

Clinical Vignette Competition

2020



UTAH ACP RESIDENTS & FELLOWS COMMITTEE

Emily Signor, MD – Chair

Marja Anton, MD

Jon Harrison, MD

Eric Moore, MD

12:00 PM	WELCOME & OPENING REMARKS Residents & Fellows Committee	JUDGES Dani Babbel, MD Roxanne Weiss, MD Matt Mulligan, MD Sonny Win, MD
12:10 PM	PRESENTATIONS	
	The Sunburn that was really Stevens-Johnson-Syndrome <i>Presented by: Blake McKinley [OSM3]</i>	Pg. 10
	COVID-19 and Clots: Using Multimodality Imaging to Uncover Mechanisms of Myocardial Infarction <i>Presented by: Joel Money, MD [PGY3]</i>	Pg. 12
	Tophaceous Gout: Red Herring or Real Herring <i>Presented by: Bianca Rich [MS4]</i>	Pg. 15
	An Unusual Case of Encephalopathy in a patient with advanced Diffuse Large B-Cell Lymphoma <i>Presented by: Scott Rizzi, MD [PGY1]</i>	Pg. 18
12:50 PM	ANNOUNCE RUNNERS-UP AND 1ST PLACE	
1:00 PM	CLOSING COMMENTS <i>Residents & Fellows Committee</i>	

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 – ASIM 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.

TABLE OF CONTENTS

FALL CLINICAL VIGNETTE PROGRAM THURSDAY, OCTOBER 08, 2020	1
Utah ACP residents & fellows committee mission statement.....	1
Where is the Malar Rash? Tyson Broadbent, MS4	3
Bradycardia and syncope: An unusual presentation of a cannot miss diagnosis Elise Brunsgaard, MD & Gina Allyn, MS3	4
A Wolf Disguised as a B (SYMPTOM) Anna Cassell, MS4	5
Stranger than Pulp Fiction: the true case of a therapeutic needle to the heart Jason Chen, MS4	6
Acute Myeloid Leukemia Causing Acute Thrombosis of Coronary ARTERIES Meganne Ferrel, MS3	7
Diagnostic Splenic RUPTURE Gibson klapthor, MD.....	8
Pancytopenia in a Middle-Aged Woman with Failure-to-Thrive Ben Harris, MD	9
The Sunburn That Was Really Stevens-Johnson Syndrome Blake Mckinley, Oms3 .	10
COVID-19 and Clots: Using Multimodality Imaging to Uncover Mechanisms of Myocardial INFARCTION Joel money, MD	12
Life is Not Always a BEACH Beck nelson OSMIV	14
Tophaceous Gout: Red Herring or Real HERRING Bianca Rich, MS4.....	15
When In Doubt, Steroids Will Fix IT Deema Sallout, MD	17
An unusual cause of encephalopathy in a patient with advanced stage Diffuse Large B-Cell Lymphoma Bryan Still, MD & Scott Rizzi, MD.....	18
Basal Cell C-aarrrrr-cinoma: A Common Cancer Presenting as Failure to Thrive and Scurvy Michael Zou, MD.....	19

Case Description: A previously healthy 18-year-old Polynesian female presented to an outside hospital with acute encephalopathy and hypoxemia in the setting of recent weakness and weight loss. She reported an abrupt onset of fatigue, poor appetite, and proximal muscle weakness approximately two months prior to admission which progressed to frequent falls, shortness of breath, and lethargy. She was subsequently admitted to the ICU for respiratory failure and encephalopathy and transferred to our institution. She was initially treated with broad spectrum antibiotics for CNS infection, however, her infectious evaluation was negative. She demonstrated improvement in her mental and respiratory status with supportive care but remained with significant proximal muscle weakness.

Pertinent Physical Exam Findings: Upon arrival to our hospital, physical examination revealed proximal and paraspinal muscle weakness, mild epigastric tenderness to palpation, hyperpigmented papules on her hands and feet, and normal mental status. She underwent extensive laboratory evaluation which was notable for pancytopenia with lymphopenia, acute liver injury, profound lipase elevation, and CK elevation. Her thorough infectious workup was negative. Her ANA was positive at 1:320 with low C3/C4. C-ANCA was positive at 1:320 with equivocal MPO and negative PR-3. Her myositis panel and autoimmune encephalitis panels were negative. APLA panel was negative. Spine MRI demonstrated diffuse paraspinal muscle edema. Skin biopsy showed intense granular deposition of multiple immunoglobulins in blood vessels, consistent with SLE. Muscle biopsy was inconclusive due to limited sample obtained. EMG was consistent with a diffuse myopathic process.

Differential Diagnosis: The presumed etiology of her muscle weakness and pancreatitis was SLE with myositis overlap as she met 2019 ACR/EULAR criteria with +ANA, fever, cytopenias, and low C3/C4 as well as evidence of SLE on skin biopsy. Other inflammatory myopathies were inconsistent with her serologic profile. Cryoglobulinemic vasculitis was considered due to her positive cryoglobulin screening but follow up testing was negative. ANCA-associated vasculitis was also considered given her positive ANCA, however, she did not demonstrate other features of ANCA-associated vasculitis. No infectious cause of myositis was identified.

Discussion: Prednisone 40mg and hydroxychloroquine 300mg daily until follow up appointment.

Conclusion: In summary, the most likely explanation of her acute presentation is SLE with myositis overlap and autoimmune pancreatitis on presentation, leading to respiratory failure and encephalopathy. Myositis and pancreatitis are relatively uncommon symptoms of SLE, occurring in 7-15% and 2-8% of patients, respectively, but may be the only manifesting symptoms of the disease.

BRADYCARDIA AND SYNCOPE: AN UNUSUAL PRESENTATION OF A
CANNOT MISS DIAGNOSIS | ELISE BRUNSGAARD, MD & GINA ALLYN,
MS3

Introduction: A 79-year-old male with a history of hypertension, right bundle branch block (RBBB), and coronary arteriosclerosis with stable angina presented following a syncopal episode. The patient woke up feeling nauseated, dizzy, and diaphoretic. Upon standing, he fainted and was unconscious for about a minute. He was fully responsive when he returned to consciousness, but had a second syncopal episode with walking prior to the paramedics arriving.

In the emergency department, he was hypotensive with positive orthostatic testing, but responded well to intravenous fluids. He endorsed continued nausea, headache, and foot pain, but denied confusion, shortness of breath, chest pain, palpitations, numbness, or weakness.

Case Description: On admission, he was afebrile with a blood pressure of 123/61, bradycardic to 49, respiratory rate of 16, and saturating 94% on room air. No cardiac murmur or neurologic deficits were appreciated on physical exam. During ambulation with pulse-oximetry monitoring, the patient had a brief period of hypoxemia to 80% accompanied by dizziness that resolved with rest.

Lab work showed mild hypokalemia and negative troponin. EKG was remarkable for sinus bradycardia and RBBB. CT head and neck was unremarkable. X-ray of the right foot showed several non-displaced fractures. Chest x-ray showed mild linear left lung base opacity, likely due to atelectasis. An echocardiogram showed mild changes consistent with hypertension. COVID testing was negative.

Discussion: The initial differential diagnosis included vasovagal syncope, orthostatic hypotension, metabolic abnormality, arrhythmia, acute coronary syndrome, or seizure. Arrhythmia was considered most likely given his sinus bradycardia and intermittent RBBB. However, this did not explain the hypoxemia with ambulation. Given his age and unexplained hypoxemia, a D-dimer was sent and returned significantly elevated. A chest CT angiogram was performed and showed multiple lobar, segmental, and subsegmental pulmonary embolisms.

Since the patient remained clinically stable and there was no evidence of right heart strain, he was determined to be appropriate for outpatient management. He was discharged on apixaban with follow-up in the anticoagulation clinic and primary care for work-up of an underlying cause.

Pulmonary embolism presenting with symptomatic bradycardia is rare, particularly in a patient with no other known risk factors for thromboembolism aside from age. However, this case demonstrates the importance of considering pulmonary embolism in patients with unexplained hypoxemia even in the absence of tachycardia or other traditional signs or symptoms of VTE given the impact on treatment and risk of morbidity and mortality if the diagnosis is missed.

Introduction: A previously healthy 25 year old female presented to the ED with two weeks of nightly fevers to 39.7 degrees.

Pertinent Physical Exam Findings: History: A previously healthy 25-year-old woman presented to the ED with two weeks of nightly fevers up to 39.7 degrees. She also reported two months of weakness, dizziness, palpitations, dyspnea on exertion, syncope, and 25 lbs. of weight loss. On the day of admission, she developed a painful mouth sore, but denied any additional sores or rashes. She endorsed mild joint pain in the mornings, but denied swelling. She denied bleeding gums and nose bleeds, but identified easy bruising.

Physical Abnormalities: Exam notable for diffuse adenopathy in her neck and bilateral axilla, with a 2x2 cm hard, nonmobile lymph node appreciated in her R axilla. She had mild erythema on her face that appeared to be acne. She had ecchymoses-like purpura on both elbows and knees and a draining oral abscess on the R lower mandible.

Lab and Imaging Results: An initial workup demonstrated the following: WBC 2.8, Hgb 11.1, platelets 108. ESR 118 and CRP 1.6. Lactate was 2.4. Fibrinogen 445 and LDH 352. UA and CMP were unremarkable. HIV, GCCT, Hep B/C, and QuantiFERON Gold were negative. Abdominal CT showed "Iliac and retroperitoneal lymphadenopathy with splenomegaly."

Differential Diagnosis: The patient's symptoms, physical exam findings, and labs were thought to be due to hematologic malignancy. Infectious etiologies including HIV and TB were also considered.

Hospital course: A lymph node core needle biopsy of an axillary lymph node was obtained, which showed a benign lymph node with reactive features. She next had a bone marrow biopsy on this day, which was negative. Due to ongoing concern for hematologic malignancy, a PET scan was completed, showing multiple hyperactive lymph nodes concerning for malignancy. An excisional lymph node biopsy of an inguinal lymph node showed a benign lymph node with reactive features.

Given the two negative lymph node biopsies and the negative bone marrow biopsy, a rheumatologic workup was pursued with ANA screening, which was positive with a titer of 1:1280. A more extensive workup for Lupus revealed positive SSA 52, SSA 60, and dsDNA antibodies and positive Anti-RNP, Anti-Smith, and Anti-La. She was diagnosed with Systemic Lupus Erythematosus.

Discussion: B-symptoms, lymphadenopathy, and pancytopenia are an important, but uncommon, presentation of Lupus.

Conclusion: When working up a young patient with B-symptoms it is important to consider at test for autoimmune and rheumatologic etiologies.

STRANGER THAN PULP FICTION: THE TRUE CASE OF A THERAPEUTIC NEEDLE TO THE HEART | JASON CHEN, MS4

Introduction: TW is a 61F with 2nd degree HB s/p pacemaker, history of cervical cancer and polio s/p below R knee amputation who presents with 1 week of progressively worsening fatigue.

Case description: She is concerned about fatigue that requires her to sleep until 3 PM. She is a current smoker and reports a 50 PY history. She notes increased cough but denies home O2. Upon ROS, she reports 2 months of diarrhea and diffuse edema. Workup for C difficile was negative.

ED course: EKG: low voltage diffusely. LDH: 497. BNP: 4300. CXR: pulmonary congestion, cardiomegaly, and findings suggestive of RLL pneumonia and pericardial effusion. O2 drops to 84% on RA, so she is given 2 L NC, later titrated to 3L. Electrolyte repletion is begun. Blood cultures are collected before starting antibiotics.

Upon further questioning, she discloses a previous SLE diagnosis and growing up in East Carbon, Utah with limited medical care. She denies a history of MI, chest trauma, renal failure, or chest radiation.

Pertinent Physical Exam Findings: T 36.8 °C, HR 60, BP 123/85, RR 13, 98% 3 L NC

AOx4, high-pitched voice. Crackles at lung bases with expiratory wheeze, increased WOB with use of accessory muscles. Distant, muffled heart sounds, 1+ pitting edema up to mid shins.

Differential Diagnosis: Pericardial effusion 2/2 Malignancy vs CHF vs Viral vs TB vs Autoimmune

DISCUSSION: A pericardial effusion is promptly confirmed on TTE. However, due to the patient's history of cardiac issues, smoking, cervical cancer, SLE, polio, and limited childhood healthcare access, the etiology remains unclear. A diagnostic pericardiocentesis drained ~1L of straw-colored fluid. Initial malignancy workup is unremarkable: pericardial fluid smear revealed only occasional benign mesothelial cells. Infectious workup is also unremarkable: pericardial fluid culture is negative, as well as the ANA IFA, respiratory virus panel, and AFB stain.

Conclusion: This patient with multiple possible underlying etiologies required a broad workup. Repeat TTE showed severely depressed LVF with an EF of 25-35%, so CHF medical therapy was maximized. She was discharged with O2, lisinopril, spironolactone, carvedilol, and colchicine. Even though approximately 1/3rd of large pericardial effusions are idiopathic, large pericardial effusions may also be the presenting sign of unrecognized malignancy in 1/5th of patients with a nonrevealing basic workup. Thus, more extensive workup should be considered at cardiology follow up.

References: Hoit, BD. Diagnosis and treatment of pericardial effusion. In: UpToDate, LeWinter, Sexton, Yeon (Ed), UpToDate, Waltham, MA, 2020

ACUTE MYELOID LEUKEMIA CAUSING ACUTE THROMBOSIS OF CORONARY ARTERIES | MEGANNE FERREL, MS3

Case Description: A previously healthy 59-year-old female presented to her PCP with a 2-week history of severe fatigue, anorexia, and generalized malaise. Patient endorsed chills, night sweats, and myalgias during this time period. Prior to her visit to her PCP, she visited urgent care and was treated for urinary tract infection. She tested negative for COVID. Complete blood count was not performed. She had no relevant medical, surgical, or family history. Physical exam revealed pallor, mildly decreased breath sounds in right base, and mild left calf tenderness without swelling.

Pertinent Physical Exam Findings: CBC showed a white blood cell count of 79.05 k/uL with hemoglobin of 11.2 g/dL and platelets of <6 k/uL. CT imaging obtained on presentation to emergency department demonstrated widespread thrombotic phenomenon with infarcts in spleen, bilateral kidneys, right lung, and liver in addition to a right segmental pulmonary embolism. Peripheral blood smear was consistent with Acute Myeloid Leukemia (AML) with blast crisis and disseminated intravascular coagulopathy.

Discussion: The patient was transferred to the intensive care unit, where she was treated for AML and a heparin drip was initiated. On hospital day 2, patient acutely decompensated. This is visualized from the normal baseline ECG at 07:35 am (Figure 1) progressing to upsloping ST depression consistent with acute myocardial infarction (MI) at 07:37 am (Figure 2). Patient experienced an episode of ventricular tachycardia (VT) at 07:39 am (Figure 3), which resolved and progressed to diffuse ST elevation at 07:48 am (Figure 4). Patient then developed VT at 07:49 am (Figure 5). During this time, brain attack was called, unfortunately, due to pulseless electrical activity in the setting of suspected ST elevation myocardial infarction and cerebrovascular accident with significant clot burden despite therapeutic heparin, reversible causes appeared limited and she was pronounced deceased shortly thereafter. Terminal arrhythmia was recorded at 07:58 am (Figure 6).

Conclusion: This case is notable as a case of AML causing acute thrombosis of coronary arteries with rapid progression from normal sinus rhythm to acute MI to terminal rhythm within a period of 20 minutes. It is uncommon to capture acute dramatic terminal cardiac changes of this quality. In addition, although this is a dramatic presentation of AML, an earlier diagnosis could likely have been made if a CBC were obtained at the time that the initial COVID was performed.

Case Description: 58 year-old woman with ESRD on HD and numerous medical comorbidities presenting with generalized weakness and acute on chronic anemia. She was at her baseline state of health until 1 week ago when she started experiencing progressive weakness, dizziness with standing, and passage of dark stools. On day of presentation, she attended HD and tolerated it well. However, her CBC revealed severe anemia prompting transfer to the ED.

Pertinent Physical Exam Findings: Chronically ill appearing and tachycardic. Rectal exam revealed dark stool with occult blood. Stat Hgb was 4.4.

Lab and Imaging Results: Given concern for GI bleed, she underwent EGD and colonoscopy. EGD demonstrated severe nodular gastritis with acute on chronic bleeding. Colonoscopy was unremarkable.

Post-procedure, she complained of abdominal pain, but serial exams were benign. She was hypotensive and remained anemic with Hgb of 5 and was given 2u PRBC. Hgb was 7.4 after transfusion.

The next day she underwent HD. During dialysis, she developed Vfib arrest. ROSC was obtained following initiation of ACLS. Stat labs showed Hgb of 4. She received 2u of PRBC, was intubated, and transferred to MSICU. CT was obtained which demonstrated a ruptured spleen with hemoperitoneum. There was also a heterogeneously enhancing retroperitoneal mass measuring 5.5 cm. She was taken for emergent splenectomy.

Differential Diagnosis: Given its appearance on CT, differential diagnosis for the mass was paraganglioma vs GI stromal tumor (GIST) with less likely considerations including: schwannoma, leiomyoma, and carcinoid tumor. MIBG scan demonstrated mild radiotracer uptake within the mass compatible with a paraganglioma. Serum metanephrine was 0.21 nmol/L (reference range: 0.00-0.49) and normetanephrine was mildly elevated to 1.69 nmol/L (reference range: 0.00-0.89).

Discussion: Given concern for a functional paraganglioma, alpha-adrenergic and beta-adrenergic blockade were administered prior to surgical en-bloc resection. Surgical resection was successful and immunohistochemical and molecular analytic techniques were performed which confirmed a diagnosis of GIST.

Conclusion: The most common mesenchymal neoplasms affecting the GI tract are collectively referred to as GISTs. These rare tumors have an estimated incidence 14.5 per million which is about 3-4x higher than the published incidence of paragangliomas. Both are highly vascular tumors that commonly present with heterogeneous enhancement on CT. Although MIBG scans have an estimated sensitivity of 88% and specificity of 84% for paragangliomas, GISTs are rarely known mimics. In addition, it is well demonstrated that patients with renal failure routinely have elevated serum metanephrine and normetanephrine due to decreased renal excretion.

Introduction: Pancytopenia has a broad differential that includes consumption, malnutrition, infection, medication side effects, and malignancy. Here we present a 58-year-old female who presented to the hospital from her care facility with a pancytopenia, fatigue, and ground-level falls. The patient had a previous history of gastric bypass 13 years prior with over 400 lbs of weight loss, 70 lbs in the last 6 months. She also had a history of atrial fibrillation, genital herpes, zinc and vitamin A deficiency, and a recent diagnosis of provoked DVT. She was on no marrow-suppressing agents. The patient was adopted, previously divorced, and currently living in Central Utah with her fiancé. She had never used intravenous drugs or any other illicit substances. She had not had any sexual partners since her ex-husband 17 years prior. Physical exam showed an anxious and extremely cachectic-appearing woman without bruising or signs of bleeding. Laboratory investigation showed a WBC of 3, hemoglobin of 6.4, and platelets of 59. Malnutrition work up showed a low vitamin C level, but was otherwise normal. A blood smear was without evidence of malignancy. DIC panel was negative. Urine culture was positive for methicillin-resistant *Staphylococcus aureus*. Due to the pancytopenia, failure to thrive, relative lymphopenia (10% of the leukocyte differential), and minimal inflammatory response in the setting of infection, a HIV test was sent and came back positive. Further physical exam showed a previously-unnoticed patch of hairy leukoplakia, as well as odynophagia. Viral load was greater than 1 million copies/mL and CD4+ T cell count was 27 cells/microliter. Patient opted against further work-up for opportunistic infections and was started empirically on fluconazole for presumed esophageal candidiasis, vancomycin for MRSA infection, as well as azithromycin and Bactrim prophylaxis. The patient followed up in AIDS clinic and was started on highly-active antiretroviral therapy (HAART). Induced sputum, which was deferred by the patient during hospitalization, was positive for PJP and the patient was treated with atovaquone. Six weeks after starting HAART her viral load was 32 copies/mL and a continued, though improved, pancytopenia with a CD4 count of 82. This case shows that HIV should be considered in patients presenting with pancytopenia or failure to thrive despite other possible etiologies or absence of "risk factors."

BLAKE MCKINLEY, OMS3

Introduction: Chief Complaint: "Blistering Sunburn"

Stevens-Johnson syndrome (SJS) is a cutaneous reaction characterized by epidermal detachment, most commonly triggered by medications.

Case Description: Patient is a previously healthy 18-year-old Polynesian female who presented with what she believed was a severe sunburn. She experienced a sudden onset of itching, burning rashes on her shoulders the day after extensive sun exposure while swimming with some friends. The rash continued to spread to her wrists, palms, trunk, lower extremities, and eventually involved the ocular and buccal mucosa. The rash progressed to form blisters, at which time she started taking oral ibuprofen and acetaminophen for pain. Extensive lesions in her buccal mucosa lead to significant odynophagia. After five days of her condition worsening, patient presented for medical treatment. At the initial onset of the rash, the patient states that she was not taking any prescriptions, over-the-counter medications, or dietary supplements. However, seven to ten days before onset, she had taken a single dose of tramadol for hip pain.

Pertinent Physical Exam Findings: Patient was febrile at 38.7 degrees Celsius. Bullae formation and skin sloughing were confirmed as noted above, lesions as large as 15 cm by 8 cm. Extensive erythematous erosive lesions involved the lips, buccal mucosa, and oropharynx. Positive Nikolsky sign upon palpitation. CRP was high at 13.7 mg/L.

Differential Diagnosis: Presumed diagnosis of SJS. Deep shave biopsy pathological report consistent with SJS.

Treatment: Patient received fluid resuscitation with 2 liters of lactated Ringer's, then transferred via LifeFlight to burn center for treatment and recovery.

Discussion: Tramadol is not one of the traditional medications that is associated with causing SJS. Nevertheless, this was the only medication that was taken prior to symptom onset. Tramadol was taken 7-10 days before symptom onset, consistent with the proposed mechanism for SJS, a delayed hypersensitivity reaction. Furthermore, tramadol was recognized in a case-control study conducted in Europe as a cause of SJS with a reported relative risk of 201. NSAIDs, like ibuprofen, are more widely recognized as a cause of SJS, however, it would be unlikely that either ibuprofen or acetaminophen were responsible for this event due to their utilization after rash onset.

Conclusion: Physicians should be aware of the possibility of SJS as an adverse drug reaction to tramadol. This reaction should be considered when a patient presents with a new rash days after taking tramadol for the first time.

References:

1. Mockenhaupt, M., Viboud, C., Dunant, A., Naldi, L., Halevy, S., Bavinck, J. N., Flahault, A. (2008). Stevens-Johnson Syndrome and Toxic Epidermal

Necrolysis: Assessment of Medication Risks with Emphasis on Recently Marketed Drugs. The EuroSCAR-Study. *Journal of Investigative Dermatology*, 128(1), 35-44. doi:10.1038/sj.jid.5701033

COVID-19 AND CLOTS: USING MULTIMODALITY IMAGING
TO UNCOVER MECHANISMS OF MYOCARDIAL INFARCTION
JOEL MONEY, MD

FINALIST

Case Description: A 59-year-old man with a history of diabetes mellitus type 2, hypertension, and class III obesity was hospitalized with COVID-19 after being in close contact with his spouse who was also infected. After 3 days of inpatient care, he was discharged to home. Two days later, he presented to the emergency department after waking up with severe substernal chest pressure that was relieved with sublingual nitroglycerin.

Pertinent Physical Exam Findings: Initial vital signs were normal. Physical exam was notable for faint upper lobe crackles but a normal cardiovascular exam. Initial cardiac troponin-I was 0.10 ng/mL and peaked at 19.03 ng/mL. Initial ECG revealed new diffuse T-wave inversions.

Lab and Imaging Results: MULTIMODALITY IMAGING EVALUATION:

A transthoracic echocardiogram revealed new basal/mid septal wall motion abnormalities corresponding roughly to a septal perforator vascular distribution.

The patient was then given aspirin, clopidogrel, enoxaparin, carvedilol, and atorvastatin. He underwent Rb-82 PET stress test one week later, which revealed a small/medium sized fixed perfusion defect in the basal/mid aspect of the septal wall, corresponding to his echocardiography findings. I evaluate his coronary arteries, a CT coronary angiography was performed, which showed no evidence of obstructive epicardial coronary disease and a total coronary calcium score of zero.

Finally, to further investigate the pathophysiology of his MI, he underwent regadenoson stress cardiac MRI at 6 weeks, which revealed residual septal hypokinesis from the base to the mid left ventricle. Importantly, perfusion during both stress and rest was normal, but late gadolinium enhancement imaging demonstrated patchy transmural pattern of enhancement of the base to mid septum, indicating the site and extent of permanent injury/scarring.

Discussion: Myocardial injury is being increasingly recognized as an important finding in patients with COVID-19. Several mechanisms have been proposed, including direct viral myocardial injury, lymphocytic myocarditis, epicardial coronary artery occlusion, and microvascular dysfunction/microthrombi.

Here, we show the ability of PET perfusion imaging to demonstrate regional perfusion defects associated with NSTEMI in the absence of obstructive coronary disease assessed by coronary CT angiography and with stress CMR showing recovered stress and rest perfusion. These findings suggest that the regional myocardial ischemia, associated with reversible microvascular dysfunction, was likely caused by microthrombi, which resolved by time of stress CMR at 6 weeks.

In support of the use of multimodality imaging in the care of COVID-19 patients with cardiovascular events, a JACC Scientific Expert Panel recently published recommendations on this topic, including proposed applications of echocardiography, PET perfusion imaging, and CMR(1).

References:

1. Rudski, L., et al., Multimodality Imaging in Evaluation of Cardiovascular Complications in Patients With COVID-19: JACC Scientific Expert Panel. J Am Coll Cardiol, 2020. 76(11): p. 1345-1357.

Case Description: A healthy sixty-six-year-old male underwent elective arthroscopic shoulder repair and remained unresponsive postoperatively. Past medical history includes hypertension controlled with metoprolol. Patient underwent routine preoperative and intraoperative phases. Patient received anesthetic block to the shoulder, versed with fentanyl for sedation, and was carefully placed in the Beach Chair Position. There were no reported intraoperative complications and patient was transferred to postoperative care in stable condition.

During postoperative care, patient had an episode of hypertension corrected with nicardipine. Patient remained unresponsive with an unknown etiology and was closely monitored by anesthesiology. Flumazenil and Narcan were administered to reverse benzodiazepine and opioid action, respectively. Patient failed to improve, neurology and hospitalist consults were obtained, and an extensive workup ensued. Initial laboratory and imaging studies were unremarkable.

Pertinent Physical Exam Findings: Exam was notable for diminished neurologic response. Patient did not follow commands, eyes opened with noxious stimuli. Fundus was without papilledema, pupils were equal and reactive, doll's eyes/corneal/cough/gag reflexes were positive. Tone and bulk were normal, but movement to noxious stimuli was absent. Reflexes were 2+ bilaterally in upper and lower extremities. We were unable to assess coordination, balance, gait, and posture.

Lab and Imaging Results: Laboratory and imaging studies were unremarkable on day 0. MRI Brain w/ Contrast on day 1 showed abnormal increased T2 signal diffusely in the left basal ganglia with associated restricted diffusion. EEG on day 1 revealed slowed activity suggestive of mild encephalopathy. MRI Brain Rapid on day 3 showed worsened appearance of diffuse ischemia involving nearly the entirety of the gray matter of bilateral frontal and parietal lobes suggesting global anoxic brain injury. Additional laboratory and imaging studies were unremarkable.

Differential Diagnosis: Presumed diagnosis was acute encephalopathy secondary to global anoxic brain injury. This was suggested by diffuse ischemia on day 3 MRI Brain. Further laboratory and imaging studies helped rule out stroke, meningitis, and syphilis, among other potential differential diagnoses.

Discussion: After reaching a final diagnosis and having a discussion with patient's family, it was agreed upon to pursue comfort measures based on patient's wishes and the fact that long-term prognosis appeared grim and would require aggressive measures. Patient subsequently developed and succumbed to aspiration pneumonia on day 6.

Conclusion: Arthroscopic shoulder surgery is a common orthopedic procedure and is routinely performed utilizing the Beach Chair Position. A rare and devastating complication of the Beach Chair Position is cerebral hypoperfusion and the potential for subsequent neurologic sequelae including stroke and brain death.

Case Description: A 51-year-old man with a history of tophaceous gout, osteoporosis with vertebral compression fractures, and recent initiation of vitamin D supplementation presented with chest pain, dyspnea, fatigue and weakness. On arrival he was mildly encephalopathic, endorsed back pain, and a rapid 32lb unintentional weight loss.

Pertinent Physical Exam Findings: Vital signs were stable. Pertinent exam findings included confusion, lethargy, facial plethora, a dorsocervical fat pad, central adiposity, diffuse gouty tophi on bilateral knees, elbows and MCP joints, and firm subcutaneous nodules scattered across the forearms and calves.

Initial serum calcium was elevated at 16 mg/dL, and iPTH was suppressed at 4 pg/mL. Cardiac evaluation for chest pain was unrevealing. He was initially treated with IV fluids and zoledronic acid for severe symptomatic hypercalcemia. While now known to be PTH-independent, the differential remained broad.

Differential Diagnosis: Hypervitaminosis D was considered with recent initiation of supplementation, however his 25-OH vitamin D was low at 17 ng/mL. We considered sarcoidosis and tuberculosis, but his 1,25-OH vitamin D was within normal limits at 46 pg/mL and serum QuantiFERON gold was negative. PTHrP returned elevated at 8.8 pmol/L, raising concern for hypercalcemia of malignancy. A PET CT was obtained and failed to show any lymphadenopathy or solid organ tumors, but did reveal increased 18-FDG uptake in his hands, wrists, and elbows where gouty tophi were located. SPEP, UPEP, and serum immunofixation were unremarkable. Given the patient's Cushingoid appearance, we performed an overnight 1mg dexamethasone suppression test, which showed a morning cortisol of 1.6 ug/dL and ACTH of 4.2 pg/mL, making cortisol excess or insufficiency unlikely as well. Serum ACE was elevated at 78 u/L.

A biopsy of a cutaneous nodule on his right thigh demonstrated gouty tophi surrounded by granulomatous inflammation, confirming our diagnosis.

Discussion: PTH-independent hypercalcemia is less common than PTH-dependent etiologies. The most common culprits were ruled out leading us to believe his hypercalcemia was related to granulomatous inflammation associated with chronic tophaceous gout in conjunction with immobility and the initiation of vitamin D therapy. While gout may cause hypercalcemia through enhanced 1 α -hydroxylation from granulomatous inflammation; rarely has a PTHrP mediated pathway been reported.¹ Interestingly, there is an existing body of literature describing PTHrP driven hypercalcemia in granulomatous diseases such as sarcoidosis² and disseminated coccidiomycosis³. Our case adds to this literature highlighting the potential of a granulomatous inflammation pathway with resultant PTHrP expression in the absence of malignancy.

References:

1. Sachdeva A, Goeckeritz BE, Oliver AM. Symptomatic hypercalcemia in a patient with chronic tophaceous gout: a case report. *Cases J.* 2008;1(1):72. Published 2008 Aug 7. doi:10.1186/1757-1626-1-72.
2. Krikorian A, Shah S, Wasman J. Parathyroid hormone-related protein: an unusual mechanism for hypercalcemia in sarcoidosis. *Endocr Pract.* 2011;17(4):e84-e86. doi:10.4158/EP11060.CR
3. Fierer J, Burton DW, Haghghi P, Deftos LJ. Hypercalcemia in disseminated coccidioidomycosis: expression of parathyroid hormone-related peptide is characteristic of granulomatous inflammation. *Clin Infect Dis.* 2012;55(7):e61-e66. doi:10.1093/cid/cis536

Case Description: A 49 year old healthy male with no past medical history presented to the ED after being referred from an urgent care facility for abnormal labs. He visited urgent care the previous day complaining of joint pain in his shoulders, wrists, knees, and ankles. Labs were drawn and he was prescribed a short course of steroids. The following day, he was asked to present to the ED because of abnormal kidney function tests.

The patient reported that he had taken 1 dose of prednisone 60mg QD which immediately relieved his symptoms. He endorsed no current joint pain, erythema, swelling, or decreased range of motion in the affected joints. He also denied any urinary or rheumatological symptoms including Raynaud's, rashes, and photosensitivity.

Pertinent Physical Exam Findings: Vitals were all within normal limits. Physical exam was largely unrevealing. All his joints appeared normal and were non-tender with normal ROM.

Lab and Imaging Results: "CBC was unremarkable. CMP showed creatinine 3.96 (previously normal), BUN 54, Calcium 12.2, and otherwise normal chemistry values. Urinalysis unremarkable. Rheumatological workup revealed positive ANA with speckled pattern and a titre of 1:80, positive RNP Ab, and negative anti-dsDNA, anti-smith, SSA/SSB, Scl-70, and anti-Jo. ESR 32. Complement levels and CRP were within normal limits. HIV, HBV, HCV, TB quant negative. ACE 126.

CT scan showed innumerable small irregular noncalcified nodules throughout both lungs with a predominantly perilymphatic distribution and mild mediastinal lymphadenopathy. Renal biopsy showed mild AIN, mild ATN, 10% fibrosis and granulomas in the interstitium."

Differential Diagnosis: Rheumatological disease, malignancy, infection and sarcoidosis were considered. Extensive laboratory workup was conducted and was mostly negative or non-specific. Given the result of the kidney biopsy and the lung CT scan, sarcoidosis was the most likely diagnosis.

Discussion: Upon admission, the patient was treated with IV fluids. His hypercalcemia resolved but his kidney function remained the same. Nephrology and pulmonology were consulted and the decision was made to treat the patient with a prolonged prednisone taper and close follow up. His kidney function improved after treatment with steroids and the patient was discharged with close outpatient follow up.

Conclusion: Sarcoidosis can present atypically. In our patient, the only symptom was joint pain although both lungs and kidneys were involved. Despite his pulmonary involvement not warranting treatment with glucocorticoids, the decision to treat with glucocorticoids was made to prevent further renal impairment.

AN UNUSUAL CAUSE OF ENCEPHALOPATHY IN A PATIENT
WITH ADVANCED STAGE DIFFUSE LARGE B-CELL LYMPHOMA
BRYAN STILL, MD & SCOTT RIZZI, MD

FINALIST

Case Description: A 69-year-old woman presented with progressive lethargy and encephalopathy of two weeks duration. Initial evaluation showed a calcium of 17.2 mg/dL and CT chest & abdomen showed bilateral neck and mediastinal adenopathy, as well as a 16cm by 16cm right lower quadrant abdominal mass. Core biopsy of the neck lymph node confirmed the diagnosis of Diffuse Large B-Cell Lymphoma (DLBCL). Brain MRI did not show acute changes or signs of leptomeningeal disease. The patient was treated for hypercalcemia with fluids, calcitonin, and bisphosphonates. Her hypercalcemia resolved to 10.8 mg/dL, but she remained encephalopathic.

Further workup for encephalopathy, including TSH, B12, urinalysis, blood cultures, ANA, and ammonia, was unremarkable. An initial lumbar puncture with administration of intrathecal methotrexate showed normal cell differential, glucose of 120 mg/dL, and protein of 48 mg/dL. Flow and cytology were negative for malignancy. Two additional lumbar punctures were performed and were negative for lymphoma. Repeat brain imaging was negative for CNS malignancy. CSF paraneoplastic autoimmune panel was negative. Her thiamine level was then found to be low at 3 nmol/L.

Pertinent Physical Exam Findings: The patient demonstrated confusion, was not oriented to time or place, and confabulated. She demonstrated significant ataxia but lacked focal neurological deficits. Her abdominal mass was firm and tender in the right lower quadrant.

Differential Diagnosis: The differential diagnosis of subacute encephalopathy included CNS involvement of lymphoma, systemic infection, viral or bacterial meningitis, prion disease, hospital-acquired delirium, paraneoplastic autoimmune phenomenon, and Wernicke's Encephalopathy (WE)/Wernicke-Korsakoff Syndrome (WKS).

Discussion: She initially received Prednisone for her lymphoma, without immediate improvement in mental status. Next, intravenous thiamine repletion was started (400mg q8hr for the first day, then 400mg daily), with rapid subsequent gait improvement. She was started on R-CHOP (Rituximab, Cyclophosphamide, Doxorubicin, Vincristine, Prednisone) with family members' consent. After receiving thiamine replacement and completing the first cycle of R-CHOP, her mental status and gait returned to baseline, with a repeat thiamine level two weeks later at 204 nmol/L.

Conclusion: Thiamine deficiency can develop in patients with malignancies and is likely secondary to increased utilization by rapidly growing cells. We recommend keeping thiamine-deficient states such as WE/WKS in the differential in patients presenting with a malignancy and altered mental status. Aggressive thiamine replacement and treating the underlying malignancy may result in improved mental status.

BASAL CELL C-AARRRRR-CINOMA: A COMMON CANCER PRESENTING AS FAILURE TO THRIVE AND SCURVY | MICHAEL ZOU, MD

Introduction: RW is an 80 year old man with a past medical history of basal cell carcinoma of the neck, severe lymphedema, and developmental delay with a chief complaint of dehydration and hypernatremia.

Case Description: The patient had been admitted to the hospital two weeks ago for encephalopathy. His encephalopathy was attributed to hypernatremia in the setting of developmental delay. The patient declined to go to a SNF as recommended.

Since that admission, the patient continued to have poor oral intake and difficulty speaking. In the interim, his neck mass was biopsied and found to be BCC. During follow up for his hospitalization, his PCP noted a sodium of 156 and told the patient to present to the ED.

Pertinent Physical Exam Findings: General: Alert and oriented but unsure why he is in the hospital. Cachectic

HEENT: 4 x 6 cm exophytic, actively bleeding plaque located at the right lateral neck and submental chin. Teeth with heavy plaque buildup. Gingiva swollen with some bleeding, purple-pink in color, with the plasticky texture of dentures. Muffled voice.

Lab and Imaging Results: Initial workup was remarkable for a sodium of 160, albumin of 3, a Vitamin C of 7 mcmol/l (range 23-114), and a creatinine of 1.69 (from a baseline of 1.2). CBC was initially normal but after hydration, hemoglobin was noted to be decreased to 9-10.

Differential Diagnosis:

Hypernatremia and failure to thrive was likely due to oral pain, dysphagia from mass effect of BCC, and limited support in the setting of developmental delay

Appearance of gingiva could be due to scurvy or processes causing gingival hyperplasia such as chronic gingivitis, medications (typically calcium channel blockers or immunosuppressants like cyclosporine), neoplasms, or sarcoidosis. Primary adrenal insufficiency may also cause the abnormal coloration observed.

Treatment:

Hypernatremia/failure to thrive: Sodium normalized with hydration. Patient was started on tube feeds and discharged to a SNFA

Scurvy: Treated with 1g daily of ascorbic acid. Improvement in gingival appearance over the course of his two week hospitalization

BCC: Excised by surgical oncology and plastic surgery

Discussion: Basal cell carcinoma rarely metastasizes but is often locally invasive. In the case of RW, his BCC contributed to poor oral intake and malnutrition, manifesting as scurvy. Scurvy is diagnosed clinically based on exam findings such as gingival bleeding and supported by vitamin C levels < 11 and anemia.

Conclusion: Though thought of as rare in the developed world, vitamin C deficiency has a prevalence of 7% in the US, with higher rates seen in the elderly, malnourished, and those of low socioeconomic status.