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Resident Poster Submissions Part 1

Bilateral Thalamic Glioma with Subsequent Malignant transformation to Glioblastoma Multiforme

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Introduction

Bilateral thalamic lesions represents an uncommon but characteristic radiographic finding. Despite this, the differential for such lesions is quite diverse and includes vascular, neurodegenerative, infective, neoplastic, metabolic, and autoimmune etiologies (1). Evaluation and proper diagnosis requires extensive history, neurological examination, and clinical investigations to determine a diagnosis. Here we present the case of a patient presenting with episodic aphasia initially ascribed to a transient ischemic attack who was found to have bilateral thalamic enhancement and enlargement. After a prolonged multidisciplinary investigation, the patient was eventually diagnosed with bilateral thalamic glioma with progressive transformation to glioblastoma multiforme (GBM).

Review of Literature

Literature on bilateral thalamic lesions categorizes the pathology into 5 major types (table below). Viral entities are most commonly flavivirus but HSV may also present this way (1). Metabolic disease, while rare, have a number of entities which may present this way. There is often characteristic T1, T2, and FLAIR findings on MRI which assists in narrowing the diagnosis significantly. This finding always represents a major pathology and every effort should be made to accurately and quickly diagnose a patient.

Mechanism	Examples
Metabolic	Wernicke encephalopathy, Fahr disease, Wilson disease, Fabry disease, GM2 gangliosidosis, Krabbe disease, Leigh disease
Demyelinating	Acute disseminated encephalomyelitis, osmotic myelinolysis
Infectious	Creutzfeldt-Jacob Disease, viral encephalitis
Neoplastic	Bilateral thalamic glioma
Vascular	Infarction of Percheron's Artery, Top of the Basilar syndrome, Cerebral venous thrombosis, hypotensive cerebral infarction

Case Presentation

A 55 year old caucasian male presented (D = 0) to an outside hospital with family due to acute onset non-fluent aphasia and confusion beginning at 10 am. The patient last known normal was the night prior at 11 pm and his initial NIH stroke scale was 6. A non-contrast CT of the head shows no acute changes however subtle left thalamic hypodensity was noted and felt to be related to possible chronic microvascular change. Telestroke for a tertiary medical center (D=0) recommended transfer for further evaluation at a dedicated stroke center however no alteplase given.

The patient arrived accompanied by his wife and children who provided much of the initial history and upon arrival (D = +1), the patient's aphasia and confusion had resolved and his NIHSS was 0 on admission. On presentation he denied any neurological, cardiovascular, or other symptomatology. His medical history was notable for uncontrolled hypertension and uncontrolled type 2 diabetes mellitus for which he was non-compliant with prescribed subcutaneous insulin. He had no prior cardiac, neurological, or cerebrovascular history. However his family history was significant for a rapidly progressive dementia which claimed his mother over the course of 6 months. His social history was notable for his work as a crab and oyster fisherman and heavy mosquito exposure.

An initial MRI with and without contrast was obtained (Figure 1). The patient was placed on empiric acyclovir and a lumbar puncture was obtained based on the imaging (Figure 2). Addition autoimmune, parasitic, infectious disease, neo- and paraneoplastic studies were ordered. A video EEG was performed which demonstrated episodes of left temporal rhythmic slowing at times associated perseverating, stuttering, and incomprehensible speech. This met criteria for recurrent left hemisphere seizures likely with a deep focus.

Patient initiated on levetiracetam.Serum and CSF WNV IgG and toxoplasmosis IgG returned positive. The patient was placed on empiric therapy for toxoplasmosis and discharged due to resolution of symptoms. At D+30, a repeat MRI was performed (figure 3) and the patient was referred to neurosurgery who recommended a brain biopsy of the accessible temporal lobe lesion. Histology from the biopsy demonstrated high grade glial proliferation with nuclear atypia associated with geographic necrosis and microvascular proliferation. Specialized staining was performed which confirmed the diagnosis as a glioblastoma multiforme, IDH-wildtype, WHO stage 4.

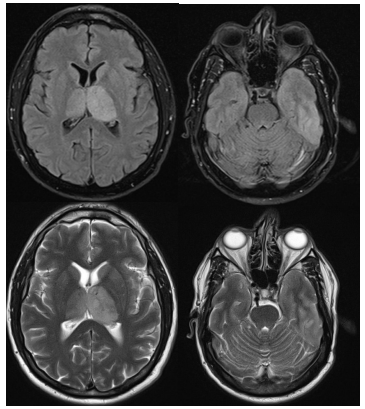

Figure 1. MRI Brain (left). Bilateral thalamic enlargement with T2/FLAIR signal hyperintensity. Additional cortical T2/FLAIR hyperintensity within the posteroinferior left temporal lobe

Lumbar Puncture	Findings
1st Aliquot Cell Differential	Color: xanthochromic and clear RBC 117 WBC 2 (40% PMN, 54% lymph, 6% monos)
3rd Aliquote Cell Differential	Color xanthochromic and hazy RBC 275 WBC 5 (64% PMN, 32%lymphocytes, 2% monos)
Cytology	Negative
Glucose	123 (mg/dL)
Protein	35 (mg/dL)

Figure 2. Basic Lumbar Puncture Results (top right)

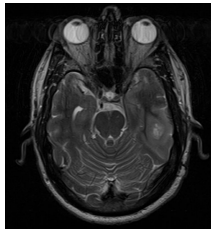

Figure 3. Repeat MRI Brain D+30 (bottom right). Peripherally enhancing temporal lobe lesion. Also noted but **not shown** was interval left thalamic enlargement with right thalamic stability.

Discussion

This patient was initially thought to have West Nile virus encephalitis given his exposure history and the presence of IgG towards the virus. His negative PCR forced us to re-evaluate. Only after repeat imaging demonstrating new peripheral enhancement and involvement of the temporal lobe was a brain biopsy pursued and a diagnosis made. Bilateral thalamic gliomas are rare in adults (2,3,4). MRI characteristics include homogenous T2 hyperintensity as seen in this patient, but the recurrence of symptoms and development of ring enhancement suggests progression of a stable lower grade glioma. Repeat imaging demonstrated stable right sided enlargement with left sided thalamic enlargement and worsening mass effect and no imaging suggestive of bihemispheric connection or communication of the tumor. IDH negativity in this case was consistent with primary glioma. This case is unusual due to the general absence of neurological signs and symptoms with the only sign of pathology being the initial episode of aphasia.

Conclusions

- Bilateral thalamic gliomas represents a rare diagnosis with unusually broad differential.
- They most commonly present in the pediatric population with signs and symptoms related to mass effect but have been known to present in adults.
- Patients may present at any WHO stage but the most commonly stage 2.
- The prognosis is generally poor regardless due to the location.

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Leukemia Cutis: An Uncommon Presentation of Acute Myeloid Leukemia

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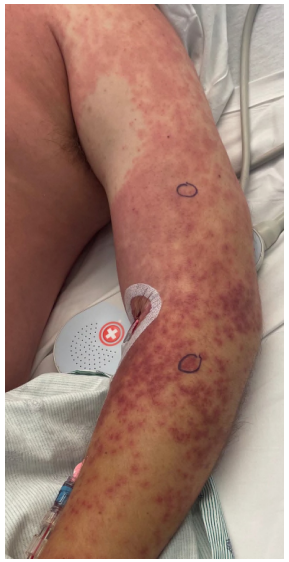
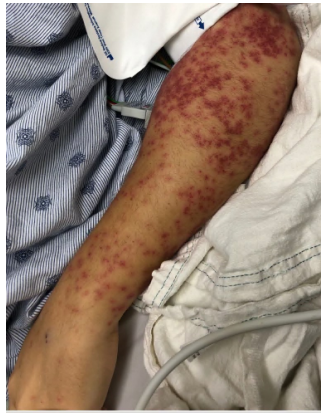
Introduction

- Leukemia Cutis is a rare condition characterized by infiltration of neoplastic cells into the epidermis and dermis with a characteristic dermatologic appearance.
- Leukemia cutis generally portends a poor prognosis in the setting of newly diagnosed blood cancers.
- We present a case of leukemia cutis as a presenting symptom in newly diagnosed acute myeloid leukemia.

Case Presentation

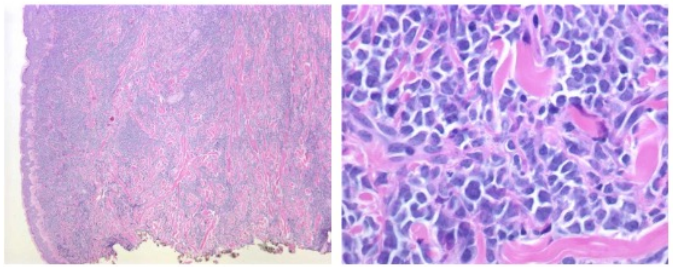
- A 31-year-old man without past medical history presented after 1 month of progressive dyspnea on exertion, worsening fevers, thirty pound unintentional weight loss, prolonged bleeding after dental procedures and diffuse rash on all extremities and trunk. Physical exam was significant for fever, tachycardia and scattered petechia and painless, erythematous papules and macules on his bilateral upper and lower extremities. Initial laboratory findings were significant for leukocytosis (222,000) with 94% immature mononuclear cells and thrombocytopenia (8,000). Punch biopsy was performed by dermatology and pathology returned leukemia cutis with myeloid features. Bone marrow biopsy was significant for acute myeloid leukemia with 86% large blastoid cells. Patient was started on induction chemotherapy with cytarabine and idarubicin resulting in slow resolution of rash and cell count recovery. Repeat bone marrow biopsy shows absence of leukemic cells after induction therapy. Patient now scheduled for allogenic bone marrow transplant after consolidation therapy.





Discussion

- Leukemia cutis is a challenging diagnosis and rare presentation of leukemia.
- When coupled with a new diagnosis of acute myeloid leukemia, as in this patient, portends a worse prognosis.
- This patient presented in blast crisis with cutaneous involvement and new diagnosis of acute myeloid leukemia was made promptly based on bone marrow biopsy results.
- Prompt dermatologic consultation resulted in diagnosis of leukemia cutis based on punch biopsy.
- Treatment is aimed at eradication of systemic disease. After initiation of induction chemotherapy (cytarabine and idarubicin), his cutaneous lesions subsided significantly with slow cell count recovery.



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CASE: Unrecognized broken heart in a hospitalized patient

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OchsnerHealth

Introduction

- Stress cardiomyopathy (CM), Takotsubo CM, is a known clinical entity presenting with chest pain, elevated cardiac markers, and wall motion abnormalities on echocardiogram.
- The presentation may become less evident in hospitalized patients with multiple comorbidities. In this report, we discuss a unique presentation of an uncommon entity while covering diagnostic signs, workup, and management in hospitalized patients.

Case Presentation

79 years old female with diastolic heart failure, paroxysmal atrial fibrillation, Papillary thyroid carcinoma status post thyroidectomy which resulted in tracheostomy. She was discharged recently after surgery when she quickly developed severe shortness of breath, productive cough, and anxiety.

On presentation, the patient was tachypneic, anxious with mid-sternal chest pain. On exam, she was cachectic and had abdominal distension. Her labs revealed elevated Troponin of 0.064, Pro-BNP 529, anemia with hemoglobin of 11.1, hyponatremia with Na of 133.

Chest x-ray was unremarkable. The CT of the neck showed subcutaneous emphysema, fluid collection in the right supraclavicular region. She was admitted and started on IV Levaquin and SoluMedrol, with continuous suctioning of recurrent mucus plugs.

The patient initially had chest pain and dynamic EKG changes while inpatient with T wave inversion in lateral then inferior leads. (Figure: EKG dynamic ST/T wave changes in I, aVL, V2-V6.) A bedside echocardiogram showed reduced ejection fraction and hypokinetic apex. Her troponin peaked at 0.174, she developed hypotension and was placed on norepinephrine. Concern was for septic vs cardiogenic shock; she was started on IV Lasix as well. The patient's left heart catheterization revealed non-obstructive coronary arteries with hypokinetic apical inferior segment. The patient transitioned to phenylephrine in the setting of stress-induced cardiomyopathy, then successfully weaned off. Additionally, she completed a 5-day course of clindamycin and aztreonam for aspiration pneumonia. The patient stabilized after 1 week and was discharged with improved cardiac function.

Discussion

- Stress or Takotsubo CM is an uncommon clinical entity with an estimated 2% incidence in the US.
- We discuss stress cardiomyopathy as an unrecognized cause for chest pain in a hospitalized patient with multiple comorbidities.
- As seen in figure 1, EKG changes include widespread and progressive deepening of T wave inversion, with progressive increase in QTc.
- Clinicians should seek out this uncommon diagnosis in patients with multiple comorbidities, recent surgical interventions, dynamic EKG changes, and new echocardiogram findings.

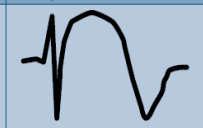
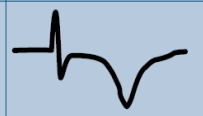
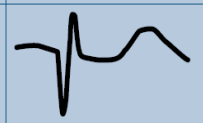
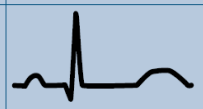
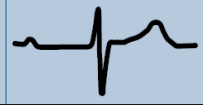
ECG change	Example	Prevalence in TTC
ST segment elevation, most commonly in praecordial leads		Common (46–100%)
Diffuse deep symmetric T-wave inversion. Resolves slowly and often partially		Common
Pathologic Q-waves that typically resolve before hospital discharge, with restoration of normal R-wave progression		37%
Prolonged QT interval (beginning of Q-wave to end of T-wave) which usually normalises in 1–2 days		
Prolonged PR (beginning of P-wave to beginning of QRS complex) interval		26%

Figure 1; ECG changes in broken heart syndrome[1]

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CASE: Serious pleuroperitoneal leak

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Introduction

- Hydrothorax can be defined as an noninflammatory collection of serous fluid in the pleural cavity most commonly as a complication of heart failure, cirrhosis, or renal failure. We present a rare but serious case of hydrothorax in the setting of peritoneal dialysis (PD).
- Once more common causes of pleural effusions are ruled out, peritoneal scintigraphy is a non-invasive method to effectively diagnose a pleuroperitoneal leak.

Case Presentation

A 57-year-old female presented to her primary care physician due to shortness of breath and nonproductive cough for three weeks. She has a history of end-stage renal disease on PD since 2019, multiple myeloma status post autonomous stem cell transplant on maintenance lenalidomide, hypertension, and type II diabetes mellitus.
On initial assessment, she was afebrile, tachypneic, and saturating at 98% on room air. There was decreased breath sounds over the entire right lung field, a 2/6 systolic murmur, and left lower extremity edema. She has a PD catheter in place and a right forearm arteriovenous fistula. Chest x-ray showed a new large, right-sided pleural effusion. Follow up CT scan showed a large volume of right-sided pleural fluid with the near complete collapse of the right lung. There was marked compressive atelectasis of the right. Thoracentesis was performed, and one liter of clear, yellow fluid was removed. The patient showed improvement in her clinical status. Repeat chest x-ray showed interval decrease in size of the effusion. Analysis of the pleural fluid showed LDH of 161 U/L, protein 3.6 g/dL, WBC 2, glucose 168 mg/dL, consistent with a transudate effusion. A nuclear medicine study with 24hr delayed imaging demonstrated tracer activity within the right-sided pleural effusion at 24hrs consistent with a right-sided pleuroperitoneal leak. Cardiothoracic surgery (CTS) was consulted following the nuclear medicine study for possible video-assisted thoracoscopic surgery (VATS) to fix the pleuroperitoneal leak. VATS drainage produced 2.6 liters of fluid. No obvious diaphragmatic defect was seen. A PleurX catheter was placed and the patient was discharged with instructions to follow up with CTS outpatient.

Discussion

- We describe a rare complication of a common entity while discussing the presentation, workup, and management.
- Pleuroperitoneal leak is a significant complication of peritoneal dialysis, prevalence estimated around 1.6%.
- Pathogenesis is theorized to be secondary to pressure gradient between the thorax and abdominal cavity. Reasons for development include leakage around the major vessels, diaphragmatic foramina, lymphatics and thoracic duct.
- 88% of pleuroperitoneal communications occur on the right side, similar to our case.
- Diagnostic thoracentesis and pleural fluid analysis are performed initially, with pleural fluid glucose level > 300 mg/ dL indicating pleuroperitoneal communication.
- Peritoneal scintigraphy is a safe and rapid method of diagnosing peritoneal cavity leaks. 3-5 milimercury of technetium 99m isotopes per 0.5 to 2 L of dialysis solution are injected abdominal cavity then multiple projections are taken, separating abdominal wall leak from peritoneal fluid posterior to it.

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Renal Wasting of Magnesium

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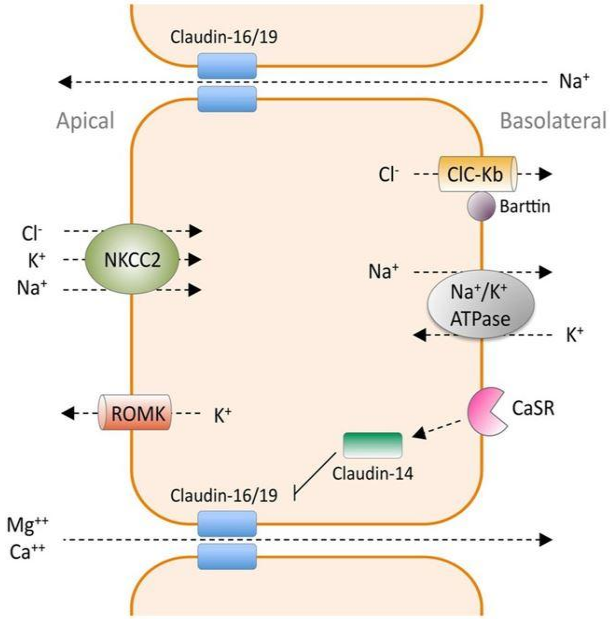
Introduction

- Hypomagnesemia commonly occurs in up to 12% of hospitalized patients with factors such as nutrition, diuretics, medications, and polygenic heritability.
- Symptomatic hypomagnesemia is difficult to ascribe to specific clinical manifestations given associations with multiple abnormalities including hypokalemia, hypocalcemia, and metabolic alkalosis.
- Typical symptoms are described as tetany, positive Chvostek and Trousseau signs, convulsions, and arrhythmias including Torsades de pointes.
- Major categories inducing hypomagnesemia are gastrointestinal and renal losses

Case Presentation

- A 43 year old woman with a history of chronic granulomatous gastritis with metaplasia s/p partial gastrectomy and Roux-en-Y, hypertension, gastric ulcers, and marijuana use who presented with 2 weeks of abdominal pain
- The abdominal pain acutely worsened 2 days prior to admission when she developed intractable nausea and vomiting with poor p.o. intake.
- She was initially hypotensive, Anion Gap of 16, potassium 3.3, calcium of 8.5, magnesium of 1.5 with an AKI that responded to fluid resuscitation and potassium repletion.

Images



Paracellular reabsorption of magnesium and calcium in the TAL of Henle's loop. This transport depends on the lumen-positive electrical potential established by the transcellular reabsorption of other cations and anions. Reabsorption of Na+, K+ and Cl− through the apical membrane occurs via the NKCC2 co-transporter. Na+ and Cl− leave the epithelial cell through the Na+/K+-adenosine 5′-triphosphatase (ATPase) and the ClC-Kb channel at the basolateral membrane, respectively. K+ is excreted to the lumen by the ROMK channel. The backflow of Na+ through the paracellular channel, as a consequence of diminishing luminal Na+ concentrations, is an additional contributor to the lumen-positive voltage that forces magnesium and calcium reabsorption. Claudin-16 and claudin-19 facilitate the paracellular transport of magnesium and calcium. Activation of CaSR by extracellular calcium upregulates claudin-14, which in turn interacts with the claudin-16/claudin-19 complex and inhibits its cation permeability

Hospital Course

- Hospital day 2, she developed profound hypomagnesemia down to 0.8mg/dl, with resolution of hypotension and AKI
- Patient required 20g of magnesium IV over several days with only moderate improvement of Mg of 1.4
- No noted ECG abnormalities noted over hospitalization
- Fractional excretion of magnesium was 23%.
- FEMg >3% with normal kidney function is suggestive for renal magnesium wasting.
- Oral supplementation with Mag-Ox TID was added while inpatient and SlowMag and amiloride were prescribed at discharge with follow up in Renal Clinic.
- PTH (86.7 pg/mL) was elevated with low calcium (7.6mg/dL) in a pattern consistent with secondary hyperparathyroidism.

Discussion

- Major categories inducing hypomagnesemia are gastrointestinal and renal losses.
- This patient had several indications for depletion including gastric bypass, PPI, volume expansion, hyperparathyroidism, ATN but the lack of response to supplementation is suggestive of renal losses.
- Patient was not hypotensive, alkalotic, or hypercalciuric to suggest Barter/Gittleman's or Familial primary hypomagnesemia with hypercalciuria and nephrocalcinosis (FHHNC).
- Some genetic mutations have been noted in literature; for example, mutations of genes encoding for Na-K-ATPase and Epidermal growth factor among others.

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Collapsing Glomerulopathy in COVID-19 Associated Nephropathy: A Case Responsive to Corticosteroids

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Introduction

Focal segmental glomerulosclerosis (FSGS) is a common histologic lesion that is found among patients with nephrotic syndrome, accounting for 35% of all cases in the United States. Collapsing FSGS, preferably termed collapsing glomerulopathy (CG), is a variant described by globular or segmental glomerular tuft collapse with hyperplasia and hypertrophy of the podocytes. This is a case of suspected collapsing glomerulopathy on histopathology in a COVID-19 infected patient that is similarly seen in COVID-19 associated nephropathy (COVAN). To our knowledge, this is one of 40 cases since the start of the COVID-19 pandemic to describe COVAN.

Clinical History

A 55-year-old African American male with a previous history of hypertension, hyperlipidemia, chronic systolic heart failure (ejection fraction 40%), nonischemic cardiomyopathy s/p ICD implant, and atrial fibrillation presented to the emergency department with chief complaint of intermittent dizziness/lightheadedness and dry cough for two days. Initial labs revealed BUN 34.8 mg/dL, creatinine 3.39 mg/dL (compared to baseline creatinine 1.00 mg/dL), and positive COVID-19 rapid testing. The patient was admitted to the hospital for acute kidney injury and acute COVID-19 infection. Nephrology services were consulted after no significant improvement in renal indices with IV fluid hydration and noted proteinuria > 600 on urinalysis. Urine protein creatinine ratio 18.78g suggesting severe nephrotic range proteinuria. Left kidney biopsy revealed the presence of severe epithelial foot process effacement by electron microscopy along with no significant glomerular lesions by light microscopy meeting diagnostic criteria for minimal change disease; however, the background tubulointerstitial changes in a patient with COVID-19 raises suspicion for unsampled collapsing glomerulopathy. It was a suboptimal biopsy with only two non-sclerotic glomeruli available for light microscopy. This patient had suspected collapsing glomerulopathy with nephrotic range proteinuria that ultimately responded to methylprednisolone 500 mg x 3 days followed by prednisone 40 mg daily x 6 days. He was prescribed a 12-week prednisone taper at hospital discharge and will require close follow-up with nephrology.

Histopathology

Two specimens 1.1 x 0.1 x 0.1 cm and another specimen 0.5 x 0.1 x 0.1 cm were obtained and underwent testing via IR CT Bx.

Electron microscopy showed widespread epithelial foot process effacement and microvillous transformation of the podocytes. Ultrastructural evaluation of glomerulus reveals wrinkled basement membranes that are uniform and are of normal thickness.

Light Microscopy showed: Mild to moderate interstitial edema is present. Some tubules are lined by a swollen epithelium with markedly increased number of cytoplasmic protein reabsorption droplets.

Tubules lined with proteinaceous casts, wrinkling of the BM in addition to widespread foot process effacement make Collapsing FSGS likely etiology of the nephrosis

Labs

Lab View	10/20/2021 3:50 CDT
IgA-ARUP	* 258
IgG-ARUP	* 800
IgM-ARUP	* 204
Total Prot-ARUP	L 4.3
Alb Lvl-ARUP	L 1.59
Alpha 1 Glob-ARUP	0.37
Alpha 2 Glob-ARUP	0.94
Beta Glob-ARUP	0.58
Gamma Glob-ARUP	0.82
Immunofix-ARUP	IFE Done

Timed Urine Chem	
U Protein Tot-LC	* >600.0
U24 Protein Tot-LC	H >2400
U Albumin LC	73.6
U Alpha 1 Glob-LC	1.8
U Alpha 2 Glob-LC	8.8
U Beta Glob -LC	6.5
U Gamma Glob -LC	9.2
U M Spike-LC	Not Observed
U24 PE Note-LC	* Comment
U24 PE Interp-LC	* Comment

Serology						
Syphilis Ab	A Reactive					
ANA Screen						* Negative
dsDNA Ab						Negative
HIV			Nonreactive			
Hep A IgM			Nonreactive			
Hep B Core IgM			Nonreactive			
Hep Bs Ag			Nonreactive			
Hep C Ab			* Nonreactive			
RPR	Non-Reactive					
MPO Abs-LC						<9.0
PR-3 Abs-LC						* <3.5
SARVS-CoV-2 PCR						

Lab View	10/19/2021 9:17 CDT
Random Urine Chem	
U Creatinine	159.5
U Osmolality	306
U Prot/Creat	H 18,777.4
U Sodium	28.0
FESodium%	
U Protein Lvl	* 2,995.0

Lab View	10/22/2021 14:24 CDT
Random Urine Chem	
U Creatinine	141.8
U Osmolality	
U Prot/Creat	
U Sodium	
FESodium%	
U Protein Lvl	1,614.5
Timed Urine Chem	

Discussion

- Collapsing glomerulopathy can be associated with genetic conditions, medications, systemic diseases, conditions related to acute glomerular ischemia, or infections (particularly viral infections like HIV).
- Direct toxic viral effect on podocytes (as occurs in HIV-associated nephropathy) and/or virus-induced cytokine injury to podocytes are two suspected mechanisms by which SARS-CoV-2 causes CG.
- Interestingly, the patient tested homozygous for APOL1, a risk factor associated with collapsing FSGS.

Conclusion

- We present a case of collapsing focal segmental glomerulosclerosis, which has been described in COVID-19 nephropathy.
- Further studies are needed to define the clinical and pathologic characteristics, prognosis, and treatment of this histologic lesion in COVAN.

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COVID-19 Infection Unmasks Underlying Pan-Hypopituitarism

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Introduction

- Hypopituitarism is defined as insufficient production of one or multiple anterior pituitary hormones secondary to damage or non-existent hypothalamus and/or pituitary gland.
- This may be a result of multiple etiologies such as acquired (mass/apoplexy), congenital (infiltration/deficiency), or iatrogenic (surgery/irradiation) insults.

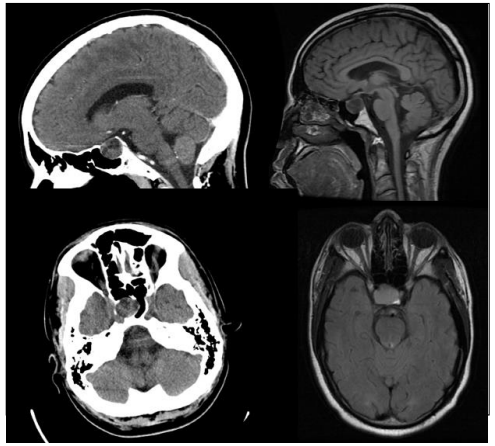
Case Description

- A 50-year-old African-American male with a history of hyperlipidemia presented to the hospital with intractable nausea and vomiting with associated non-bloody diarrhea, generalized body aches, fatigue, and headache for three days.
- He was initially hypotensive and was adequately resuscitated with IV crystalloids. Remaining vitals were within normal limits and physical exam was remarkably benign.
- The patient endorsed difficulty with deep inspirations, however maintained saturations above 95% on room air. Presenting symptoms were tempered with antiemetics and antipyretics.
- Initial lab work was significant for positive COVID-19 rapid test, acute renal insufficiency, transaminitis, and elevated inflammatory markers (Ferritin and CRP).

Case Description (Continued)

- Thyroid function studies signified a euthyroid sick pattern. Further workup for adrenal insufficiency was pursued after a low random cortisol level resulted. A cosyntropin stimulation test was performed which showed insufficient cortisol elevation at both 30 and 60 minutes.
- Further history revealed symptoms related to hypopituitarism for the past ten years including fatigue, dry scalp, hypothermia, and diffuse body aches. This progressed to include increased urination, decreased libido, and progressive blurry vision.
- He reported being prescribed muscle relaxants and steroids on multiple occasions over this time frame, with intermittent resolution, but unfortunately the symptoms returned.
- CT head showed an expansile seller/suprasellar mass and MRI brain suggested a 2.5 cm heterogenous cystic pituitary macroadenoma with mass effect on the optic nerves and optic chiasm.
- Ophthalmology was consulted for formal visual evaluation. Further lab work revealed low T3/T4, LH/FSH, ACTH, testosterone, and IGF-1. TSH and Prolactin levels were within normal limits.
- He was stabilized and discharged home with endocrinology and ophthalmology follow-up. He presently awaits neurosurgical evaluation for tumor resection.

CT/MRI Images



Conclusion

- Sellar masses may present with symptoms related to hormonal systems or neurological pathways, but simply may present asymptomatically and be found incidentally on CT/MRI imaging.
- Pituitary Macroadenomas require close surveillance as insufficient hormones can lead to increased risk of morbidity and mortality.

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A Curious Case of Meigs-like Syndrome

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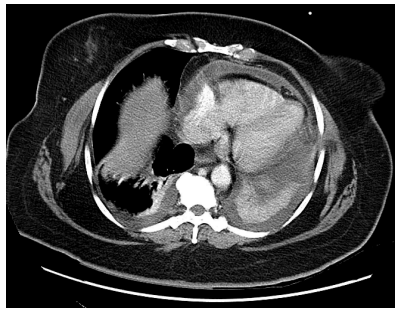
Introduction

- Meigs syndrome is defined as a benign ovarian tumor (II) associated with ascites and pleural effusion (I) that resolves after tumor is removed.
- Represents only 1% of all ovarian tumors
- Diagnosis of exclusion once malignancy is ruled out
- Most common in post-menopausal woman and peaks in the 7th decade
- Pathophysiology is unknown; thought to be abdominal tumor putting pressure on abdominal lymphatics → ascites → transudation into pleural fluid

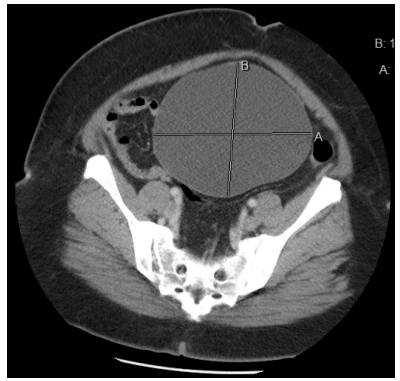
Hospitalization #1

- A 42-year-old female with history of hypertension, hypothyroidism, abnormal uterine bleeding presents with a complaint of shortness of breath and tachycardia.
- Noted to have moderate bilateral pleural effusions in the setting of community-acquired pneumonia.
- Underwent thoracentesis revealed exudative effusion, with reactive mesothelial and inflammatory cells
- Subsequently underwent video-assisted thoracoscopy with chest tube placement and Talc pleurodesis.
- Additionally, MRI Pelvis was significant for a 15mm right ovarian mass with mild ascites.
- CA-125 level was elevated (93) with negative autoimmune and infectious workup
- The patient was started on Provera on discharge with plans for outpatient surgical intervention as initial diagnosis was endometriosis.

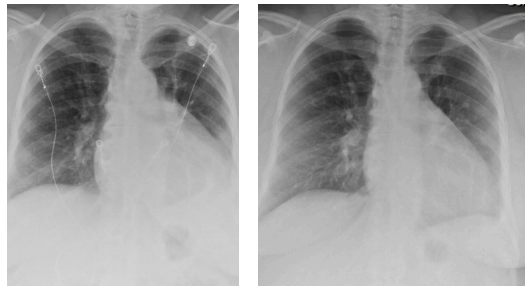
Case Description (labs/imaging, etc.)



I) CT Thorax image showing both pericardial and pleural effusion



II) CT abdomen and pelvis image showing both presence of cyst (A) and free fluid



III) Progression of chest x-ray changes from day before surgery (left) versus 5 months after hospitalization (right)

Hospitalization #2

- On readmission, CT Thorax W/O contrast showed reoccurrence of bilateral pleural effusions and new-onset pericardial effusion, confirmed by echocardiogram.
- Cardiology was consulted and a left and right heart catherization was performed without evidence of coronary obstruction or evidence of pericardial constriction.
- Then underwent a laparoscopic right salpingo-oophorectomy and pathology was consistent with a paratubal cyst and no evidence of malignancy
- Her symptoms improved post-op and eventually CA-125 normalized.
- Discharged to follow up with serial chest x-rays (III)

Discussion

- The presence of pericardial effusion in the setting of Meigs syndrome is extremely rare - roughly 3 published cases known
- In this patient, the resolution of the pericardial effusion following the removal of the ovarian mass does point to a link.
- Good prognosis with early detection and intervention
- Our case underlines that in the setting of unexplained pericardial effusion, the possibility of Meigs Syndrome should be considered.

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Resistant Hypertension and Hypokalemia in the Setting of Primary Aldosteronism

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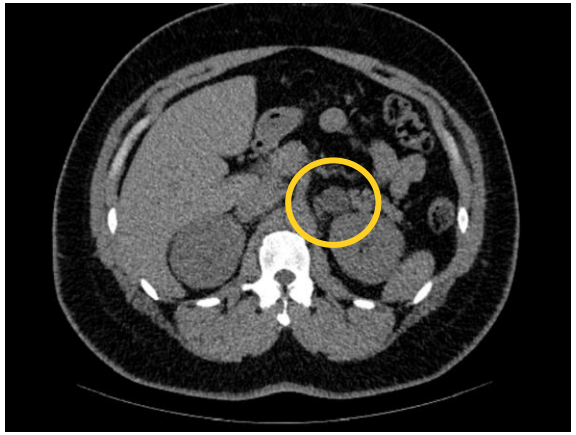
Introduction

- Primary Aldosteronism is a common yet underdiagnosed cause of resistant hypertension- affecting as much as 10% of this patient population
- This disorder is characterized by hyper-secretion of aldosterone outside of normal feedback mechanisms resulting in sodium retention, increased potassium excretion and hypertension via volume overload
- Diagnosis can be confirmed via measurements of serum aldosterone and renin activity
- Treatment is focused on aldosterone antagonist therapy as well as surgery if a discrete tissue source is identified

Case Presentation

- A 39-year old African American man with history of heart failure with reduced ejection fraction <15% (HFrEF), chronic kidney disease stage III (CKD 3), resistant hypertension (HTN), Primary Aldosteronism (PA), multiple prior admissions for HTN emergency presented with volume overload consistent with HFrEF exacerbation
- Over a ten-year span, CT angiograms had demonstrated a progressing left adrenal mass with corresponding aldosterone/renin ratio increase over the last 2 years PTA.
- Despite therapy with an aldosterone antagonist and multiple other anti-hypertensives, his blood pressure and hypokalemia had remained poorly controlled
- Current medications included spironolactone, hydralazine, amlodipine, carvedilol, and bumetanide

Images



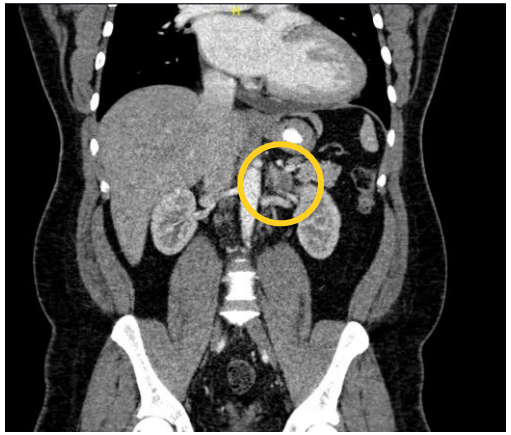


Image 1-2: 2.6cm adrenal nodule seen on CT imaging

Labs

Renin Activity	<0.1 ng/mL/hr (0.2-1.6 ng/mL/hr)
Aldosterone/Renin Ratio	126 ng/dL (<31ng/dL)
Potassium on admission	3.1 mmol/L (3.6-5.2 mmol/L)

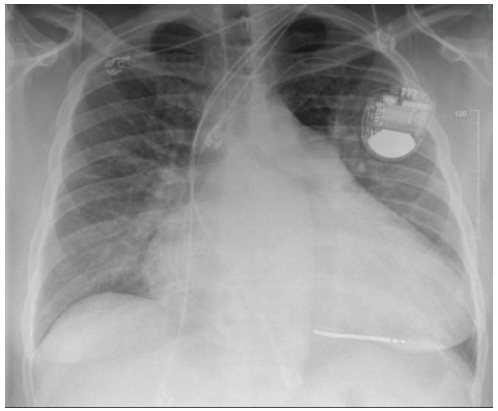


Image 3: Appearance of enlarged cardiac silhouette on chest xray consistent with patient's hypertension-induced cardiomyopathy

Hospital Course

- The patient was admitted and treated with IV diuretics in addition to up-titration of blood pressure medications.
- Normokalemia was maintained with frequent potassium supplementation in addition to the eventual titration of spironolactone to 200mg daily
- He was kept inpatient to optimize volume status in anticipation of adrenal vein sampling and subsequent laparoscopic adrenalectomy
- After confirmation of single aldosterone hyper-secreting mass via adrenal vein sampling, patient opted to pursue medical management due to his high surgical risk
- He was discharged on 200mg spironolactone daily, 25mg carvedilol BID, sacubitril-valsartan 24/26 BID, isosorbide dinitrate 10mg TID, and bumetanide 1mg daily. At his outpatient follow-up 2 weeks later both blood pressure and potassium had normalized

Discussion

- Primary Aldosteronism (PA) is a common cause of resistant hypertension; high-risk patient should be screened with a plasma aldosterone/renin activity ratio
- Initial management is medical therapy with an aldosterone antagonist in addition to other anti-hypertensives as needed
- In those with a discrete aldosterone-secreting mass confirmed on CT and adrenal vein sampling, laparoscopic adrenalectomy can be curative
- Left untreated, an aldosterone-secreting mass can cause serious co-morbidities due to chronic hypertension

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THE CROWN OF MISFORTUNE: A CASE OF AN UNDER-DIAGNOSED RHEUMATOLOGIC RARITY

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INTRODUCTION

- Crowned dens syndrome (CDS) is an under-recognized and often misdiagnosed condition that mimics multiple neurologic and rheumatologic diseases. It is an uncommon presentation of Calcium Pyrophosphate Deposition or "Pseudo-Gout" that manifests as acute attacks of neck pain with fever, nuchal rigidity, and elevated inflammatory markers related to radio-dense deposits of CPPD around the odontoid process.

CASE PRESENTATION

- 83-year-old male presented with a 7-day history of confusion, progressive weakness, severe head, neck and shoulder pain along with a maximum documented temperature of 103F.
- On examination, he was found to be confused and in distress with severe posterior cervical tenderness, tenderness to palpation of the trapezius muscle and peri-scapular region, and pain with neck flexion.
- Swelling, warmth, and tenderness of the right ankle and knee joints were noted.
- Initial labs revealed ESR 88, CRP 18.2, Cr 3.84, BUN 61, WBC 7.96, and normal serum uric acid.
- CT report of the head and neck showed degenerative changes of the cervical region

HOSPITAL COURSE

- Given the history and presentation, we suspected meningitis and initiated the patient on empiric antibiotics.
- However, a lumbar puncture was performed and meningitis work-up was negative.
- With no clinical improvement after receiving antibiotic therapy, Polymyalgia Rheumatica and Reactive Arthritis were considered.
- Antibiotics were discontinued and the patient was started on Prednisone.
- The patient reported significant clinical improvement after which CPPD was considered in the differential.
- Synovial fluid analysis revealed no crystal deposition.
- On further evaluation of imaging with the radiologist, calcification of the transverse ligament around the odontoid process with surrounding swelling was identified on the CT scan. Also noted chondrocalcinosis within the left wrist and right knee on the x-ray.
- These radiographic findings in conjunction with the patient's clinical presentation confirmed the diagnosis of Crowned Dens Syndrome.

DISCUSSION

- CDS is a radio-clinical diagnosis characterized by the calcification of the cruciform ligament around the odontoid process and usually presents as acute neck pain, stiffness, fever along with signs of systemic inflammatory reaction.
- It is more common in elderly population over the age of 60.
- Although the exact pathophysiology of CDS is not fully known, it involves chondrocytic transformation of fibroblasts in the ligaments at the atlantoaxial joint with eventual microcrystalline deposition leading to inflammation of the periodontoid ligaments and surrounding tissues.
- Some of the common predisposing factors include age, osteoarthritis, hyperparathyroidism, hemochromatosis, hypomagnesemia and low bone mineral density.
- Although it is not always symptomatic, patients can remain asymptomatic and proceed to present in an acute fashion similar to a gout flare up.
- The onset is usually acute, but sometimes can be chronic as well.
- It is usually suspected in patients with diagnosed Calcium pyrophosphate deposition disease, but like our patient, CDS could be the initial manifestation of CPPD.
- The typical presentation of CDS often mimics other conditions like Meningitis, Polymyalgia Rheumatica, Giant Cell Arteritis or Spondylitis which often leads to delay in the diagnosis. The other common manifestations include jaw claudication, occipital and temporal pain, shoulder pain and even hemiplegia and hypoesthesia.
- Cervical CT scan focusing on C1 and C2 is considered the gold standard for CDS diagnosis.
- NSAIDS are considered as the first line and gold standard for the treatment of CDS.
- Majority of CDS patients fully recover within a week of high dose NSAIDs, corticosteroid, colchicine or combination therapy.
- In patients who are either intolerant or unresponsive to NSAIDs, low dose corticosteroids is a good alternative.

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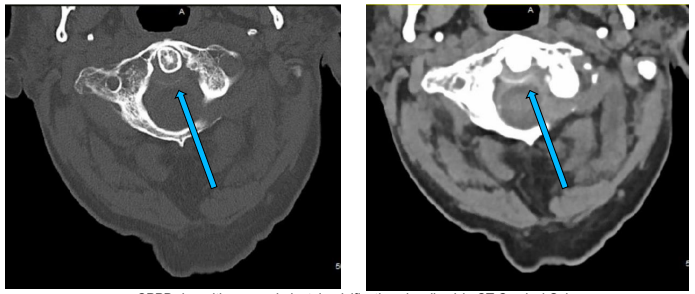
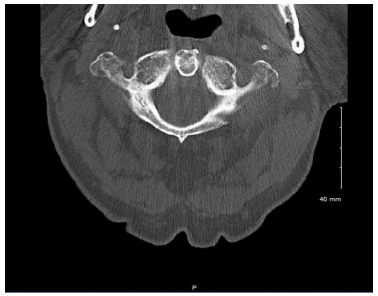
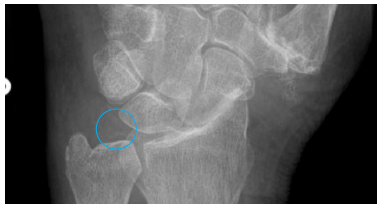


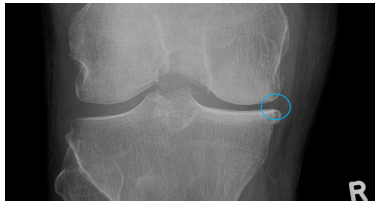
CPPD deposition or periodontal calcification visualized in CT Cervical Spine



CT Cervical Spine from two years prior to the current presentation



CPPD deposition in the left wrist



CPPD deposition in the right knee

Post Renal Transplant Temporal Lobe Abscess

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2. Ochsner Lafayette General Medical Center- Lafayette, LA

Introduction

- A concerning complication that may arise in patients with solid organ transplants is developing an opportunistic infection within the first six months post-transplant. Although the lung is the most common site of infection, many organ transplant patients have a disseminated infection and about 1/3 of these will have central nervous system involvement.
- Patients who have received solid-organ transplants are at risk for brain abscesses caused by fungi and nocardia species, with fungi being implicated in the majority of these cases.
- Nocardia is commonly found in water and soil, thus infection is typically acquired through inhalation and subsequent hematogenous spread. Brain abscesses are often asymptomatic in severely immunocompromised patients, and fever may not be present. Patients most commonly present with headaches, mental status changes, and seizures.

Case Description

- A 44-year-old female with past medical history of ESRD s/p renal transplant on chronic Tacrolimus was brought to the emergency room after experiencing a witnessed seizure in her home.
- She was intubated by EMS en route and was afebrile on presentation. Her BP was 120/75 mmHg with a heartrate of 83 bpm, and she was saturating 100 % on ventilation.
- Her home medications include tacrolimus 5 mg, cinacalcet 90 mg, labetalol 200 mg, and famotidine 20 mg.
- Her initial labs showed unremarkable electrolytes, BUN/Creatinine of 25.6/1.46, PT/PTT of 14.7/36.4, Hb/Hct of 9/27.9, WBCs 9.9 and platelets 226
- Non-contrast CT revealed a focal hypodensity within the left parietotemporal lobe most consistent with an acute infarct, although malignancy was not excluded
- Subsequent MRI of the brain with and without contrast exhibited 1.4 cm and 1 cm intra-axial ring-enhancing lesions with central diffusion restriction favoring abscesses in the left temporal lobe.
- The patient was then placed in the ICU for closer monitoring. She underwent stereotactic craniotomy for evacuation of the abscess, and cultures grew Nocardia africana.
- She was treated with IV Xyvox and meropenem for 4 weeks and subsequently survived.

Case Description (labs/imaging, etc.)

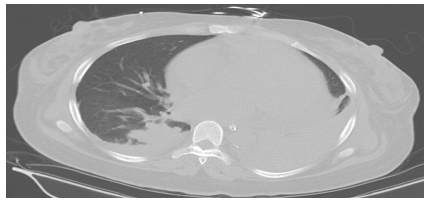

Figure 1 shows bilateral pleural effusion and basilar consolidation

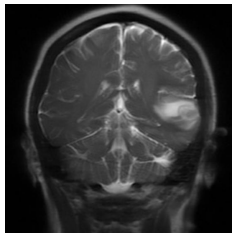

Figure 2 MRI brain shows left temporal lobe intra-axial ring enhancing lesion

Case Description (labs/tables, etc.)

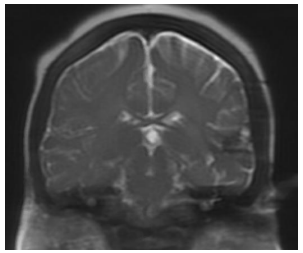

Figure 3 MRI brain shows improving findings in left temporal lobe after stereotaxic craniotomy for abscess evacuation

Conclusion

- Close follow up post-renal transplant and surveillance for the development Nocodiasis is necessary to avoid development and systemic dissemination.
- Subtle clinical clues may be of help, followed by pulmonary and head CT imaging if suspected.

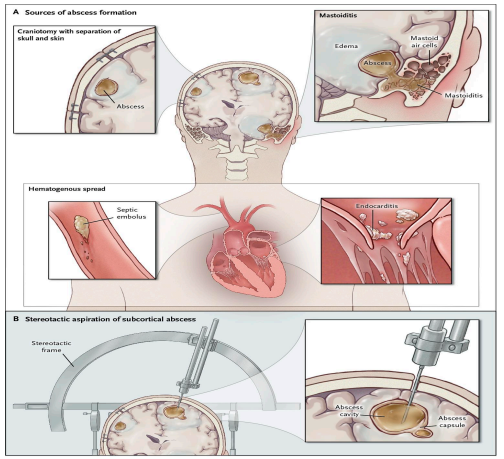

Figure 4 demonstrates different sources of abscess formation in A and stereotactic aspiration of subcortical abscess in B.

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Vacuolated Acellular Casts are a Distinct Type of Urinary Cast Associated with Severe Nephrotic Glomerulopathy

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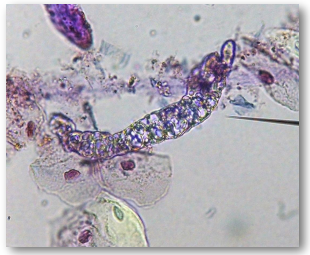
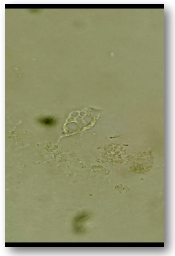
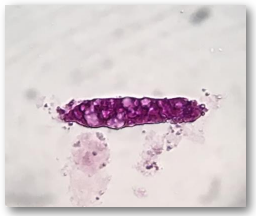
Background

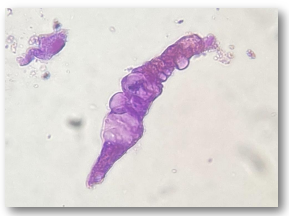
Urinary casts identified through microscopic examination of the urinary sediment (MicrExUrSed) constitute clinically useful elements for the diagnosis of acute and chronic kidney pathologies. Granular, waxy and cellular casts are well characterized. However, a unique type of casts containing nonpolarizable lipoid-like granules immersed within a lightly granular cast matrix is occasionally found. These casts have been labeled as vacuolated acellular casts (VAC). The clinical significance of VAC is not known. Herein, we present a case series of patients with specimens containing VAC





Non polarizable lipoid-like granules immersed in a granular cast





Methods

We utilized an educational social media platform (twitter) to probe for individual cases of VAC.

We surveyed known educators who frequently post micrographs of MicrExUrSed asking for files of VAC

4 urine Microscopists from Brazil (2), India (1), and USA (1) contributed to the Case Series

Demographic and Clinical Characteristics were Extracted and Representative Images were Compiled for correct identification of VAC

We tried to identify cases in which a kidney biopsy was either performed or being performed within one month of image collection.

Results

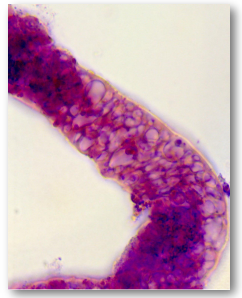
Baseline characteristics of patients with Vacuolated Casts present in Urine Sediment Microscopy (n=17)

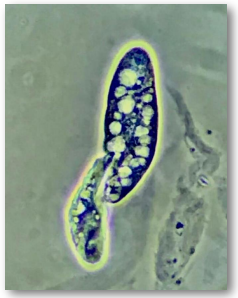
Age (years)	56 (15-81)
Race	3 (18%) Asian 9 (53%) Hispanic 2 (11%) African American 3 (18%) White
Gender	4 (24%) Female 13 (76%) Male
Baseline Serum Creatinine (mg/dl)	3.4 (1.2-6.5)
3+ Dipstick Protein	16 (94%)
Urine Protein-Creatinine Ratio	6.7 (1.3-11.7) N=10

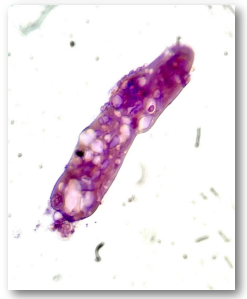
Histopathological Findings (n=11)

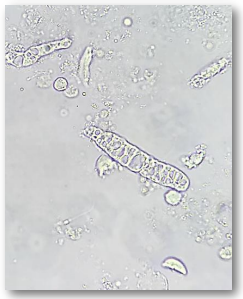
Diabetic Glomerulopathy	3
Focal Segmental Glomerulonephritis	3
Transplant Glomerulopathy	2
Membranous Nephropathy	1
Thrombotic Microangiopathy	1
Advanced Arterionephrosclerosis	1

Case Examples at Ochsner Medical Center









Conclusions

-VAC are a distinct type of casts that can be found in specimens of patients with advanced proteinuric glomerulopathy.

-The specific origin and composition of these casts remains unknown and requires further study.

Recurrent Presyncope During Hemodialysis Following Coronary Artery Bypass Grafting: A Tale of Pathophysiology

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Baton Rouge General
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Introduction

We present a case of tamponade-like physiology secondary to concurrent left pleural effusion presenting with pre-syncope in a gentleman with end stage renal disease on hemodialysis that recently underwent coronary artery bypass grafting and was found to have post-pericardiotomy syndrome.

Case Description

A 56-year-old male with a past medical history of diabetes mellitus, hypertension, end stage renal disease stable on nocturnal in-center dialysis presented to the emergency department after inability to tolerate dialysis on numerous occasions due to hypotension, pre-syncope, and chest discomfort. He also complained of near-syncope with coughing. He had undergone coronary artery bypass grafting approximately 3 weeks prior to presentation. Vital signs were significant for hypotension and no abnormalities otherwise. Physical examination was positive for decreased breath sounds to the left lower lung base with otherwise normal heart sounds and no evidence of a pericardial friction rub. Chest radiography was with evidence of a moderate to large left sided pleural effusion. Echocardiography was obtained, which displayed a small volume pericardial effusion. Cardiology was consulted to review this to rule out cardiac tamponade, which was without findings to suggest this on static echocardiogram. Laboratory analysis was positive for elevated ESR and CRP, and postpericardiotomy syndrome was suspected. Cardiac enzymes and EKG were found to be normal. He underwent sustained low-efficiency dialysis during his hospitalization, which was complicated by syncope during dialysis with no noted arrhythmia or evidence of acute coronary syndrome. He continued to have a non-productive cough with left sided chest discomfort with near-syncope symptoms. Cardiothoracic surgery was consulted, and a thoracentesis was performed, which displayed an exudative effusion consistent with post-pericardiotomy syndrome. He was initiated on colchicine and symptoms completely resolved following two weeks of therapy. He then tolerated dialysis without further episodes.

Discussion

Our case illustrates that the combination of a large volume pleural effusion with a concurrent small pericardial effusion can induce tamponade like physiology, which has been well documented in previous studies. Increased venous return to the heart is limited due to inability to generate a more negative intrathoracic pressure in the setting of both pleural and pericardial effusions, however the increase in RV filling during inspiration is still the driving force. The increase in RV filling during inspiration in combination with the synergistic effect of the pleural and pericardial effusion restricts LV volume and thus LV filling (see Figure 2). This will ultimately lead to decreased left ventricular stroke volume with a decrease in mean arterial pressure (1). The combination of the above scenario resulted in recurrent pre-syncope and hypotension in this case. A review of the pathophysiology of tamponade physiology can be seen in Figure 1.

Cough syncope as a symptom of cardiac tamponade has been previously described (2). Continuous coughing or Valsalva maneuver will lead to increased intrathoracic pressure with increased peripheral pooling leading to decreased ventricular filling and thus decreased stroke volumes. Baroreceptors are therefore exposed to diminished pulsations and mediate peripheral vasodilation, which will lead to decreased mean arterial pressure with decreased cerebral perfusion (3). Patients who have pericardial/pleural effusions with raised intrathoracic pressures to start with demonstrate an exaggerated response to coughing. The delivery of hemodialysis creates a similar scenario by the initial displacement of blood in the circuit even before ultrafiltration. This decreases venous return and may be sufficient to cause hypotension in the subset of patients with high intrathoracic pressures as in our patient.

Echocardiography may be utilized in a non-traditional standard to aid in accurate diagnosis. A dynamic echocardiography rather than standard is crucial in individuals on dialysis as physiology is altered. It has been demonstrated that a decreased transmitral E/A ratio in addition to an increased transtricuspid E/A ratio are more sensitive to display tamponade physiology in this setting (4). While an echo did not demonstrate evidence of tamponade-like physiology in our patient, the timing of the study would have been of more utility if performed during dialysis when the patient continued to have symptoms.

Our case displays that tamponade physiology can be induced by a large pleural effusion in the setting of a small pericardial effusion given evidence of symptom resolution following thoracentesis and effective treatment with colchicine for post-pericardiotomy syndrome. The pathophysiology is crucial for the physician to recognize in order to achieve timely care and accurate diagnosis.

Figures

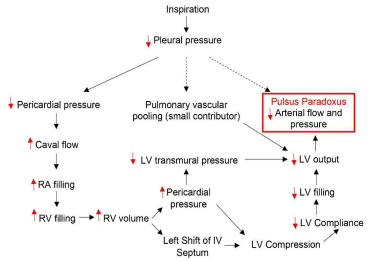

Figure 1: Physiology Flow Diagram of Cardiac Tamponade

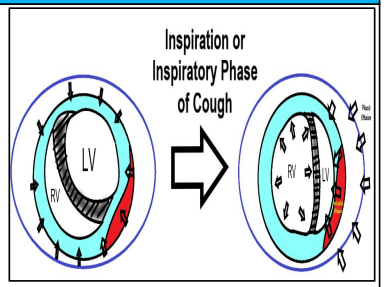

Figure 2: Diagram of Heart in Pathologic State Described in Case Report

Conclusion

We introduce this case to highlight the pathophysiology of a concurrent pericardial effusion coupled with a pleural effusion synergistically causing cardiac tamponade-like physiology presenting as intolerance to dialysis and cough syncope. We also propose that the clinical history would lead to this diagnosis even in the absence of collaborating echocardiographic evidence.

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Acute Ischemic Stroke as the Initial Presentation for TTP

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Introduction

- Thrombotic thrombocytopenic purpura (TTP) is a rare blood disorder and is a type of microangiopathic hemolytic anemia
- Classically characterized by the pentad: Fever, hemolytic anemia, thrombocytopenia, renal dysfunction, and neurological dysfunction.
- Pathogenesis is due presence of unusually large von Willebrand factor (vWF) multimers that lead to platelet clumping and subsequent microvascular thrombosis

Case Presentation

- 50-year-old woman with a past medical history of hypertension was transferred to our hospital for thrombocytopenia
- Initially presented with worsening severe headache and partial vision loss
- Patient continued to complain of a throbbing occipital headache and partial vision loss
- On physical exam, patient was found to have right temporal visual defect
- Labs significant for thrombocytopenia (Fig. 1) and microangiopathic hemolytic anemia (Table-1)
- CT head (Fig. 2) shows no acute intracranial hemorrhage and no acute ischemia
- Patient was admitted for management of Thrombocytopenia. Hematology was consulted.
- MRI brain w and w/out contrast was significant PCA territory acute infarcts including the left occipital lobe, posterior parietal lobe, left thalamus (Fig 3-A and 3-B)

Labs and Images

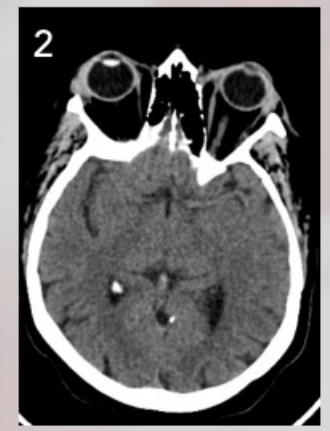
Labs

1

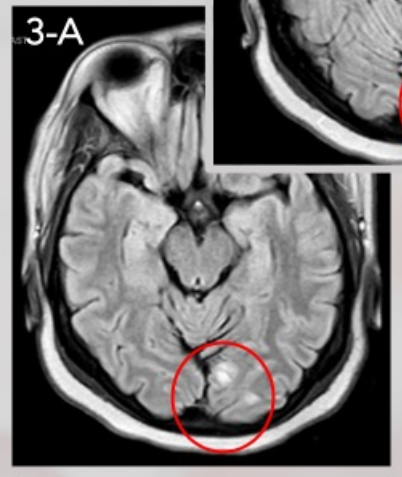
Table 1	
Fibrinogen	640 ↑
D-Dimer	703 ↑
LDH	633 ↑
Haptoglobin	<30 ↓
DAT	neg

Images

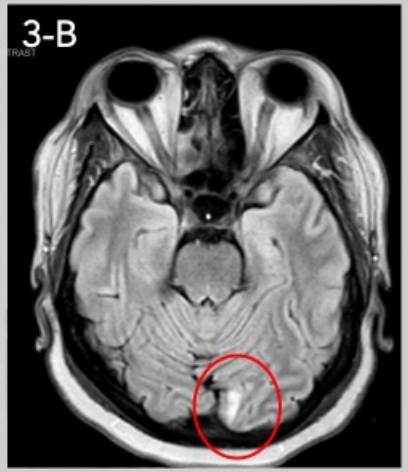
2



3-A



3-B



Hospital Course

- ADAMTS-13 activity (absent at 0%) and ADAMTS-13 antibodies (elevated) confirmed TTP
- TTP is the likely cause of patient's stroke and embolic phenomenon
- Our approach to the management of acute stroke in the setting of TTP was to treat the TTP itself
- Patient underwent therapeutic plasma exchange (TPE) emergently and was started on a high dose steroids. She also received Rituximab infusion weekly.
- Continued to have temporal hemianopia , but did not experience any additional neurological symptoms
- Completed TPE course and was discharged with follow up with hematology for continued weekly Rituximab and to taper steroids therapy

Discussion

- The classic pentad is not the usual presentation for all patients with TTP, which makes it difficult to distinguish TTP from other causes of thrombocytopenia in the acute setting.
- Neurological findings such as headache, seizure are common initial symptoms
- In the setting of thrombocytopenia, stroke management can be challenging as it is difficult to identify the underlying pathology
- TTP diagnosis and treatment should be considered as treatment of acute stroke in setting of thrombocytopenia

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Treating What's Under the Hood: Empiric Antibiotic for Clitoral Abscess

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Introduction

Clitoral prepuce abscess formation is rare, but can cause significant pain and discomfort for patients. In this case study, we seek to add to the small body of literature discussing clitoral prepuce abscesses, in order to improve empiric antibiotic treatment choice.

Diagnostic imaging is important to evaluate if edema is secondary to abscess formation from bacterial infection vs from foreign body. When considering empiric antibiotics for a clitoral abscess literature supports including MRSA and anaerobic coverage. Base on this case we recommend including streptococcus species that are commonly found in the vaginal tract.

Case

Twenty-one year old female patient, with Trisomy 21, presents with 2 days of vaginal swelling noticed by her mother. Patient's mother takes care of most of her ADLs. Mother allows the patient to take care of her genital hygiene, but follows behind her to ensure she is clean. Two days prior to presentation, Mom noticed a red swelling in the patient's vaginal area. The following day, Mom noticed the swelling was larger and that patient had started to scratch at her suprapubic and mons area.

The only past medical history for this patient was a patent ductus arteriosus that was repaired at 2 years old. She took no daily medications. Mother and patient deny fevers, abdominal or flank pain, dysuria, cloudy or foul smelling urine, or unusual vaginal discharge.

Vital signs at time of our exam included blood pressure of 121/79, pulse 108, temperature 98 °F (36.7 °C), respiratory rate 20, weight 55 kg (121 lb 4.1 oz). Cardiopulmonary and abdominal examination were benign. Genitourinary exam revealed scant pubic hair over the pubis mons, Tanner Stage 3, normal looking labia minora and majora. The clitoral prepuce was erythematous and edematous, mildly tender to palpation, no vaginal discharge and no odor.

Urine pregnancy test was negative, no leukocytosis on CBC, Urine Analysis was negative.

Soft tissue ultrasound of clitoral prepuce revealed a loculated 1.7 x 0.9 x 1.3 cm fluid collection with surrounding edematous tissue; with concern for small foreign body.

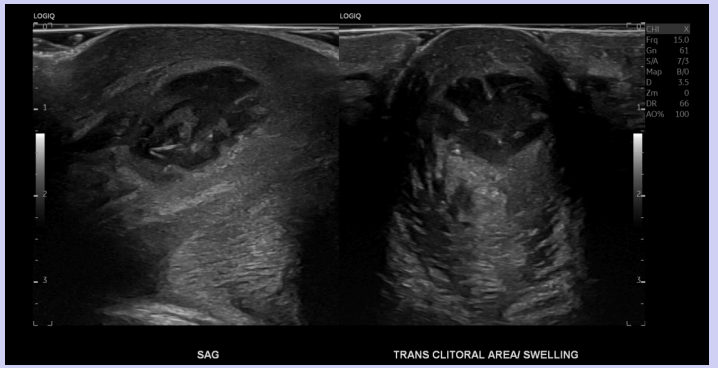

Figure 1. Soft Tissue Ultrasound of clitoral hood at time of presentation; Radiology Impression Ultrasound of the clitoral region demonstrates the presence of a well delineated complex collection measuring 1.7 x 0.9 x 1.3 cm with surrounding hyperemic tissue. There are linear increased echo within the collection which may represent possible small foreign body. Differential includes small foreign body with surrounding granulomatous/fluid collection, epidermoid /sebaceous cysts and abscess.

Pt was taken to the operating room by Gynecology for incision and drainage source control. Incision of a 3 cm by 2 cm by 3 cm fluctuant area resulted in white purulent discharge was drained. No foreign body was found. Perioperative 2 g dose of Ancef was given after intraoperative cultures were taken. Patient was discharged with a ten day course of metronidazole 500 mg BID and Bactrim 800-160 mg BID.

Gram staining showed gram positive cocci in pairs, gram positive negative bacilli, and gram negative coccobacilli. Cultures grew Streptococcus anginosus and Prevotella disiens. Susceptibilities showed sensitivity to penicillins. The patient's family was called and antibiotics changed to Augmentin for a 5 day course.

Patient followed up with Gynecology four days after initial presentation. At that visit, she had a new complaint of a breast abscess with purulent drainage from the nipple. No fevers, tenderness, erythema or warmth were reported. A pea sized nodule was palpated on physical exam in the right areola. Cultures from expressed discharge were unsurprisingly negative, given concurrent antibiotic therapy. No change was made to antibiotics at this visit. Hibiclens washes were recommended.

Pt has additional follow up visit two weeks from initial presentation and one week from initial follow up visit. Mom reported resolution of nipple discharge. Imaging of the breast revealed a structure most consistent with benign fibroadenoma.

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Discussion

Prepuce and clitoral abscesses are rare with equally little documented on culture data; however based on this case report, common bacteria of the vagina should be covered with empiric treatment for streptococcus species. Diagnostic imaging is important to evaluate if edema is secondary to abscess formation from bacterial infection vs foreign body. Soft tissue ultrasound proved to be adequate for this patient.

Genital abscesses are common, and commonly of the vulva, Bartholin, Skene glands and folliculitis. Empiric antibiotic coverage for these abscesses, after source control, include coverage for MRSA and enteric gram-negative aerobes. Antibiotics should be tailored based on culture data, which is not always available. The few case reports of clitoral prepuce abscess indicated these abscess can sometimes lead to necrotizing fasciitis in elderly patients, and that early surgical intervention is beneficial. Zeitoun et all in 2020 presented a review of current cases and found that polymicrobial infections with Enterobacteriaceae were most common. The less encountered infections were frequently caused by beta hemolyzing Streptococci. Based on our case, we recommend including antibiotic coverage for streptococcus species that are commonly found in the vaginal tract.

Culture data from our patient grew Streptococcus anginosus organisms. S. anginosus, S. constellatus, and S. intermedius,are catalase negative, gram-positive cocci. They are normal components of gut and oropharynx flora. When pathologic growth occurs as pyogenic infections, abscess formation or endocarditis is most common. This group is commonly susceptible to ß-lactam antibiotics. Prevotella disiens is part of the Provotells species that are common to the vaginal microbiota, overgrowth of which has been associated with bacterial vaginosis. This species tends to susceptible to penicillin. Generally, both isolated species are common to the vaginal microbiota and the Strep species has been implicated in abscess formation.

When encountering a clitoral prepuce abscess source control is of utmost importance, but until that can be achieved empiric antibiotics is critical. This case supports ultrasound to evaluate for abscess that would require surgical source control to cure the infection. We found streptococcal and prevotella species of the vaginal tract, supporting empiric antibiotic coverage for these species.

CNS Nocardiosis in AIDS Patient

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LSU Health NEW ORLEANS

LSU Health Internal Medicine

Introduction

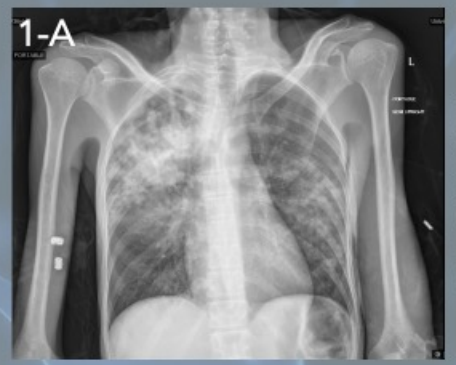
- Nocardia is an opportunistic pathogen, occurring in patients with depressed cell-mediated immunity
- Pulmonary nocardiosis is the most common clinical presentation. The central nervous system (CNS) is the most common extrapulmonary location for nocardiosis.
- HIV patients with very low CD4 and high viral load are especially vulnerable to nocardia and disseminated disease

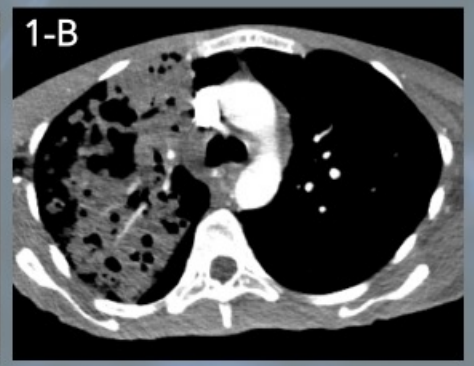
Case Presentation

- A 36-year-old man with a history of HIV/AIDS presented to the ED in November 21 for progressively worsening fever, sweat, and chills and new onset altered mental status, urinary incontinence, and seizure.
- Patient's absolute CD4 count 13 and Viral load of > 1 million
- He was recently (in July 2021) diagnosed with pulmonary Nocardiosis. CXR showed Multifocal bilateral coalescent and nodular airspace opacification throughout the lungs, greatest within the right upper lobe and extensive right upper lobe solid aeration, with cavitation is seen on CT chest (fig. 1-A and 1-B)
- CT (fig 2-A) and MRI (fig. 2-B/C) of the head showed multiple ring enhancing lesions concerning for CNS nocardiosis. 6-mm Midline shift was additionally noted on both.
- LP was deferred due to 6 mm midline shift

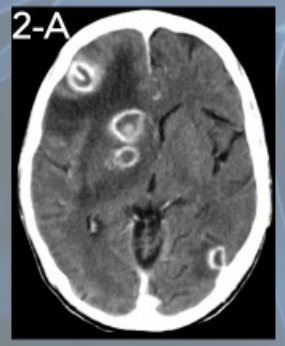
Images

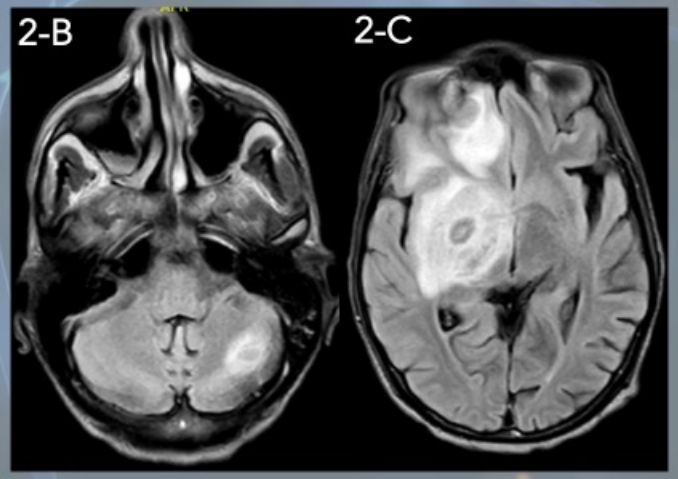
Pulmonary Nocardiosis





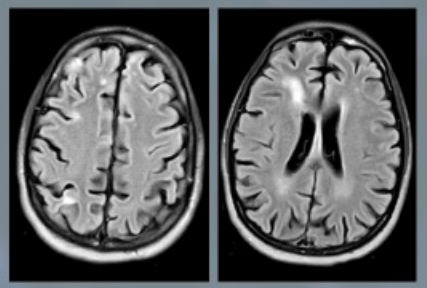
CNS Nocardiosis vs. Toxoplasmosis





Hospital Course

- Patient was initiated steroid for vasogenic edema and midline shift
- Antibiotics: trimethoprim-sulfamethoxazole and imipenem → later switched to Ceftriaxone (based on pulmonary cultures susceptibilities)
- Repeat MRI (below) shows **marked improvement** in the numerous ring-enhancing lesions throughout both cerebral hemispheres and the left cerebellar hemisphere



Discussion

- Differentiating between CNS nocardiosis and CNS toxoplasmosis is difficult in patients who are vulnerable to both, such as in HIV infected patients with CD4 count < 50
- LP and even lesion biopsy might confirm the diagnosis but may not be easy to obtain in patient with extensive brain lesion especially in the setting of high ICP with midline shift
- Tailoring treatment to cover both pathogens is not problematic

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Metformin-Associated Lactic Acidosis in the setting of pre-renal Acute Kidney Injury

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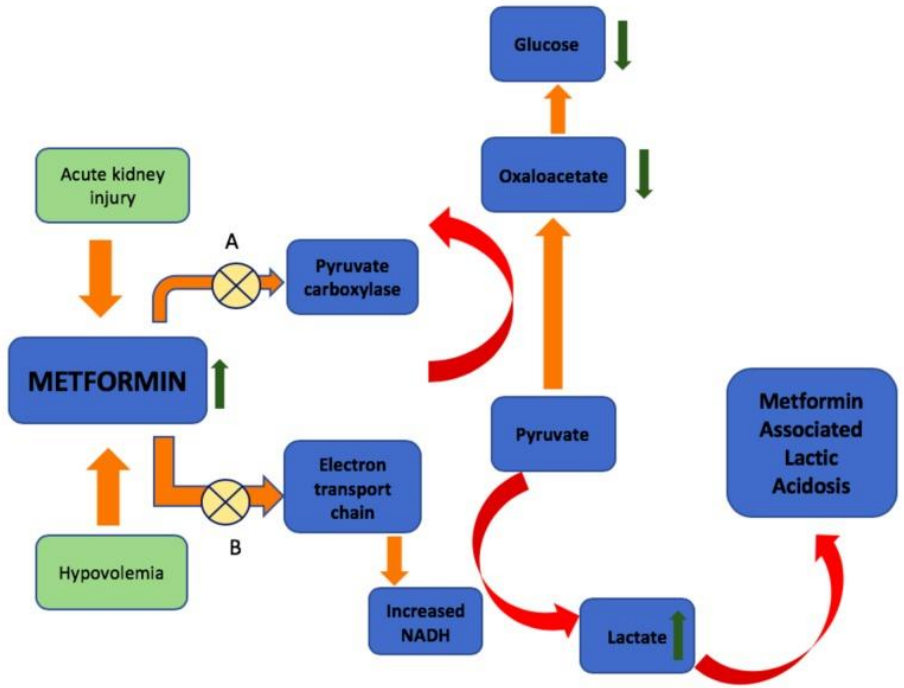
Introduction

- Metformin-Associated Lactic Acidosis (MALA) is a rare but serious adverse effect of metformin therapy often associated with acute kidney injury or hypovolemia
- This event occurs in the setting of increased metformin accumulation either via overdose or decreased renal clearance
- MALA is thought to result in acidosis via the inhibition of two pyruvate-utilizing pathways, forcing pyruvate metabolism via lactic acid production
- Despite aggressive therapy, overall mortality in patients with MALA is estimated at 36% or higher
- Current treatment is only supportive and focuses on maintaining blood pressure while resolving the acidosis and metformin accumulation with renal replacement therapy

Case Presentation

- A 66-year old man with a history of diabetes mellitus, hypertension, hyperlipidemia, and coronary artery disease presented to the Emergency Department with nausea, vomiting, and generalized weakness.
- The patient had been working on exterior home repairs in the aftermath of Hurricane Ida in temperatures that exceeded 90 degrees Fahrenheit.
- His home medications included metformin, glipizide, lisinopril, aspirin, clopidogrel, and atorvastatin.
- At the time of admission he was found to be in acute renal failure with a creatinine of 11.23 mg/dL, potassium of 6.0 mg/dL, anion gap of 27, lactic acid of 11.8 mmol/L, and a venous blood gas with pH 6.99. EKG showed peaked T-waves.

Proposed mechanisms of increased lactic acid production after metformin accumulation



Asif, Bennett, Marrakath, 2019 PMID: 31245187

Hospital Course

- The patient was treated with insulin, calcium gluconate, and volume resuscitation in the Emergency Department before emergent hemodialysis was performed.
- Two sessions of hemodialysis were required to resolve his acidosis and hyperkalemia. The patient's renal function recovered to his baseline after 3 days of admission.
- Patient was discharged with outpatient follow-up and new diabetes regimen of glipizide and linagliptin.

Discussion

- MALA is associated with decreased renal excretion of metformin during acute kidney injury, and mortality has been estimated at 36% or higher.
- The process of metformin-induced lactate production is uncertain but thought to be related to disruption of pyruvate-utilizing pathways, causing the metabolism of pyruvate into lactic acid.
- This can amplify lactic acidosis caused by another disease process, such as inadequate tissue delivery in this patient with volume depletion.
- While metformin is commonly used as a first-line diabetic agent, prescribers should be aware of its rare but serious adverse effects-particularly in patients at risk of renal dysfunction or hypovolemia.

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Thyroid Storm Leading to Cardiogenic Shock

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Introduction

Thyroid storm is rare, acute hypermetabolic state that presents with a variety of constitutional, GI, cardiac, and neurological symptoms. Tachyarrhythmias are a common early manifestation. Quick diagnosis and familiarity with complications of this state is imperative to prevent a fatal outcome, which has been noted to be as high as 30%.

Case Description

A 65 yo woman with PMH of Grave's Disease, hypertension, and medication non-adherence presented with lower extremity edema and dyspnea ongoing for three weeks. Other relevant symptoms included feeling overheated, fatigue, nausea, and orthopnea. She had not taken her prescribed medications in over a year. On physical exam, temperature 97.9 F, HR 164, other vitals wnl. She had an irregular rhythm, bulging eyes, 3+ pitting edema of the legs bilaterally, and was axo x3. Her extremities were warm to the touch. Patient had no recent cardiac workup.

Diagnostic Studies

Day 1:
- EKG – afib RVR
- Creatinine: 0.6 mg/dL
- AST/ALT: normal
- Total bilirubin: 1.5 mg/L
- BNP: 670 pg/mL
- Troponin: normal
- Lactic acid: 2.0 mmol/L
- TSH: <0.01 uIU/mL
- Free T4: 2.50 ng/dL

Day 2:
- Free T4: 1.84 ng/dL
- Lactic acid: 6.0 mmol/dL
- Total bilirubin: 3.2
- AST/ALT: 1354/326 U/L
- Creatinine: 1.3 mg/dL

TTE: EF 20%, global hypokinesis, moderate RV dysfunction, biatrial enlargement, RVSP 45 mmHg, CVP 15 mmHg

Imaging

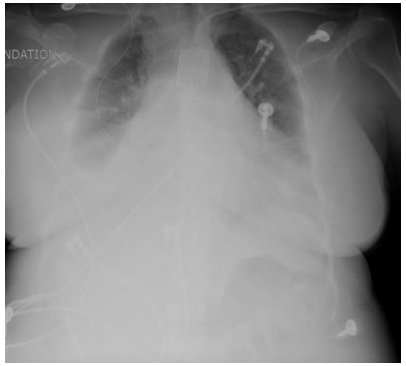

Fig 1 (left): AP X-ray chest Demonstrating enlarged cardiac silhouette, right pleural effusion, coarse interstitial attenuation, suspicious for pulmonary edema

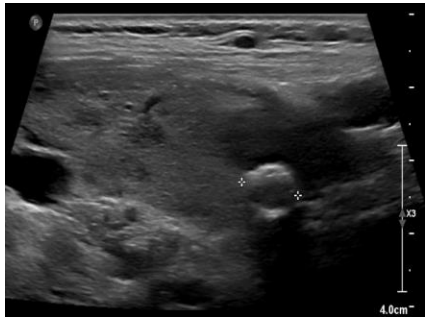

Fig 2 (right): US soft tissue head/neck/ thyroid Thyromegaly with heterogeneous echotexture, multiple thyroid nodules (right lobe seen here)

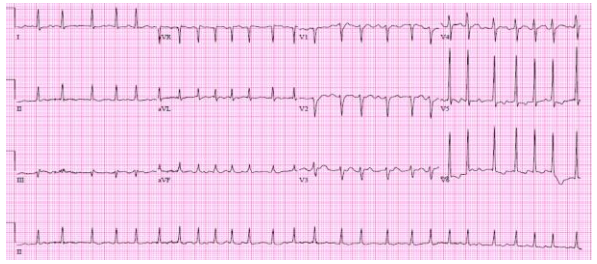

Fig 3 (left): Initial ECG demonstrating atrial fibrillation with rapid ventricular response

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Management & Outcome

Patient was started on propranolol, propylthiouracil (PTU), steroids, apixaban (CHADSVasc 2), and IV furosemide. 24 hours later, she had acute decompensation with rising lactate, LFTs, and creatinine. On exam, she was hypoxic and cold with signs of low flow state. Hemodynamics revealed cardiac index of 1.5 L/min/m2.. Propranolol was discontinued & she was started on dobutamine, and transferred to ICU. Dobutamine was switched to milrinone. Endocrine switched PTU to methimazole given her ischemic hepatitis. Esmolol added for heart rate control. Clinically, patient responded and was transitioned to an oral regimen of digoxin, apixaban, metoprolol succinate, sacubitril/valsartan, and methimazole.

Discussion

This case highlights the potential for cardiopulmonary collapse in thyroid storm. In this case, early recognition of thyroid storm was made and guideline therapy was started. Beta blockers are recommended to treat thyroid storm to decrease hyperadrenergic symptoms, and medications such as propranolol also have been shown to decrease peripheral conversion of T4 to T3. It is also well known that initiation of beta blockers in acute heart failure can worsen outcome due to the negative inotropy. Familiarity with decompensated HF as a manifestation of severe thyrotoxicosis is imperative in order to make a clinical decision on whether to start or hold beta blockers. A bedside echo, not done here, is a cheap and effective way to assess cardiac function given the prolonged tachyarrhythmias these patients tend have. Prompt treatment of thyroid storm is crucial, however starting a beta blocker should be done only after acute heart failure is ruled out due to the risk of cardiac compromise, as seen in our patient.

Gleich Syndrome

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2. Ochsner Lafayette General Medical Center- Lafayette, LA

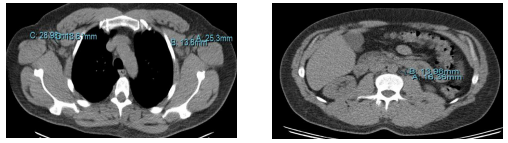
Introduction

- Episodic angioedema with eosinophilia, also known as Gleich Syndrome is a rare disorder characterized by monthly intervals of spontaneously resolving urticaria, fever, angioedema, and dramatic eosinophilia.
- Gleich Syndrome generally responds very well to systemic corticosteroid treatment, with affected patients usually having a good clinical prognosis.

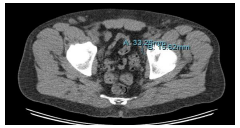
Case Description

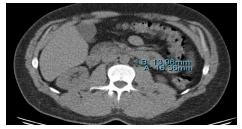
- A 36-year-old male presented to clinic with intermittent joint pain and swelling, subjective fevers, and chronic non-productive cough for the past three years.
- Of note, he started a job as a construction site manager in West Texas and Arizona approximately 4 years prior to presentation.
- Initial labs revealed significant leukocytosis with eosinophilia, mildly elevated CRP, and normal sed rate.
- Rheumatologic workup and imaging exhibited axillary lymphadenopathy, moderate splenomegaly, and bilateral alveolar consolidations of the mid lobes.
- Infectious disease evaluation revealed negative HIV and hepatitis screening, negative stool ova and parasites, and a positive coccidiomycosis IgM antibody, although cultures and IgG were negative.
- Biopsy of an axillary lymph node exhibited reactive lymphoid hyperplasia with sinus histiocytosis and no signs of atypia or evidence of malignancy.
- After further evaluation, the patient admitted to experiencing frequent episodes of night sweats and occasional episodes of rash and generalized edema.
- After seven months of extensive infectious disease, hematology, oncology, allergy, and immunology workup, bone marrow biopsy, a diagnosis of Gleich Syndrome was finally achieved for this patient.
- He responded very well to corticosteroid therapy during disease flares.

Imaging

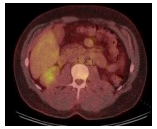


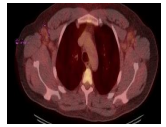
Images 1. & 2. CT abdomen and pelvis scan showing lymphadenopathy

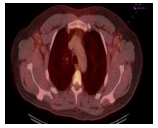




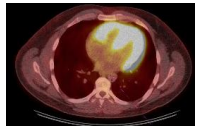
Images 3. & 4. CT abdomen/pelvis showing lymphadenopathy

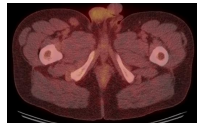






Images 5., 6., & 7. PET CT Thorax showing lymphadenopathy





Images 8. & 9. PET CT Thorax showing lymphadenopathy

Bone Marrow Biopsy Results

BONE MARROW FINAL REPORT

Diagnosis
PERIPHERAL BLOOD:

MILD LEUKOCYTOSIS. SEE NOTE.

NOTE: The eosinophils represent 6% of the circulating white blood cells.

RIGHT POSTERIOR ILIAC CREST, BONE MARROW ASPIRATE (PART 1) AND BIOPSY (PART 2):

NORMOCELLULAR MARROW WITH MARKED EOSINOPHILIA. SEE NOTE.

NOTE: The eosinophils and their precursors represent 37% of all nucleated bone marrow elements and 47% of the myeloid series (based on a differential count). No clinically significant mutations have been identified in a comprehensive myeloid NGS panel. The bone marrow is also negative for FIP1L1-PDGFRalpha gene

Conclusion

- Episodic angioedema with eosinophilia (EAE) is a multilineage cycling disorder which is rare with less than 50 reported cases.
- Defined by periodic cycling eosinophilia and angioedema and preceded by rise in serum IL-5.
- Previously reported patients with definite EAE had a rise in IgM levels which is uncommon in Idiopathic Hypereosinophiilc Syndromes (HES).
- EAE is associated with the presence of aberrant and/or clonal T-cell populations and eosinophil-driven pathology.
- Aberrant clonal lymphocytes $CD3^{-}CD4^{+}$, were shown to produce increased type II cytokines driving eosinophilia.
- Previously studied subjects had increased $CD3^{-}CD4^{+}$ aberrant T cells at peak eosinophilia coinciding with symptoms.
- Although the intracellular cytokine profile in T-lymphocytes suggests that the eosinophil activation may be lymphocyte-driven, the etiology of the cycling in EAE is still unknown.

References

P. Khoury et al. Episodic angioedema with eosinophilia (Gleich syndrome) is a multilineage cell cycling disorder. Haematologica. 2015 Mar; 100(3): 300–307. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4349267/

A Case Report of Neuro Sarcoidosis

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Department of Internal Medicine- Lafayette, LA

Introduction

- Neuro sarcoidosis is a diagnostic consideration in patients with a diagnosis of sarcoidosis who develop neurological complaints.
- Neurological complications are seen in only 5-10% of patients with established diagnosis of sarcoidosis.
- Generally neuro sarcoidosis responds well to high-dose glucocorticoids in the majority of patients, but relapse is common.

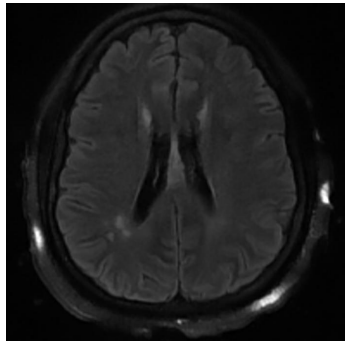
Case Description

- A 31-year-old male presented with chief complaints of double vision and binocular diplopia for 1 day. He also reported that his left eye was pulled inwards 1 day prior.
- He denied any headaches, dizziness, lightheadedness, balance issues, and had no other cranial nerve or motor/sensory deficits.
- The patient was seen in pulmonology clinic a few months prior for ongoing worsening SOB with nonproductive cough for over 2 years. Ct Thorax revealed bilateral hilar opacification with scattered nodules throughout the lungs.
- An extensive autoimmune/infectious work-up along with a bronchoscopy and subsequent biopsy revealed chronic granulomatous inflammation suggestive of pulmonary sarcoidosis.

Case Description cont.

- He was prescribed prednisone 40 mg; however, the prescription was never filled.
- In the background of pulmonary sarcoid his new symptoms of binocular diplopia with left lateral rectus palsy was concerning for neuro-sarcoid prompting an MRI of the brain.
- MRI results revealed multifocal enhancing cerebral cortex nodules, scattered leptomeningeal, optic chiasm distal optic nerve enhancement along with chronic appearing non-enhancing T2 hyperintense foci of cerebellar white matter consistent with neuro sarcoid.
- Neurology and Rheumatology recommended pulse dose steroids followed by a slow steroid taper with plans to start on biologic DMARDs in the outpatient setting. His symptoms slowly but steadily improved.

Imaging



MRI Brain revealing contrast enhancing lesions

Conclusion

- Neuro sarcoidosis is a noncaseating granulomatous disease of unknown etiology that can involve central, peripheral and autonomic nervous system.
- It's fairly uncommon with manifestation of sarcoidosis with <10% patients developing neurologic involvement.
- Neurological features usually develop within 2 years of diagnosis and can range from mono/polyneuropathies to paraparesis/milieu of multiple neuro symptoms.
- Histological analysis of tissue provides definite diagnosis , however lab or radiological support in the right clinical setting is sufficient.
- Corticosteroids are still the main stay of treatment for neuro-sarcoidosis with duration varying with severity of disease.
- Immunomodulators can be considered in addition to steroids in cases of severe/uncontrolled disease or can be used by themselves when steroids are not an option due to intolerance /side effects.

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Bathla G, Singh AK, Policeni B, Agarwal A, Case B. Imaging of neurosarcoidosis: common, uncommon, and rare. Clin Radiol. 2016; 71:96–106.

Tavee JO, Stern BJ. Neurosarcoidosis. Clin Chest Med. 2015;36: 643–56.

Feigning Weakness: Lessons from a Stranger Out of Town

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Learning Objectives

1. To practice holistic review and thorough history-taking to identify possible external secondary gain.
2. To recognize the harm of overtreatment in patients with factitious disorder.

Case Presentation

48-year-old man with CAD s/p PCI x 6 & 3-vessel CABG, cardiac arrest s/p CABG, pericardial window, CVA at 14 years old due to cerebral aneurysm and multiple sclerosis presented with BLE weakness with difficulty walking. He claimed that his symptoms were similar to prior MS flares that required high-dose IV steroids. He was visiting from out of town and was unable to provide other details of his medical history or current living situation.

He was started on steroids for MS flare, but imaging and workup revealed signs of prior MS flares but no acute flares.

He also complained of chest pain, and coronary angiogram revealed patent native vessels, stents, and grafts.

Quickly identifying factitious disorder and external secondary gain can prevent unnecessary, potentially harmful treatment.

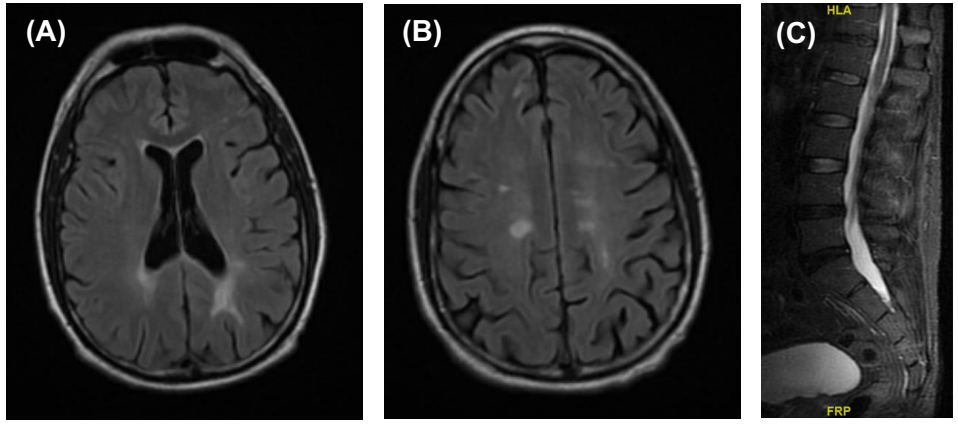

Figure 1. (A) MRI brain consistent with old MS flares. (B) MRI orbit with no optic neuritis (C) MRI spine with no demyelination

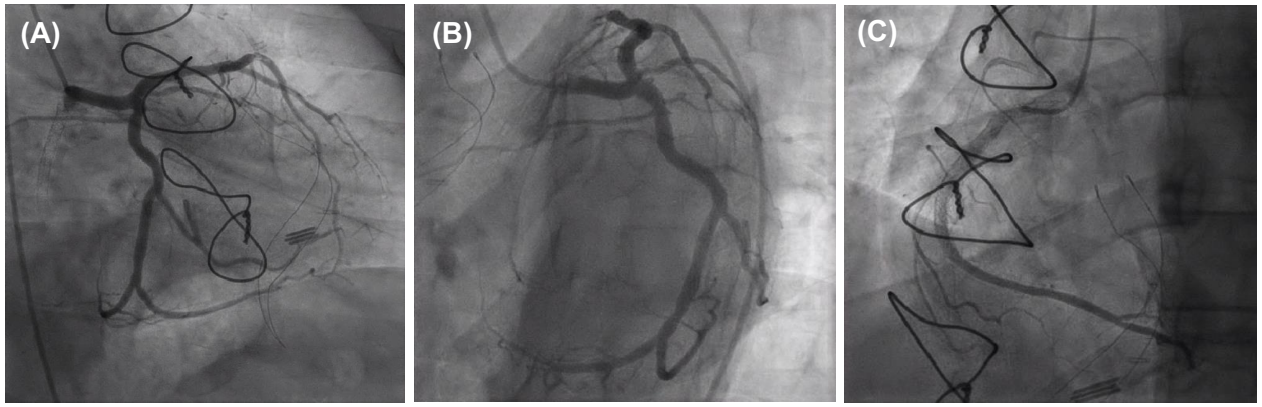

Figure 2. (A&B) Coronary angiogram with patent left coronary vessels, grafts, and (C) stent

Discussion

- He continued to have neurological complaints and chest pain, despite negative workup and maximum treatment.
- Upon psychiatric evaluation, the patient became angry and walked out of the hospital against medical advice.
- Due to the patient's homelessness and uncertain living situation, external secondary gain was believed to be housing.
- Factitious disorder with cluster B personality traits can lead to misdiagnosis and unnecessary treatment.
- Holistic review and through history-taking can elucidate non-medical needs that can drive external gain.

References

1. Bass C, Halligan P. Factitious disorders and malingering: Challenges for clinical assessment and management. *The Lancet*. 2014;383(9926):1422-1432. doi:10.1016/s0140-6736(13)62186-8
2. Direk N, Kulaksizoglu IB, Alpay K, Gurses C. Using personality disorders to distinguish between patients with psychogenic nonepileptic seizures and those with epileptic seizures. *Epilepsy & Behavior*. 2012;23(2):138-141. doi:10.1016/j.yebeh.2011.11.013.

Severe Noncirrhotic Hyperammonemia: What Urea-lly Should Consider

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Learning Objectives

1. To review the workup and management of hyperammonemia.
2. To learn about etiologies of hyperammonemia when liver pathology is unlikely.

Case Presentation

57-year-old man with HFrecEF, atrial fibrillation, and cervical neck fracture s/p recent corpectomy and fusion presented with worsening dysphagia and found to have a cervical fluid collection. He had no known underlying liver disease and denied significant alcohol history. He received a dexamethasone taper and a lumbar drain. He was admitted to the ICU for unstable atrial fibrillation with RVR. In the ICU, he became more somnolent and was unable to follow commands. Encephalopathy workup revealed hyperammonemia. Liver workup was normal. Workup for inborn errors of metabolism showed mildly decreased citrulline. His hyperammonemia resolved and his mentation improved with lactulose and rifaximin. Urea cycle disorder gene panel and further genetic workup is ongoing and to be continued outpatient.

Inborn errors of metabolism should be on the differential when hyperammonemia is not the result of liver disease.

Labs

WBC 7, **Hb** 9.6, **Plt** 313
Sodium: 142
Potassium: 4.2
Chloride: 103
Carbon dioxide: 34
BUN 91, **Cr** 0.53
Hepatitis A, B, C negative

TPro: 6.7, **Albumin** 1.9
AST/ALT: 136, 149
Alk Phos: 89
TBili: 0.3, **DBili** <0.1
PT/INR: 16.7/1.4
Ammonia 772

Urine and plasma amino acids:
Glutamine – normal
Ornithine – normal
Arginine – normal
Citrulline – 8 (normal 12-55)
Urine organic acids – normal
Urine orotic acid – normal

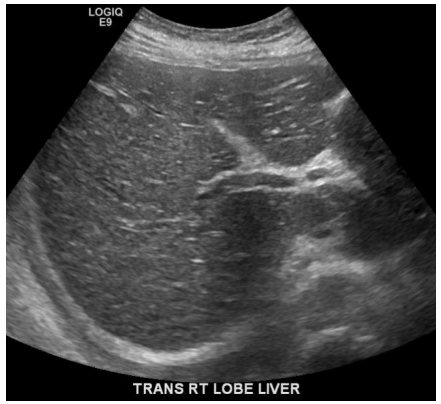

Figure 1. RUQUS with nonspecific heterogenous hepatic parenchyma bu no portal vein thrombosis.

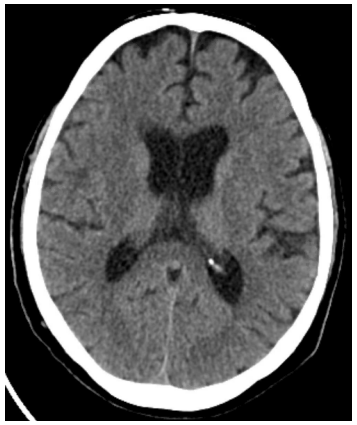

Figure 2. CT head with no cerebral edema

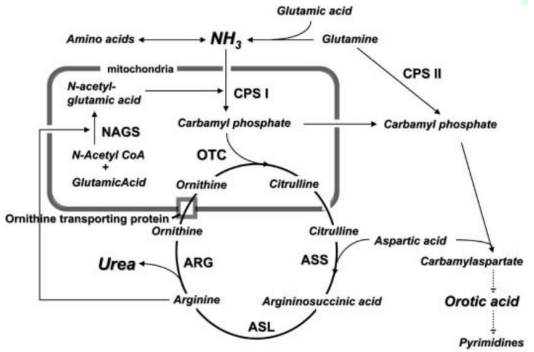

Figure 3. Urea cycle. ASL, argininosuccinate lyase; ARG, arginase; NAGS, N-acetylglutamate synthetase.

Discussion

- Patients with elevations of ammonia present with encephalopathy, which may progress quickly to cerebral herniation.
- Liver disease, medications, degradation of blood products, and high protein tube feeds can lead to hyperammonemia and elevated BUN.
- A workup for inborn errors of metabolism (IEMs), like a urea cycle disorder, should be considered in unexplained hyperammonemia.
- IEMs may be unmasked by steroid therapy.
- Treatment for a potential IEM begins prior to confirmation of an etiology.
- Geneticists should be consulted early on for evaluation and management.

References

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2. Clay AS, Hainline BE. Hyperammonemia in the ICU. *Chest*. 2007;132(4):1368-1378. doi:10.1378/chest.06-2940

Have You Seen Your Dentist Lately?

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2. Ochsner Lafayette General Medical Center, Lafayette, LA

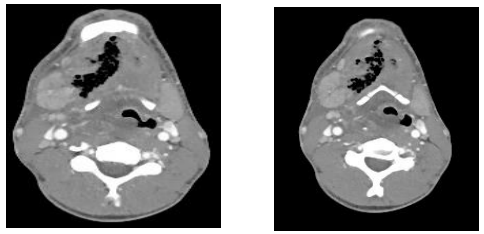
Introduction

- Although more common in children between the ages of two and four, retropharyngeal and parapharyngeal abscesses can occur in adults secondary to trauma or untreated oropharyngeal infection.
- Risk factors include poor dentition, diabetes, and immunocompromise. Bacteria most commonly implicated in retropharyngeal abscess include Group A Streptococcus pyogenes, Staphylococcus aureus, Fusobacterium, Hemophilus, and other respiratory anaerobes.
- Parapharyngeal abscesses are among the most dangerous oropharyngeal conditions due to their ability to spread to the anterior and posterior portions of the mediastinum with complications such as airway obstruction, which is the most common cause of death in these cases.

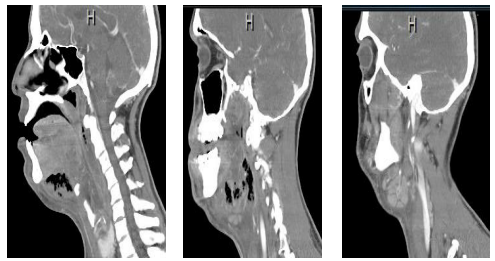
Case Description

- 39-year-old gentleman with no significant past medical history who presented to the ED with complaints of intermittent dyspnea, jaw swelling, and right molar tooth pain that started approximately two years ago.
- This past week however, he developed progressively worsening swelling, shortness of breath, a low-grade fever, change in voice, and dysphagia. He had an appointment with his dentist one day prior to presentation and was started on metronidazole without improvement.
- ENT was consulted and took the patient emergently to the OR after CT neck soft tissue revealed gas production with severe cellulitis/gangrenous changes involving the submental, right submandibular, and right parapharyngeal regions and loculated appearance of fluid collection at the right supraglottic paravertebral region resulting in mass-effect and localized compressive airway occlusion at the hypopharynx.
- While in the OR, minimal purulent drainage was evacuated via right lateral neck incision and he required an emergency tracheostomy for airway compromise as couldn't be intubated due to the oropharyngeal mass effect. He was subsequently admitted to the ICU for postoperative care and ventilatory support where he was started on Unasyn and clindamycin.

Imaging



Images 1. & 2. Demonstrate loculated gas and abscess collection in the patients parapharyngeal area.



Images 3., 4., & 5. Demonstrate loculated gas and abscess collection in the patients parapharyngeal area.

Lab Results- Microbiology

- Anaerobic culture was positive for Peptostreptococcus and Prevotella denticola
- Body fluid culture was positive for Streptococcus anginosus

Conclusion

- Parapharyngeal abscesses secondary to gas forming organisms pose a potentially life-threatening illness if not promptly recognized and addressed surgically as well as with appropriate broad poly-microbial coverage of the oropharyngeal flora.
- Further adding to the burden of disease is that patients without antecedent medical history or risk factors can suddenly find themselves in the path of danger once seeding has occurred in the danger space before further dissemination.
- Patient responded well antimicrobial therapy with Unasyn and Clindamycin and improved significantly after washout and drainage with ENT. Subsequently downgraded from ICU and weaned to room air then discharged just 4 days after admission.

References

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Liver Abscesses in the Context of Entamoeba Histolytica Infection

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Department of Internal Medicine- Lafayette, LA

Introduction

- Entamoeba histolytica is an anaerobic parasitic amoebozoan which is transmitted primarily through the fecal-oral route via contaminated water and in areas with poor sanitary conditions or lack of indoor plumbing. Major manifestations include amoebic dysentery and liver abscesses.
- Infection is uncommon in the United States, however there are reported cases in Texas and California due to close geographic proximity to areas endemic with E. Histolytica (ie Mexico, Central and South America).
- We will discuss a case of E. Histolytica hepatic abscesses in an immigrant from Mexico.

Case Description

- A 26-year-old male presented after a seven-day history of fever and chills followed by right sided abdominal pain for 3 days. The patient immigrated to New Iberia, LA from Northern Mexico; having last visited Mexico 1.5 years ago. On admission, he was febrile and tachycardic. Physical exam revealed right upper quadrant tenderness without guarding or rebound tenderness. Labs revealed WBC 29.3, AST/ALT 86/77, and direct bilirubin 0.6. An abdominal ultrasound and CT scan showed four hypoechoic, right-sided liver lesions. He was empirically treated with IV ciprofloxacin and Flagyl with improvement in symptoms and stabilization of vital signs.
- Extensive infectious workup in the hospital was unrevealing, including abscess fluid pathology, gram stain, and cultures obtained from interventional radiology drainage of a liver abscess.
- After discharge, a stool PCR parasite panel returned positive for Entamoeba Histolytica. He followed up in ID clinic, and because his previous treatment regimen had not included an intraluminal agent, he was prescribed metronidazole 500mg TID x10 days followed by oral paromomycin 1g TID x10 days.

Lab Workup

- **Extensive workup included Serum serologies, cultures, and PCR tests; urine antigen tests; Abscess fluid cultures; and stool cultures which were all negative.**
- **After discharge stool PCR resulted positive for Entamoeba Histolytica**

Serum, Urine Studies

Serum – all negative
Syphilis Aby
Brucella Aby
Coccidioides IgM, IgG
Histoplasma Ag
Bartonella PCR
Echinococcus Aby
Strongyloides IgG
Hepatitis Panel, T-Spot
Blood Culture x2
Urine Histo, Blasto- Neg.

Stool Studies

Entamoeba Histolytica PCR - positive

Negative Stool Studies:
Ova & Parasites
Fecal Leukocytes
Cryptosporidium PCR
Giardia PCR
Dientamoeba frag. PCR
Cyclospora PCR
Stool Culture

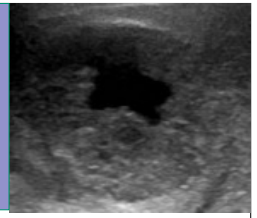
Abscess Fluid

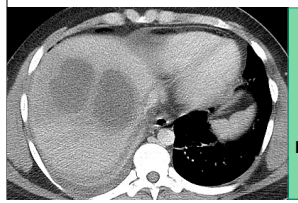
WBC 59,000
51% Neutr, 47% Lymph.
RBC 43,000
Pathology:
--Sparsely cellular sample

AFB Cx – neg.
Fungal Cx – neg.
Gram Stain
--Rare G+ Cocci
Anaerobic Cx – neg.
Aerobic Cx – neg.

Liver Imaging

US Liver:
1 of the 4 Hypoechoic lesions, 6cm, with anechoic center





CT A/P with Contrast:
2 of the 4 lesions in right lobe of the liver

Conclusion

- In this case, we illustrate a pathognomonic case of Entamoeba Histolytica liver abscesses in an immigrant from Mexico. Diagnosis was made via stool PCR and the patient was started on the appropriate treatment regimen, which included tissue and intraluminal agents.
- Entamoeba Histolytica is transmitted through the fecal oral route and often spread through contaminated water sources. The life cycle of Entamoeba Histolytica consists of ingestion of mature cysts from fecally contaminated food, water, or hands. Excystation occurs in the small intestine and trophozoites are released, which migrate to the large intestine. Trophozoites can then invade the intestinal mucosa or blood vessels and reach extraintestinal sites (i.e. liver, brain, and lungs).
- Amebic liver abscesses are the most common manifestation of extraintestinal amebiasis which was seen in this case report. In the United States the majority of reported Entamoeba Histolytica infections occur in high risk groups which include the MSM community, travelers, recent immigrants, immunocompromised individuals, and institutionalized populations.
- This report serves as a reminder of the importance of considering infectious etiologies which are uncommon in the United States but which have higher prevalence in immigrants from endemic regions.

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LSU Health NEW ORLEANS

Dermatomyositis: The Great Predictor

Department of Internal Medicine

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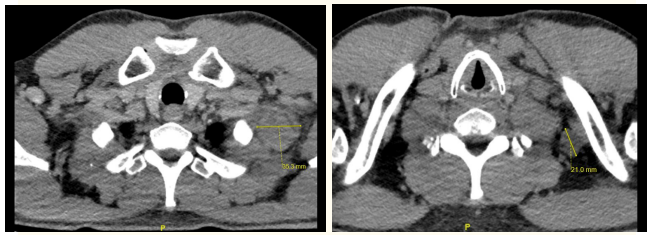
Introduction

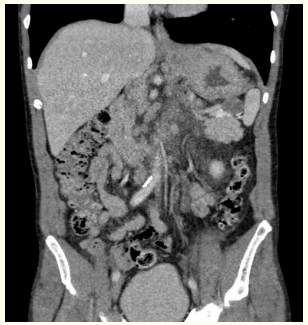
- Dermatomyositis (DM) is an idiopathic inflammatory connective tissue disease with varying muscle and cutaneous involvement.
- It typically presents with progressive proximal muscle weakness and classic cutaneous manifestations (e.g. gottron's papules and heliotrope rash).
- It has a well established association with malignancy.
- Patients with DM have a 6-fold higher risk of malignancy compared with the general population.
- This risk is particularly evident in the first 2 years after DM diagnosis.

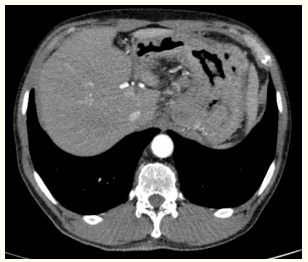
Case Presentation

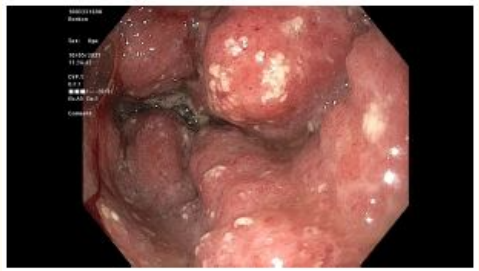
- A 59-year-old male with newly diagnosed DM four months prior presented with a complaint of left neck swelling.
- It was first noticed the morning prior to presentation.
- The swelling was non-tender and did not affect his swallowing or breathing.
- Other complaints at that time included weight loss, skin rash, and proximal muscle weakness.
- Notably, he had a screening colonoscopy one month prior that revealed 3 non-cancerous polyps.
- Vital signs were normal, and his physical examination was significant for left sided supraclavicular lymphadenopathy that was non-tender, firm, and mobile.
- Preliminary lab-work was non-revealing expect for a mild thrombocytosis.
- CT imaging of the neck was obtained which revealed lymphadenopathy of the left supraclavicular and infraclavicular regions.
- He was admitted to the internal medicine service for further work-up.

Imaging









Hospital Course

- Follow-up CT chest and CT abdomen/pelvis were obtained revealing a proximal gastric mass with extensive regional adenopathy.
- Gastroenterology was consulted for further investigation and EGD was performed in which a large, fungating, ulcerated, and partially circumferential mass was found extending from the gastroesophageal junction to the posterior wall of the stomach.
- Pathology revealed invasive gastric adenocarcinoma.
- He followed-up with H/O, and further staging imaging was done which revealed bony metastasis.
- He was eventually started on systemic chemotherapy with capecitabine and oxaliplatin.

Discussion

- Despite increased risk of malignancy in DM there are no established guidelines for cancer screening in these patients.
- Some cancers with increased risk in DM patients include: lung, ovarian, colorectal, cervical, bladder, nasopharyngeal, esophageal, kidney, and lymphatic/hematopoietic.
- Our patient received age-appropriate cancer screening with colonscopy following his diagnosis of DM; however, this age-appropriate screening did not reveal his underlying gastric cancer.
- Because the cancers linked to DM involve many different organs systems, many cancers can go undiagnosed by only following age-appropriate screening guidelines.
- Therefore, in patients with newly diagnosed DM, blind cancer screening may be appropriate; however, if the patient has clinical symptoms of a particular cancer, then more focused testing should be pursued.

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A Case of the Malabsorbed Thyroid Hormone

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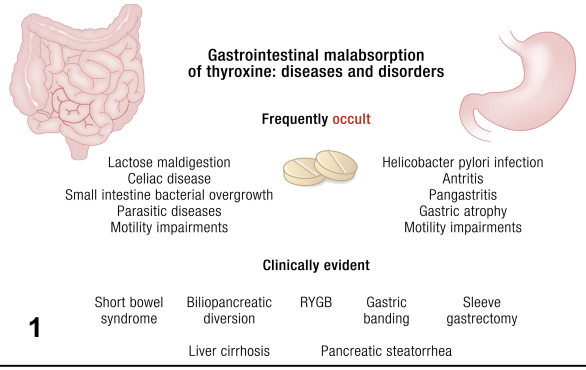
Introduction

- Hypothyroidism affects 1 in 300 Americans, making it one of the most common endocrinological disorders we encounter.
- Hormone replacement with levothyroxine is the mainstay of treatment
- For patients with refractory hypothyroidism the most common differential to be considered is poor patient compliance with levothyroxine
- Gastrointestinal malabsorption of oral levothyroxine is becoming a more frequent cause of refractory hypothyroidism as previously reputed

Case Presentation

- A 79-year-old woman with history of type 2 diabetes mellitus, atrial fibrillation on Eliquis, hyperlipidemia, hypothyroidism and recent ascending cholangitis secondary to choledocholithiasis status post biliary drain placement presented with 1-week history of weakness, hypotension and tachycardia.
- On evaluation her temperature was 97.4 F, BP 90/46, HR 100, RR 18, O2 Sats 100% on RA.
- Physical exam was remarkable for tachycardia with irregularly irregular rhythm, fine crackles of lung bases on auscultation and a soft nontender abdomen.
- Laboratory studies were notable for WBC 13.4K/uL, Cr 2.36 mg/dL, TSH 104.31 uIU/mL, Free T4 0.11 ng/dL. Chest x-ray was significant for basilar atelectatic changes with no pleural effusion or pneumothorax.
- Patient was admitted for worsening acute kidney injury and treatment resistant hypothyroidism

Images



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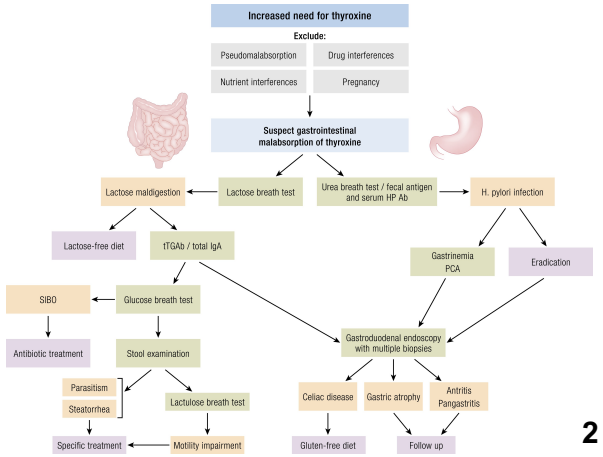

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Table 1: Trend of TSH and FT4 through Hospital Course

	Hospital Day 1	Hospital Day 3	Hospital Day 10	Hospital Day 17
TSH (uIU/mL)	104.31	102.76	73.24	17.06
FT4 (ng/dL)	0.11	0.41	0.90	0.88

Hospital Course

- Cholangiogram showed choledocholithiasis of distal common bile duct with dilation of extrahepatic and intrahepatic bile ducts and subsequent exchange and upsize of biliary drain.
- Given the drastic increase in TSH despite consistent oral levothyroxine use and complicated cholestatic history, the patient was transitioned from oral to intravenous levothyroxine after hospital day 1.
- Over the course of 2 weeks, the patients TSH decreased to 17.06 uIU/mL and Free T4 increased to 0.88 ng/dL (Table 1). Patient underwent exploratory laparotomy with common bile duct exploration, cholecystectomy and sphincteroplasty.

Discussion

- Hypothyroidism is a common disease, easily treated with thyroid hormone replacement therapy (levothyroxine).
- The tablet formulations of levothyroxine contain a stable salt, sodium that requires dissolution by gastric acid prior to absorption in the small intestine. We present a case in which hypothyroidism did not respond to therapy due to possible impaired gastrointestinal absorption.
- Levothyroxine is absorbed along the whole small intestine (~15% in duodenum, ~30% in upper jejunoileum, ~24 % in lower jejunoileum)
- GI malabsorption etiology (Figure 1)
  - Lactose maldigestion, celiac disease, SIBO, H. pylori, gastric atrophy, *biliopancreatic diversion,* gastric sleeves, liver cirrhosis, pancreatic steatorrhea
  - Drugs causing malabsorption: PPIs, calcium carbonate, beta blockers, raloxifene, TCAs, bile acid sequestrants
- Exclusion of pseudomalabsorption, drug interference, nutrient interference, and pregnancy is suggested prior to further gastrointestinal testing. (Figure 2)
- Levothyroxine absorption test is used to differentiate between malabsorption and pseudo-malabsorption (intentional nonadherence)

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When Normal Is Not Normal: A Case of Rasburicase-Induced Hemolytic Anemia

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Background

- Glucose-6-phosphate dehydrogenase (G6PD) deficiency is the most common enzymatic disorder of red blood cells in humans worldwide.
- G6PD deficiency is an X-linked disorder predominantly affecting males of African, Southern European, Middle Eastern, Oceanic and Southeast Asian descent.
- The gold standard for diagnosing G6PD deficiency is by obtaining G6PD enzyme levels via quantitative analysis.

Case Presentation

- A 71-year-old African-American male with a history of diffuse large B cell lymphoma (DLBCL) was admitted to the hospital with generalized weakness, falls, and concern for disease recurrence.
- Restaging CT scan of the abdomen and pelvis revealed a distended bladder, thickened rectal wall and soft tissue fullness in between these structures c/w locally advanced disease.
- Radiologic findings combined with the patient's poor performance status led to the decision to pursue palliative chemotherapy with rituximab and five days of high dose prednisone.
- Due to his high risk of tumor lysis syndrome, he received rasburicase and aggressive hydration prior to treatment.
- Given his African American descent, he was screened for G6PD deficiency using quantitative analysis with his G6PD level being in the low normal range (Figure 3)
- Four days after receiving rasburicase, he developed substernal chest pain with dyspnea.
- Labs revealed a drop in his hemoglobin of 4 grams to 6.5; total bilirubin of 2.7; LDH of 2,363; and an undetectable haptoglobin.
- Peripheral smear revealed evidence of Heinz bodies, microspherocytes and bite cells c/w G6PD associated hemolysis(Figures 1 and 2).
- Supportive measures including blood transfusions and avoidance of offending agents lead to complete resolution of hemolysis within a week.

Images

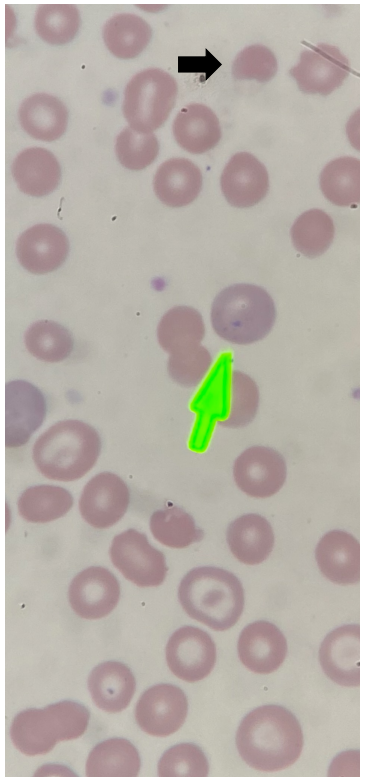

Figure 1: Peripheral smear showing Heinz bodies (green) and bite cell (black)

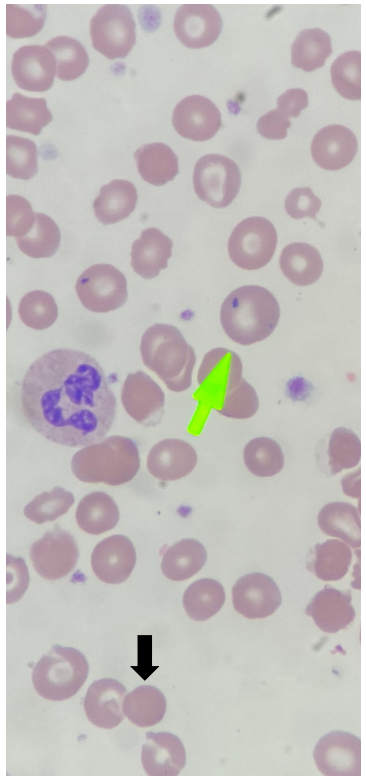

Figure 2: Peripheral Smear showing Heinz bodies (green) and microspherocytess (black)

RBC	4.14 - 5.80 x10E6/uL	3.85 ⌄
G6PD Qn	127 - 427 U/10E12 RBC	133

Figure 3: G6PD Quantitative analysis revealed levels at lower-limit of normal.

Discussion

- With an estimated 400 million carriers of a G6PD deficiency gene and prevalence of up to 20% in tropical and subtropical areas of the world, it is one of the best-characterized examples of genetic polymorphism in the human species
- Rasburicase is a well-known trigger for G6PD associated hemolysis and this patient was appropriately screened as a male of African descent.
- Quantitative analysis is the gold standard in diagnosing G6PD deficiency, and this patient was found to have G6PD level in the lower limits of normal.
- Yet, as was discovered in our patient, patients with G6PD levels in the low normal range remain at risk of developing G6PD associated hemolysis.
- Thus, close monitoring is required in this setting after exposure to potentially offending agents such as rasburicase.
- Patients at risk for tumor lysis syndrome may benefit from more definitive testing techniques in this situation.

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A PRESing Neurologic Sequela of COVID19 Infection

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INTRODUCTION

- A crucial component in the management of delirium is to identify the underlying causes, particularly reversible causes.
- One less-common, reversible cause of delirium in severely ill patients is the Posterior Reversible Encephalopathy Syndrome (PRES).
- PRES is thought to develop as a result of a failure in cerebral blood flow autoregulation at the endothelial level and is associated with uncontrolled hypertension and pregnancy.[1,3,4]
- Both PRES and AIDP have been described in association with COVID19 infection.[4,6]

CASE HISTORY

Patient: 29-year-old African-American female. ~3 weeks postpartum from a C-section delivery (G4P4).

Chief Complaint: Transfer from OSH for "seizure activity" with recent ascending weakness and encephalopathy.

Medical History: Anemia of Pregnancy, COVID-19 (~5 wks. prior)

Physical exam: Notable for profound weakness of both the upper and lower extremities with diffuse hyporeflexia. Confused and disoriented at presentation. BP 158/87 mmHg. Tmax 97.7 °F

EVALUATION

- Developed COVID-19 at 30 weeks gestation and was admitted to the OB/GYN service. That hospital stay was complicated by uterine rupture (prompting emergency C-section), wound dehiscence, and concern for intrabdominal infection.
- Following discharge, she developed ascending weakness, hyperalgesia, and multiple falls at home. Initial clinical evaluation was concerning for AIDP, and she subsequently developed worsening hypertension, and seizure like activity around the time of transfer.
- A Lumbar puncture had been performed immediately prior to transfer (6 WBCs/μL, 49 mg/dL glucose and protein, and no organisims).
- MRI at our facility demonstrated findings concerning for PRES (*Figure 1*) prompting transfer to the ICU for IV antihypertensive therapy. [2]

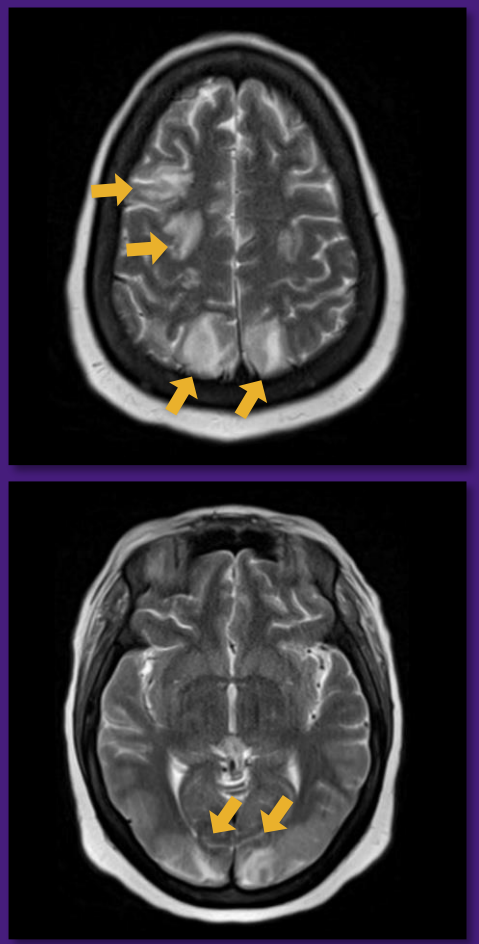

Figure 1. MRI Brain
Areas of T2 hyperintensity in the bilateral occipital and parietal lobes noted on MRI obtained shortly after admission. Also present on this scan (not pictured here) was diffusion restriction extending caudally into occipital lobes and the posterior temporal lobes.

TREATMENT

- Her mental status rapidly improved with IV antihypertensives, although she ultimately required five oral antihypertensives to transition to PO therapy (despite no prior history of hypertension or history of antihypertensive use in the past).
- Her strength slowly improved over the next 3 weeks with physical therapy, supportive care, and a trial of IVIG.

DISCUSSION

This case demonstrates a rare cause of delirium, likely triggered by a cascade of events following COVID-19 infection. We suspect she developed AIDP in the aftermath of COVID-19 during her pregnancy. In addition to causing motor and sensory changes, AIDP has also been known to cause autonomic dysfunction.[5] We believe this dysautonomia then precipitated her rapid elevation of blood pressure, leading to PRES.

Although patients hospitalized for COVID-19 most typically present with respiratory illness, this case underscores the broad array of pathophysiology which can be induced by SARS-CoV-2 infection, leading to less common clinical syndromes such as PRES and AIDP.[4,6]

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LSU Health Baton Rouge

Our Lady of the Lake Regional Medical Center
Franciscan Missionaries of Our Lady Health System

Late Presentation of Rheumatoid Arthritis in Sickle Cell disease: a case report

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Introduction

- Rheumatoid arthritis (RA) is a chronic inflammatory disease involving the small joints of the hands and feet that typically presents in the third to fifth decade of life
- Classic RA causes symptoms of polyarthritis that affects the small synovial joints of upper and lower limbs, including elbows, shoulders, ankles, and knees
- Based on guidelines from the American College of Rheumatology, diagnosis of RA requires at least six of a possible 10 in the following four domains: involved joints (range 0–5), rheumatoid factor (RF) or anti-cyclic citrullinated peptide antibody (anti-CCP) (range 0–3), elevated erythrocyte sedimentation rate (ESR) or C-reactive protein (CRP) (range 0–1), and symptom duration of six weeks or more (range 0–1) (1)
- Joint pain can develop as a sequela of sickle cell disease (SCD), likely secondary to acute synovitis, avascular necrosis, or gout
- Although RA and SCD can occur independently and both affect synovium of joints, their coexistence may be attributable to more than just coincidence
- We are presenting an elderly male with a longstanding history of sickle cell disease who developed rheumatoid arthritis in his 60s

Case Description

- A 66-year-old African American male with a history of SCD with associated avascular necrosis of the hip and recurrent deep vein thromboses presented to the emergency department with diffuse joint pains in his bilateral shoulders, arms, neck, and back
- He had multiple presentations over a period of about 2 years to the emergency department related to joint pains that were attributed to his sickle cell crises that had minimal improvement despite being treated with standard therapy
- On physical exam he had exquisite pain with both active and passive movement of all extremities
- Workup for inflammatory arthropathy yielded a positive anti CCP IgG, Rheumatoid factor IgM, and elevated inflammatory markers
- The patient was seen by rheumatology and initiated on methotrexate for treatment of rheumatoid arthritis

Case Description (labs/imaging, etc.)

Labs on Presentation	Lab value	Reference
ANA Pattern	Speckled	*
ANA Titer	1:320	
Rheumatoid Factor IgM	>100 units	0-6
ESR	69 mm/hr	0-15 mm/hr
Cyclic citrullinated peptide IgG	179 units	0-19
C Reactive Peptide	9.62	<=0.50

Fig 1: Given nonimprovement with standard treatment for sickle cell crises, a screening rheumatoid factor was ordered which revealed elevated inflammatory markers, a positive CCP, and a positive antinuclear antibody

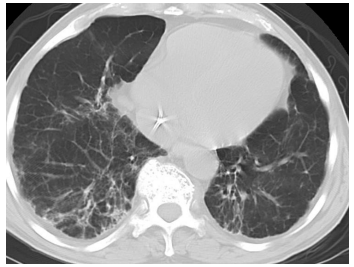

Fig 2: CT of the lungs showed chronic interstitial changes suggestive of UIP

Hemoglobin	Percentage
HbF	3.0
HbA	12.1
HbS	80.0
HbC	0
HbA2	4.9

Fig 3: Hemoglobin electrophoresis performed confirmed presence of sickle cell disease

Conclusion

- Although the exact mechanism is unclear, proposed mechanisms of overlap include endothelial dysfunction from vasoocclusive crises, inflammatory cytokines, and oxidative stress (2)
- Given irreversible joint damage from untreated RA, although symptoms of RA and SCD may overlap, persistent joint involvement in sickle cell disease should prompt workup of rheumatic etiology
- Treating SCD coexisting with RA is a clinical dilemma, due to having to weigh the benefits of pain relief with the risks of immunosuppression and infection
- In many cases, disease modifying anti-rheumatic drugs as monotherapy in patients with both RA and SCD were shown to be insufficient in providing complete relief of symptoms, often requiring immunobiologic therapy (2)
- In conclusion, with the advent of new therapies and better access to care, the longer life expectancy of patients with sickle cell disease correlates with a greater increase in the prevalence of RA, warranting further attention

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Fungal PetroClival Osteomyelitis due to Aspergillus in an Immunocompetent Patient

Ayesha Malik, MD; Sahith Thotamgari, MD; Shreedhar Kulkarni, MD

INTRODUCTION

- Skull base osteomyelitis (SBO) is a rare entity with severe life-threatening consequences.
- Numerous pathogens, such as Streptococci, Pseudomonas, Staphylococcus aureus, and less commonly, fungal or mixed bacterial infections have been recognized (1).
- Fungal skull base osteomyelitis is uncommon and is usually seen in immunocompromised patients and is mostly caused by Aspergillus and Candida species (2).
- The overall mortality in skull base osteomyelitis is around 9.5% (3) and can go up to 50% in fungal skull base osteomyelitis due to Aspergillus.

CASE PRESENTATION

- A 62-year-old man with a history of hypertension and well controlled type 2 diabetes (HgA1c 6.4%) presented with progressive worsening of left ear pain, neck pain and double vision for the past two months.
- Ear problems started around 10 months ago when he had right sided ear pain. He was found to have otomastoiditis and felt better after undergoing mastoidectomy.
- About 2 months ago, he reported having pain in the left ear for which a tympanostomy tube was placed and was started on oral antibiotics in an outlying hospital.
- Physical examination revealed erythematous tympanic membrane and minimal purulent drainage in left ear. Left lateral gaze palsy was present on neurologic examination.
- CBC and CMP were unremarkable. Nasal endoscopy showed normal nasal mucosa.
- CT scan of temporal bone showed left petroclival hypodensity and cortical erosion.
- MRI further confirmed findings suggestive of petroclival osteomyelitis. Gallium scan showed increased uptake in the left petroclival area suggesting infectious/inflammatory process.
- Culture from left ear purulent discharge grew Aspergillus species.
- Patient underwent left sided mastoidectomy and intraoperative cultures from left ear again grew Aspergillus species.
- Patient was treated with intravenous voriconazole

DISCUSSION

- Aspergillus and Candida are the most commonly reported fungal pathogens causing central or atypical SBO, although the infection is very rare in immunocompetent patients.
- Our patient does not have any immunodeficiency conditions, his diabetes is well controlled, and HIV is negative.
- Fungal SBO can have significant morbidity and mortality rates up to 50%. Early diagnostic sampling along with high suspicion towards fungal infection should be considered in diabetic patients who do not improve with antibiotics

CONCLUSION

- PetroClival osteomyelitis due to Aspergillus species is a serious condition seen even in immunocompetent patients with high mortality rate and treatment includes aggressive surgical debridement, long-term culture-directed systemic antimicrobial therapy.

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A case report of fulminant myocarditis after recovery from COVID-19

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Introduction

- Myocarditis remains a rare complication of viral infections, with recent CDC data suggesting an association between COVID-19 infections and myocarditis hospitalizations (Fig 5 (1))
- Among all patients hospitalized for myocarditis between March 2020 and January 2021, 41.7% had a recent COVID-19 infection
- A clinical diagnosis may be made with electrocardiogram (ECG), which may show characteristic changes of diffuse ST elevations and T wave abnormalities as well as elevated cardiac enzymes
- When feasible, cardiac magnetic resonance imaging (CMRI) should be done to confirm the diagnosis
- Although this is frequently asymptomatic and self resolving, the myocarditis can be associated with life threatening sequelae including arrhythmia, and fulminant heart failure and cardiogenic shock with ventricular dysfunction
- We present a young otherwise healthy male with no comorbidities who developed fulminant myocarditis requiring a ventricular assist device

Case Description

- **A 27 year old healthy male presented to the emergency department complaining of diarrhea, generalized weakness, and fatigue**
- Of note, he had tested positive for COVID-19 approximately 6 weeks prior with only mild body aches that had completely resolved
- He presented to the emergency department on day 40 after his positive test complaining of diarrhea and was treated with intravenous hydration and discharged
- He returned two days later with worsening of his weakness and now dizziness, he was hypotensive requiring initiation of vasopressors
- At this time, a polymerase chain reaction for COVID-19 was negative
- An echocardiogram performed revealed an ejection fraction of 15-20% with global hypokinesis, and a dilated right ventricle and right atrium. (EKG in Fig 1)
- He was urgently transferred to the catheterization lab, which revealed normal coronary arteries, with biventricular failure likely secondary to myocarditis
- He remained on ventricular and ventilatory support, but was able to be extubated and weaned off ventricular support after 4 days
- Cardiac function promptly returned to baseline (fig. 2) within a week and the patient was able to be discharged to home in stable condition

Case Description (cont'd)

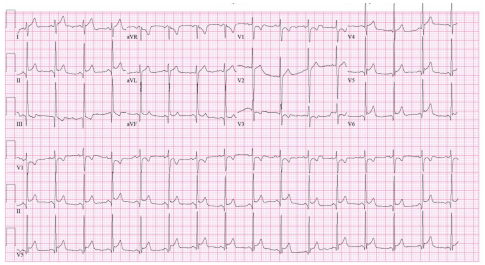

Figure 1: EKG upon presentation showed sinus tachycardia with diffuse ST elevations, T wave abnormalities and ST depressions

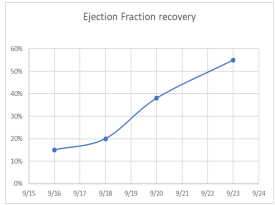

Fig 2: Serial echocardiograms were performed showing prompt recovery of ventricular function with standard supportive therapy

Labs on admission	Lab value	Reference
Troponin	8.012 ng/mL	0-0.045
CK-MB	17.2 ng/mL	<=7.2
CRP	29.60 mg/dL	<0.50
BNP	528.3 pg/mL	<=100

Fig 3: Cardiac enzymes and inflammatory markers on admission, troponin peaked at 30.509 ng/mL, but subsequently trended downward

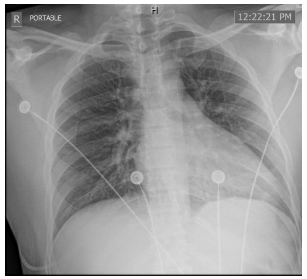

Fig 4: Chest X Ray on admission showed a slightly enlarged cardiac silhouette

Conclusion

- Myocarditis is a rare condition both in patients with and without COVID-19 infection
- In this patient, a clinical diagnosis of fulminant myocarditis was observed approximately 6 weeks after full recovery from a COVID-19 infection
- Among the 2,116 patients with COVID-19 and myocarditis, 1,895 (89.6%) received a diagnosis of COVID-19 and myocarditis during the same month; the remaining patients received a myocarditis diagnosis 1 month (139; 6.6%) or ≥2 months (82; 3.9%) after their COVID-19 diagnosis (1)
- COVID-19 must be recognized as a strong risk factor for developing myocarditis, warranting implementation of prevention strategies such as vaccination
- Workup of COVID-19 related fulminant myocarditis often warrants ruling out other de novo causes such as ischemic heart disease or acute heart failure
- Management primarily consists of supportive therapy including ventricular assist devices; there is no role for non steroidal anti inflammatory drugs or corticosteroids in management of viral myocarditis (3-4)

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A Case Report of Gullian-Barre Syndrome following SARS-CoV-2 Viral infection

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Introduction

- With the evolution in different strains of novel corona virus (SARS-CoV-2) significant non-pulmonary complications were reported and neurological consequences are one among them.
- We present a case of Guillain-Barré syndrome (GBS) post COVID-19 viral infection.

Case Description

- Patient is a 50-year-old Caucasian male with recent Covid-19 viral pneumonia 2 weeks prior to presentation comes with complaints of dyspnea and ascending paresthesias of bilateral lower extremities.
- Examination revealed a motor strength of 5/5 in upper extremities, 3/5 in lower extremities with areflexia and decreased sensations.
- Imaging revealed no abnormality and CSF analysis revealed elevated protein at 154, total cell count of 3 indicating albumino-cytological dissociation supporting the diagnosis of GBS.
- Patient was started on IVIG and transferred to ICU for respiratory muscle weakness and ultimately intubated. During the ICU course, he experienced severe dysautonomia leading to labile blood pressures, severe bradycardia, asystole requiring vasopressor support and biventricular pacemaker placement prior to discharge.

Tables

- Table showing parameters of the patient during initial course

Parameters	Day-1	Day-2	Day-3
Respiratory rate	17	22	32
Saturation (O2)	94%	92%	86%
NIF (Negative inspiratory force)	>48	40	30

Discussion

- GBS classically starts within 4 weeks of preceding infection with symptoms peaking between 2-4 weeks with variable recovery period. The diagnosis mostly is clinical that can be supported by nerve conduction studies and lumbar puncture for CSF.
- **Common presentations:** Sensory symptoms, ascending weakness that progress to paraplegia/quadriplegia.
- **Unusual presentations:** like cranial neuropathy, autonomic disturbances, ophthalmoplegia.
- Autonomic disturbances like arrythmias and labile blood pressures are severe consequences which can present early in the disease process that warrant an ICU admission with continuous monitoring. These were seen only in 16.7% of COVID related GBS (1).
- As per literature, severity of COVID infection corelates with severity of GBS and could be an indicator for intensity of immune response (2). In contradiction, our patient had a milder COVID infection followed by severe GBS with complicated hospital stay.

Discussion

- When compared to non-COVID GBS there in not much variation in management of COVID related GBS, IVIG still remains the mainstay treatment (1). No role of second dose IVIG and it further exposes patient to more risks than benefits
- Monitoring these patients for disease progression is very important and studies have strongly recommended to use various parameters like respiratory rate, oxygen saturation, FVC, negative inspiratory force, and maximal expiratory pressure (3).
- These need to be monitored 2-4 hours initially and every 6-8 hours in stable patients. We as physicians should emphasize these parameters and take needed actions appropriately.

Conclusion

- In conclusion, there is no much variation between the classic post-viral GBS and Covid related-GBS in terms of clinical presentations, diagnostic approaches treatment modalities and prognosis.
- However, pathophysiology behind this neurologic sequela of COVID-19 infection is still unclear.

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A Pain In The Neck

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Introduction

- Lemierre's syndrome (also termed postanginal sepsis and necrobacillosis) is a rare condition that usually begins with tonsillopharyngitis and progresses to involve internal jugular vein thrombosis, adjacent soft tissue inflammation, persistent bacteremia, and even septic embolization.[2]
- Lemierre's syndrome most often occurs in healthy young adults, and some studies have shown that it might occur more in men. This condition remains relatively rare, and it should be noted that there are no standardized criteria to define Lemierre's syndrome.
- The most common pathogen isolated in this condition is the anaerobe *Fusobacterium necrophorum*, but other organisms including *Streptococcus pyogenes* have been reported.
- Interestingly, this patient reported an altercation with her boyfriend where he reportedly choked her one week prior to hospital presentation.

Case Description

- 43-year-old Caucasian female with a PMH of HTN and drug abuse presented with dyspnea on exertion, chills, sore throat, cough, increased fatigue, and right neck swelling x2 days.
- She was afebrile, hypertensive and mildly hypoxic on arrival.
- CTA head neck and chest showed right IJ thrombus.
- The patient was started on heparin drip, blood cultures drawn and empiric vancomycin and Unasyn were started.
- 2 days later blood cultures returned positive for GAS.
- She was given long-term antibiotics and followed up as outpatient.

Imaging

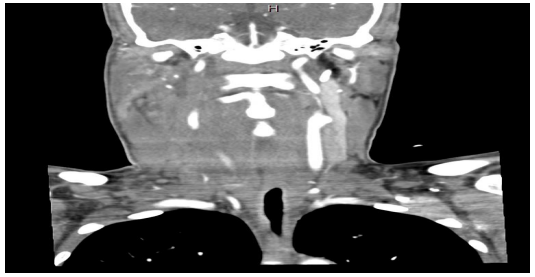

Figure 1: CTA neck, coronal, 2.0 mm showing right IJ thrombus (on 1/21/21).

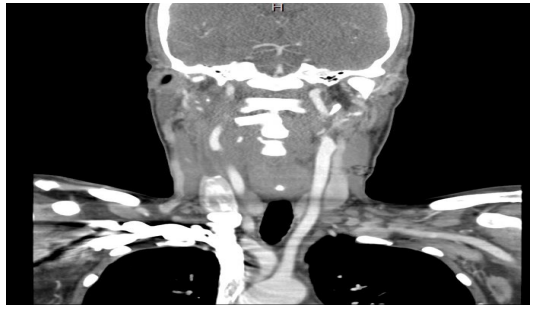

Figure 2: CT soft tissue neck with IV contrast, coronal, 2.5 mm thk showing right IJ thrombus (on 1/28/21).

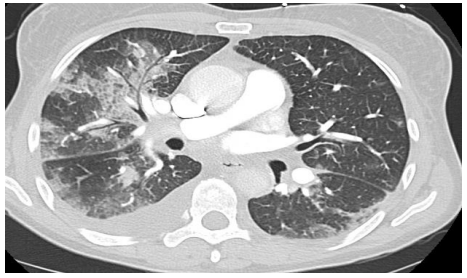

Figure 3: CT Angio Chest PE with IV contrast, axial, 2.5 mm thk showing bilateral multifocal infiltrates with more confluent infiltrate at the right lower lobe concerning for septic emboli.

Conclusion

- Though the differential diagnoses can be broad, physicians should consider this condition when patients present with fever, pharyngitis/sore throat, and unilateral neck pain.
- Currently there are no significant studies on the pathophysiology, management, and prognosis of Lemierre's syndrome.
- This case highlights the importance of recognizing the clinical manifestations of Lemierre's. We believe more case reports are needed to aid in the understanding of this syndrome.

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Reactive Arthritis: A Joint Effort by *Haemophilus* and Syphilis?

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Introduction

- Reactive arthritis, previously called Reiter syndrome, is an inflammatory arthritis that typically presents several days after a gastrointestinal or genitourinary infection
- It is grouped in the subclass of seronegative spondyloarthropathies that affect the axial skeleton
- It is believed to be an immune-mediated syndrome: T lymphocytes are induced by lipopolysaccharide and nucleic acids when the bacteria enter systemic circulation
- Common manifestations include the triad of arthritis, urethritis and, conjunctivitis
- Treatment with non-steroidal inflammatory drugs are the initial treatment of choice, in addition to anti-microbial therapy for the infectious trigger (1)
- Of note, reactive arthritis has been documented to be more common in HIV individuals (2,3)

Case Presentation

- A 37-year-old male with past medical history of HIV (CD4 464) presented to the ED with a 3-day history of severe right shoulder and right hip pain
- Four days prior to admission, he experienced fever to 103F and became progressively immobile due to 10/10 rated pain
- Preceding his admission, he was immobilized due to the pain in both his right shoulder and right hip
- On presentation, BP 139/93, Pulse 105, Temp 99.5, leukocyte count was 23,800, ESR 27
- Blood cultures were positive for *Haemophilus influenzae*
- Chest X-ray imaging with bibasilar pulmonary opacities
- MRI joint imaging of right shoulder (Fig. A) and hip (fig B) showed asymmetric joint effusions
- Patient was admitted for bacteremia with concerns of septic arthritis of right hip and right shoulder joint

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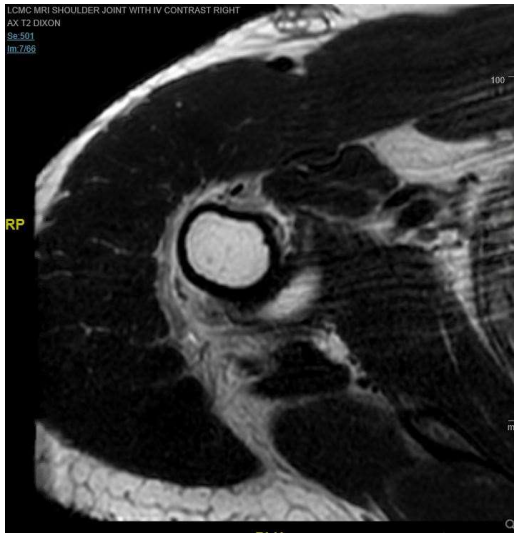
Images

A



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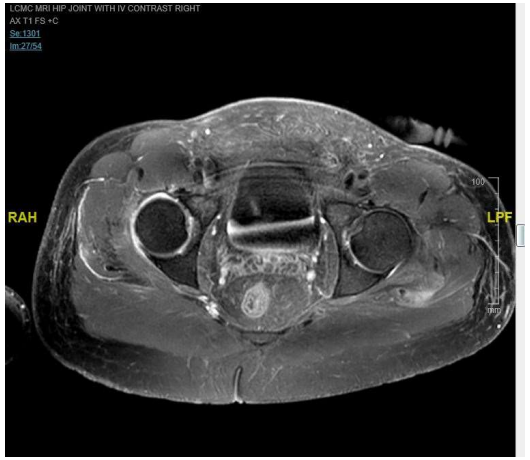

Hospital Course

- Patient's joint aspiration had no bacteria and <15,000 WBCs, making it not consistent with septic arthritis
- He spiked intermittent fevers and his joint pain remained severe despite antibiotic treatment for his *Haemophilus influenzae* bacteremia
- A broader work-up of infection sources was conducted given HIV status and lack of improvement over 4 days
- RPR titer was 1:64 - positive for secondary syphilis infection; his prior infection was in 2010 with documented treatment
- CSF VDRL and ophthalmological exam were negative
- Initiation of NSAIDs significantly improved joint inflammation and enhanced mobility
- Given confirmed secondary syphilis, he was treated with Penicillin G 2.4M units IM for 3 doses
- On day of discharge, he was ambulating and achieved functional use of his previously immobile right arm

Discussion

- Given the severe joint pain with limited range of motion, fever, and bacteremia, septic arthritis was initially at the top of the differential diagnosis
- However, the joint aspiration results were more consistent with the diagnosis of reactive arthritis
- The patient lacked the typical associated findings of reactive arthritis: enthesitis, urethritis, or uveitis
- Reactive arthritis is supported by the significant improvement of his joint pain with NSAIDs late in the hospital course
- Secondary syphilis is typically associated with tenosynovitis, but there are some case reports of it causing a reactive arthritis syndrome, often with associated dermatologic manifestations that resemble classic autoimmune rheumatologic disease (4,5,6)
- *Haemophilus influenzae* is not often associated with reactive arthritis, but data support its association with HLA-B27 independent reactive arthritis (7,8)
- It is unclear whether *Haemophilus influenzae* or secondary syphilis was the primary etiology of the reactive arthritis, and the possibility remains that they contributed in a joint effort

Hurricane Hypersensitivity Pneumonitis

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Introduction

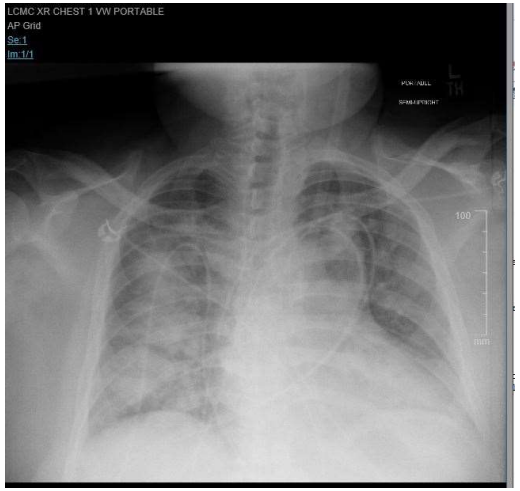
- Hypersensitivity pneumonitis is a condition that is often acquired with through occupational exposure
- The acute form is typically acquired via heavy exposure to a specific antigen and is associated with dyspnea, malaise, cough, and fever
- Chronic exposure can progress to pulmonary fibrosis, however early diagnosis and allergen avoidance typically result in full recovery to baseline
- The lung inflammation is due to a mix of type III and type IV hypersensitivity reactions
- The common treatments are avoidance of the antigen and systemic glucocorticoid administration (1,2)

Case Presentation

- A 57-year-old male with past medical history of heart failure with preserved ejection fraction, chronic kidney disease stage III, obstructive sleep apnea, obesity, and hypertension who presented with shortness of breath
- His home was damaged by Hurricane Ida 1 month prior to admission, and he was performing renovation on the building
- His renovations included sawing through wall and insulation; he did not use a particulate mask during the renovation
- He denied any recent illnesses and was not COVID +
- In the ED, the patient was saturating 45% on room air and was placed on a non-rebreather mask
- In the ED, he was afebrile and his blood pressure was 221/144 with a pulse of 107
- ABG in ED was pH 7.28, PaCO2 56, PaO2 58, HCO3- 26.31 significant for hypoxic hypercapnic respiratory failure
- The patient was admitted to the ICU for hypoxic hypercapnic respiratory failure presumed due to pulmonary edema 2/2 underlying heart failure

Images

A



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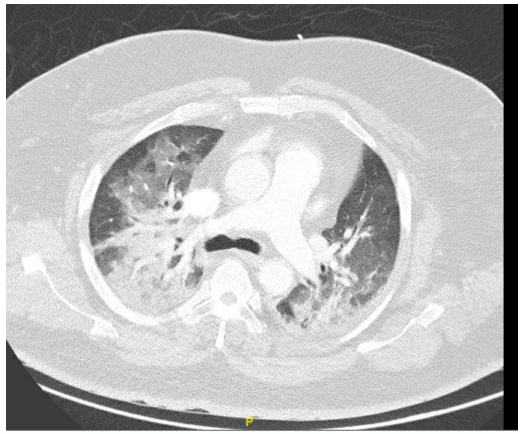

Hospital Course

- Patient was aggressively diuresed due to presumed pulmonary edema 2/2 heart failure being the driver of his hypoxic respiratory failure
- He failed to improve with continued diuresis, and required intubation in the ICU
- Chest X-ray at the time showed diffuse, patchy interstitial opacities without effusion (fig A)
- CT scan was significant for multifocal airspace consolidation with ground-glass opacification (fig B)
- Steroids were initiated, which resulted in rapid improvement of hypoxia then extubation after 12 hours
- Within 2 days after the initiation of steroids, he was saturating 96% on 3L NC
- At discharge he was on room air and was tapered on steroids for a 4-week total taper

Discussion

- The patient's rapid improvement with steroids makes the diagnosis consistent with hypersensitivity pneumonitis given his recent exposure to inhaled dust from the home renovation
- Bronchiolar lavage was not performed due to rapid improvement, but it would have suggested the diagnosis if it were to show lymphocytosis with low CD4:CD8 ratio
- Wood dust pneumonitis is the most likely contributor, caused from dust from oak, cedar, or pine woods
- Organic toxic dust syndrome is also a possible contributor since the hurricane damaged walls were damp and likely moldy from the water exposure (3,4)
- This case is significant for hurricane region of the United States as citizens performing home renovations may not have adequate respirators and will be exposed to mold and wood dust
- This case also is significant for the importance of broadening a differential and not anchoring on a certain diagnosis if the patient is not improving on a treatment, such as this patient's diuresis

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An Unusual Presentation of IgA Vasculitis with Cardiac Involvement

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INTRODUCTION

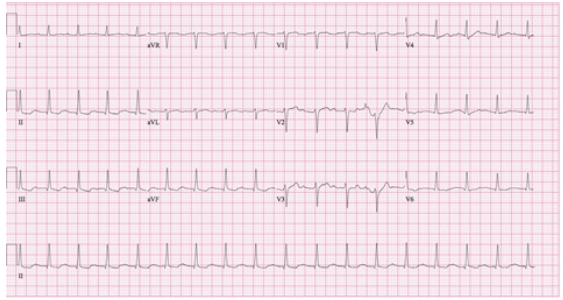
IgA vasculitis is a systemic inflammatory disease that affects the small vessels. This case report describes IgA vasculitis in an adult with cardiac involvement and a modified approach to therapy.

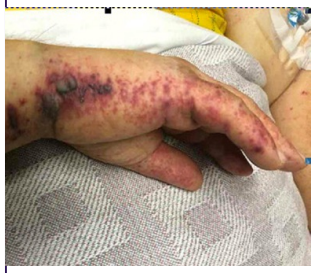
CASE PRESENTATION

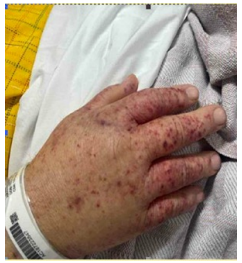
A 68-year-old male with a history of diabetes and paraplegia with chronic foley presented as a transfer for evaluation due to duodenal thickening on CT scan. The patient was experiencing fevers, abdominal pain, nausea, hand swelling, and rash to bilateral arms and legs. During the hospitalization, the patient developed hematuria, palpable purpura, worsening leukocytosis, and acute kidney injury. EGD showed ulcers in the duodenum. Over the course of a week, the hemoglobin decreased from 13 to 8. A repeat EGD and a colonoscopy did not show any bleeding, but biopsies showed transmural hemorrhagic and edematous inflammation. Further work up showed elevated serum IgA and IgE levels. A skin biopsy with a direct immunofluorescent exam revealed IgA and fibrinogen in the small arterioles and arteries of the superficial dermis. The patient was diagnosed with IgA vasculitis and started on prednisone.

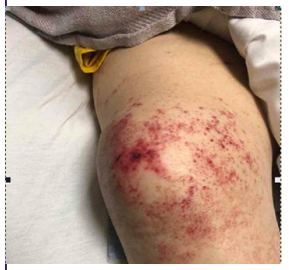
The patient developed chest pain later during the hospitalization. His EKG showed sinus tachycardia with first-degree AV block and slight ST depressions in leads II, III, aVF, V5-V6. His troponin peaked from 0.3 to 4.5. Cardiology recommended a modified acute coronary syndrome therapy with aspirin and low dose heparin for 48 hours because of the patient's recent gastrointestinal bleed. As the patient was not a good candidate for dual antiplatelet therapy, plavix and heart catheterization were deferred. An echocardiogram showed an ejection fraction of 60% with no abnormalities. The patient's symptoms eventually resolved and his prednisone was tapered. Of note, the patient was readmitted for a NSTEMI 6 months later, with cardiac catheterization showing multivessel CAD.

EKG/IMAGES









DISCUSSION

This case is notable for cardiac involvement in adult IgA vasculitis. A review of treatment options shows the benefit of high dose steroids, but no specific guidelines exist. In adult patients, differentiation of the causes of NSTEMI may be more complex, especially in those with multiple comorbidities. In conclusion, there needs to be further exploration of proper management of NSTEMI in the setting of vasculitis.

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ACKNOWLEDGEMENTS

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KSHV Inflammatory Cytokine Syndrome (KICS)

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Introduction

Kaposi Sarcoma-associated Herpesvirus (KSHV) or Human Herpesvirus-8 (HHV-8) is a DNA virus that is most associated with the vascular malignancy, Kaposi Sarcoma

KSHV can also cause :

- Primary Effusion Lymphoma (PEL)
- Multicentric Castleman Disease (MCD)
- KHSV Inflammatory Cytokine Syndrome (KICS)

Case Presentation

33 y/o male with newly diagnosed HIV, (CD4 332 cells/mm3), ocular syphilis, and Kaposi Sarcoma (KS).

He was discharged a few days prior after a 12-day hospital course associated with ocular swelling, tearing, and redness -> newly diagnosed ocular syphilis and HIV.

- He was also treated for sepsis pneumonia, AKI, thrombocytopenia, and diagnosed with KS.
  - discharged with ART and dexamethasone

The patient presents 3 days later with:

- progressive dyspnea
- productive cough
- pleuritic chest pain

Physical exam was notable for coarse lung sounds, KS lesions, and diffuse lymphadenopathy.

Admitted to the MICU for respiratory distress requiring NIPPV

Diagnosis

- The diagnostic criteria shown below is adapted from *Polizzotto et al*
- Little available data therefore based on expert opinion
  - To diagnose KICS, patient must exhibit:
    - 3 findings featured in figure 1a +
    - At least 2 criteria in each category (A-C) depicted in figure 1b.

Findings specific to our patient are noted in the arrows in figure 1a and are bolded in figure 1b.

Figure 1a

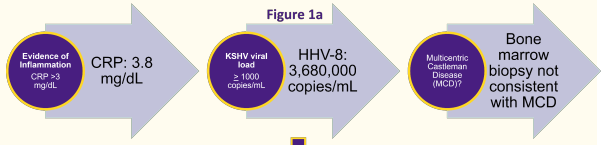
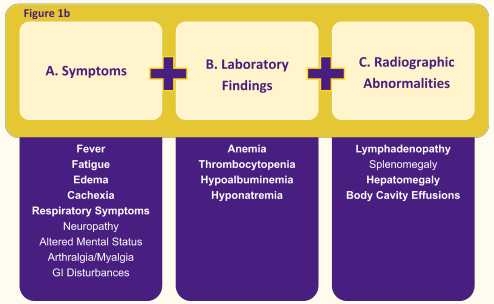


Figure 1b



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Hospital Course

A few hours after admission, he quickly decompensated and was intubated. Broad empiric antibiotics were initiated.

- The next day, his abdomen became distended in the setting of oliguria.
  - A chest tube was placed after imaging showed abdominal and pleural effusions.
  - During this time, the patient became hypotensive requiring three pressors with no improvement.
  - Empiric antifungal coverage was added.
  - He remained anuric, so CRRT was initiated.
- Day 5 of admission, in response to the patient's progressing multiorgan failure, his family chose to pursue comfort care. He was palliatively extubated and quickly deteriorated into asystole. His family declined autopsy.

After his death, his infectious workup was confirmed to be negative.

- A cytokine panel was remarkable for elevated IL-2, IL-2R, interferon gamma, IL-10, IL-13, and IL-6 suggestive of KICS as the etiology of the patient's presentation.

Although, KICS was suspected early, the patient was too unstable for chemotherapy to be initiated.

Discussion

KSHV Inflammatory Cytokine Syndrome (KICS) is a newly-described complication of HHV-8 infections/Kaposi Sarcoma that is poorly described due to its 60% mortality rate.

- Presents in AIDS patients with a low CD4 count even if the patient is being treated with ART.
- Clinical presentation often resembles sepsis, but these patients will not respond to antibiotic therapy.

Better outcomes reported in cases with early diagnosis and subsequent treatment with chemotherapy.

CMV-associated Splenic Infarction in an Immunocompetent Patient

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Introduction

Splenic infarctions can occur from either arterial or venous occlusion.

- The most frequent causes are thromboembolic and hematologic diseases.
  - Other causes are trauma, infection, abdominal pathology, malignancy, atherosclerosis of the celiac or splenic arteries

Common presentations include splenomegaly, leukocytosis, nausea, vomiting, and left-sided pain. Some patients can also be asymptomatic

- CT is the preferred imaging modality to visualize an acute splenic infarction.
  - Ultrasound can also be used to identify an infarct

- Treatment is based on etiology and ranges from supportive care to splenectomy

Case Presentation

A 31-year-old woman with a past medical history of endometriosis and anxiety presented with left flank pain, chills, malaise, polydipsia, nausea, and vomiting for 3 weeks.

Prior to presentation, she experienced transient sore throat and nasal congestion. No known sick contacts, recent travel or prolonged immobility

The patient had visited the emergency department on multiple occasions in the past year for abdominal pain. *Past imaging was unremarkable.*

- Most recently, she sought care at an urgent care where she was treated for pyelonephritis with antibiotics and a muscle relaxant for the pain

- Left upper quadrant tenderness on physical exam.
- CT abdomen on admission demonstrated multiple new splenic infarcts. See figures A and B.
- Lower extremity ultrasound showed no evidence of DVT.

Because the etiology of the splenic infarcts was unclear, patient was admitted for further evaluation.

Images

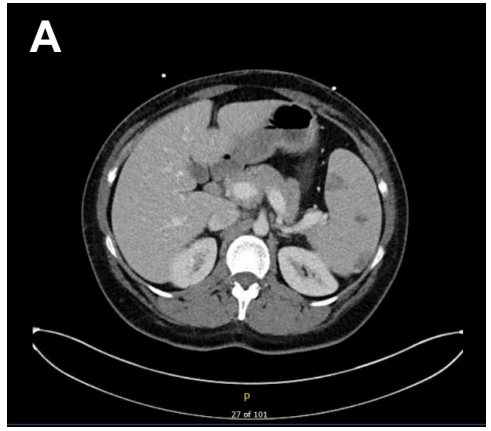

Figure A: CT Abdomen on admission showing multiple new splenic infarcts

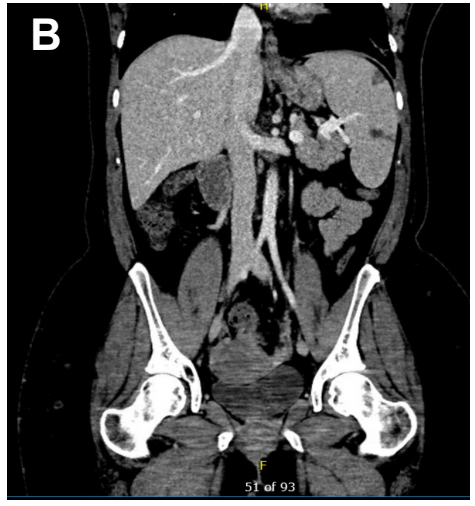

Figure B: Coronal view

Hospital Course

Upon admission, patient was evaluated extensively for potential causes of the splenic infarct

- Negative autoimmune workup
- Negative HIV, RPR and hepatitis panel
- Biofire positive for rhinovirus/enterovirus. COVID-19 negative, EBV IgM levels wnl
- CMV IgG and IgM elevated. CMV DNA viral load 1020 IU/mL

Other labs and physical exam findings were unremarkable.

Supportive care during admission with pain and nausea control. IV fluids were administered while patient was unable to tolerate oral intake.

- Empiric antibiotics were initiated and later discontinued after cultures were negative
- The patient was discharged with Zofran a few days later once her symptoms improved

Discussion

Cytomegalovirus (CMV) is a ubiquitous and highly prevalent human herpes virus.

- Common presentations include fever, sore throat, muscle pain, and fatigue.
  - Most immunocompetent adults will not have any symptoms

CMV-associated thrombosis and splenic infarction are uncommon in immunocompetent patients but have been reported extensively in immunocompromised patients.

- Anticoagulation therapy is not indicated for immunocompetent patients unless there is another hypercoagulable condition.
- Antiviral treatment is recommended in cases with severe multi-organ involvement.

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Systemic lupus erythematous as a mimicker of lymphoproliferative disease

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Introduction

- Systemic lupus erythematous (SLE) is a known mimicker of many other conditions because of its diverse multi-system presentation.
- When evaluating for a lymphoproliferative disease, consider SLE as a coexisting or alternative diagnosis.

Case Presentation

- A 42-year Latin American female with no known past medical or family history presented to the hospital with three days of chest pressure, shortness of breath and fever, and five days of throat pain.
- The patient also reported weakness, arthralgias, myalgias, fatigue, night sweats and a 20lb weight loss over the prior two months.
- Infectious workup was negative; however, patient was noted to have pancytopenia with a WBC count of 2.0 K/uL, HgB of 7.6 gm/dL and platelets of 120 k/uL.
- Imaging showed lymph nodes in both sides of neck, supraclavicular region, axilla, mediastinum, and scattered enlarged lymph nodes in periportal, perisplenic area, retroperitoneum, pelvis, and groin.
- The patient had an elevated serum creatinine of 1.27 mg/dL with nephrotic range proteinuria and mild elevation in transaminases.
- Other relevant labs included an elevated ESR and a positive EBV IgG.

Images

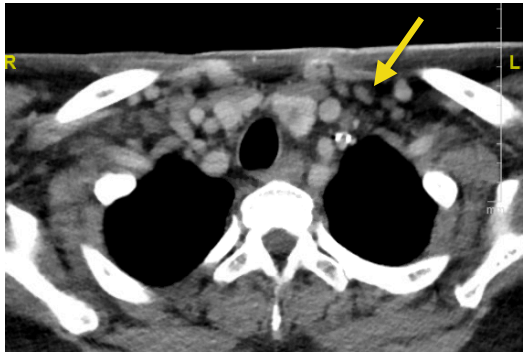

Image 1:
CT Chest showing lymphadenopathy

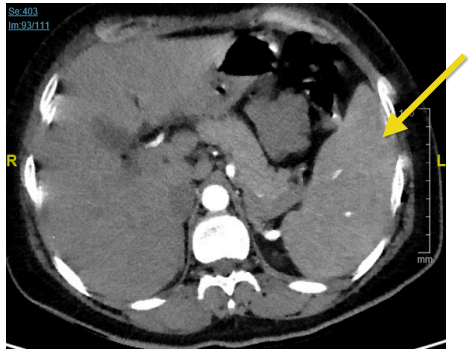

Image 2:
CT Abdomen and Pelvis showing splenomegaly

Anti-Ribosomal P	<1.0 AI	1.4 ^	Anti-SSB	<1.0 AI	2.1 ^
Anti-SmRNP	<1.0 AI	>8.0 ^	Anti-SSA	<1.0 AI	>8.0 ^
Anti-SM	<1.0 AI	>8.0 ^	Anti-Chromatin	<1.0 AI	>8.0 ^
Anti-ds DNA	<=4.0 IU/mL	>300.0 ^			

Hospital Course and Follow Up

- Oncology was consulted due to concern for lymphoma and the patient underwent excisional lymph node biopsy and bone marrow biopsy.
- Lymph node biopsy revealed reactive lymphoid hyperplasia, but was negative for malignancy, as was the bone marrow biopsy.
- A concurrent autoimmune workup eventually revealed a positive ANA and low complement levels.
- Further autoimmune workup was positive for anti-ds DNA, as well as anti-ssa, anti-ssb, anti-sm, anti-smrnp, anti-chromatin, anti-ribosomal P, anti-RNP.
- Patient was seen by rheumatology who diagnosed her with systemic lupus erythematosus.
- Nephrology was consulted for management of lupus nephritis, with renal biopsy showing lupus nephritis class IV.
- The patient's symptoms improved with prednisone and she was eventually put on hydroxychloroquine and mycophenolate for management of lupus and lupus nephritis.
- Follow-up labs revealed significant improvement in her anemia and renal function.

Discussion

- This case highlights the importance of keeping a broad differential when approaching a patient to ensure a prompt and accurate diagnosis.
- Because this patient lacked the cutaneous manifestations commonly seen with SLE, her B symptoms, pancytopenia, and diffuse lymphadenopathy suggested a diagnosis of lymphoma.
- During her workup it became apparent that her fever, lymphadenopathy, anemia, and kidney injury were also common presenting sings of SLE and warranted testing autoimmune antibodies[1,2].

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A Rare Etiology of Elevated Anion Gap Metabolic Acidosis: Methanol Toxicity

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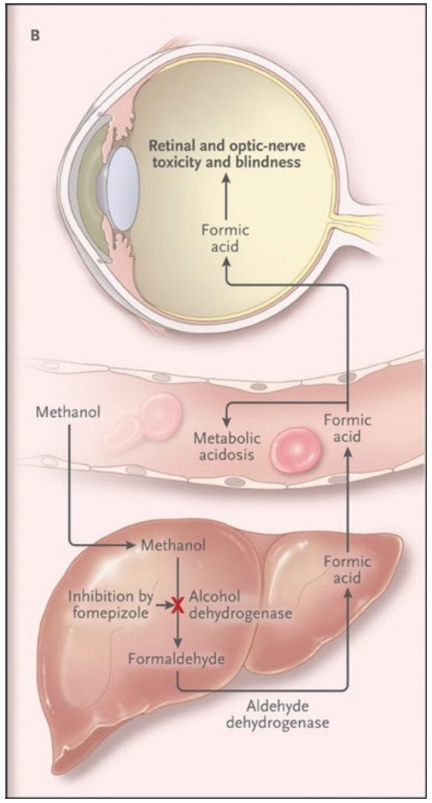
Introduction

- When approaching elevated anion gap metabolic acidosis (AGMA), many etiologies are commonly taught but not frequently seen in practice. The acronym "MUDPILES" is commonly learned by medical students as a memory tool for recalling the common causes of AGMA: methanol, uremia, diabetic ketoacidosis, paraldehyde, isoniazid, iron, lactic acidosis, Ethanol/Ethylene Glycol, Sailcylates.
- We present an unintentional methanol ingestion as the underlying etiology of elevated AGMA.

Case Presentation

- 45-year-old male with past medical history of CAD and polysubstance use disorder presented with chest pain and worsening blurry vision after consuming 10 24-oz beers and 1.5 fifths of liquor over 1 day. His chest pain was sharp and substernal without radiation. He denied any other substance ingestion. Physical exam was significant for tachycardia, tachypnea, left conjunctival hemorrhage and mid-sternal tenderness to palpation. Initial lab values showed thrombocytopenia, hyponatremia, hypochloremia, and acidosis with an elevated anion gap. Toxicology screen showed an elevated methanol level (33; normal <4mg/dL) and normal ethanol level. Serum osmolality was elevated with an elevated serum osmolar gap. Electrocardiogram showed normal sinus rhythm. Head imaging was negative for acute abnormality. Toxicology, ophthalmology, and nephrology were consulted and performed urgent dialysis with administration of fomepizole and leucovorin. Patient reported vision improvement after treatment.



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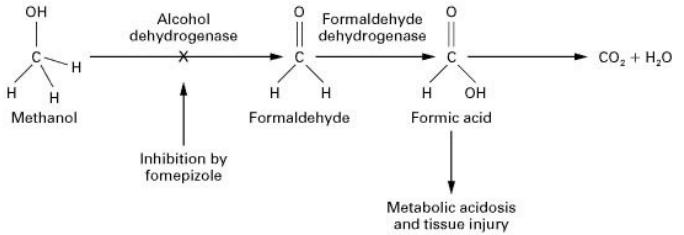
Discussion

- Methanol toxicity is a rare cause of elevated AGMA and can present a diagnostic challenge.
- Methanol toxicity can mimic ethanol intoxication and may progress to end-organ damage (renal failure, vision changes) if not promptly recognized.
- Diagnosis relies upon serum osmolality, blood gas and excluding other etiologies of elevated AGMA
- Treatment inhibits breakdown of methanol by alcohol dehydrogenase with fomepizole and Leucovorin and may include hemodialysis in severe toxicity defined as metabolic acidosis, methanol levels greater than 50 mg/dL or evidence of end-organ damage.



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Maternal Outcomes in Subsequent Pregnancies after Peripartum Cardiomyopathy

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LSU Health SHREVEPORT

Introduction

- Peripartum cardiomyopathy (PPCM) is a rare cardiomyopathy associated with pregnancy and has a high maternal morbidity and mortality during both index and subsequent pregnancies
- Data to help guide women with subsequent pregnancies is sparse
- The purpose of this large retrospective study is to perform a comparative analysis of maternal outcomes in women with recovered left ventricular ejection fraction (LVEF) and persistent left ventricular (LV) dysfunction during subsequent pregnancies after PPCM

Methods

- We identified 46 patients who had a subsequent pregnancy and an echocardiogram prior to their subsequent pregnancy in our registry of 121 patients with PPCM
- Data in the registry was gathered by retrospective chart review
- LVEF recovery was defined as improvement to >50%
- We divided patients into a recovered group (RG) (n=16) and non-recovered group (NRG) (n=30)

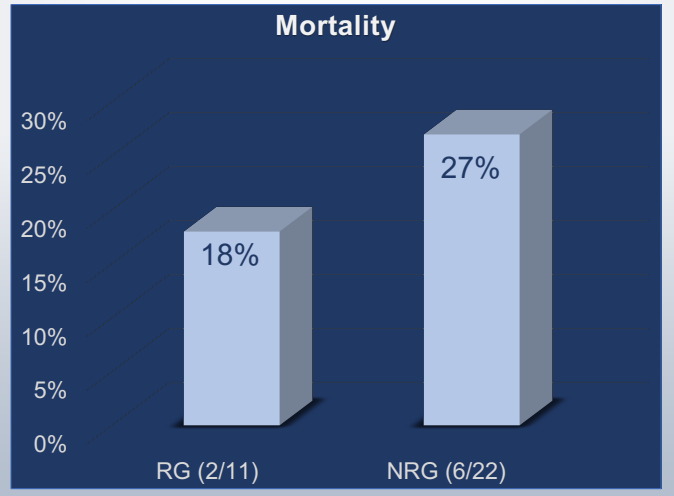
Results

- Total maternal mortality among women with PPCM and subsequent pregnancy was 24%
- Mortality in the RG was 18% compared to 27% in the NRG
- Mortality among African Americans (AA) was higher when compared to other ethnic groups

Discussion

- Maternal mortality related to PPCM in the US ranges from 7% to 20%
- In our study, we found that the risk of maternal mortality among women with PPCM who had one or more subsequent pregnancies was 24%
- It is well known that women with non-recovered LV function before subsequent pregnancy carries a worse maternal outcome when compared to women who enter the subsequent pregnancy with recovered LV function
- Our findings reaffirm this, as we found that mortality rate in the NRG was higher when compared to the RG
- Our findings also align with previous studies in that, AA women have a higher mortality rate when compared to other ethnic groups

Figure 1



Conclusions

- Recovery of LVEF predicts a better prognosis but is not an absolute protection from the risk of mortality and/or recurrent PPCM
- Hence, close monitoring of LVEF with serial echocardiograms is still indicated during a subsequent pregnancy for women with PPCM
- African ethnicity carries a worse maternal prognosis in women with PPCM

A Case of Nontyphoidal Salmonella Bacteremia, Disseminated Histoplasmosis, and Disseminated MAC in an Immunosuppressed Patient

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Case Presentation

- 51-year-old man with HIV/AIDS (CD4 count 15), HCV without cirrhosis, nasopharyngeal DLBCL (in remission), cryptococcal meningitis (s/p induction therapy), presented for fever associated with dyspnea, productive cough, and pleuritic chest pain for three days superimposed upon a one-week history of fatigue and generalized body aches
- The patient had undergone induction therapy for cryptococcal meningitis approximately four months previously and had been on maintenance therapy since then with oral fluconazole. He had recently been restarted on his antiretroviral (bictegravir/emtricitabine/tenofovir alafenamide) approximately two weeks prior to this hospital presentation; he had also been adherent with his PJP prophylaxis (atovaquone, which was chosen for his G6PD deficiency)
- Initial vitals: 104.8°F (40.4°C), HR 135, BP 118/69, RR 32, SpO2 100% room air, Ht 185.4cm (6'1"), Wt 56kg (124lb), BMI 16.3kg/m^2
- Exam: Cachectic advanced-aged Black man, chronically ill-appearing, with bitemporal wasting. Poor dentition; clear oropharynx. Regular rate and rhythm; no murmurs. Tachypneic with mildly diminished breath sounds bilaterally with mild coarseness; no rales or wheezes. Benign abdomen. Skin survey non-contributory. Neurologically intact. No leg edema.
- A broad sepsis evaluation, broad-spectrum antibiotics, and IV fluid resuscitation were initiated. The patient was admitted to Internal Medicine. Pulmonary and Infectious Disease were consulted early into his hospital admission for assistance.
- His presumed source was initially believed to be a primarily right-sided pneumonia that was complicated by a right paratracheal mediastinal abscess vs. necrotic lymphadenopathy (see Figures 1 & 2).
- Within 12 hours of his initial lab work, his blood cultures grew Gram negative rods (4/4 bottles). Surprisingly, he was found to have a pan-sensitive *Salmonella enterica* bacteremia with no evidence for recent or current gastrointestinal symptoms. Further evaluation found no evidence for associated endocarditis.
- As pulmonary disease due to enteric nontyphoidal Salmonella is rare and uncommon, there became concern for another underlying etiology of the patient's lung findings. Considering his persistent leukopenia and a positive Histoplasma antigen test, the patient underwent bronchoscopy and a bone marrow biopsy. His bronchoscopy biopsy results ultimately demonstrated histoplasmosis, and he was initiated on induction therapy with liposomal amphotericin B.
- He was also initiated on clarithromycin and ethambutol after AFB blood cultures grew *Mycobacterium avium* complex (MAC).
- His final outpatient regimen included the following: antiretroviral treatment with bictegravir/emtricitabine/tenofovir alafenamide, prophylaxis with atovaquone, oral levofloxacin for Salmonella bacteremia, oral voriconazole for dual coverage of disseminated Histoplasmosis and cryptococcal meningitis, and clarithromycin with ethambutol for disseminated MAC.

Discussion

- Invasive nontyphoidal Salmonella is not common in the continental USA. Although only approximately 1% of cases of enteric infections due to nontyphoidal Salmonella results in concurrent bacteremia, primary bacteremia is even less common and is of concern for underlying immunologic dysfunction.[1,2]
- Histoplasmosis is a mycotic infection endemic to North and Central America; within the continental USA, cases are often seen in midwestern and central states along the Ohio and Mississippi River Valleys. In patients who manifest symptoms, histoplasmosis often presents with primary pulmonary involvement and symptomatology. Dissemination of this mycotic infection beyond the lungs can be seen in certain patient populations such as those with immunosuppression.[3,4]
- The diagnosis of common and uncommon diseases in the immunosuppressed HIV patient can be difficult to establish. Often, diagnostic reasoning tools such as Occam's razor do not apply as in this case. For the immunosuppressed HIV patient, a low threshold to start a broad and thorough evaluation is necessary for the correct diagnosis of disease(s).

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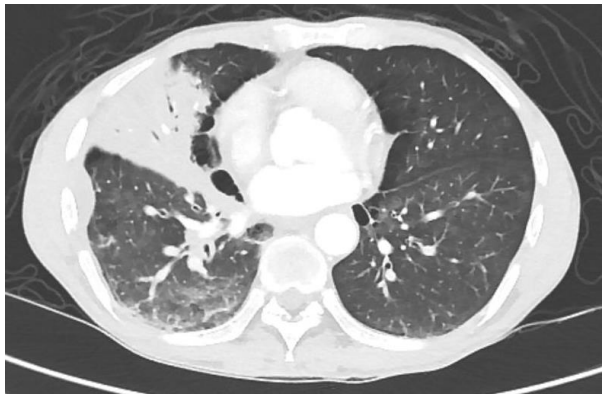

Figure 1. CT chest with contrast (transverse view) with right middle and lower lobe consolidation with air bronchograms, multifocal cystic changes, and thickening of the right-sided fissure.

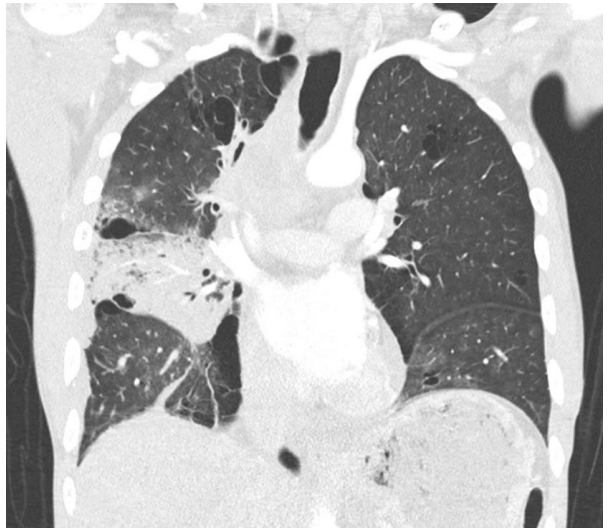

Figure 2. CT chest with contrast (coronal view) with right middle and lower lobe consolidation. Right paratracheal mediastinal peripherally enhancing fluid collection (2.2x1.3x2.5cm) with foci of gas concerning for empyema with mediastinal abscess.

Diaphragmatic Shunt Associated with Peritoneal Dialysis

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Introduction

- Pleuroperitoneal shunts are pathologic connections between the pleural and peritoneal spaces and are an uncommon complication seen in PD: incidence rate of 1.6-10% [1, 2]
- Unclear pathology, but potentially due to congenital or acquired diaphragmatic defects [3,4]
- More common among female PD patients and in patients with PCKD [5]

Case Presentation

- A 55-year-old woman with a past medical history of HTN, DM-II, CAD with CABG, ESRD secondary to PCKD on PD presented with 4 days of worsening shortness of breath and dyspnea after minimal exertion
- On arrival, CXR revealed a large right-sided pleural effusion
- Thoracentesis was performed, which revealed a transudate with a glucose count of 593mg/dL concerning for peritoneal fluid. The remainder of the fluid studies were within normal limits, and culture of the fluid was negative.
- CT scan of the chest without contrast demonstrated a large right-sided pleural effusion occupying half the volume of the right hemithorax (see Figure 1.)
- Cardiothoracic Surgery service was consulted for evaluation for a diaphragmatic defect. They performed video-assisted thoracoscopic surgery (VATS) with talc pleurodesis to prevent peritoneal fluid from entering the thoracic cavity again. Per their operative evaluation, adhesions were noted between the right lower lobe and the diaphragm, but no other obvious pathology was noted on inspection of the entire diaphragm
- The patient was transitioned from PD to HD during her hospitalization and recommended to stay on HD for at least 8 weeks following her VATS while her diaphragm healed. She was discharged on HD with plans to resume PD at 8 weeks post-operatively.

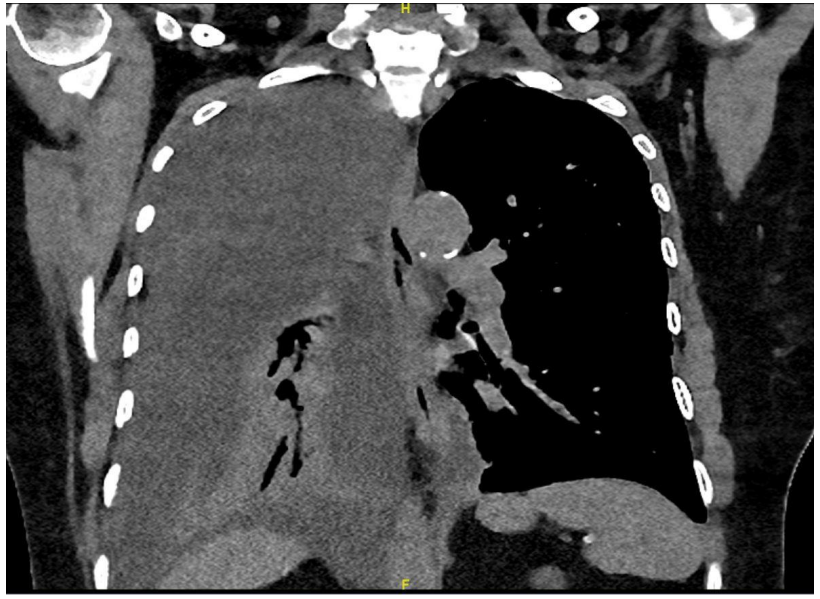

Figure 1. Computed tomography imaging of the chest without contrast showing a right pleural effusion occupying at least one half of the volume of the right hemithorax with complete atelectasis of the right lower lobe with air bronchograms.

Discussion

- This patient had several factors that predisposed her to this rare and uncommon PD complication: female sex and history of PCKD
- However, the development of PD-associated hydrothorax typically occurs more acutely (within the first month of initiation) whereas our patient had been on PD for approximately 18-20 months. This is suggestive of an acquired diaphragmatic defect.[5]
- No apparent diaphragmatic defect was found during her VATS procedure. However, in PD patients with transudative pleural effusions, a pleural fluid-to-serum glucose ratio >1 is consistent with a pleuroperitoneal shunt.[1] Our patient's transudative pleural effusion had a calculated pleural fluid-to-serum glucose ratio of 1.27
- Our patient's hydrothorax was also right-sided, which is typical of how PD-associated pleuroperitoneal shunts present.[3]
- Management of PD-associated hydrothorax can proceed conservatively (withholding PD to allow spontaneous resolution of the hydrothorax and diaphragmatic shunt) vs. surgically (e.g., chemical pleurodesis). Successful resumption of PD after pleurodesis is seen in approximately 50% of patients.[6]
- It is unclear if our patient would have had recurrence of her hydrothorax. Due to personal preferences, this patient chose to stay on HD after her VATS/pleurodesis rather than re-try PD.

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A Presentation of Profound (8200mL) Urinary Retention

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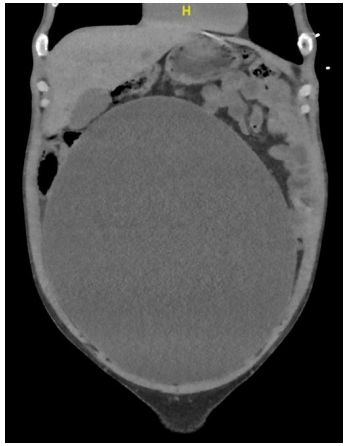
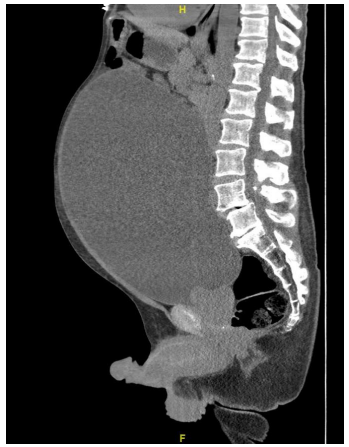
Introduction

- Acute urinary retention (AUR) often presents in men as the inability to urinate coupled with acute lower abdominal/suprapubic pain.[1]
- AUR in men is most commonly due to outflow obstruction secondary to BPH, but other etiologies including neurologic dysfunction, medication adverse effects, and infection can also be attributed to this.[1]
- In the case of chronic urinary retention (CUR), however, patients often present without any pain symptoms.[1]

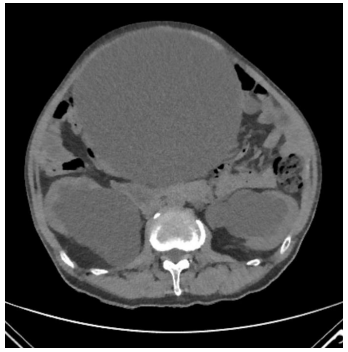
Case Presentation

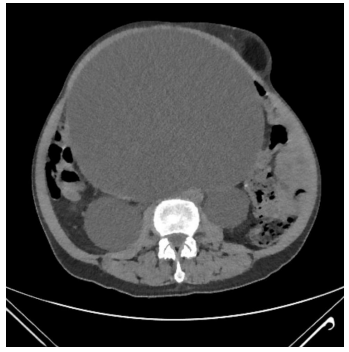
- 60-year-old man with HTN presented for 1.5 months of nausea, vomiting, and fatigue associated with PO intolerance and a 10-lb weight loss over that time period
- He had no abdominal pain or urinary complaints including no decreased urination or reported incontinence
- His exam was concerning for a firm, non-tender, and grossly distended abdomen with protuberance that stretched from his xiphoid process to his pubic symphysis
- Initial bloodwork was notable for elevated renal indices (BUN 136mg/dL and creatinine 10.3mg/dL); his urine studies did not show evidence for infection
- CT imaging of his abdomen/pelvis was concerning for a large intraabdominal mass (approximately 31cm in diameter) with bilateral hydronephrosis and hydroureter
- A foley was placed, and 1L of urine was initially drained before clamping. Urology cleared the patient for complete bladder emptying: 8.2L of urine was drained in total
- The patient was admitted to Medicine with both Urology and Nephrology following. He was discharged following resolution of his post-obstructive diuresis and with improvement in his post-renal AKI from his obstructive uropathy. A foley was left in place upon discharge for close outpatient follow-up with Urology.





Figures 1 & 2. CT imaging of the abdomen and pelvis without contrast. 1) Coronal view of massive urinary bladder distention. 2) Sagittal view of massive urinary bladder distention secondary to prostatomegly.





Figures 3 & 4. CT imaging of the abdomen and pelvis without contrast. 3 & 4) Transverse views of massive urinary bladder distention with severe hydroureteronephrosis.

Discussion

- Due to its gradual nature, chronic urinary retention can present without pain symptoms and without significant urinary complaints. Most patients will present with urinary retention on the scale of 1-1.5L.[1] In our case, our patient had gross abdominal distention and was found to be retaining 8.2L of urine, which appears to be the largest volume of retained urine documented at this time.[2]
- Hematuria, circulatory collapse, and post-obstructive diuresis are some common complications that can arise after bladder decompression in patients with large-volume urinary retention. Post-obstructive diuresis can occur in up to 50% of patients with urinary retention but is typically not of concern unless the patient is retaining >1.5L.[1,3]
- Urologic literature varies between the risks and benefits of rapid decompression vs gradual decompression of the bladder. Rapid emptying is generally considered safe and less time-consuming. Recent studies have also supported that gradual decompression does not reduce the knowns risks of emptying the bladder.[4]
- Our patient underwent rapid emptying after consultation with Urology. While he did develop post-obstructive diuresis, he did not experience circulatory collapse. He was ultimately discharged home with a foley in place after a 1-week hospitalization.
- Depending on your patient's clinical presentation, it would be appropriate to consider urologic consultation before proceeding with bladder decompression; however, rapid complete bladder emptying (versus gradual decompression with periodic clamping) is considered a safe and effective way to decompress the bladder.[4]

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Herpetic Colitis Causing Lower GI Bleeding in a Patient Undergoing Chemotherapy

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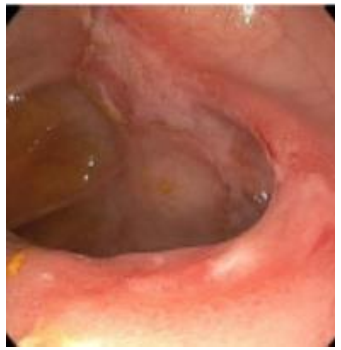
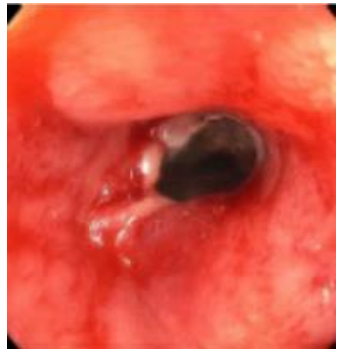
Introduction

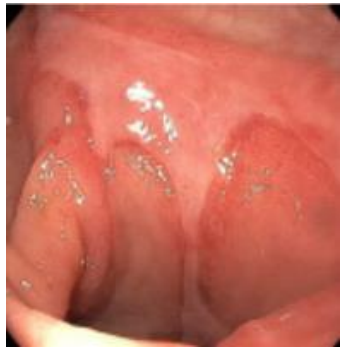
- Herpes simplex virus is a ubiquitous double-stranded DNA virus that is capable of causing a multitude of pathological manifestations.
- It is estimated that in 2016, 13.2% of the world's population aged 15-49 years were living with HSV-2, and that 66.6% of the world's population aged 0-49 were living with HSV-1.[4]
- The clinical manifestation is broad, with documented cases of oral, genital, ocular, neurological, and gastrointestinal presentations.
- The most common mode of transmission is via mucocutaneous secretions or exposure.[3] Once introduced to a mucosal surface or cutaneous surface via abrasion, the virus will initially replicate in cells in the dermis and epidermis. Latency is achieved once sufficient replication occurs and results in the virus traveling retrogradely via neurons into the ganglia.[1,2] Reactivation of the virus erupts in the setting of various stimuli, including co-infection with another pathogen, stress, and immunosuppression.
- Herpetic colitis is a rare manifestation of the herpes simplex virus that has been mostly documented in patients suffering from inflammatory bowel disease who are being treated with immunosuppressive therapy.[5,6]
- Use of medications, such as steroids, cyclosporine, azathioprine, and more can cause an exogenous immunosuppression that leads to reactivation.

Case Description

A 66-year-old male with a past medical history of colorectal cancer treated with resection and chemotherapy presents with acute encephalopathy. On admission, the patient was febrile and tachypneic. A urine culture grew Candida albicans and blood cultures were positive for Staphylococcus epidermidis. The patient was treated broadly with antimicrobials and had his port-a-cath replaced. During the hospitalization, the patient experienced bloody output in his colostomy bag and a decrease in hemoglobin requiring a blood transfusion. The patient underwent an esophagogastroduodenoscopy and had two angioectasias cauterized. Approximately two weeks later, the patient experienced another episode of bloody ostomy output. The patient underwent a small bowel enteroscopy and had a duodenal angioectasia treated. Unfortunately, the patient continued to express blood-tinged stool via his ostomy and underwent a video capsule endoscopy. After eight hours, the capsule failed to advance further than the esophagus and was deemed inconclusive. The patient then underwent a colonoscopy. During the procedure, multiple areas of nodularity, friability, and ulceration were discovered and biopsied. The collected biopsy specimens demonstrated ulcerated mucosa with enlarged amorphous inflammatory cells suggestive of a cytopathic effect that was positive for HSV. This finding was consistent with herpetic colitis. The patient was treated with valacyclovir twice daily for fourteen days. During the remainder of his hospitalization, the patient did not suffer any more episodes of bloody ostomy output and was eventually discharged to a skilled nursing facility.







Imaging: Nodular mucosa, friability with contact bleeding, and mucosal ulceration.

Discussion

- Lower gastrointestinal bleeds have an annual incidence rate of 20.5 – 27 out of 100,000 adults. Even though 80-85% of cases resolve spontaneously, they have a reported mortality rate of 2-4%.[7]
- This case highlights the need for recognition of opportunistic infectious causes of colitis in susceptible patients. Since the physical exam and laboratory findings may be unrevealing, practitioners must maintain a clinical level of suspicion. It is crucial to consider uncommon causes of colitis in patients experiencing gastrointestinal bleeding who have primary or secondary immunodeficiencies,.
- Cytotoxic chemotherapy has the propensity to induce immunosuppression that results in reactivation of a latent HSV infection. This can manifest in a classic oral or genital presentation, or can present atypically as a more severe infection or with visceral involvement.[9]
- Symptoms of herpetic colitis are variable, and can include bloody diarrhea, abdominal discomfort, fever, arthralgias, and weight loss.[6,10]
- Recognition of infection in colonic tissue via immunofluorescence staining or PCR is the most reliable method of diagnosis.
- Treatment consists of administration of an antiviral nucleoside analog such as acyclovir, valacyclovir, and famciclovir.
- Healthcare providers should familiarize themselves with the visceral manifestations of HSV, susceptible populations, diagnosis, and management.

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Chylothorax Secondary to Nephrectomy and Lymph Node Dissection

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Introduction

Chylous ascites is a rare form of ascites that results from the leakage of lymph into the abdomen usually due to trauma or obstruction of the lymphatic system. In rare cases, chylous ascites can ascend into the thorax causing pulmonary symptoms. The most common sources of chylothorax secondary to abdominal ascites include pancreatectomy and liver cirrhosis; there are few reported cases of chylous ascites secondary to nephrectomy with lymph node dissection.

Case Presentation

History of Present Illness

A 53 year old female with recent diagnosis of renal cell carcinoma of the left kidney status post left nephrectomy with lymph node dissection 3 weeks prior presented to the emergency department with one week of progressive shortness of breath. Immediately following surgery, the patient noted a milky-white discharge from the incision site which resolved after two days. Over the following two weeks the patient noted abdominal fullness and bloating followed by progressive shortness of breath that was worse with exertion and lying flat. She stated she was not able to follow up sooner due to evacuating for Hurricane Ida. Patient denied any fever, cough, lower extremity edema, or hemoptysis.

Past Medical History

Hypertension, Hyperlipidemia, Obesity, Type 2 Diabetes Mellitus without Neuropathy, Renal Cell Carcinoma of the L Kidney

Social History

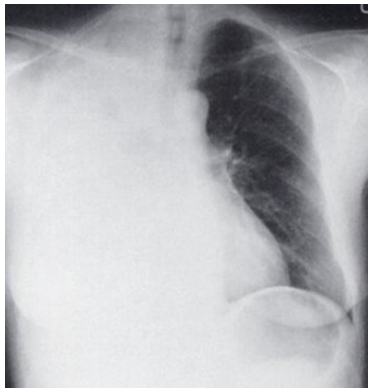
Lives in Metairie with Husband, Retired School Teacher, 30+ Pack Year Smoking History, Current Everyday Smoker, Moderate Alcohol Use, No illicit drug use history

Medications/Allergies

Amlodipine 10mg qd, Lisinopril 40mg qd, Metformin 1000mg bid, Insulin Detemir 20U nightly, Atorvastatin 40mg qd; NKDA

Physical Examination

Vital Signs: HR 105, RR 24, Sp02 96% on room air, BP 160/100, T 97.9 F
Constitutional: **Obese, uncomfortable**
Cardiovascular: **Tachycardia** with regular rhythm, JVP estimated to 5 cm, no lower extremity edema
Pulmonary: Clear to auscultation on the left, **absence of breath sounds on the right and dullness to percussion on the right up to the level of T2**; no crackles or rales
Abdomen: **Distended, well healed surgical scars to L upper/mid/lower abdomen**, soft, non-tender, no organomegaly, **tympany to percussion in upper quadrants, dullness to percussion in lower quadrants**, normal bowel sounds
Neuro: Alert and oriented x3, following all commands, no motor or sensory deficit
Labs:
CBC: WBC 7.5, RBC 4.4, Hgb 12.4, Hct 37, MCV 81, MCH 33, Plt 211
CMP: Na 137, K 4.0, Cl 101, C02 30, **BUN 16, Cr 1.96**, Glu 108, Ca 9.3, Total Protein 6.3, Albumin 4.1, T Bili 0.8, ALP 42, AST 28, ALT 31
FENa: 0.7%
BNP: 22
Imaging:
CXR: Opacification of the right lung field
Bedside US: Large volume ascites and right pleural effusion



Thoracentesis fluid analysis – SAAG 0.6, WBC 189 (ANC 4), RBC 7,888, Glucose 55, LDH 226, Protein 4.1 Triglycerides 3,945, Cholesterol 183

Cytology – Benign effusion of reactive mesothelial cells, macrophages, and chronic inflammatory cells

Discussion

The differential diagnosis of white fluid on thoracentesis includes chyle, cholesterol effusion, empyema, and leakage of tube feeds. Chylothorax is often attributed to obstruction or trauma to the regional lymphatic system. However, chylous ascites has been cited as the cause of chylothorax in 8% of cases. In these cases, pancreatectomy and cirrhosis of the liver are the most common cause of chylous ascites with few reported cases related to nephrectomy. It is important to consider chylous ascites as the cause of chylothorax in any patient with recent history of abdominal surgery. In these patients, ascites generally resolves with conservative management.

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Stevens-Johnson Syndrome/Toxic Epidermal Necrolysis in the Setting of Cirrhosis

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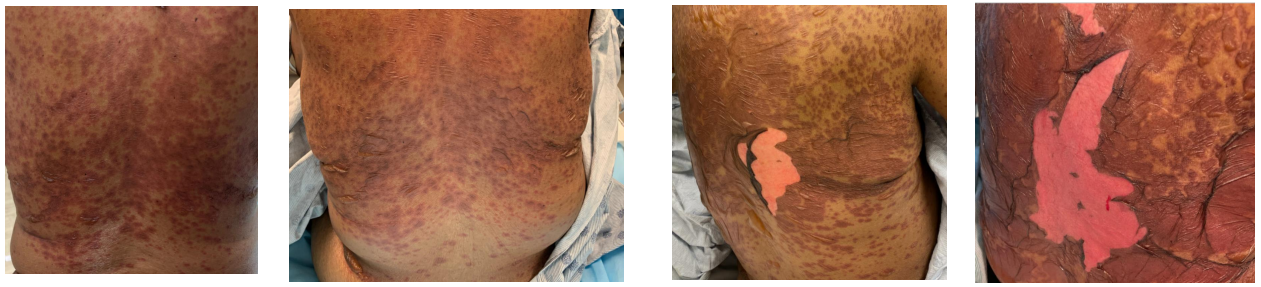
Introduction

- Stevens-Johnson syndrome/toxic epidermal necrolysis is an adverse mucocutaneous reaction that is mediated by T cells. The pathogenesis is not completely understood, but is thought to involve drug and drug metabolites interacting with HLA either directly or indirectly, and causing the activation of T cells.
- The reaction can have triggers including medications, pathogens, and genetics.
- Cutaneous manifestations include macular lesions with a positive Nikolsky sign, bullae, and skin sloughing. Patients can also experience ocular, oral, and genital involvement.
- Withdrawal of the offending agent and supportive treatment remains the standard of care.
- SJS, SJS/TEN, and TEN have a low incidence rate of approximately 1 to 5 per 1,000,000, and a reported mortality rate of 5-30%.
- Prompt diagnosis and specialized involvement is imperative to improve mortality.

Case Description

A 62-year-old female with a past medical history of hypertension and cirrhosis presents to the hospital complaining of dysphagia, oral discomfort, and a skin rash for 2-3 days. One month prior to admission, the patient presented to an outside hospital where she was treated for cirrhosis and a urinary tract infection. She received ceftriaxone and was discharged with spironolactone, furosemide, and propranolol. On physical exam, the patient presented with skin sloughing of her chest, back, upper bilateral arms, abdomen, vulva, and oral mucosa. Her labs were significant for pancytopenia, and elevated lactic acid, ammonia, and HSV1/2 antibody titers. A biopsy consistent with SJS/TEN was obtained, an intravaginal dilator was inserted, and the patient underwent amniotic membrane grafting of both eyes. The patient was initially treated with cyclosporine, morphine, and fluids. The patient's medications from her previous admission were held in the absence of a causative agent. Subsequent fluids were administered with albumin in an attempt to mitigate intravascular loss. The patient developed worsening pitting edema on physical exam, but appeared dry as she was most likely experiencing insensible losses due to her mucocutaneous lesions. The patient progressively became more encephalopathic and hypoxic as a result of her decompensated liver cirrhosis. Ethacrynic acid was used in an attempt to diurese the patient, as furosemide was a medication she was taking when her symptoms began. She underwent a paracentesis, and was treated with lactulose and rifaximin. Unfortunately, the patient's status continued to decline. Her mental status worsened, and her cirrhosis made fluid management difficult. The patient grew positive blood and wound cultures, her blood pressure continued to drop, and her cutaneous involvement made her a poor candidate for a central line. The patient succumbed to her illness on day 7 of her hospitalization.



Imaging: From left to right, progression of macular lesions, bullae, expanding involvement, and skin sloughing over a four day period.

Discussion

- SJS, SJS/TEN, and TEN are T cell-mediated mucocutaneous reactions of the same disease spectrum that are separated by severity of body surface area involvement. SJS affects <10% body surface area, SJS/TEN 10-30%, and TEN >30%.
- Commonly reported associated medications include antibiotics, allopurinol, NSAIDs, and antiepileptics. Studies have also shown an association with certain HLA haplotypes.
- The efficacy of various treatments of SJS/TEN, such as corticosteroids, TNF-α antagonists, plasmapheresis, IVIG, and cyclosporine, remains debatable. However, removal of the suspected offending agents and supportive care, such as fluids, nutrition, pain control, and hygiene are absolutely necessary to reduce morbidity and mortality.
- Patients presenting with co-morbidities, such as cirrhosis, make basic supportive care difficult and can negatively impact the outcome of this disease.

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Discrepancy between Finger Probe SpO2 readings when placed on Finger VS Forehead

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Introduction

- Monitoring of oxygen saturation in the hospital is a key vital sign
- O2 saturation is most commonly measured with a pulse oximeter placed on the fingertip. Other common locations include the earlobe and forehead.
- Continuous pulse oximetry is commonly monitored in patients admitted to the hospital requiring supplemental oxygen
- Although less accurate than an arterial blood gas measurement, pulse oximetry is beneficial because of its ease of application, noninvasive method, and real time constant readings.

Case Presentation

- A 25-year-old female G3P2 at 23w6d with no past medical history presented to the Emergency Department for one week of progressive cough and shortness of breath.
- This was the patient's third presentation with these symptoms over a 5 day period. She had previously been discharged directly from the ED with an Albuterol inhaler, Prednisone, Nasal Spray, and Antibiotics. Symptoms continued to progressively worsen.
- Patient was febrile to 101F, tachypneic RR 30s-40s, tachycardic HR 110s, and oxygen saturation 98% but with desaturation to 88% during minimal exertion.
- Admission ABG pH 7.38 | pCO2 27 | pO2 78
- Patient tested positive for COVID-19
- Chest x-ray revealed bilateral airspace disease consistent with viral pneumonia
- CT PE study with no pulmonary embolism
- The patient was admitted for acute hypoxic respiratory failure secondary to COVID-19 pneumonia
- Given pregnancy, patient's goal oxygen saturation was ≥ 95%

Images

Fig. 1: Continuous pulse oximetry readings with forehead probe (top monitor) vs. fingertip probe (bottom monitor)

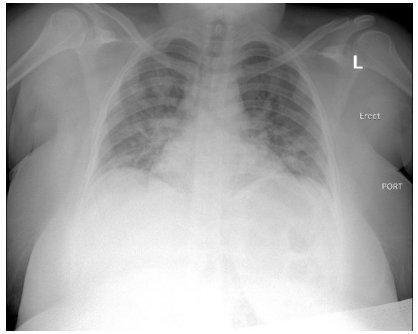

Fig. 2: Patient's admission CXR

Hospital Course

- The patient was started on Dexamethasone 6 mg every 12 hours for fetal lung maturation and Remdesivir
- Due to escalating oxygen requirements by day 2 of the hospital course, the patient was stepped up to the Intensive Care Unit for BIPAP
- By day 8 of the hospital course, the patient's oxygen requirements had steadily improved and she was able to step down to the floor on high-flow nasal cannula
- On day 9 of admission, the patient was noted to have an SpO2 of 97% on room air with a disposable finger probe applied to the forehead, which is a practice not uncommon when unable to obtain an adequate waveform on the finger. A separate evaluation that day using a disposable finger probe on the finger revealed a markedly different oxygen saturation in the low 90's. Confirmatory ABG showed pH 7.46 | pCO2 31 | pO2 48
- The patient remained admitted for several additional days pending resolution of her hypoxemia and oxygen requirements greater than 6 L/min

Discussion

- After determining hypoxemia, disposable finger probes were placed on both the finger and forehead at the same time using two separate machines. The probe on the forehead showed an SpO2 of about 10% higher than the reading on the finger. The probe connectors were switched and continued to show a 10% higher reading on the probe attached to the forehead
- Given the degree of hypoxemia confirmed on ABG, we concluded that the disposable finger probe used on the forehead provided a falsely elevated SpO2 reading
- One small study comparing disposable finger probes on the finger vs the forehead showed a discrepancy of >5% in over half the patients
- Critical management decisions are made based on the SpO2, and inaccurate readings pose significant risk to the patient

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One not-so salty lady

Tulane University Health Sciences Center

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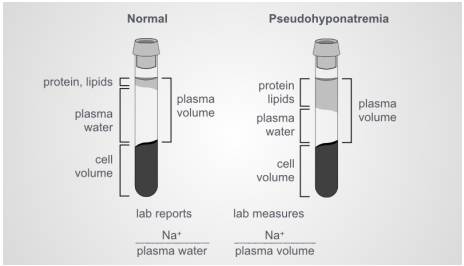
Learning Objectives:
1.Develop an approach to hyponatremia.
2.Recognize roles of aldosterone and ADH in fluid balance.

Case Presentation:
69 yo woman with stage 0 CLL, hypothyroid, and depression was admitted for progressive weakness found to be profoundly hyponatremic to 105 mEq/L with evidence of altered mental status.

Value	Day 1	After restarting fluoxetine	DC
WBC	160	-	-
Na	105	126	134
K	Too high	4.4	5.0
Cl	74	93	99
HCO3	25	26	30
BUN	19	11	10
Cr	0.7	0.7	0.6
Glu	91	129	117
OsmS	231	273	278
OsmU	513	365	-
NaU	<12	38	-

Confirmed with lab, sodium count not affected by WBC. Serum osmolality studies initially consistent with hypotonic hyponatremia. Sodium derangements improved with large total volume IVF resuscitation, yet, she re-developed hyponatremia with low serum osmolality and high urine osmolality possibly related to with increased ADH. Presumed culprit medications were held, and sodium normalized with fluid restriction.

Diagnosis	Serum osms Normal ~ 285-295	Urine osms Low <<300 mOsm High: > 300 mOsm	Urine sodium Low < 20 mM High > 40 mM
Pseudo-hyponatremia -Hypertriglyceridemia -High protein (MM, IVIg) -Hyperglycemia (DKA, HHS)	Normal or High	Variable	Variable
Water > solute intake -Psychogenic polydipsia -Beer potomania -Low-solute diet	Low	Low	Variable
Hypovolemia; non renal -GI losses -Reduced PO intake	Low	High	Low
Hypovolemia; renal -Renal salt wasting -Current diuretic use	Low	High	High
Euvolemic hyponatremia -SIADH -Adrenal insufficiency -Hypothyroidism	Low	High	High
Edematous states -Heart failure -Cirrhosis -Nephrotic syndrome	Low	High	Low



The unusually high protein or lipid fraction (seen in leukemia or hypercholesterolemia) results in the sample being over diluted generating in a false report of hyponatremia. The serum sodium is normal and must be compared to serum osms[1].

Discussion:
Our patient had hypotonic hypovolemic hyponatremia on admission, her serum osmolality was truly low and her urine sodium was low, indicating aldosterone was increased and trying to increase her total body sodium. Her urine osmolality was high showing that ADH was also at work trying to increase her total body water. She improved with IVF, however she later developed hypoxic respiratory failure from volume overload. While she was hypervolemic, she likely had low effective circulating volume due to cardiac decompensation. So, her body likely would have been secreting ADH to increase her total body water. She was diuresed with improvement in her condition. Also, she had been nauseous, which can also increase ADH secretion in the body.[2] There was also the concern that she had syndrome of inappropriate antidiuretic hormone secretion from medication as her serum sodium decreased from normal a few days after restarting her home medication of fluoxetine, which had been stopped on admission. Her sodium eventually returned to normal with discontinuation of fluoxetine and fluid restriction.

Take Home Points:
- *Use serum osmolality to assess true hyponatremia*
- *Use urine sodium to assess if aldosterone is at work*
- *Use urine osmolality to assess if ADH is at work*

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Breast Cancer Metastasis to Bone Marrow, Initially Diagnosed as Myelofibrosis

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INTRODUCTION

Micrometastasis of breast cancer to bone marrow is not uncommon[1]; however, development of symptoms secondary to bone marrow involvement is relatively rare[2]. Additionally, patients with breast cancer treated with radiation and/or chemotherapy are at an increased risk for developing myelodysplastic syndromes (MDS)[3]. This case reviews a patient with a history of breast cancer who presented to bone marrow transplant (BMT) clinic for treatment of myelofibrosis after a recent bone marrow biopsy, but upon re-analysis of the bone marrow a new diagnosis of metastatic breast cancer was made.

CASE DESCRIPTION

A 58-year-old female with a history of HTN, HLD, CAD, hypothyroid, and locally advanced hormone receptor-positive (ER+, PR+, HER2-) invasive lobular breast carcinoma presented to BMT clinic for evaluation of a stem cell transplant for the treatment of myelofibrosis. Her treatment for breast cancer consisted of bilateral mastectomy, chemotherapy, radiation, and adjuvant endocrine therapy. Her baseline hemoglobin had been around 14 g/dl; however, in the months prior to the BMT clinic visit her Hg was consistently <10 g/dl with accompanying shortness of breath, fatigue, and weakness. Her symptomatic anemia was initially thought to be due to her chronic blood loss anemia secondary to diverticular bleeds. Her MCV, however, was elevated (112 fL) with no clear etiology upon workup, which was concerning for a MDS, therefore, a bone marrow biopsy was obtained. The biopsy revealed hypercellular bone marrow with mild megakaryocytic hyperplasia and increased reticulin fibrosis. These bone marrow changes along with splenomegaly that was seen on recent CT were consistent with a myeloproliferative neoplasm, favoring primary myelofibrosis, therefore, she was referred to BMT clinic. Before a final decision was made whether to pursue a stem cell transplant or not the bone marrow biopsy was re-reviewed to confirm the diagnosis. Upon re-analysis of the bone marrow there were cells consistent with metastatic lobular carcinoma which stained positive for GATA3, which confirmed a new diagnosis of metastatic lobular carcinoma of the breast.

DISCUSSION

Re-analysis of the bone marrow was pursued due to her myelofibrosis risk varying between high risk and low risk depending on which scoring system was used. Her case was also difficult due to her chronic blood loss anemia, which made some answers in the scoring systems unreliable (e.g., is she transfusion dependent). Metastasis to bone marrow can lead to extramedullary hematopoiesis with resulting splenomegaly and fibrosis in the bone marrow which can often be confused with myelofibrosis[4]. If there is any concern about an official diagnosis of myelofibrosis after a bone marrow biopsy in a patient with a history of cancer, consider a second opinion or re-analysis of the biopsy.

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Penile Calciphylaxis

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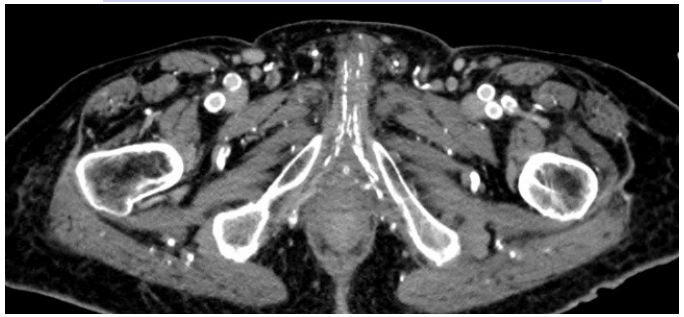
Introduction

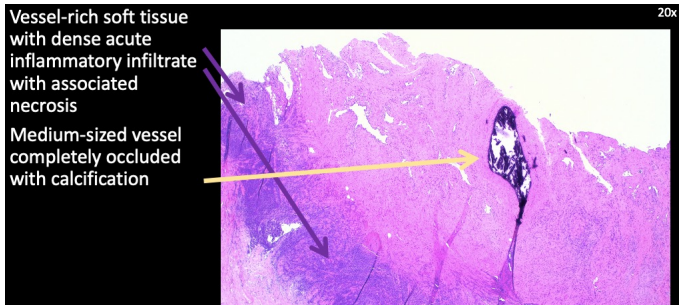
- Calciphylaxis is a rare skin disorder that presents as intensely painful areas of skin ischemia and necrosis
- Most commonly involves the abdomen and thighs, in rarer cases the penis
- Usually associated with ESRD and is exacerbated by warfarin use
- Diagnosis requires skin biopsy showing intravascular calcification and thrombosis
- Treatment includes sodium thiosulfate, thorough wound care, and aggressive pain management
- Carries a high mortality rate

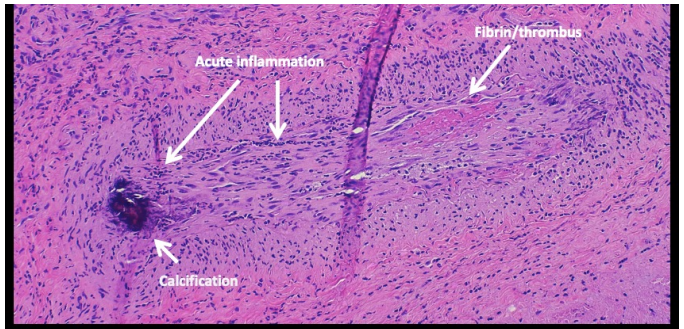
Case Presentation

- A 50-year-old man with past medical history of ESRD on HD and mechanical mitral valve replacement on warfarin presented with one month of progressive penile pain and swelling.
- Per the patient, the area worsened over time to an ulcerated lesion at the glans penis with resultant phimosis.
- CT pelvis demonstrated marked diffuse atherosclerotic disease with wall calcification of all major vessels, as well as questionable mild soft tissue edema and skin thickening of the penis.
- Infectious workup for HIV, HSV, chlamydia/gonorrhea, and syphilis was negative.

Images







Hospital Course

- Pt was evaluated by urology and taken to the OR for circumcision and penile biopsy.
- Frozen section for cancer was negative.
- Final surgical pathology results revealed necrotic ulcer with thrombosis and intravascular calcification at the ulcer base, findings most consistent with calciphylaxis.
- Treatment plan was coordinated with nephrology to begin sodium thiosulfate with dialysis.
- Pt continued to complain of intense pain despite continuous up-titration of pain meds.
- Palliative care and wound care were consulted for symptom management.
- Pt was offered penectomy given refractory pain, however he declined.
- And although it is recommended to discontinue warfarin as it can worsen skin necrosis, the decision was made to continue warfarin given his mechanical valve and otherwise limited options due to ESRD.

Discussion

- Penile calciphylaxis is particularly rare because of the rich vascular network in the area.
- If extensive enough, it can be devastating. Microvascular calcification leads to a cascade of vascular endothelial injury and consequent narrowing and thrombosis of the vessels, which ultimately leads to tissue necrosis from reduced blood flow.
- There is no approved treatment for calciphylaxis. A trial of sodium thiosulfate is suggested but with uncertain efficacy and make take weeks to months to observe clinical response.
- In treatment resistant penile calciphylaxis, penectomy is warranted to potentially prevent months of pain and poor quality of life.
- Still, in spite of maximal efforts to contain this disorder, the prognosis is poor and mortality risk remains very high.

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Do You Hear That?: A Case of Vancomycin Ototoxicity

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Introduction

- Ototoxicity is the damage of ear-related structures (auditory nerve or vestibular system) due to a substance, often a medication.[1]
- Vancomycin ototoxicity is a rare side effect of vancomycin treatment whose mechanism of action is unclear, but suspected to be due to direct damage to CN VIII's auditory branch and is non-dose dependent.[1,2]
- Predisposing factors: increasing age, renal dysfunction, co-administration with additional ototoxic agents, prolonged exposure[1]
- Symptomatology: sensorineural hearing loss (often irreversible), tinnitus, dizziness, vertigo[1,2]

Case Information

- 42 year-old African American female admitted for amputation of the right 5th digit and IV administration of antibiotics following a diagnosis of diabetic wet gangrene.
- Developed bilateral hearing loss 26 days after initiation of IV Vancomycin.
- Hearing loss described as "whooshing noise" in bilateral ears that progressed to near-total hearing loss.
- PMHx: HTN, DM2, ESRD on home PD, PAD
- Physical Exam: (+) Rinne test bilaterally, air > bone. Tympanic membranes pearly-white and intact bilaterally, no signs of effusion.

Table 1. Random vancomycin levels (ug/mL) throughout hospitalization.

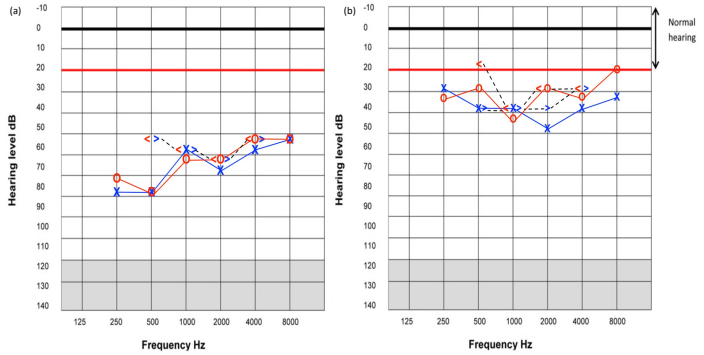
Minimum	16.3
Maximum	28.8
Mean (Average)	21.2
Median	20.9

Clinical Course

- Following initial amputation, patient was discharged home on vancomycin and cefepime as per culture sensitivities for Acinetobacter baumannii, Pseudomonas aeruginosa, Enterococcus faecalis, and Staphalococcus haemolyticus.
- Patient returned to the hospital 5 days later with worsening gangrene of her right foot, now requiring amputation of the 4th digit followed by right femoral to distal arterial bypass.
- Patient 1st reported impaired hearing on day 26 of vancomycin therapy. ENT was consulted for evaluation. Within 1 week, symptoms progressed to near-total hearing loss.
- ID recommended transitioning from IV vancomycin to IV daptomycin.
- Extensive discussion between the primary team, vascular surgery, ENT, and the patient determined systemic steroid administration for treatment of sudden sensorineural hearing loss carried greater risks than benefits to her leg healing and potential need for full leg amputation. Patient desired to avoid systemic steroid treatment.
- On discharge, patient was taken to ENT clinic for audiometry testing and bilateral intratympanic dexamethasone injection. Hearing significantly improved at her 2 month evaluation.

Results

Figure 1. Audiometry results (a) before and (b) 2 months after bilateral intratympanic dexamethasone injections, confirming the diagnosis of moderate-to-severe bilateral sudden sensorineural hearing loss. (O) Right Ear, Air Conduction. (O) Left Ear, Air Conduction. (<) Right Ear, Bone Conduction. (>) Left Ear, Bone Conduction.



Discussion

- We present a patient with vancomycin ototoxicity whose treatment was complicated by comorbidities of ESRD and PAD.
- Predisposing factors contributing to ototoxicity include kidney dysfunction, supratherapeutic drug levels, and prolonged exposure to vancomycin.
- Patient preference for peritoneal dialysis before hemodialysis limited the ability to remove vancomycin quickly from her body.
- The patient's strong desire to avoid limb amputation prevented the use of the first line therapy of high dose systemic steroids as treatment for her sudden sensorineural hearing loss.
- Important learning points:
  - Identification of high risk patients for drug-induced pathology may improve patient outcomes.
  - Partial reversibility of vancomycin ototoxicity is possible with prompt treatment response.
  - Informed decision making by the patient is a critical component to any treatment regimen.

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Abbreviations: Cranial Nerve (CN), Hypertension (HTN), Diabetes mellitus, type 2 (DM2), End Stage Renal Disease (ESRD), Peritoneal Dialysis (PD), Peripheral Arterial Disease (PAD), Infectious Disease (ID)

Comparing the Biopsy Yield and Stenting Efficacy of Endoscopic vs Percutaneous procedures for Biliary & Pancreatic Cancer

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Introduction

The most common modalities used in the management of pancreatic cancer and cholangiocarcinoma (CCA) are endoscopic retrograde cholangiopancreatography (ERCP), endoscopic ultrasound (EUS), CT guided biopsy, and ultrasound-guided biopsy. This study's aim was to examine differences in biopsying and stenting efficacy based on a tumor's anatomic location. We hypothesized endoscopic procedures would have a greater diagnostic and therapeutic yield for more distal cholangiocarcinoma and pancreatic cancer while percutaneous procedures would have a greater yield for more proximal and intrahepatic lesions.

Methods

This study was a retrospective chart review of a single academic hospital. A total of 96 patient charts were identified from the hospital tumor registry. Patients that received multiple biopsies and stenting modalities were examined. Endoscopic procedures included both ERCP and EUS and percutaneous guided biopsy (PCGB) included both CT guided biopsy and ultrasound-guided biopsy. A successful diagnostic yield was defined as a biopsy that lead to a definitive diagnosis for the patient's cancer. Tumors were subdivided into six different groups based on their anatomic location. These groups included intrahepatic cholangiocarcinoma (iCCA), perihilar cholangiocarcinoma (pCCA), distal cholangiocarcinoma (dCCA) , pancreatic head, pancreatic uncinate process, and pancreatic body and tail. Stenting efficacy was determined by examining a 50% bilirubin reduction within three weeks of the procedure and a bilirubin reduction to less than 2.5mg. Adverse outcomes were examined for all procedures. Adverse outcomes included pancreatitis, cholangitis, bleeding requiring transfusion within one week, stent occlusion, and drain occlusion.

Images

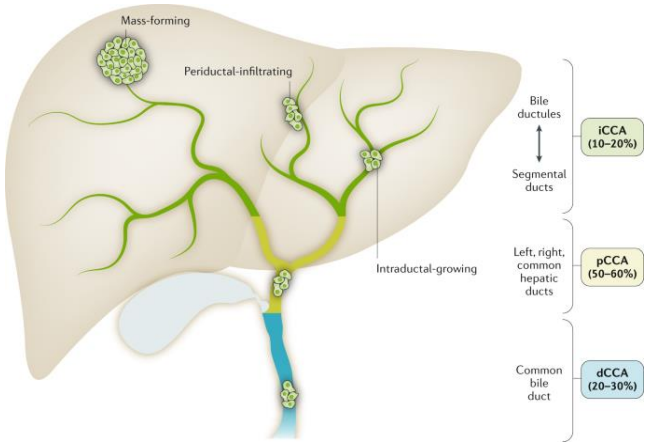

Figure 1: iCCAs are intrahepatic lesions located proximal to the left and right hepatic ducts. pCCA are lesions located in the area distal to the left and right hepatic ducts but proximal to the intersection of the common bile duct and cystic duct. While dCCA encompass lesions found in the distal remaining biliary system up until the ampulla of Vater.

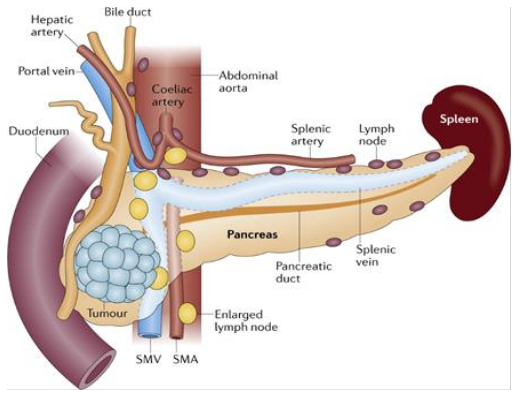

Figure 2: Illustration of pancreatic anatomy in setting pancreatic tumor

Results

96 patient charts were obtained which included 77 cases of pancreatic cancer and 19 cases of cholangiocarcinoma. There were 104 endoscopic procedures and 38 PCGB with the intent to biopsy. When considering all tumor locations, endoscopic procedures yielded a diagnosis 71% of the time and percutaneous procedures successfully yielded a diagnosis in 76% of the cases examined (p-value 0.416). Subgroup analysis of 15 cases of iCCA and pCCA showed endoscopic procedures yielded a diagnosis 50% of the time and percutaneous cases 100% of the time (p-value 0.101). 31 patients received stenting procedures including a total of 38 endoscopic procedures and 6 percutaneous procedures. Bilirubin reduction to less than 2.5mg occurred in 66.7% of the endoscopic cases and 33% of the percutaneous cases (p-value 0.182) and reduction of bilirubin by 50% occurred in 88.3% of percutaneous cases and 70.4% of endoscopic cases (p-value 1). Additionally, there was no statistical difference in adverse outcomes among all procedures.

Conclusion

Tumor location did not effect the biopsying or stenting efficacy of either percutaneous or endoscopic procedures for pancreatic and cholangiocarinomas at the single academic institution. Limitations to this study include sample size and examining operators from a single hospital.

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The importance of recognizing growing teratoma syndrome: A rare complication of non-seminamatous germ cell tumors

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Introduction

Growing teratoma syndrome (GTS) is a rare complication of non-seminamatous germ cell tumors (NSGCT) seen in testicular and ovarian cancers. According to the diagnostic criteria first described in 1982 GTS is defined by the following criteria: normalization of elevated serum alpha fetal protein (AFP) and human chorionic gonadotropin (HCG) levels, tumor growth during or after appropriate chemotherapy for NSGCT, and the exclusive presence of mature teratoma in the resected specimen. The incidence of GTS has been reported to be 1.9 - 7.6% in testicular cases and around 12% in ovarian cases (1,2).

Case Presentation

A 32-year-old male with a past medical history of schizophrenia and polysubstance abuse was evaluated for a right testicular nodule. The ensuing workup showed a complex testicular mass concerning for malignancy, numerous pulmonary nodules in bilateral lung fields concerning for metastatic disease, and lymphadenopathy in the left supraclavicular region, mediastinum, and retroperitoneum. Initial alpha fetal protein was elevated at 43 ng/ml, beta-HCG, and LDH were within normal limits. The patient underwent a right radical orchiectomy. The pathology revealed mixed germ cell tumor in 60% of the specimen and the remaining 40% was showed teratoma. The patient received an initial cancer staging of Stage IIIC (PT1aNXM1b).

The patient underwent three cycles of bleomycin, etoposide, and cisplatin. Repeat imaging showed worsening pulmonary nodules as well as retroperitoneal and supraclavicular lymphadenopathy. Due to disease progression, the patient then received two cycles of cisplatin, etoposide, and ifosfamide. Repeat CT imaging revealed worsening retroperitoneal lymphadenopathy measuring 8.6 x 11.5 cm with evidence of compression on the inferior vena cava and worsening left supraclavicular lymphadenopathy measuring 12 x 9 cm. AFP and HCG levels remained within normal limits throughout treatment. Biopsy of the left neck mass on showed benign teratoma. Upon meeting the patient and reviewing the case, GTS was highly suspected and further chemotherapy was not recommended at this time. Instead, surgical management was pursued. Repeat core needle biopsy of the left neck mass showed mature teratoma. At this time of writing this the patient is currently undergoing workup for surgical intervention.

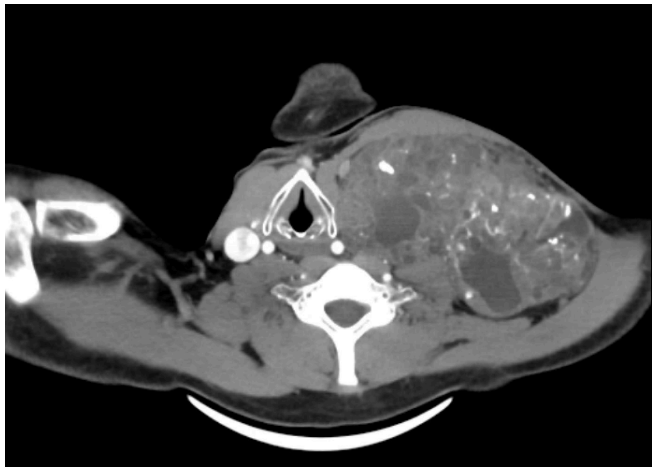

Figure 1: Large heterogeneous left supraclavicular mass measures 13.8 x 7.5 cm axial plane by 9.2 cm craniocaudal. There are multiple fluid density and calcified components evident in the mass. Biopsy later revealing mature teratoma. The mass is displacing the trachea to the right.

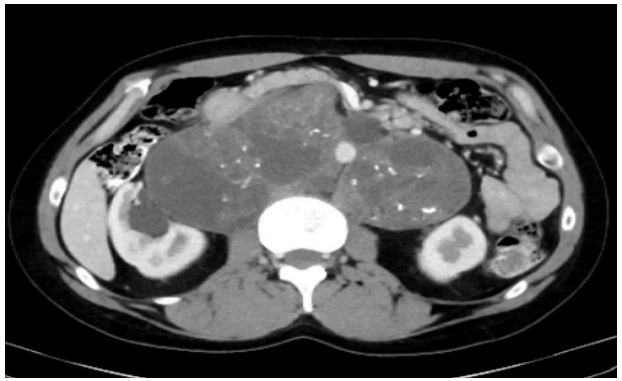

Figure 2: Large mass measuring 18.8 x 8.4 cm in the axial plane. The aorta and vena cava are displaced anteriorly by 2.5cm and 7cm. The right kidney is displaced laterally with moderate hydronephrosis.

Discussion

This case highlights the importance of having a high level of suspicion for GTS in any patient with NSGCT with tumor size progression and with normalization of tumor markers after systemic chemotherapy. GTS is generally resistant to standard chemotherapy regimens. Surgical resection of residual tumor burden is the primary treatment (1,2). Early recognition is important to avoid unnecessary chemotherapy toxicities and to allow for early surgical evaluation.

GTS exerts its detrimental effects through mechanical obstruction of adjacent tissues, and limited data suggest a malignant transformation of GTS in around 3% of cases (2). GTS recurrence rate ranges from 72 to 83% after partial resection and 0 to 12.7% after complete resection (2). No consensus guidelines exist on management GTS, and care should be evaluated with a multidisciplinary team of physicians. In conclusion, this patient was spared further unnecessary chemotherapy by recognizing GTS. Appropriate management was pursued quickly to optimize patient's outcome.

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