (CD). The associated aortitis can cause aortic aneurysm, regurgitation and mural thrombosis. Weakening of the arterial wall secondary to aortitis may also predispose to aortic dissection. Few case reports documented the association between CD and Takayasu arteritis suggesting the possibility of a common pathogenetic mechanism. However, one case report presented aortitis and subsequent aortic dissection as an extraintestinal manifestation of CD in the absence of concomitant Takayasu arteritis.

Case Presentation:
A 48 year-old African American woman, previously healthy, presented with a three week history of intermittent, progressive, colicky abdominal pain in the left upper and lower quadrants, worse with food and associated with nausea and vomiting. During this period of time, she lost eight pounds but denied having any diarrhea or hematochezia. For the past two years, she has been complaining of a left axillary tender nodule that fluctuates in size with periodic discharge of pus. On examination, the abdomen was tender to palpation at the left upper and lower quadrants. There was a tender nodule in the left axilla draining pus through a sinus tract consistent with hidradenitis suppuritiva. She had no pulse deficits, arterial bruits or audible murmurs. Pertinent lab findings were: erythrocyte sedimentation rate = 68, C-reactive protein = 91.9, normal lipid panel and negative rapid plasma reagin test. The antinuclear, antimitochondrial, anti-smooth muscle, antineutrophil cytoplasmic and anti-saccharomyces cerevisiae antibodies were all negative. CT scan of the abdomen revealed inflammation of the proximal sigmoid colon, small intestine and adjacent mesentery with presence of a 2.6 cm abscess. Incidentally, two aortic aneurysms were detected; a 4.1 cm infrarenal abdominal aortic aneurysm and a 3.8 cm distal thoracic aortic aneurysm. Chronic aortic dissection was evident in the distal abdominal aorta extending to the level of the aortic bifurcation and associated with mural thrombosis. Other than the distal thoracic aneurysm, the remaining aorta, including the celiac and superior mesenteric arteries, was normal on CT chest without evidence of dissection or intramural hematoma. Colonoscopy revealed granular, friable and erythematous mucosa extending from the terminal ileum to the transverse colon with involvement of a segment of the sigmoid colon. Pathology showed chronic colitis and non-caseating granuloma consistent with CD. She was treated with antibiotics, mesalamine and prednisone with resolution of her symptoms. Vascular surgery found no need for surgical intervention since the aortic dissection was chronic. Carvedilol was given to maintain systolic blood pressure below 120 and pulse below 60. She was discharged upon resolution of her symptoms.

Conclusion:
Aortitis is a rare yet potential extraintestinal manifestation of CD which may predispose to the development of aortic aneurysm and dissection.
NOT JUST A DVT - AN INTRIGUING CASE OF MAY THURNER SYNDROME (MTS)

Rumaisa Hameed, MD; Aditya Chada, MD; Nejda Lugo Mandes, MD; Victor Herrera, MD
Florida Hospital Orlando

Introduction:
May Thurner Syndrome (MTS), also known as iliac vein compression syndrome, is a condition diagnosed in 2-5% of all patients being evaluated for lower limb DVT. In MTS, left common iliac vein is compressed between the fifth lumbar vertebrae and the right common iliac artery, as it crosses in front of the vein. Failure to recognize this condition could result in serious complications such as chronic leg pain, worsening DVT, leg ulcers and Pulmonary Embolism.

Case Presentation:
A 34-year-old Hispanic female presented with 2-day history of sharp pain in the left buttock radiating to the left leg; associated with swelling, redness, and tenderness predominantly in the calf. The patient denied any recent history of travel, smoking, or use of OCP. Upon examination, the left leg was erythematous and measured 1.5 inches more in diameter than the right leg. Doppler of the left leg revealed acute extensive DVT of the left leg. A CTV confirmed extensive DVT of left common iliac, external iliac, femoral, and popliteal vein throughout the tributaries in the calf. Left common iliac vein was small and compressed; and was associated with inflammatory stranding surrounding DVT in the left pelvis. Workup for hypercoagulable disorder was negative. A diagnosis of May Thurner syndrome was made and vascular surgery was consulted. The patient underwent a venogram and an ‘Ekos treatment’ (a left lower extremity catheter directed thrombolysis with tpa and heparin infusion). After 24 hours, a repeat venogram showed complete resolution of thrombosis up to the level of the distal iliac vein. However, the proximal external iliac vein and common iliac veins were still occluded. A balloon angioplasty was performed and two bare metal stents were deployed from the IVC bifurcation to the left external iliac vein. A follow-up venogram revealed complete resolution of thrombosis. The patient was eventually sent home on Coumadin. On follow up appointment, the leg pain and swelling had completely resolved. The patient was advised to continue Coumadin for at least 6 months.

Conclusion:
This case highlights the classic form of MTS with no evidence of thrombophilia. It is important to consider MTS in the differential diagnosis of extensive unilateral DVT especially in young patients. If diagnosis is missed, the recurrence of thrombosis, post thrombotic syndrome, and PE could lead to significant morbidity and mortality. The mainstay of treatment is removal of the clot; repair of anatomic defect by stents and balloon venoplasty; and anticoagulation for at least six months.
"AM I HAVING A STROKE?"

Debby Sentana, MD
Florida Hospital Orlando

Introduction:
In clinical practice, neurological symptoms are one of the most common clinical encounters. When an adult patient presents with hemiplegia, hemiparesthesia, and gait disturbance, we often think of stroke as the most likely cause. We report a case of a less common etiology which may lead to permanent neurologic sequelae if left untreated warranting prompt intervention.

Case presentation:
A 42 year old female presented with right sided weakness for the past 2 years, along with numbness and tingling of her arms, worsening in the past several days. Patient has frequent falls due to gait difficulty in the past two years, occipital headache, and thoracic vertebral pain. Neurologic examination showed decreased motor strength in the right upper and lower extremities, normal motor strength in the left side, and no clonus noted. Sensory examination was unremarkable. Patient had hyperreflexia in her lower extremities, Babinski sign was present bilaterally, and had staggering gait. Lhermitte’s sign was present. On reviewing her record, patient had head CT scan 2 years ago which showed equivocal low lying cerebellar tonsils. On admission, CT scan of head did not show acute hemorrhage or infarct. MRI of brain showed 15 mm of tonsillar ectopia with effacement of basal cisterns at the level of foramen magnum and associated syringomyelia consistent with Chiari 1 malformation. MRI of the cervical and thoracic spine showed presence of syrinx formation extending from the cervical to thoracic cord with significantly expanded cervical and thoracic cord and thinning. The patient then underwent surgical decompression with suboccipital craniectomy and C1 laminectomy on day 2 of hospitalization. Within days after the procedure patient noted improvement of her back pain, with mild gradual improvement of her strength and gait.

Discussion:
Chiari syndrome is a developmental malformation of the craniocervical junction leading to the herniation of the lower cerebellar structures through the foramen magnum, and may cause compression of the spinal canal. Chiari I malformation (CM-1), which is the most frequent subtype, is often associated with syringomyelia. Most cases present in adolescence to adulthood. Our patient’s symptoms started in her adulthood for the past 2 years, with clinical features suggestive of spinal cord lesion, and MRI findings of CM-1 along with significantly thinned cervical and thoracic cord due to syringomyelia. Craniocervical decompression is an effective method for treatment of CM-1 and syringomyelia, and in some cases may be combined with syringosubarachnoid shunt placement. CM-1 may spontaneously resolve, however if left untreated, it may also lead to worsening syrinx and permanent neurological sequelae. This case highlights the importance of including Chiari I malformation with syringomyelia in the differential diagnosis of patients who present with hemiparesis, hemiparesthesia, and gait disturbance to ensure early recognition and treatment of this potentially reversible condition.
VIBRIO PARAHAEAMOLYTICUS INDUCED NECROTIZING FASCIITIS: AN ATYPICAL ORGANISM CAUSING AN UNUSUAL PRESENTATION

Asim Ahmad, MD; Mike Maniachi MD, Lisa Brumble MD
Mayo Clinic Florida

Background:
Necrotizing fasciitis (NF) represents a life-threatening bacterial infection characterized by rapid necrosis of deep subcutaneous tissue and facia underlying the skin. Despite its lethal nature, NF occurs infrequently, leaving many physicians unfamiliar with the disease process, common pathogens, and treatment strategies. Here we present a case of NF caused by an unlikely organism, Vibrio parahaemolyticus. We highlight the innocuous nature of initial presentation and the potentially devastating sequela.

Case Report:
A 45 year-old lady presented to the emergency department with a chief complaint of left leg pain. Past medical history was significant for diabetes mellitus type 2, morbid obesity, and chronic Hepatitis C infection. She noted that she had been jet skiing the day prior in brackish water and had sustained a small laceration on her left posterior calf.

Upon presentation, physical exam performed in the emergency department and revealed an afebrile woman in modest distress. There was noted to be a small abrasion over the left calf with associated erythema and tenderness. This area was marked with a skin marker. Given concern for a salt water infection, doxycycline and ceftazidime were administered. In order to cover against Streptococcus, cefazolin was also employed.

The patient was admitted to the Internal Medicine service. Upon reexamination, the left leg was now noted to be 3 times the size of the right. Numerous large bullae were noted, the area of involvement had spread well beyond the area initially demarcated by the skin marker.

Findings were concerning for NF with distributive shock and multi organ failure. Sepsis management was employed, and patient was taken for emergent fasciotomy.

Afterwards, the patient’s condition remained critical. Later on that morning, she went into asystolic cardiac arrest. Despite maximum heroic efforts, she was unable to be resuscitated. Later on that day, both blood cultures and cultures from the leg would grow out Vibrio parahaemolyticus

Conclusion:
This patient suffered from DNA sequencing proven Vibrio parahaemolyticus. NF septicemia and death are quite infrequent with Vibrio parahaemolyticus infections when compared to the more virulent strains of vibrio like vulnificus and cholerae. It usually manifests rather nonspecifically with symptoms such as fever, chills, malaise, and aches. The skin generally becomes warm, tender, and edematous with localized pain described as “out of proportion to physical exam”.

NF caused by Vibrio parahaemolyticus represents a medical and surgical emergency, requiring aggressive sepsis management and prompt surgical intervention. Despite the devastating consequences, NF occurs infrequently enough that most providers may not be comfortable with the diagnosis. Through this vignette we hope to have highlighted possible presenting characteristics, the importance of serial physical exams, and how laboratory data as well as patients comorbidities can be used to create an optimum treatment plan.
SECONDARY CUTANEOUS DIFFUSE LARGE B CELL LYMPHOMA INITIALLY DESCRIBED AS A T CELL HISTIOCYTE RICH B CELL

Jason Hew, MD; Robert A. Zaiden Jr., M.D.
University of Florida Jacksonville

Diffuse Large B cell lymphoma (DLBL) is an aggressive, high grade subtype of Non – Hodgkins Lymphoma (NHL), accounting for about 30% of these tumors annually. Genetic analysis confirms significant heterogeneity within this population. The skin is the second most common extranodal site of involvement behind the gastrointestinal tract. T cell/Histiocyte Rich Large B cell Lymphoma (THRLBCL) is a rare pathologic entity, listed as an uncommon variant of DLBL in the 2008 WHO reclassification. It is characterized by the presence of <10% neoplastic B cells in an inflammatory background of non-neoplastic T-cells and histiocytes. It infrequently primarily involves the skin. We present the case of a 64 year old male evaluated for a large ulcerating tumor of the back. Skin biopsy performed at an outside institution was initially interpreted as a THRLBCL. Second opinion review of the biopsy at our institution concluded that this was cutaneous DLBL, given the non-dispersed nature of neoplastic B cells. This case highlights the subtle differences, and difficulties, often with therapeutic and prognostic implications in accurate subclassification of this disease.
Elevated troponins are not synonymous of myocardial damage, and patients with low pretest probability for coronary artery disease should undergo workup for alternative etiologies including myopathy as part of the differential. We describe the case of a 39 year old man who presented with generalized weakness and muscle pain and was admitted for workup of elevated troponins. He denied any past medical history, however, he stated he had been experiencing these symptoms for about 6 years and they had recently worsened without exacerbating factors. He denied chest pain but did note the worst pain was present in his thighs and knees. He denied fever, chills, or weight loss. He was able to perform activities of daily living without difficulties. He had had several prior visits with similar complaints that reportedly resolved with administration of fluids and anti-inflammatory medications, however further workup was never pursued. Upon physical examination, he was noted to have male pattern frontal baldness and bitemporal wasting with a long, narrow face, an arched palate, and sagging of the jaw. He was also found to have bilateral ptosis, wasting of the sternocleidomastoid, bilateral hand interosseus muscle atrophy, gynecomastia, and atrophy of his testicles. On neurological exam he had grip and percussion myotonia, with a slow relaxation phase, as well as difficulty getting up from the supine and sitting positions. Initial labs showed normal TSH, creatinine kinase (CPK) of 1085u/L, and CPK-MB of 17 u/L. Since CPK MB was elevated, three set of troponins were ordered which remained elevated at 0.07. ECG demonstrated sinus bradycardia with 1st degree A-V block, left ventricular hypertrophy (LVH) with QRS widening. Because of the abnormal ECG, a two dimensional echocardiogram was performed, showing concentric LVH with preserved ejection fraction. Ultimately, the patient underwent an EMG which showed typical myotonic discharges and early recruitment with minimal contraction in several muscles, confirming the diagnosis of myotonic dystrophy (DM) type 1, also called Steinert’s Disease. Since he had evidence of AV conduction disease on ECG, he underwent pacemaker placement as recommended by current guidelines due to risk for sudden cardiac death. Myotonic dystrophy is an autosomal dominant myopathy characterized by progressive skeletal muscle wasting and cardiac conduction abnormalities with multisystem manifestations. Structural heart disease is also frequently observed in approximately 20% of patients. As in other types of muscular dystrophies, cardiac arrhythmias may occur early in the absence of symptoms and may progress to high grade AV heart block. For this reason, per ACC/AHA guidelines, a permanent pacemaker is indicated in patients with confirmed myotonic dystrophy and any degree of AV block on baseline ECG. As with our patient, persistently elevated troponins in an otherwise asymptomatic patient, warrants further workup of other non-cardiac etiologies.
A PUZZLING CASE OF FEVER AND TRANSAMINITIS

Stacy Lieberman, MD; Karlo Lizarraga, MD
University of Miami / Jackson Memorial Hospital

Case Presentation:
A 71 year-old woman with advanced NK/T-cell EBV-positive lymphoma despite chemotherapy plus radiation developed facial erythema and pain one month prior to admission. She received amoxicillin/clavulanate for presumed cellulitis. She continued receiving zidovudine and bortezomib with allopurinol for tumor lysis prophylaxis—the last dose of these given one week prior to admission. She developed neutropenia and transaminitis at a routine outpatient follow up and was started on filgrastim. The following day she developed fevers, rigors and altered mental status and was admitted to the hospital. Broad-spectrum antibiotics were started according to the Infectious Disease Society of America (IDSA) Guidelines for febrile neutropenia.

The patient’s hospital course was complicated by persistent fevers, transaminitis (a 5-fold increase) and hyperbilirubinemia of unknown etiology. She continued to spike very high fevers despite appropriate antibiotic coverage and negative blood cultures. Viral hepatitis panels were negative, too. An abdominal CT showed only hepatic steatosis. The patient progressively worsened, developed fulminant liver failure with transaminases in the thousands, hyperbilirubinemia and loss of synthetic function with associated coagulopathies and cytopenias. Corticosteroids were initiated but she developed acidosis with respiratory distress. The family requested no further invasive measures and the patient passed away.

The differential diagnosis of her decompensation ranged from liver metastasis to drug-induced liver failure secondary to amoxicillin/clavulanate versus allopurinol or zidovudine. On the date of her death, labs were drawn to evaluate for possible hemophagocytic lymphohistiocytosis (HLH). The ferritin level was 350,326 ng/mL and triglycerides were 645 mg/dL. The patient had four of the eight criteria for HLH: fever, cytopenias, hyperferritinemia, and hypertriglyceridemia. Although bone marrow or liver biopsies were not done, the hyperferritinemia at this level is virtually pathognomonic for the diagnosis of HLH.

Discussion:
HLH is a multisystem inflammatory syndrome characterized by an uncontrolled and poorly understood activation of T-helper 1 lymphocytes and macrophages. There are two forms of HLH—a familial form (seen in infants) and an acquired form as a result of intensive immunological activation due to severe infections, autoimmune disorders or malignancies, especially NK/T-cell lymphomas. Although many viruses can cause HLH, Epstein-Barr Virus (EBV) is by far the most common. Diagnosis is made by molecular and/or genetic defects consistent with HLH, or with at least five of the following: fever; splenomegaly; cytopenias affecting two or three lineages; hypertriglyceridemia and/or hypofibrinogenemia; hemophagocytosis in bone marrow, spleen, or lymph nodes; hyperferritinemia (>500 μg/l); low/absent NK-cell activity; or elevated interleukin-2 receptor levels. Supportive evidence includes additional diagnostic criteria: encephalopathy; transaminitis; hyperbilirubinemia; and elevated LDH. Diagnosis of HLH is often delayed leading to adverse events, however its prompt recognition lends to the early intervention with corticosteroids and chemotherapy may decrease mortality.
ALTERED MENTAL STATUS IS NOT ALWAYS A DRUG OVERDOSE. PANCREATIC ENCEPHALOPATHY: A CASE REPORT

Shivam Kalan, MD; Karan Vyas, MD; Brijesh Patel, MD; Viraj Modi, MD; Akhil Patel, MD; Satyam Kalan, MD; S, Taylor, MD; M. Rumbak, MD
University of South Florida

Introduction:
Pancreatic Encephalopathy (PE) is a rare and potentially fatal complication of acute pancreatitis (AP). PE, first described by Lowell in 1923, manifested with mental status changes in patients with AP. The common symptoms include spatial disorientation, agitation, delusions and hallucinations. We present a 29-year-old female who developed PE secondary to AP after experiencing a fall. Rapid intervention with intravenous thiamine led to her successful recovery. PE poses a challenge, diagnostic and therapeutic, given its poorly understood pathogenesis and high mortality rate.

Case Description:
A 29-year old female with no significant past medical history had a fall while horseback riding and presented to an outside hospital for shoulder injury. Fracture was ruled out and she was prescribed opioids for pain. A few days later, she returned to the same hospital with abdominal pain, nausea, vomiting and diagnosed with AP by CAT-scan. She was promptly discharged home; however, within a few days her family mentioned behavioral changes including hallucinations and confusion. Her neurological function worsened and she was found unresponsive. She was taken by EMS to a different hospital, and was presumably diagnosed with opioid overdose. She was then transferred to our facility for a second opinion and critical care management. Upon arrival, the patient was non-responsive to voice and pain. Laboratory findings showed a metabolic acidosis, amylase and lipase levels of 256 and 3,270 respectively. Brain MRI revealed infarctions in the globus pallidus bilaterally, right cerebellum and signs of anoxic brain injury. EEG revealed severe diffuse encephalopathy without epileptiform changes. Management consisted of respiratory and nutritional support, given her critical state. Our research and review of her case led to the diagnosis of PE given her history of recent AP. On hospital day 2, the patient was started on intravenous thiamine 100 mg therapy. By day 3, for the first time, the patient’s neurologic function significantly improved and she started to follow commands. Our patient advanced clinically each day after thiamine therapy, soon after that she was extubated and had regained full cognitive function and was discharged approximately 3 weeks later.

Discussion:
This case demonstrates the benefit of using thiamine in the management of PE. In patients with AP with neuropsychiatric symptoms, PE is overlooked and often misdiagnosed. Early intervention with thiamine can reduce the high mortality associated with PE and can potentially reduce hospital stay. Pathophysiology of PE remains unclear; most literature describes its relation to phospholipase A (PLA) activation. PLA-2 damages the brain cell membrane and platelet activating factor increases vascular permeability leading to brain edema and demyelination. This is thought to cause the deteriorating mentation in patients with PE. Additional awareness to recognize PE is essential because its timely diagnosis with thiamine treatment can be life-saving.
I'M NOT CRAZY! A CASE OF ACUTE INTERMITTENT PORPHYRIA DIAGNOSED AS SOMATOFORM DISORDER

Amit Patel, MD; Sunil Medidi, MD; Candice Mateja, MD
University of South Florida

Acute intermittent porphyria (AIP) is a rare disorder that results from a deficiency in the enzyme porphobilinogen deaminase in the heme pathway. It typically presents with waxing and waning symptoms of constipation, abdominal pain, neuropathies and psychiatric manifestations. Because of this unusual constellation of symptoms, diagnosis can be difficult. Here we present a case of a 26-year-old female who was diagnosed with AIP after multiple hospitalizations for abdominal pain.

A 26-year-old female presented to our institution with a 6-day history of intermittent abdominal pain. She was admitted and treated with enemas and motility agents with improvement in her symptoms. However, 6 months later the patient returned the hospital with similar complaints of abdominal pain but now with associated back pain, myalgias and bilious vomiting. She was diagnosed with viral gastroenteritis and treated supportively. 2 months later the patient once again presented to the hospital, this time for generalized pain. In addition to the diffuse abdominal pain she also experienced left chest, arm and bilateral leg pain as well as decreased sensation to light touch and temperature in her thighs and pelvic area. She also reported a 20-pound unintentional weight loss over the past month. Given her recurrence of symptoms gastroenterology was consulted and performed an EGD and colonoscopy which were both normal. Since her medical work up failed to produce a cause for the pain, psychiatry was consulted to evaluate for a possible psychiatric cause. She was diagnosed with generalized anxiety and somatoform disorder and was started on Klonopin and Sertraline. Before discharge, a 24 hr urine porphobilinogen level was ordered. A few days after discharge her level came back elevated at 105 mg (normal

Diagnosing AIP can be a challenge to the medical profession given its rarity and its similarities more common conditions. In addition, once the basic work up is complete but failed to give a diagnosis, it is easy to diagnosis these patients with functional abdominal pain syndromes, fibromyalgia, pain seeking behavior or other psychiatric disorder. It is wise to rule out common conditions first but if the patient has recurrence of symptoms a detailed history and physical with questions specific for AIP should be done. For clinicians, we believe that it is appropriate to include obtaining a 24 hr urine porphobilinogen level in all patients with chronic abdominal pain when other more common etiologies are ruled out to avoid costly re-hospitalizations, potential surgeries, and invasive medical procedures.
A COMMON COMPLAINT WITH AN UNCOMMON CAUSE

Brijesh Patel, MD; Jose Lezama, MD, FACP; Lucy Guerra, MD, MPH, FACP, FHM
University of South Florida

49yo Caucasian male veteran with PMH significant for HIV (last CD4/viral load 597(ng/dl)/undetectable) was admitted for chronic diarrhea, a 15lb weight loss and joint pain. He reported having diarrhea for the past 6 months with diffuse abdominal tenderness and 6-10 watery, non-bloody stools daily. Given a negative infectious disease work-up, his complaints were attributed to his HAART medications. His c/o arthralgia were attributed to arthritis. Routine eye exam revealed an incidental finding of papilledema of the right eye.

A decision for colonoscopy was made and it showed multiple, small white-speckled areas along the entire small bowel. Multiple biopsies from the duodenal bulb and 2nd portion of the duodenum showed macrophages with PAS positive and diastase resistance. Tissue was negative for acid fast bacilli, fungal organisms and H. Pylori. With findings supportive for the diagnosis of Whipple’s disease, the patient was started on Bactrim®. His diarrhea resolved within a week and he regained nearly 15lbs.

This case is very unique with endoscopy suggestive of Whipple’s disease and a dramatic improvement in symptomology including, complete resolution of joint pain within a week of Bactrim® initiation. Our patient had a classic presentation of abdominal pain, weight loss, arthralgia and chronic diarrhea with remote optic manifestations. Duodenal biopsy is the gold standard in conjunction with the pathologic diagnosis. Though the bacterial isolate and PCR results were not obtained, the pathology of jejuna aspirate supports the diagnosis of Whipple’s disease: large number of PAS-positive and diastase resistant macrophages in the lamina propria, with prominent lymphangectasia.

Chronic diarrhea is defined as passage of loose stools lasting > 4 weeks. In HIV disease, chronic diarrhea is a common complaint especially when the CD4 count is < 50 ng/dl. The work-up for diarrhea is extensive and can be categorized into infectious and malabsorbtive causes. A rare but important cause of infectious diarrhea includes Whipple’s disease (WD). WD is caused by the bacterium Tropheryma whippelii, a member of Actinomycetales order found in soil. Symptoms of WD include chronic abdominal pain with lipodystrophy, weight loss, fatigue and chronic diarrhea. The incidence is very rare with only 696 reported cases between 1907-'87. Worldwide the incidence since 1980 has been approximately 30 cases/year. Cases of WD in AIDS patients have been sparsely reported because it is very hard to differentiate the causes of diarrhea in AIDS patients whether it is due to: HIV enteropathy vs. other opportunistic infections, namely those with Mycobacterium avium complex and Rhodococcus equi. The later have been reported to mimic the histological appearance of Whipple’s disease by the presence of PAS-positive inclusions of macrophages. In conclusion; this case demonstrates a rare cause of diarrhea in HIV patient with classic presenting symptoms.
Emphysematous cholecystitis (EC) is a potentially life-threatening infection which carries a mortality rate of approximately 20% as compared to 1.4% for acute cholecystitis. Emphysematous cholecystitis presents similarly to acute cholecystitis, with right upper quadrant pain and fevers, however it may also have an insidious onset with mild clinical findings which can progress to a rapid clinical deterioration and death, if not diagnosed promptly. Herein, we present a case which illustrates a deceptive presentation of this disease and the importance of maintaining a broad differential in the care of patients. A 70 year-old gentleman with a history of diabetes mellitus type II, hypertension, hyperlipidemia, peripheral vascular disease and chronic kidney disease presented to the hospital with a one day history of acute onset right sided, non-radiating pleuritic chest pain. He denied any trauma to the area, shortness of breath or cough. He denied any relation to food ingestion or changes in his bowel habits. He did report an episode of non-bloody emesis prior to arrival to the emergency department. On the initial exam the patient was noted to be afebrile, normotensive and was saturating 89% on room air. The physical exam on admission was unremarkable. Labs were notable for acute kidney injury with a creatinine of 1.9. Electrocardiogram and chest radiographs were obtained did not show any acute findings. The patient was subsequently admitted to the Internal Medicine service and started on heparin weight based protocol for suspicion of pulmonary embolism. Overnight, the patient decompensated with increasing hypoxia, tachycardia, tachypnea, fever of 102.2F and altered mental status. He was transferred to the intensive care unit and an arterial blood gas revealed pH 7.34, pCO2 34 mmHg, pO2 68 mmHg and a lactic acid of 4.7. Repeat labs obtained in the intensive care unit showed improvement in the acute renal failure. A CT angiogram of the chest was obtained that was negative for pulmonary embolus, however it was notable for emphysematous cholecystitis. Soon after, the patient became hypotensive. Blood cultures were obtained and he was started on broad spectrum antibiotics and vasopressors. Later that day he underwent a successful laparoscopic cholecystectomy. The blood cultures later grew Escherichia Coli and Clostridium Perfringens. This case demonstrates how emphysematous cholecystitis initially presented without fever, leukocytosis or signs of infection and then rapidly progressed to hypotension and a very ill patient. Considering a broad differential of what may cause pleuritic chest discomfort will allow for the prompt diagnosis of serious conditions such as emphysematous cholecystitis.
A UNIQUE CASE OF LYME CARDITIS

Michael Schnaus, MS IV; Jennifer Yehl, MD; James Austin Follett, MD; Asima Cheema, MD; Alexander Reiss, MD
University of South Florida Morsani College of Medicine

A 29-year-old Caucasian man presented to the hospital per his primary physician, stating: “something is wrong with my heart.” He presented with an EKG showing first-degree AV block, a PR interval of ~350 ms (image).

A thorough history was obtained: In early June 2013, the patient traveled to NY and New Jersey, where he hiked and camped. He denied any tick or bug bites during the trip. He endorsed multiple unprotected female sexual encounters during the same trip. He denied any sick contacts or IV drug use.

One week after returning, he experienced constitutional symptoms: chills, fevers, diaphoresis, and neck stiffness. He simultaneously discovered bilateral lymphadenopathy in the inguinal, axillary, and cervical regions. All symptoms then resolved.

However, one week after resolution of symptoms he experienced new, asymmetrical, migrating joint pains. Both the proximal and distal joints of the upper and lower extremities were affected. He also noted the presence of three new rashes, described as flat and warm with a centrally cleared area that progressively expanded. These appeared on the outside of his left arm, the anterior right forearm, and the inside of his right thigh (image). He also endorsed a more recent episode of painful urethral discharge.

His past medical history was non-contributory. Family history was significant for rheumatoid arthritis. Social history was as documented in his history.

The patient appeared well and was without distress. He was tachycardic (HR: 100-120), his other vital signs were stable. He had swollen, erythematous tonsils without exudates. No significant lymphadenopathy was found. The distal joint on the third metatarsal of the right foot was slightly erythematous, swollen, and tender. The rest of the exam was normal.

Initial labs revealed a leukocytosis (WBC: 10,800). Hemoglobin, platelets, electrolytes, and LFTs were within normal limits. His ESR (65) and CRP (3.734) were elevated.

Cardiac monitoring and infectious disease screening for Lyme disease, Chlamydia, Neisseria gonorrhoeae, and HIV were performed. One gram of azithromycin once and 2 grams IV ceftriaxone daily were given. His PR interval progressively shortened (images) to approximately 232 ms. Neisseria gonorrhoeae, Chlamydia, and HIV screens were negative. Lyme screening and Lyme IgM and IgG western blots were positive. A final diagnosis, Lyme carditis, was made.

This case represents a unique case of disseminated Lyme disease, or Lyme carditis. This disease is rare, especially outside of the northeastern United States where the Lyme vector is more prominent. This case presents physical proof of the effectiveness of ceftriaxone in the treatment of Lyme carditis per EKG findings and clinical improvement. It is our hope that this case enhances the clinical recognition of, and the appropriate treatment selection for Lyme disease and Lyme carditis amongst internal medicine physicians in Florida.