Clinical Vignette Competition

2022

UTAH ACP RESIDENTS & FELLOWS COMMITTEE
Emily Signor, MD – Chair
Ben Drum, MD
Eric Moore, MD
Sumi Mishra MD

FALL CLINICAL VIGNETTE PROGRAM | THURSDAY, OCTOBER 20, 2022

University of Utah | School of Medicine | Internal Medicine Grand Rounds

12:00 PM **WELCOME & OPENING REMARKS**
Residents & Fellows Committee

JUDGES
Stephanie Chan MD
John Christensen MD
Sumi Mishra MD
Karen Stenehjem MD

12:10 PM **PRESENTATIONS**

12:50 PM **ANNOUNCE RUNNERS-UP AND 1ST PLACE**

1:00 PM **CLOSING COMMENTS**
Residents & Fellows Committee

UTAH ACP RESIDENTS & FELLOWS COMMITTEE | MISSION STATEMENT

To improve the professional and personal lives of Utah Residents and Fellows and encourage participation in the American College of Physicians.

1. **Foster Internal Medicine Resident's interest in the ACP – ASIM.**
   - Encourage ACP associate membership and a lifelong interest in ACP – ASIM.
   - Encourage representation on National and Local ACP subcommittees.
2. **Foster educational Opportunities for Internal Medicine Residents.**
   - Encourage participation in local and national ACP – ASLIM Associates Clinical Vignette and Research opportunities.
   - Organize the local competitions. Provide information on board review courses. Publicize local and national educational opportunities. Work with residency programs to improve residency education.
3. **Identify practice management issues for Internal Medicine Residents.**
   - Provide information for residents as they prepare to enter practice, such as practice opportunities and contract negotiation.
4. **Identify public policy concerns of residents.**
   - Monitor local and national health policy and how it relates to Internal Medicine and residency training.
5. **Encourage an interest in community service.**
   - Identify ways associates can become involved with community service in Utah.

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Identification: 49 y/o male with a past medical history of hypertension and diabetes. Patient worked as a general contractor performing heavy manual labor.

Chief Complaint: Bilateral (left greater than right) hand and arm numbness and weakness.

History: Patient initially presented to his PCP with mild neuropathic pain and weakness of the left 4th and 5th digit and mild back pain nine months prior. Imaging, including C-spine and left extremity X-ray, at that time did not demonstrate clear etiologies for the pain resulting is presumed peripheral radiculopathy. He received multiple treatments of prednisone that temporarily resolved the symptoms. He managed his symptoms until an acute decline over the span of one week where his strength and sensation decreased significantly. He lost his ability to button his jacket or zip up his pants due to weakness over just one week.

Physical Abnormalities: On physical exam, the patient did have a positive Spurling's sign and tenderness over the lower C-spine. Repeat MRI noted an "acute or subacute burst fracture of the T1 vertebra plana of the central vertebral body," a fractured right pedicle and facet resulting in a "perched right C7/T1 facet," and "retropulsion of the T1 vertebral body measuring 4 mm with associated moderate spinal canal stenosis and posterior displacement of the cervical cord." Patient was also found to be hypertensive, thrombocytopenic, and have a normocytic anemia. Patient had a mildly elevated kappa free light chain, significantly elevated lambda free light chain, and low J/L ratio.

DDX: The differential diagnosis includes ulnar nerve peripheral neuropathy from cubital tunnel syndrome, compression in the wrist, or irritation from repetitive movements or stress, cervical radiculopathy from stenosis, trauma, or fracture, or plexus radiculopathy.

Treatment: Patient was immediately transferred to the hematologist service. He was enrolled in a study that used 8 cycles of carfilzomib, lenalidomide, and dexamethasone and completed this study.

Conclusion: Multiple myeloma presenting symptoms are well recognized as hypercalcemia, renal insufficiency, anemia, and bone lesions (CRAB). Peripheral neuropathies are a less well-known symptoms of multiple myeloma. This symptom can be present prior to the typical CRAB symptoms and facilitate for further complications, like in this patient resulting in burst fracture and a progressed disease. With time between presenting symptoms and diagnosis being a key prognostic indicator for multiple myeloma, ensuring proper management of a patient with signs and symptoms can possibly improve clinical outcomes.

HISTORY: The patient is a 57-year-old man with no significant comorbidities, who presented to the emergency room on his primary care physician's advice for progressive dyspnea. A few weeks previously, he reported feeling unwell with several days of sore throat, malaise, and a mild fever. His symptoms continued to progress, and he developed a cough that was occasionally bloody. A chest x-ray obtained prior to his admission was unremarkable, and a subsequent CT chest was read as bilateral, multifocal tree-in-bud opacities with superimposed subsegmental consolidation. He was started on empiric antibiotics for pneumonia, but continued to have symptoms and presented to the ED.

PHYSICAL EXAM/LAB ABNORMALITIES: The patient had normal vitals without hypoxia. Our exam was notable only for minimal bilateral crackles. CBC on admission was remarkable for leukocytosis to 14 and normocytic anemia. CMP noted a creatinine of 4.52 from a normal baseline, and hyperkalemia to 7.2. ESR was 76, hsCRP was 17. His urinalysis was negative for protein and RBCs but demonstrated muddy brown casts. COVID-19 and infectious workup was negative. A bronchoscopy during his admission was consistent with diffuse alveolar hemorrhage, cultures negative for organisms. ANA was positive with a speckled pattern and a titer of 1:320. ANCA vasculitis panel was negative. Anti-glomerular basement membrane antibody was positive to a value of 80 AU/mL.

DIFFERENTIAL DIAGNOSIS: Presumed diagnosis was Goodpasture's syndrome based on the presence of highly specific anti-GBM antibodies. Patient's AKI was attributed to ATN from extensive NSAID use, as no protein or RBCs were ever identified in the patient's urinalyses. Renal biopsy completed one-month post admission did not demonstrate findings consistent with anti-GBM disease.

TREATMENT: The patient was started on methylprednisolone for 3 days and then a prolonged prednisone taper. With nephrology assistance, he underwent plasmapheresis for 5 treatments, and was started on cyclophosphamide prior to discharge.

CONCLUSION: Goodpasture's syndrome is a rare disease that occurs in roughly one in a million people, and is characterized by the triad of pulmonary hemorrhage, rapidly progressive glomerulonephritis, and anti-glomerular basement membrane antibodies. Of these patients, only individual case reports exist that highlight "atypical" Goodpasture's syndrome, which often lacks pulmonary involvement. Here is a case of "atypical" Goodpasture's syndrome that lacked classical renal findings despite the presence of a concomitant kidney injury. It is important to recognize that in an uncommon disease, there are atypical presentations that are even rarer.

HOW DO YOU KNOW WHEN YOU HAVE KNOCKED OVER THE FIRST DOMINO? | JOHN BOSAK OMS3

Case Presentation: Patient is a 42-year-old woman with a history of PCOS, Hypothyroidism, and anxiety who initially presented in 2015 for sexual assault with vaginal bleeding after rough intercourse with her husband. She had just told her husband she was 5 days late on her menstrual cycle and believes he had intentional rough sex to induce a miscarriage. After a negative HCG and a lack of pain or distress she was discharged without follow-up.
This event appeared to be the first domino in a chain reaction that led her our care in the Uintah County Jail, where she is receiving counseling for daily auditory hallucinations, anxiety, and guilt after the murder of her son and an attempted suicide.

Lab Results: Medical workup was negative outside of a UTI on several occasions. This included consistently serum HCG, negative drug screens, alcohol quantity, metabolic panels, and blood counts. No imaging was ever completed.

Psychiatric Treatment: She had seen psychiatry intermittently for 3 years prior to her first visit with well controlled anxiety. However, at the end of 2015, she presented again with delusions and an altered mental status believing that her father had killed several people and her niece was being molested by the LDS church. She was started on Ziprasidone after that visit, but 4 months later returned to the emergency department for suicidal ideation and was again transferred to Provo Canyon for psychiatric care. She was then lost to follow up for the next 4 years with no history of what happened during this time, and finally returned on no medications with worsened psychosis.

Discussion: This patient's case calls into question where the medical system failed in treating her psychiatric conditions. After her initial sexual trauma, she never received counseling. She was a "known schizophrenic" to the emergency department physicians, yet never carried a diagnosis of schizophrenia. She originally showed good adherence to medication until she was lost to follow-up without social worker involvement. She frequently sought help from medical providers until the end when she stopped seeking care after nothing was being done to help with her complaints. Towards the end, the physician on call typed into the note saying, "I am concerned that she might have a full psychotic episode soon even though tonight she does not seem to be dealing with one. If she really has not slept for a month, that will be a problem."

A STAGGERING CASE OF SIADH | ADELINE BROWNE MD, PGY2

FINALIST

Case presentation: Patient is a 73-year-old female with a history of CAD, CKD, COPD and pulmonary hypertension s/p lung transplant five years prior to presentation immunosuppressed on tacrolimus, prednisone, and everolimus who presented with one day of acute onset ataxia and confusion. Two days prior to presentation the patient was seen at urgent care for a rash, was diagnosed with shingles, and prescribed valacyclovir. She was otherwise in her usual state of health. On arrival to the ER a code stroke was called, with subsequent brain MRI revealing no acute intracranial process. Initial laboratory evaluation was significant for hyponatremia to 123. Patient was admitted to pulmonology for further evaluation.

Physical exam: Initial vital signs were unremarkable. Patient was A&O to self. Her neurologic exam was notable for +Romberg, dysmetria, and asterixis. Vesicular rash with overlying crust was present in a dermatomal distribution on her back.

Lab and imaging results: Serum osmolality was 257, UOsm was 411 and UNa was 30 consistent with SIADH. Tacrolimus, Everolimus, and ammonia levels were within normal limits. Toxicology screen was negative. CT head and MRI brain were negative.

Clinical course: Valacyclovir was held due to concern that supratherapeutic dosing given CKD may be causing AMS. Patient's sodium improved to 128 with fluid restriction, and her mental status improved. She was discharged home with close follow up, however the underlying etiology of her SIADH remained unknown. After two days, she re-presented with worsening ataxia and confusion. A lumbar puncture was performed, and CSF revealed a pleocytosis with high normal protein and normal glucose. Encephalitis panel was positive for VZV by PCR.

Treatment: Patient was diagnosed with Varicella-zoster virus infection with acute cerebellitis and encephalitis with associated SIADH and started on IV Acyclovir. Although 25% of patients with disseminated VZV have positive VZV PCR in CSF and confirmatory antibody testing was not obtained, Neurology agreed with the clinical diagnosis given neurologic findings. At follow up, her mental status had returned to baseline.

Conclusion: We found case reports of disseminated VZV as a rare cause of SIADH. VZV encephalitis should be considered in the immunocompromised patient who presents with recent shingles and neurologic symptoms. This case underscores the need to maintain a broad differential in transplant and immunocompromised patients. It also provides a cautionary tale against premature closure; in this case, cognitive bias led to misattributing the patient's symptoms to hyponatremia, which resulted in delayed diagnosis.

A CASE OF SILENT MALLIGNANCY | MIN KYUNG CHUNG OMS3, KATIE OFFNER OMS3, JOHNNY CHENG DO

Chief Complaint/Identification: The patient is a 76-year-old male presented with chest pain and repeat shocks from his automatic implantable cardioverter-defibrillator (AICD).

History: The patient history includes ventricular tachycardia and an inferior STEMI which lead to an AICD placement in 2017. Additional history includes congestive heart failure, hypertension, and a 50 pack-year smoking history.

Physical Abnormalities/Labs: The patient has chronic hyponatremia which persisted throughout this hospitalization resulting in multiple ICU placements for treatment with hypertonic saline (3%). Elevated urine sodium was recorded on multiple occasions while urine osmolality remained within normal range. The patient had multiple pulmonary findings throughout the hospitalization including left pleural effusion, numerous consolidations bilaterally, and left lung mass.

Differential Diagnosis: Given the chronic hyponatremia, elevated urine sodium, tobacco use history, and evidence of malignancy from radiology reports, the primary differential included SIADH secondary to malignancy. Small cell lung carcinoma is most likely due to the tobacco history and its classic presentation with SIADH.

Case Presentation: To prevent further arrhythmia, the patient was started on amiodarone. A potential lung pathology was incidentally identified on chest X-ray during the chest pain evaluation. This instigated a workup for potential malignancy including repeat chest X-rays, multiple chest CTs, thoracocentesis, bronchoscopy, and multiple biopsies. Left cervical lymph node biopsy identified benign mantle zone/ follicular hyperplasia. A bone scan showed evidence of metastasis in the upper extremities and femur. The metastatic finding in the absence of a positive lung or lymph node biopsy created an ambiguous clinical picture regarding the source of malignancy. With his stable condition, he was discharged with plans for follow-up with oncology to determine an appropriate treatment plan with cancer detection.

Discussion: What appeared to be a standard ventricular tachycardia case turned into an SIADH case, secondary to undetermined malignancy. While multiple imaging studies suggest the potential for cancerous growth, all biopsies returned negative with the exception of cervical lymph node hyperplasia. This demonstrates a potential interplay between three conditions, which encourages clinicians to look beyond what they believe is a cause, to find the main stem these conditions branch from.

Conclusion: Hospital medicine often presents with multiple medical conditions that require further evaluation to identify whether they are interrelated or independent issues. This case exemplifies how a root problem may propagate other conditions and emphasizes the importance of a thorough workup.

A WARM WELCOME | THOMAS GEORGE MD, PGY2

Case Presentation: AN, a 31-year-old female, presented with her husband to the emergency department with a three-week history of worsening vaginal bleeding. AN is a Dari-speaking refugee, recently resettled in SLC with her husband and three children after leaving Afghanistan amidst political unrest. Additional history revealed that AN was 3 months pregnant. A bedside ultrasound in the emergency department showed a viable intrauterine pregnancy with a chronic subchorionic hemorrhage, likely the source of patient's bleeding; the obstetrician was comfortable with outpatient follow-up. As the patient was being readied for emergency department discharge, she was noted to be hypoxemic requiring 2 liters of oxygen. Upon closer inspection, a 4/6 systolic ejection murmur and clubbing were noted. AN was subsequently admitted to the medicine service for further workup.
Additional procedural history in which a "spring" was placed in the patient's heart at the age of 15 was uncovered. Prior to this procedure, patient had daily syncopal episodes accompanied by cyanosis. Subsequent inpatient TTE showed severe pulmonic stenosis with right to left shunting across a secundum atrial septal defect. The patient underwent a balloon valvuloplasty with reversal of flow across the ASD resulting in normalization of oxygen saturations. 6 months later the patient gave birth to a healthy baby girl; both she and mom are doing well.

Differential diagnosis: Leading diagnoses included pulmonary embolism, infection (HIV/PJP, tuberculosis), congenital heart disease, acute decompensated heart failure.

Physical examination/lab results/imaging: 4/6 systolic ejection murmur best heard at the left upper sternal border in addition to clubbing. CBC notable for a hemoglobin of 14.6. Uterine ultrasound as mentioned above. VQ scan normal. TTE with severe pulmonic valve stenosis, moderate pulmonic valve regurgitation, normal pulmonary artery pressures, large right to left intra-atrial shunt.

Treatment: Patient underwent a balloon valvuloplasty with subsequent improvement in the pulmonary valve gradient from 71 mmHg to 22 mmHg. As patient's pulmonary vasculature was not affected by the stenotic lesion, the pressures in the RV went from 80% to 31% of systemic pressures with no evidence of RV failure on subsequent echocardiograms.

Conclusion: Patients with severe pulmonary hypertension associated with presyncope or syncope will at times undergo atrial septostomy's to allow for adequate left ventricular filling. This patient thankfully had a secundum atrial septal defect which provided her with adequate preload until her stenotic lesion could be ballooned.

CC: Shortness of Breath

Patient is a 41-year old pregnant woman, currently at 34 weeks of gestation, with a history of 2 years of shortness of breath and a right-sided, painless cervical lymph node now admitted by obstetrics for respiratory failure. The patient denies chest pain and endorses worsening difficulty breathing particularly at night and insomnia. She has a history of essential hypertension, preeclampsia, gestational diabetes, and obesity. Initial workup of SOB 2 years ago was suggestive of asthma and potential tracheal stenosis and the patient was scheduled for a future tracheal widening surgery. CXR showed some scarring in left midlung but otherwise unremarkable. Focused ultrasound of the right mid neck revealed an elongated teardrop shaped hypoechoic 2.06 x 0.44 x 0.98 cm lesion. The node is immediately lateral to and adjacent to the right internal jugular vein and "probably represents a lymph node." Results of a sleep study suggested obstructive sleep apnea hypopnea syndrome.
Upon acute presentation of respiratory failure and stridor, ENT was consulted. Direct laryngoscopy with bronchoscopy and biopsy of tracheal mass were performed in conjunction of cesarean delivery for evaluation of possible malignancy.

EXAM ABNORMALITIES: Physical exam was notable for hoarse voice, stridor, and BP of 137/68. Direct laryngoscopy showed subglottal mass with stenosis and initial pathology of throat tissue was positive for CK5/6, p63, and CD117 antibodies. Histology revealed adenoid cystic carcinoma.
Patient was noted to have significant hypokalemia during hospitalization (also throughout pregnancy) of unsure etiology.

DIFFERENTIAL DIAGNOSIS: Initial differential included tracheal stenosis secondary to mass or infection and asthma exacerbated by pregnancy. Biopsy of the tracheal mass confirmed adenocystic carcinoma, likely of minor salivary gland origin.

TREATMENT: Patient underwent tracheostomy and is scheduled for surgical resection of the tumor with total laryngectomy and possible total thyroidectomy due to 10 mm T2 hyperintense cyst/nodule seen in right thyroid lobe. Hypokalemia managed with potassium replacement.

CONCLUSION: No new abnormalities or evidence of progressive or nodal metastatic disease in the soft tissue neck. The patient was educated about post-op changes to voice, swallowing, and breathing as well as the possibility of both hypothyroidism and hypoparathyroidism. Future steps include alaryngeal vocal rehabilitation with an electrolarynx and tracheoesophageal prosthesis.
Despite multiple visits for shortness of breath and concern of neck mass, this patient's malignancy was not identified until acute presentation of respiratory failure. Patient's history of obesity may have played a role in the prolonged evaluation of the cause of tracheal stenosis.

WHAT'S WORSE, DYSPNEA OR DIARRHEA? | BENJAMIN GOW-LEE MD, PGY2

Identification: Our patient is a 66-year-old man with a history of coronary artery disease, atrial fibrillation, and type two diabetes who presented in septic shock with dyspnea and diarrhea.

History: Our patient felt well until the day before presentation when he began feeling nauseous, feverish, and weak. He developed chills that night. The next morning, his fatigue worsened and he developed a nonproductive cough, dyspnea, diarrhea with watery stools, and confusion prompting his presentation. He had a sick contact about 10 days prior to admission, but both wore a mask and sat far from each other. He had recently traveled to Idaho for a fishing trip and to Arizona.

Physical abnormalities: Initial exam was notable for a temperature of 39 degrees Celsius, blood pressure of 92/49 mmHg, and respiratory rate of 35. Exam was significant for a nonfocal neurological exam and bilateral basilar crackles. Labwork was remarkable for a leukocytosis to 13,500 g/dL with a neutrophil predominance, sodium of 132 mmol/L, creatinine of 1.6 mg/dL, and a lactate of 2.3 mmol/L. A respiratory virus PCR panel was negative. Chest x-ray revealed a patchy opacity over the left midlung.

Differential diagnosis and course: Our differential diagnosis included community acquired pneumonia and gastroenteritis. Given his dyspnea, diarrhea, and hyponatremia, legionella pneumonia was especially considered. He was empirically treated with ceftriaxone and azithromycin. In the emergency room, his blood pressure fell requiring vasopressors and he developed a 3L oxygen requirement. He was admitted to the ICU for septic shock. His pressure and oxygen requirement quickly improved after ICU admission and he was soon downgraded. His urine legionella antigen test returned positive, and he was prescribed 2 additional days of azithromycin for a 5-day total course.

Discussion: *Legionella pneumophila* infection often presents as a rapidly progressive severe pneumonia often accompanied by hyponatremia, thrombocytopenia, diarrhea, and relative bradycardia. It is transmitted via water aerosols and has been detected in rainwater and other freshwater sources; case rates have been seen to increase after rain. However, cooling towers and other water systems still produce the most frequent sources of *Legionella* in community-acquired outbreaks.

Conclusion: Clinicians should be aware of the various symptoms of Legionella pneumonia and *Legionella*'s presence outside of human made water systems.

OLMESARTAN INDUCED ENTEROPATHY | DHARMIK JADVANI MS4, PATEL MD, PGY2, MEENU SINGH MD

FINALIST

Identification: Patient is a 58-year-old male

Chief Complaint: watery non-bloody diarrhea, abdominal pain, weight loss

History: Patient is a 58-year-old male with PMH of hypertension, GERD, and hypothyroidism presented with watery, non-bloody diarrhea and abdominal pain for three months. Diarrhea started three months prior while the patient was vacationing in Greece. He used tap water for drinking, denied any sick contacts. The stool was watery, yellowish and nongreasy with associated weight loss of 35 lb over three months.
EGD(with biopsy) a week ago showed mild erosive gastritis, ruled out celiac sprue and Colonoscopy showed diverticulosis and a single 3mm sessile polyp. Empiric metronidazole did not provide much relief. Diarrhea was acutely worse a day before admission. Patient denied any fever, vomiting or melena. Home medications included Olmesartan, levothyroxine and metoprolol.

Physical Abnormalities: Vitals stable, exam pertinent for dry mucous membranes and hyperactive bowel sound without any abdominal tenderness.

Labs/imaging: Abnormal: K-2.9, Cr-1.59, TSH-86mU/L, Free T4-0.7 ng/dl, Lactoferrin-positive, Stool osmolarity-365, Calprotectin-331 ug/g, AST-58, ALT-67. **Normal:** Hb-16, WBC-5.23, Thyroxine-4.83ug/dl. Stool studies for C-difficile, Ova and parasite, Salmonella, Shigella, Campylobacter jejuni, E-coli, Vibrio, Aeromonas, Plesiomonas, Giardia antigen were negative. VIP, Gastrin, Calcitonin, 5HIAA, Pancreatic-elastase, TTG IgA- Ab, CRP, fecal fat, LFTs WNL. CT abdomen pelvis-no significant findings.

Differential Diagnosis/Treatment: Workup ruled out infection, inflammation, pancreatitis, secretory tumour, celiac sprue and allergy. None of the interventions provided any relief except holding Olmesartan, which provided significant relief.

Discussion: Olmesartan medoxomil, the prodrug of Olmesartan, is an angiotensin II receptor blocker (ARB). In 2013, the United States FDA reported that Olmesartan can produce a "sprue-like enteropathy" characterized by severe chronic diarrhea and weight loss, occurring months to years after initiation of the drug, with higher risk after 2 years of use[1]. In a French cohort of 4,546,680 patients who initiated therapy with Olmesartan, or a different ARB, or an ACE inhibitor[1]. Intestinal malabsorption severe enough to cause hospitalization occurred substantially more often among patients taking Olmesartan for one to two years (adjusted risk ratio 3.7, 95% CI 1.8-7.3) and among those taking Olmesartan for more than two years (adjusted risk ratio 10.6, 95% CI 5.0-22.5) compared with ACE inh[1]. Secondary to Olmesartan being a common anti-hypertensive medication, it is important to spread awareness of this side effect to patients and providers.

References:

1. Basson M, Mezzarobba M, Weill A, Ricordeau P, Allemand H, Alla F, Carbonnel F. Severe intestinal malabsorption associated with olmesartan: a French nationwide observational cohort study. Gut. 2016 Oct;65(10):1664-9. doi: 10.1136/gutjnl-2015-309690. Epub 2015 Aug 6. PMID: 26250345.

WHEN OCCAM'S RAZOR DEVOLVES INTO HICKAM'S DICTUM | ROMA
KOVTUN MS4, ETHAN KRAUSPE MD, PGY2, BENJAMIN HARRIS MD, C

FINALIST

Case Description: A 73-year-old male with myasthenia gravis and rheuma arthritis (on intravenous immunoglobulin, eculizumab, azathioprine, prednisone) and interstitial lung disease presented to the ED complaining of six months of dyschezia and intermittent watery diarrhea that had become bloody over the last two weeks. He reported >3 daily episodes, noting intermittent passage of clots with abdominal pain and subjective fevers. Physical exam, review of systems, and initial work-up with basic labs and imaging were unrevealing, and the patient was discharged from the ED with a plan for outpatient colonoscopy. Stool cultures returned positive for *Campylobacter upsaliensis/helveticus* several days later, and patient was instructed to return to the ED, where he reported worsening symptoms, was hypotensive with mild lactic acidosis and pancytopenia. He was admitted to general medicine for management.

Differential: History, acuity, and immunocompromised state direct a wide infectious differential. The presumptive diagnosis was campylobacter enteritis, given his stool cultures and positive PCR. Non-infectious etiologies were considered but thought to be unlikely given the unremarkable physical exam and CT scan.

Treatment and Trajectory: Infectious disease recommended IV meropenem resulting in several days of improved symptoms. However, the patient continued to have intermittent fevers with negative CT chest, abdomen, pelvis, and blood cultures. Coverage was broadened to caspofungin and ivermectin. Cytomegalovirus (CMV) was considered, but empiric ganciclovir was deferred for fear of worsening pancytopenia. Two days later, the CMV viral load resulted with >1 million copies/ml and ganciclovir was started. Unfortunately, the patient acutely decompensated during this timeframe, requiring ICU transfer and intubation. Bronchoalveolar lavage samples were positive for CMV suggesting viral pneumonitis, although CT imaging was inconsistent. Colonoscopy demonstrated friable and inflamed mucosa throughout the colon with areas of ulceration in the rectum and biopsy positive for CMV colitis. He responded to ganciclovir with decreasing viral load and was clinically improving towards extubation. However, he self-extubated, electing for comfort care and passing away several days later.

Discussion: Given the relatively high incidence of campylobacter infections (19.5/100,000[1]), positive PCR studies, and positive stool cultures, the patient was certainly suffering from this infection. However, given the initial response and later worsening in the setting of correct treatment, a second disease was highly likely. CMV is a common cause of bloody diarrhea in the immunocompromised and, in hindsight, starting empiric treatment when the patient was worsening was likely the best course of action.

Sources:

1. Centers for Disease Control and Prevention. Foodborne Diseases Active Surveillance Network (FoodNet). https://wwwn.cdc.gov/foodnetfast/ (Accessed on October 1, 2022).

WHAT'S THAT IN YOUR BRAIN? | JOHN MARSIGLIO MD, PGY2, B. HARRIS MD, CMR

FINALIST

Case Presentation: Patient is a 27-year-old female with no past medical history who began to develop significant fatigue and dizziness along with progressive shortness of breath over a 4-week period. At her initial presentation a week prior to our encounter with her, she was told she had a blood clot in her IVC and fluid in her lungs. She was sent home on rivaroxaban but her shortness of breath worsened and she began to develop leg and abdominal swelling. She followed up with her PCP, bloodwork showed elevated Cr and LFTs, ultrasound suggested possible abdominal mass. She was subsequently referred to the emergency department. Labwork confirmed acute liver and kidney injury. She was admitted to medicine for workup and management.

Physical Abnormalities: Exam was notable for reduced breath sounds in bilateral lungs, R>L. Tachycardic. Abdomen distended, right flank pain. Pitting edema in lower extremities bilaterally.

Lab and Imaging Results: CT chest confirmed the presence of IVC thrombus extending into the right atrium, along with bilateral pleural effusions. A CT abdomen showed ascites along with a 9.7 x 14.8 x 17.6 cm right retroperitoneal mass plus enhancing liver lesions. Initial labs notable for hyponatremia, hyperkalemia, elevated creatinine, elevated AST/ALT/alk phos with normal bilirubin, elevated LDH.

Differential Diagnosis: High concern for malignancy, review of scan indicating likely adrenal origin. Adrenocortical carcinoma likely based on radiological appearance, with pheochromocytoma on differential. Workup notable for normal metanephrine/normetanephrine, modest elevation in androgens and modest elevations in cortisol/24-hour cortisol. PET CT showing hypermetabolic activity in thrombus, liver lesion biopsies without malignancy.

Treatment: Her IVC thrombus was treated with heparin, after stabilization she underwent resection of right kidney along with mass. Thrombectomy also performed, malignant cells in thrombus. Pathology confirmed diagnosis of adrenocortical carcinoma with tumor thrombus, negative atrial margins. Treated with hydrocortisone taper post-operatively. She was discharged in stable condition with Oncology follow-up to discuss chemotherapy.

Conclusion: IVC tumor thrombosis is rare but known complication of adrenocortical carcinoma and can, along with mass effect symptoms, be the primary presenting issue of this tumor as only 50-60% are have enough hormone secretion to cause symptoms. Although data is limited, resection of the mass and tumor-associated thrombus is important in management in a similar fashion to renal cell carcinoma due to improvement in outcomes.

WHEN ITCHING AND "ECZEMA" FORESHADOW SOMETHING BIGGER | ALLISON MCCLAIN MD, PGY3

Identification and Chief Complaint: The patient is a 53-year-old man with no significant past medical history, with 10 months of worsening itching relieved only by spending 23 hours a day in the bathtub.

Case presentation: He initially presented to his PCP for itching and a rash on his chest in November 2021. He trialed topical steroids, oral steroids, antihistamines, antibiotics, Fluconazole, permethrin, gabapentin, without improvement. He saw dermatology May 2022, was diagnosed with prurigo simplex, and was given gabapentin, triamcinolone, most recently cyclosporine.
At presentation (September 2022) he reported about 50 lbs of weight loss and admitted to having night sweats for several months. He then mentioned a swollen lymph node in his R axilla, which appeared sometime in April, and was recently biopsied at OSH, but did not have the results for yet.

Physical Abnormalities: Our exam was notable for hundreds of excoriated papulonodules and ill-defined plaques spread diffusely over most skin surfaces, sparing the mucosa surfaces, genitals and lower back. R lateral chest wall had a large, non-tender, mass with poorly defined borders. No other palpable lymph nodes.

Results: WBC 13.77 with neutrophil predominance, abs eos of 580, Hgb 15.9, Plt 454. Chemistry revealed normal electrolytes, kidney function, LFTs <2x ULN with nl bili. Protein of 8.5, Albumin of 3.3. Uric acid of 9.5, LDH of 1254. PET CT showed avid adenopathy in the right axilla extending into the supraclavicular and right subpectoral region. Skin biopsy showed prurigo simplex. Core biopsy of mass revealed

Differential Diagnosis: Initial history was suspicious for malignancy with paraneoplastic itching. We considered soft tissue sarcoma, osteosarcoma; hematologic such as lymphoma; or lung cancer with local invasion. We also considered separate diagnoses such as a large irregular lipoma, with some poorly controlled allergic skin reactivity. Chest wall mass showed immature non-hodgkins lymphoma, further classified at peripheral t-cell lymphoma.

Treatment: Initially started on prednisone 100mg x5 days as well as allopurinol. Then transferred to HCH and had induction chemo with BVCHP. The itching and lesions never responded to multimodal therapy and per chart review, appears to be finally improving with chemotherapy.

Conclusion: Although commonly discussed with malignancies such as polycythemia vera, T-cell lymphoma, late onset itching with skin lesions, can be a warning sign of malignancies, presenting up to months before first diagnosis. Skin biopsy on its own was unrevealing, showing prurigo simplex an otherwise idiopathic/inflammatory disease process in many cases, but was paraneoplastic in this case.

A LITTLE MORE THAN SHORTNESS OF BREATH | APARNA NAIK OMS2, MARIA AVAKOVA OMS2

Identification: 22-year-old male
Chief Complaint: Acute dyspnea
History: Patient traveled to Park City from Mississippi for skiing. Patient had trouble sleeping the night before and developed dyspnea and productive cough in the mountains. In a local clinic, his saturation was in the 70s, which prompted a trip to the ED. Patient experienced a similar episode previously while hiking at high altitude. PMH/FH non-contributory, denied smoking/vaping.
Physical Abnormalities: CT scan notable for bilateral ground glass opacities, absent right pulmonary artery, and hypoplastic right lung.
Lab Results: HR 123; RR 32; SpO2 74%, WBC 18.2 K/mcl, Hb 19.4 g/dL, Hct 57.8%
Differential Diagnosis: On admission, the symptoms were consistent with pneumothorax. Patient was set up for a chest tube, but the ED physician proceeded with a CT scan, which came back negative for pneumothorax. Elevated WBCs and CT findings raised suspicion for an infection. Negative for COVID- 19.
Case Presentation: Patient was airlifted to Utah Valley Hospital for higher level of care. ICU physician determined that the CT findings could indicate high-altitude pulmonary edema. TTE showed longstanding pulmonary hypertension. Patient was transferred to the floor on 2nd day of admission. He was still experiencing significant hypoxia on room air and was kept on 1L of O2.
Treatment: Patient received ceftriaxone, azithromycin in the ED and started on IV diuretics, nifedipine for high-altitude pulmonary edema. Discharged home on day 6 with DME oxygen therapy and nifedipine. Advised to follow up with a pulmonologist and avoid high altitude.
Discussion: Unilateral absent pulmonary artery (UAPA) is a rare congenital anomaly (1 in 200,000 cases). UAPA results from the sixth aortic arch failing to connect with the pulmonary trunk (1). Isolated absence of the right pulmonary artery is twice as common as that of the left pulmonary artery. Since patients are asymptomatic at birth, most UAPA cases are adults. Due to its rarity and variety of clinical presentations, UAPA poses a diagnostic challenge (2). As patients become older, they can develop dyspnea, high- altitude pulmonary edema, and pulmonary hypertension (2). 44% of patients have pulmonary hypertension, which can lead to heart failure (2). UAPA remains a therapeutic challenge. For pulmonary hypertension, vasodilator therapies such as calcium channel blockers (i.e., Nifedipine) and prostacyclin have been used (3).
Conclusion: Early recognition of isolated UAPA is essential to avoid the devastating effects of longstanding pulmonary hypertension. When a newborn exhibits signs of cardiorespiratory concern, prompt cardiological and respiratory evaluations are needed.

References

1. Mohan V, Mohan B, Tandon R, Kumbkarni S, Chhabra ST, Aslam N, Wander GS. Case report of isolated congenital absence of right pulmonary artery with collaterals from coronary circulation. Indian Heart J. 2014 Mar-Apr;66(2):220-2. doi: 10.1016/j.ihj.2013.12.032. PMID: 24814121; PMCID: PMC4017382.
2. Raymond, A., Pedretti, E., Privitera, G. *et al.* Neonatal diagnosis of isolated absence of the right pulmonary artery: a case report and review of the literature. *Ital J Pediatr* 44, 27 (2018). https://doi.org/10.1186/s13052-018-0465-1
3. Seedat F, Kalla IS, Feldman C. Unilateral absent pulmonary artery in an adult - A diagnostic and therapeutic challenge. Respir Med Case Rep. 2017 Sep 14; 22:238-242. doi: 10.1016/j.rmcr.2017.09.004. PMID: 28951831; PMCID: PMC5604951.

SOMETIMES YOU HAVE TO DO SOME PALM-READING | CASSIDY NGUYEN MS3, GUILHERME KUCEKI MS3

Identification: 67 year old female

Chief Complaint: Elbow lesions

History: A 67 year old female here for a skin exam. She is concerned about scar-like lesions on her bilateral elbows that she has been chronically dealing with for several years. Patient has a history of essential hypertension. Family history is notable for high cholesterol in her father and mother.

Physical Abnormalities: On examination, nodular xanthomas are present on elbows, soles of feet, and along the lateral nail folds of feet. Additionally, she presents with palms with interdigital planar xanthomas on the palmar/finger creases.

Lab Results: Lipid panel resulted in cholesterol 595 mg/dl. LDL Cholesterol 147 mg/dl. HDL 24 mg/dl. Triglycerides 885 mg/dl.

Differential Diagnosis: Presumed diagnosis was dysbetalipoprotenemia. Labatory findings supported this diagnosis with elevated serum cholesterol and triglyceride levels as well as a strong family history. However, myeloma and amyloidosis could also present with nodular xanthomas.

Treatment: Tricor 45 mg was prescribed. However, LDL apheresis should be considered if refractory to fenofibrate treatment.

Case Presentation: Patient is a 67-year old woman. She is concerned about scar-like lesions on her bilateral elbows that she has been chronically dealing with for a few years. Patient has a history of essential hypertension. Family history is notable for high cholesterol in her father and mother. Nothing notable on a review of systems. At her last PCP visit, patient was diagnosed with dyslipidemia, and had tried several statins unsuccessfully.

Discussion: Familial Dysbetalipoproteinemia, or Type III hyperlipoproteinemia, is a genetic disorder that predisposes patients to early onset atherosclerosis, acute pancreatitis, and premature cardiovascular disease. Patients with FD have an estimated ten-fold increase in risk compared to controls of developing cardiovascular disease. Additionaly, severe hypertriglyceridemia is estimated to increase the risk of acute pancreatitis twelve-fold. The pathogenesis is caused by an accumulation of lipoprotein remnants due to an erroneous expression or deficiency of apoliporotein E (apoE), which serves as a ligand for LDL receptor. The clinical presentation of FD is variable with approximately half of patients developing cutaneous xanthomas. Pathognomic findings include lipid deposition in palmar creases. Lipoprotein remnants induce inflammation, which increases the permeability of vessels causing eruptions of tuberous xanthomata.

Conclusion: Familial dysbetalipidemia should not be overlooked when screening and ordering routine lipid panels. It is important to screen for xanthemotous skin presentations, which can detect dyslipidemia and dysbetalipoproteinemia even with lipid measurements that are in an acceptable range.

KNOCKING OUT TWO VERTEBRAL ARTERIES WITH ONE GIANT CELL | MARY PENG MS3

Case Presentation: A 77-year-old woman presented to the emergency department with a 5-day history of feeling unsteady. Computed tomography angiography (CTA) showed the string-of-beads sign classically associated with fibromuscular dysplasia (FMD) in her bilateral internal carotid arteries (ICAs) and a left vertebral artery dissection (VAD). Magnetic resonance imaging (MRI) of the brain revealed acute left thalamic ischemic stroke. The patient was subsequently started on dual antiplatelet therapy (DAPT) with aspirin and clopidogrel. Three weeks later, the patient returned with acute onset left-sided hemiplegia and ataxia, and a stroke code was initiated on arrival. Imaging showed she had a new dissection and occlusion of the right vertebral artery and redemonstration of multifocal beading of the bilateral ICAs. DAPT was switched to a heparin drip, and the patient was admitted for further evaluation.
Upon further inquiry, she reported having abdominal pain with 15-pound weight loss 3 months prior and bitemporal headaches 2 weeks before presentation. She denied any recent fevers, night sweats, vision changes, or arthralgias.

Physical exam: Left-sided hemiparesis.

Lab and imaging findings: Hemoglobin 11.7 mg/dl, Platelets $486\times10^3/\mu L$, Erythrocyte Sedimentation Rate (ESR) 56 mm/h. White blood cell count, comprehensive metabolic panel, and cerebrospinal fluid (CSF) analysis were unremarkable. Digital subtraction angiography revealed multifocal areas of narrowing involving the basilar artery and bilateral intradural vertebral artery segments. These vessels showed postcontrast circumferential vessel wall enhancement on vessel wall MRI.

Differential diagnosis: The presumed diagnosis was giant cell arteritis (GCA); other diagnoses under consideration included varicella zoster virus vasculopathy and herpes simplex virus vasculitis. The patient met three of five criteria listed by the American College of Rheumatology 1990 (age ≧50 years, new headache, and elevated ESR).[1] Together with the imaging findings, and negative CSF results, she was diagnosed with GCA.

Treatment: The patient received IV methylprednisolone 1g for 5 days, followed by tocilizumab and oral prednisone taper starting at 60mg. Heparin was discontinued and DAPT was re-initiated with the plan to continue clopidogrel for 30 days. She was discharged in stable condition after 1 week of hospital stay to a rehabilitation facility.

Discussion: GCA, typically seen in adults older than 50 years, is a rare cause of VAD that can progress to vertebrobasilar ischemia.[2] This case of GCA, which initially mimicked the findings of FMD, highlights the importance of broadening differentials when patients develop unexpected clinical courses. In conclusion, GCA should be considered in elderly patients presenting with spontaneous VAD.

References

1. Hunder GG, Bloch DA, Michel BA, Stevens MB, Arend WP, Calabrese LH, et al. The American College of Rheumatology 1990 criteria for the classification of giant cell arteritis. *Arthritis Rheum* 1990; 33:1122-8.

2. Zwicker J, Atkins EJ, Lum C, Sharma M. An atypical presentation of giant cell arteritis. CMAJ. 2011 Mar 22;183(5):E301-5. doi: 10.1503/cmaj.100380. Epub 2011 Feb 7. PMID: 21324853; PMCID: PMC3060216.

NOT YOUR FATHER'S ENDOCARDITIS | ERIC SHEFFIELD MD, PGY2

Case Presentation: The patient is a 76-year-old male with a PMH including atrial fibrillation, HFpEF, tachy-brady syndrome status-post dual-chamber pacemaker, severe OHS/OSA, pHTN, T2DM, HTN, and CAD with an extensive hospitalization history.

On initial admission, he had blood cultures that grew ESBL *Klebsiella pneumoniae* thought to be due to parotitis. His blood cultures cleared on carbapenems; he had a PICC placed and underwent a 2-week course of ertapenem as an outpatient.

He re-presented 7 days after completion of these antibiotics with a heart failure exacerbation. Blood cultures revealed recurrence of ESBL *Klebsiella pneumoniae* bacteremia. CT neck showed parotid enlargement with no inflammation, abscess, or other evidence of acute infection, making the source unclear. Repeat cultures after initiating meropenem were without growth. The initial plan was for 6 weeks of meropenem.

The patient had a prolonged hospitalization requiring extensive diuresis. As the patient approached a euvolemic state, he underwent TEE with cardioversion at which time we asked the echo team to also evaluate his pacemaker leads. TEE showed a mobile, 1 cm long, filamentous echo-density attached to right atrial lead and another highly mobile, 0.6 cm long, filamentous mass on the right ventricular lead at the ostium of the superior vena cava.

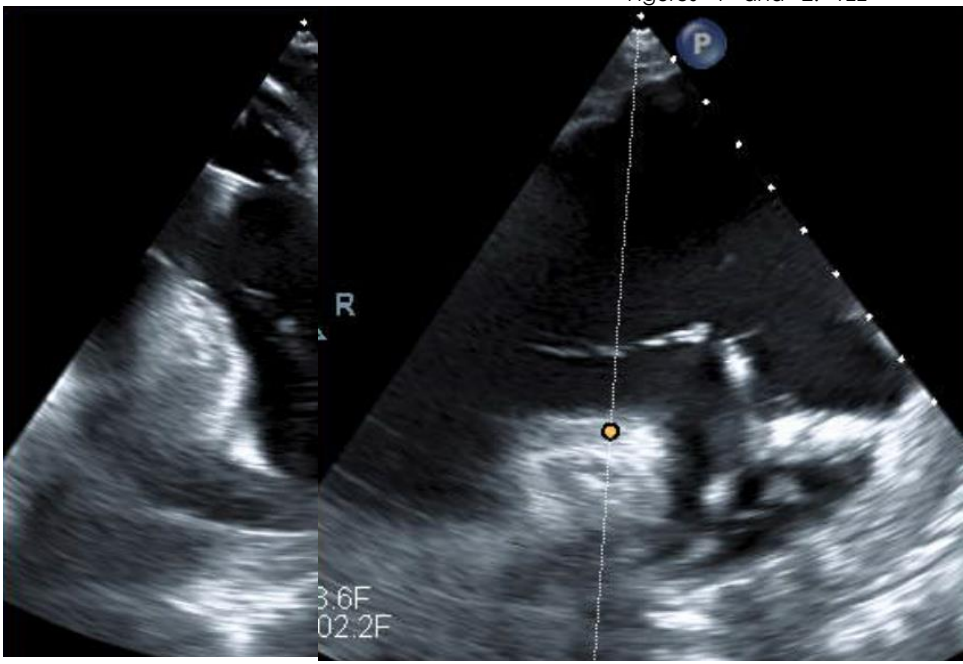
Treatment: Electrophysiology and Infectious Disease were re-consulted at the time of pacemaker lead endocarditis discovery. Infectious disease recommended continuing meropenem. After a discussion with the consulting medical teams and patient, a consensus was reached to pursue pacemaker explantation. The current plan is for pacer lead extraction and re-implantation of a leadless pacer. There are no clinical guidelines regarding antibiotic therapy after pacemaker lead removal [1], so the tentative plan is for 2 weeks of meropenem therapy after pacemaker removal.

Discussion: ESBL *Klebsiella Pneumoniae* is a gram-negative organism that has become more prevalent throughout the United States, especially in frequently hospitalized populations [2], but is rarely found to be the cause of infective endocarditis. ESBL *Klebsiella pneumoniae* endocarditis of pacemaker leads is a clinical picture that has not yet been reported in medical literature. While ESBL Klebsiella endocarditis already presents a challenging clinical scenario, involvement of the pacemaker leads further complicates management. As we see more patients with cardiac devices it is very likely that we will see an increased prevalence of unusual or resistant infections involving these devices, specifically in frequently hospitalized patient populations.

Sources:

1. Sohail, Muhammad R., et al. "Management and Outcome of Permanent Pacemaker and Implantable Cardioverter-Defibrillator Infections." Journal of the American College of Cardiology, vol. 49, no. 18, 5 May 2007, pp. 1851–1859., https://doi.org/10.1016/j.jacc.2007.01.072.
2. Thaden, Joshua T., et al. "Increasing Extended-Spectrum β-Lactamase-Producing Escherichia Coli in Community Hospitals throughout the Southeastern United States." Open Forum Infectious Diseases, vol. 2, no. 1, 2015, https://doi.org/10.1093/ofid/ofv131.152.

Figures 1 and 2: TEE



showing filamentous vegetations on the pacemaker leads within the right atrium

WHEN GUT HEALTH BECOMES HEART-UNHEALTHY: A CASE OF *LACTOBACILLUS RHAMNOSUS* ENDOCARDITIS | LOGAN SHELNUTT MD, PGY2

Identification: 68-year-old male with history of amphetamine use, Hepatitis C, and COPD

Chief Complaint: 3 days of lower extremity rash, feet/hand pain & swelling

History: The patient initially presented as a direct admission from clinic with a painful and progressive bilateral rash. Further questioning revealed the patient had experienced 3 other intermittent episodes of this rash over the last year. These were treated with prednisone on the emergency basis without PCP follow up.

Accompanying the rash, he noted bilateral foot and hand swelling, as well as dyspnea with exertion. He had no fevers. He had no known prior cardiac history. He had been treated for *S. Pneumoniae* bacteremia at an outside hospital 6 months prior to presentation, but TTE was negative at that time.

Physical Abnormalities: 1+ lower extremity edema, purpuric non-blanchable macules and papules on bilateral lower extremities. Linear hemorrhagic streaks were noted on the fingernails. A partially reducible direct inguinal hernia was additionally noted.

Lab Results: ESR 60, CRP 2.8. WBC 5.8.

Differential Diagnosis: Initial differential included infectious endocarditis, large and small-vessel vasculitis, TTP, and ITP.

Case Presentation: Blood cultures revealed *Lactobacillus rhamnosus* bacteremia on 4/4 cultures. TTE demonstrated bulky vegetation on the septal leaflet of the tricuspid valve. Follow-up TEE revealed TV and PV vegetations with questionable AV abscess vs. inflammation.
After consultation, his rash was ultimately felt most consistent with IgA Vasculitis, likely triggered by underlying Hepatitis C infection. The differential diagnosis also included a drug reaction to prior fluoroquinolone use, though he declined biopsy of the affected lesions.
Workup for a source of bacteremia revealed gum disease and nonspecific tissue enhancement in the hepatic vein. HIV was negative. The primary team's hypothesized *L. rhamnosus* translocation into the bloodstream from his inguinal hernia. He denied any recent consumption of over the counter probiotics, as well as any recent GI procedures.
Patient was initially treated with IV Penicillin G and Gentamicin. Penicillin was planned to be continued for 6-8 weeks from discharge. Cardiothoracic Surgery evaluated the patient for valvular replacement and ultimately determined he was not a surgical candidate due to comorbidities.

Conclusion: While *L. Rhamnosus* is most commonly a probiotic strain with multiple health benefits, it is uncommonly pathogenic and may trigger serious infections in immunocompromised hosts with significant comorbidities. Treatment options include high dose penicillins with or without aminoglycosides. It is associated with a high mortality in susceptible patients and its presence on blood cultures should guide further investigation.

WHEN "SATURDAY NIGHT PALSY" ENDS UP AS INVASIVE PULMONARY ASPERGILLOSIS | LOGAN SHELNUTT MD, PGY2

Identification: 54-year-old male with history of heart transplant, CKD 3, Hepatitis C s/p treatment, DMII

Chief Complaint: 1 day of L hand weakness, fever, chills, productive cough
History: This patient presented to the emergency department for 1 day of hand weakness. He reported going to bed the night before feeling more cold than usual, then waking up with a numbness and weakness in his left wrist. He additionally reported a measured fever to 100.5. This fever was accompanied by a new, productive cough of brownish sputum. He endorsed complete adherence to his post-transplant regimen.

He did note a potentially related event with nausea and vomiting the week prior. He had attributed this to food poisoning. He had no chest pain, edema, palpitations, blurry vision.

He had been staying locally in a hotel since his transplant. He lived in Oregon prior to transplant, but had worked as a trucker with travel everywhere in the continental US. He did have recent dental work 2 weeks prior to presentation without antibiotic prophylaxis.

Physical Abnormalities: Regular rate and rhythm, L-sided 3/5 weakness to wrist extension with numbness to sensation in radial distribution, coarse breath sounds bilaterally.

Lab Results: WBC 4.65 without eosinophilia. 1,3-BDG >500, Aspergillus Galactomannan serum Ag POSITIVE

Differential Diagnosis: Initial differential included infectious endocarditis, community acquired pneumonia, fungal/bacterial/viral CNS infection, atypical bacterial infection, community acquired pneumonia, tuberculosis

Case Presentation: Chest radiography was obtained and revealed round opacities in the left upper and left lower lung. Follow up CT chest was obtained and revealed new, multifocal bilateral round opacities concerning for atypical infection, TB, or neoplastic process.
Bronchoalveolar lavage was performed and respiratory fungal cultures (and serum studies) were positive for *Aspergillus fumigatus.* He was discharged on Voriconazole with planned treatment until resolution.
With patient's hand weakness, after negative workup for central causes including MRI brain, he was diagnosed with a "Saturday night palsy" resultant from a flexed posture during sleep and unrelated to his *Aspergillus* pneumonia.

Conclusion: In the immunocompromised, post-transplant patient presenting with upper respiratory symptoms, full workup is crucial to identify invasive pathogens early. Here, we present a case of invasive *Aspergillus* pneumonia presenting with an acute onset and rapid diagnosis and treatment. This case illustrates the importance of maintaining a broad differential and not anchoring prematurely on a patient's initial complaint (in this case, hand weakness).

SUMMERTIME SICKNESS | JARROD SMITH MD, PGY2

Introduction/Chief Complaint: A 63-year-old man with a history of anoxic brain injury, colon cancer, polysubstance use disorder, and recurrent UTI presented in early September with altered mental status and generalized weakness.

Case Presentation/History: History was obtained from the patient's wife, as his neurologic baseline is orientation only to self and location. She described one week of somnolence, decreased interactivity, and poor appetite. He had generalized weakness leading to a ground-level fall. He often has similar symptoms with UTIs, but had no urologic symptoms. He was started on prophylactic trimethoprim 2 months ago. ROS was notable for occasional headaches, a non-pruritic rash, and a recent bug bite.

Physical Exam: He was febrile to 38°C. He was lethargic and disoriented. Strength was diffusely diminished without focal deficit. Mild nuchal rigidity was present. Petechiae were present on all extremities on a background morbilliform eruption. There was a small erythematous papule on the left forearm.

Lab Results: CBC showed mild thrombocytopenia. CMP and TSH were normal. Lumbar puncture drained 13 mL of clear CSF with opening pressure of 27 mmHg. CSF studies showed 115 WBC (50% neutrophils, 36% lymphocytes), protein 112, and normal glucose. Culture, gram stain, and meningitis/encephalitis PCR panel were negative. CSF West Nile virus (WNV) IgM level was elevated, and WNV meningitis was diagnosed.

Discussion: WNV is endemic in the US. It causes febrile illness in 25% of patients and neuroinvasive disease in < 1%. Patients 60 and older have increased risk for neuroinvasive disease and increased mortality. WNV is underdiagnosed; one study found only 5% of patients seeking medical care were correctly diagnosed. Diagnosis is with CSF or serum IgM, not IgG or PCR. Typical CSF studies show a mild lymphocytic pleocytosis, normal glucose, and mildly elevated protein. Warmer temperatures correlate with increased incidence, which is highest from July through September. Treatment is supportive, and no vaccine is approved. Utah has seen a rapid increase in WNV incidence. In 2020, 0.008% of mosquito trap sites were positive; this figure increased to 8% in 2021, leading to 21 cases of neuroinvasive disease. Rising temperatures and rainfall fluctuations may play a role in this increase.

Conclusion: WNV is an underdiagnosed pathogen, and older patients are at increased risk for neuroinvasive disease. Providers in endemic areas such as Utah, which has recently experienced a rapid rise in WNV incidence, should maintain a high index of suspicion for WNV infection, particularly during summer months.

References:

1. Petersen LR, Brault AC, Nasci RS. West Nile virus: review of the literature. JAMA. 2013 Jul 17;310(3):308-15.
2. Paz S. Climate change impacts on West Nile virus transmission in a global context. Philosophical Transactions of the Royal Society B: Biological Sciences. 2015 Apr 5;370(1665):20130561.
3. https://epi.health.utah.gov/west-nile-virus/
4. https://epi.health.utah.gov/wp-content/uploads/2021/11/WNV_WeeklyReport_103021.html

Identification: A 27-year-old male with no significant past medical history presented with 5 weeks of yellowish discolouration of skin, persistent right upper quadrant abdominal pain, nausea and vomiting. He also reported generalized fatigue, diarrhea, mental cloudiness and an inability to follow conversations. He had been incarcerated for six months and received a tattoo in prison ~5 weeks prior with a "dirty needle". He denied any prior or current use of IV illicit substances, alcohol, personal or family history of liver diseases, or sexually transmitted diseases.

Physical Abnormalities: Patient appeared diffusely jaundiced with icteric sclerae. He also reported tenderness to deep palpation diffusely throughout his abdomen, was bradycardic, and had vitiligo in both hands. He did not have any asterixis and was alert and oriented to person, place, time, and situation.

Initial Lab Results: Labs were notable for total bilirubin 18.7, AST 1704, ALT 2582, ALP 171, Lipase 21, and INR 1.8. WBC, Hgb and Platelets were within normal limits. CT abdomen pelvis showed small volume ascites, periportal/gallbladder wall edema, and periportal lymphadenopathy indicative of acute hepatitis. Viral hepatitis panel was notable Hep C PCR quantification of 291K IU/mL and positive Hep C Ab but otherwise negative for Hep A, B, and E serology.

Differential Diagnosis: New-onset acute liver failure has a broad differential, including infectious causes (EBV, (EBV, CMV, Adenovirus, VZV, and HSV), autoimmune, genetic (Wilson's and Hemochromatosis) and toxin-related causes.

Further testing: A liver biopsy was performed. Findings showed pseudosepta formation from hepatitis, dropout of hepatocytes and reticulin framework collapse. There was severe panlobular lymphocytic hepatitis with marked lobular hepatocyte swelling, cholestasis, spotty hepatocyte necrosis, and marked Kupffer cell hyperplasia. No iron overload was seen, and no alpha-one anti-trypsin inclusions. Pathology was also negative for CMV, Adenovirus, and EBV stains. ***Findings were consistent with acute liver failure due to Hepatitis C.***

Treatment: Hep C treatment is predicated on genotype. In the absence of genotype information, a 12-week regimen of Sofosbuvir-Velpatasvir (Epclusa) is preferable, which is what this patient was started on.

Conclusion: Acute liver failure due to Hepatitis C without any pre-existing liver disease or Hepatitis B is an exceedingly rare diagnosis, not been seen by most internists during their career. It is estimated that the rates of Hep C-induced acute liver failure are < 0.5% globally. This is because Hepatitis C virus typically tends to clear spontaneously, with more than two-thirds of patients being asymptomatic during the acute episode and others having very mild symptoms.

Case Presentation: A 76-year-old woman with a history of tobacco use presented to the Emergency Department with increasing weakness and fatigue. The patient endorsed unintentional falls, weight loss, constipation, and back pain. Vitals were significant for tachycardia and a room air saturation of 86%. On exam, the woman was thin and disheveled. Lung sounds were clear. Heart was tachycardic without murmurs. No abdominal masses were appreciated. No lymphadenopathy was appreciated.

Laboratory evaluation was notable for Sodium of 132, Creatinine of 1.05, Calcium of 14.0, Total protein of 9.0, and Albumin of 4.5. CBC was unremarkable. CT Chest with IV contrast demonstrated multiple sub-segmental Pulmonary Emboli and multiple thoracic compression fractures.

Further investigation revealed a PTH of 12, PTHRP of 3.6 (ULN 3.4), normal Vit D 25 OH, and low Vit D 1,25 OH. Given high concern for malignancy, a PET scan was ordered and revealed no obvious underlying malignancy. SPEP was significant for an elevated serum Kappa Light Chain of 34.6, serum Lambda Light Chain of 0.24, and an elevated serum Kappa/Lambda ratio of 144.17. M-spike 1.17 g/dL of 1.3 g/dL in gamma region. Bone Marrow Biopsy showed 50% monoclonal plasma cells and hypercellular marrow 60%. Multiple Myeloma MRI showed diffuse micronodular bony disease.

The patient was treated inpatient with aggressive fluid resuscitation with subsequent improvement in hypercalcemia. Her pulmonary emboli were treated with therapeutic subcutaneous enoxaparin. She was discharged home, and soon started on Cyclophosphamide, bortezomib, and dexamethasone.

Discussion: In this case, PE was suspected and diagnosed early in the course given her hypoxia and tachycardia on presentation. The diagnosis of multiple myeloma may have been overlooked if not for the incidental hypercalcemia.

The rate of VTE in MM independent of treatment is likely due to a procoagulant state caused by abnormal production of cytokines and acute phase reactants and inhibition of normal fibrin structure in the setting of elevated immunoglobulins [1]. Given the growing data and case reports of MM presenting as PE, we suggest considering a MM workup when the etiology of PE is unclear, as it is treatable and can make a substantial difference in patient morbidity and mortality.

[1] Zangari M, et al. (2003) The Blood Coagulation Mechanism in Multiple Myeloma. Seminars in Thrombosis and Hemostasis; 29(3): 275-282.

CYTOMEGALOVIRUS VIREMIA-ASSOCIATED HEMOPHAGOCYTIC LYMPHOHISTIOCYTOSIS | HEIDI WELLENSTEIN MS4

FINALIST

Case Presentation: A 47-year-old female with Crohn's disease, treated with azathioprine, presented to an outside hospital with abdominal pain, diarrhea, bilious emesis, and progressively worsening lethargy. She was found to be febrile and pancytopenic, with a lipase of 1200. She was started on broad-spectrum antibiotics and intravenous fluid resuscitation for sepsis of unknown source and suspected azathioprine-induced pancytopenia and pancreatitis. Despite these interventions, she remained persistently febrile and hypotensive, prompting her transfer to a tertiary hospital.

Upon arrival, the patient was febrile, tachycardic, and hypotensive (78/48). Physical exam was notable for diffuse abdominal tenderness. Initial laboratory evaluation revealed pancytopenia (WBC 2.99, Hgb 9.0, platelets 79), hypertriglyceridemia (triglycerides 341), LDH of 585, albumin of 2.6, and negative HIV and viral hepatitis screenings.

The patient remained hypotensive despite aggressive intravenous fluid resuscitation. She was transferred to the intensive care unit and started on intravenous steroids, and broad-spectrum antimicrobials (vancomycin, meropenem, and caspofungin).

Additional laboratory analysis revealed: fibrinogen of 106, ferritin of 7,558, positive CMV IgM and IgG, and CMV viral load of 2,490,000.

The patient's persistent fevers, markedly elevated ferritin, hypertriglyceridemia, anemia, thrombocytopenia, and hypofibrinogenemia raised concern for hemophagocytic lymphohistiocytosis (HLH) in the setting of severe CMV viremia. Bone marrow biopsy revealed rare hemophagocytosis, low natural killer cell activity, and an elevated soluble CD25 receptor level of 66,400, further supporting the diagnosis of HLH.

The patient was started on intravenous ganciclovir and continued on high-dose intravenous steroids. The patient's blood pressure normalized, fevers gradually resolved, and serum ferritin declined. She was ultimately transitioned to oral ganciclovir and her most recent CMV viral load was undetectable. Azathioprine was replaced with vedolizumab, which has adequately controlled her Crohn's disease.

Discussion: The patient was diagnosed with CMV viremia with associated hemophagocytic lymphohistiocytosis (HLH). It was believed the patient's azathioprine, in addition to her Crohn's disease, predisposed her to develop an active CMV infection and associated viremia, leading to secondary HLH[2]

Thiopurines are an effective drug class in inducing and prolonging remission in patients with IBD. However, thiopurines increase the risk of developing opportunistic infections and patients should be routinely monitored for drug toxicity, including lymphopenia. HLH should be considered in patients with IBD with persistent fevers that are unresponsive to antimicrobials. Viral infections, including CMV, are the most common triggers of HLH in patients with IBD[3].

[1]Qiu Y, Mao R, Zhang SH, Li MY, Guo J, Chen BL, et al. Safety profile of thiopurines in Crohn disease: analysis of 893 patient-years follow-up in a Southern China cohort. Medicine. (2015) 94:e1513. 10.1097/MD.0000000000000151

[2]Li Y, Xia X, Zhang J, Song Z, Zhou L, Zhang Y, Huang Y, Shi Y, Quigley EM, Ding S. Haemophagocytic lymphohistiocytosis in inflammatory bowel disease with virus infection. Prz Gastroenterol. 2015;10(2):78-82. doi: 10.5114/pg.2015.48995. Epub 2015 Oct 2. PMID: 26557937; PMCID: PMC4631275.

CAMPING WITH A SIDE OF ELEVATED CREATININE | KAYLA WOODRING MS2

Introduction: A 23-year-old previously healthy male college student presented to Utah Valley Hospital with a two-week illness that began after camping in Moab, Utah. Initial symptoms of cough and myalgia progressed to include fevers, night sweats, extreme fatigue, headache, arthralgias, mouth sores, pleuritic chest pain, nausea/vomiting, and episodes of shortness of breath with cyanosis of the digits.

Case Presentation: A few days prior to admission, he was seen by his primary care provider who suspected a viral syndrome for which the patient took ibuprofen. When symptoms continued, he went to InstaCare and tested negative for influenza, COVID-19, EBV, and RSV. Labs then showed elevated BUN and creatinine and decreased GFR. He was instructed to increase fluid intake due to suspected dehydration. Despite doing so, symptoms worsened.

Eventually, the patient presented to the emergency department where labs revealed acute renal failure with a creatinine of 4.5 mg/dL for which he was admitted to the hospital. Throughout admission, systemic markers of inflammation were consistently elevated, including WBC of 15-16.5K/uL, ESR of 40-50 mm/hr, and CRP values of 14-21 mg/dL. Chest imaging showed bilateral pleural effusions and bibasilar opacification/atelectasis. Social history was notable for part-time work transporting deceased bodies for a local mortuary, always with PPE.

Consultations with ID, nephrology, and rheumatology services were arranged. Studies for HIV, viral hepatitis, hantavirus, legionella, toxoplasmosis, fungi including coccidiodes, and various autoimmune antibodies were all negative. Continuous IV fluids led to improvement in his creatinine, which was 1.97 at discharge. Following negative infectious workup, steroids were initiated. A renal biopsy revealed necrotizing, granulomatous interstitial nephritis. He was discharged with instruction to follow up with ID, nephrology, and rheumatology.

Discussion: At discharge, a rheumatologic process was considered most likely since the infectious workup was unrevealing. While autoimmune studies were negative, the consulting rheumatologist emphasized that antibody serologies can become positive after initial presentation. Thus, anti-glomerular basement membrane disease, sarcoidosis, systemic lupus erythematosus or other autoimmune/autoinflammatory disorders were entertained since the patient demonstrated multi-organ (including pulmonary) symptoms. Though ibuprofen may have contributed to his renal injury, drug-induced nephropathy would not have explained his myriad symptoms. Due to follow-up received outside our hospital system, the final diagnosis is unknown. Nevertheless, this case exemplifies a multidisciplinary approach in response to a broad differential arrived at through thoughtful clinical reasoning critical for all internists.

FINALIST

HPI: The patient is a 63 y/o male with a PMH of RA on MTX, HTN, insomnia who presented with agitation, aggression, poor sleep, and chest pain. He was challenging to communicate with and, at times, delirious and confused. He notes that he recently started zolpidem 2-3 days ago for insomnia. He also states he has had multiple episodes of intermittent chest and jaw pain. Upon further questioning, he has been unable to sleep longer than 2-3 hours per night, developed new grandiose thinking, and completed many projects around the house. He has a history of occasional alcohol consumption, daily marijuana use for 10-20 years, and a 40-pack year smoking history, quit in 2003. He does not use any other illicit drugs.

Physical Abnormalities: Vital signs were notable for hypertension (160s/100s), otherwise hemodynamically stable. Physical exam was significant for anxious and agitated affect, with grandiose thinking. Neurological exam was unremarkable. No other pertinent exam findings were identified.

Lab/Imaging Results on admission: The patient was found to have hypokalemia, hypernatremia, leukocytosis, and elevated creatinine. High sensitivity troponin was elevated, and TSH was low. The urine tox screen was positive for marijuana and no other substances. Brain CT and MR imaging were unremarkable.

Hospital Course: The patient was initially worked up for neurological causes of agitation and delirium, including LP and viral testing, which all came back negative. Psychiatry was consulted, and he was started on anti-psychotic medications, which seemed to improve his mood overall. However, he remained persistently hypokalemic and developed hypernatremia with persistent hypertension. Chest CT showed a solitary lung nodule unchanged in size since the previous CT. On initial laboratory testing, the Aldosterone level was elevated, possibly suggesting hyperaldosteronism. Endocrine was consulted, and 24-hour urine cortisol is 9948 with ACTH elevated to 226. The diagnosis of ACTH-dependent Cushing's disease secondary to ectopic ACTH production was then established. The patient was started on ketoconazole for profound hypercortisolism and discharged to follow up for definitive management with lung nodule resection.

Discussion: This is an interesting case presentation of overt hypercortisolism secondary to ectopic ACTH presenting as metabolic disturbances and manic episodes. Furthermore, his poor sleep before admission could also have been secondary to these findings—common psychiatric complaints associated with hypercortisolism range from mild mood disturbances to manic activity. Interestingly, psychiatric symptoms are not always resolved after correcting excess cortisol levels, suggesting possible long-term neurological damage if not quickly treated.