January 22, 2022

Resident Poster Submissions
Part 1
Bilateral thalamic glioma represents an uncommon but characteristic radiographic finding. Despite this, the differential for such lesions is quite diverse and includes vascular, neurodegenerative, inflammatory, neoplastic, 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 prior 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 diseases, while rare, have a number of entities which may present this way. There is often characteristic TI, 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.

**Mecanisms**

**Metabolic**
- Heritable encephalopathies; Fahr disease; Wilson disease; Fabry disease; GHD mutations, Kufor disease; Leigh disease

**Demyelinating**
- Acute disseminated encephalomyelitis, systemic lupus erythematosis

**Infectious**
- Neurotropic Bacillus, Viral encephalitis

**Neoplastic**
- Gliomatous thalamic glioma

**Vascular**
- Infarction of lateral thalamus, territory of Aristotle's artery, thalamic vascular malformations, subependymal arteriovenous malformation

**Case Presentation**

A 55 year old caucasian male presented (D = 3) to an outside hospital with family due to acute onset non-fluent aphasia and confusion beginning at 10 am. The patient had known normal was the right prior at 11 pm and his initial NIH stroke scale was 6. Non-contrast CT of the head showed no acute changes however subtle thalamic hypodensity was noted and felt to be related to possible chronic microvascular change. Telestroke for a tertiary medical center (D=4) recommended transfer for further evaluation at a dedicated stroke center however no admissions given.

The patient admitted 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 symptomology. His medical history was notable for uncontrolled hypertension and uncontrolled type 2 diabetes. His mother had a history of Alzheimer’s disease. His sister had a diagnosis of brain metastases from lung cancer.

The patient’s imaging revealed bilateral thalamic enhancement with periventricular hypodensity. Further evaluation at a tertiary center revealed a complex lesion involving both thalami with heterogeneous signal intensity on T1 and T2. Glioblastoma multiforme was ruled out on cytology and immunohistochemistry.

**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 supports 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 hemorrhagic 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.

**References**


References


**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.

**References**


Introduction

- Hydrothorax can be defined as a 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 placed 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 millimercury 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.

References

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, hypocapnia, 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 luminal-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 luminal-positive voltage that forces magnesium and calcium reabsorption. Claudin-16 and claudin-19 facilitate the paracellular transport of magnesium and calcium. Activation of CatSBR 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%.
- FEtMg >3% with normal kidney function is suggestive for renal magnesium wasting.
- Oral supplementation with Mag-Ox TID was added while inpatient and SlowMag and amlodipine 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 hypercalcic to suggest Barter/Gillerman’s or Familial primary hypomagnesemia with hypercalcuria 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 receptor among others.

References

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 25% 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 ischemic 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 weeks. 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 infection. Nephrology consultation was arranged. 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 mg/dL. Urine protein creatinine ratio 18.78 g/mmol, compared to baseline creatinine 1.80 mg/dL, and positive COVID-19 rapid testing.

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 homozgyous for APOL1, a risk factor associated with collapsing FSGS.

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 the glomerulus revealed wrinkled basement membranes that are uniform and are of normal thickness.

Clinical History
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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.

References
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 associated with 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).

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 sellar/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

Sellar masses may present with symptoms related to hormonal systems or neurological pathways, but simply may present asymmetrically 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.

Conclusion

References

A Curious Case of Meigs-like Syndrome
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Department of Internal Medicine - Lafayette, LA

Introduction
- Meigs syndrome is defined as a benign ovarian tumor (I) associated with ascites and pleural effusion (II) 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

Case Description (labs/imaging, etc.)

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 catheterization 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

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 catheterization 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

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.

References

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.

iii) Progression of chest x-ray changes from day before surgery (left) versus 5 months after hospitalization (right)
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 HF/EF 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, amiodipine, carvedilol, and bumetanide

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 bumeataline 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

Labs

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
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<tbody>
<tr>
<td>Renin Activity</td>
<td>&lt;0.1 ng/mL/hr (0.2-1.6 ng/mL/hr)</td>
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<tr>
<td>Aldosterone/Renin Ratio</td>
<td>126 ng/dL (&lt;31ng/dL)</td>
</tr>
<tr>
<td>Potassium on admission</td>
<td>3.1 mmol/L (3.6-5.2 mmol/L)</td>
</tr>
</tbody>
</table>

Images

- Image 1-2: 2.6cm adrenal nodule seen on CT imaging
- Image 3: Appearance of enlarged cardiac silhouette on chest xray consistent with patient’s hypertension-induced cardiomyopathy

References

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 103°F.
- 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-scalpular 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, BIUN 61, WBC 7.66, 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 isolated the patient on empiric antibiotics.
- However, a lumbar puncture was performed and meninges 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 tissues was noted.
- CT scan of the head and neck showed degenerative changes of the cervical region.

REFERENCES

Case Description (labs/imaging, etc.)

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/tables, etc.)

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 (labs/tables, etc.)

Post Renal Transplant Temporal Lobe Abscess
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Introduction

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Case Description (labs/tables, etc.)

Conclusion

Close follow up post-renal transplant and surveillance for the development of Nocardiosis is necessary to avoid development and systemic dissemination.

Subtle clinical clues may be of help, followed by pulmonary and head CT imaging if suspected.

References

Coussement J. et al., Nocardia infections in solid organ and hematopoietic stem cell transplant recipients. Current Opinion in Infectious Diseases: December 2017 - Volume 30 - Issue 6 - p 545-551
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.

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)

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>59 (15-81)</td>
</tr>
<tr>
<td>Race</td>
<td></td>
</tr>
<tr>
<td>3 (18%) Asian</td>
<td></td>
</tr>
<tr>
<td>9 (53%) Hispanic</td>
<td></td>
</tr>
<tr>
<td>2 (11%) African American</td>
<td></td>
</tr>
<tr>
<td>3 (18%) White</td>
<td></td>
</tr>
<tr>
<td>Gender</td>
<td></td>
</tr>
<tr>
<td>4 (24%) Female</td>
<td></td>
</tr>
<tr>
<td>13 (76%) Male</td>
<td></td>
</tr>
<tr>
<td>Baseline Serum Creatinine (mg/d)</td>
<td>3.4 (1.2-6.5)</td>
</tr>
<tr>
<td>3+ Dipstick Protein</td>
<td>16 (94%)</td>
</tr>
<tr>
<td>Urine Protein-Creatinine Ratio</td>
<td>6.7 (1.3-11.7)</td>
</tr>
</tbody>
</table>

Histopathological Findings (n=11)

- Diabetic Glomerulopathy: 3
- Focal Segmental Glomerulosclerosis: 3
- Transplant Glomerulopathy: 2
- Membranous Nephropathy: 1
- Thrombotic Microangiopathy: 1
- Advanced Arteriophrasclerosis: 1

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

Dylan H. West M.D.; Adarsh Jones MS4; Jones Samuel M.D.

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 venricular filling and thus decreased stroke volumes. Baroreceptors are thereby exposed to diminished pulsations and mediate peripheral vasodilatation, 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 transmirtal 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.

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.

References


Figure 1: Physiology Flow Diagram of Cardiac Tamponade

Figure 2: Diagram of Heart in Pathologic State Described in Case Report
Acute Ischemic Stroke as the Initial Presentation for TTP

Vivian Jaber MD PhD, Kiara Fahimipour MD, Naseem Khorrarm MD, Seema Walvekar MD, William Imsais MD

Department of Internal Medicine, Louisiana State University Health Sciences Center, New Orleans, LA

**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 to 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/o 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**

|  | 
|---|---|
| Fibrinogen | 640
| D-Dimer | 703
| LDH | 633
| Haptoglobin | <30
| DAT | neg

**Images**

**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.

**References**

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 choices.

Diagnostic imaging is important to evaluate if edema is secondary to abscess formation from bacterial infection vs foreign body. When considering empiric antibiotics for a clitoral abscess literature supports including MRSA and anaerobic coverage. Based 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 ducus arteriosus that was repaired at 2 years old. She took no daily medications. Mother and patient deny fevers, chills, or flank pain, dysuria, cloudy or foul smelling urine, or unusual vaginal discharge.

Vital signs at time of our exam were blood pressure of 121/79, pulse 108, temperature 98.1°F (36.7°C), respiratory rate 20, weight 55 kg (121 lbs), and height 1.5 m. Cardiopulmonary and abdominal examination were benign. Genitourinary exam revealed a 3 cm x 3 cm x 3 cm collection in the suprapubic area, most consistent with an abscess, and edematous prepuce. The prepuce was erythematous and edematous, mildly tender to palpation, no vaginal discharge and no odor.

Ultrasound of the prepuce revealed a complex structure measuring 1.7 x 0.8 x 0.9 cm, with surrounding hypoechoic edematous tissue. There was linear increased echogenicity within the collection which may represent possible small foreign body. Differential includes small foreign body with surrounding granulomatous fluid collection, epididymo-orchitis, and an abscess.

Pt was taken to the operating room by Gynecology for incision and drainage source control. Incision of a 3 cm x 3 cm x 3 cm collection resulted in white purulent discharge. 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 meticillin/azide 500 mg BID and Bactrim 800-160 mg BID.

Gram staining showed gram positive cocci in pairs, gram positive bacilli, and gram negative cocccobacilli. 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.

When encountering a clitoral prepuce abscess, reported in the literature, the patient was taken to the operating room by Gynecology for incision and drainage source control. At that visit, a new complaint of a breast abscess with purulent drainage from the nipple. No fever, 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.

Summary

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 choices.

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

Preoperative cultures showed sensitivity to penicillins. The patient’s family was called and antibiotics changed to Augmentin for 5 days.

References

1. Zaidman BA, R дет все 2020 presented a review of current cases and found that polymicrobial infections with Enterobacteriacaea 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.

2. 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 the gut and oral cavity flora. When pathologic growth occurs as pyogenic infections, abscess formation or endocarditis is most common. This group is commonly susceptible to beta-lactam antibiotics. Prevotella disiens is part of the Prevotella species that are common to the vaginal microbiota, overgrowth of which has been associated with bacterial vaginosis. This species tends to susceptible to penicillins. Generally, both isolated species are common to the vaginal microbiota and the Streps species has been implicated in abscess formation.

3. 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 follow 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
Vivian R. Jaber MD PhD, Logan S. Ledet MD, Victoria E. Burke MD
Department of Internal Medicine, Louisiana State University Health Sciences Center, New Orleans, LA
Department of Infectious Diseases, Louisiana State University Health Sciences Center, New Orleans, LA

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 nodule, 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

References
Metformin-Associated Lactic Acidosis in the setting of pre-renal Acute Kidney Injury
William Kemp MD, Fernanda Correa MD, Jorge Martinez MD
Department of Medicine, LSU Health Sciences Center, New Orleans LA

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.

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 lactate 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.

References

Thyroid Storm Leading to Cardiogenic Shock  
Anjali Kakkar, DO  
Department of Internal Medicine, Ochsner Medical Center, LA, USA

**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**

<table>
<thead>
<tr>
<th>Day 1:</th>
<th>Day 2:</th>
</tr>
</thead>
<tbody>
<tr>
<td>EKG – afib RVR</td>
<td>Free T4: 1.84 ng/dL</td>
</tr>
<tr>
<td>Creatinine: 0.6 mg/dL</td>
<td>Lactic acid: 6.0 mmol/L</td>
</tr>
<tr>
<td>AST/ALT: normal</td>
<td>Total bilirubin: 3.2</td>
</tr>
<tr>
<td>Total bilirubin: 1.5 mg/L</td>
<td>AST/ALT: 1354/326 U/L</td>
</tr>
<tr>
<td>BNP: 670 pg/mL</td>
<td>Creatinine: 1.3 mg/dL</td>
</tr>
<tr>
<td>Troponin: normal</td>
<td>TTE: EF 20%, global hypokinesis, moderate RV dysfunction, biatrial enlargement, LVSP 45 mmHg, CVP 15 mmHg</td>
</tr>
<tr>
<td>Lactic acid: 2.0 mmol/L</td>
<td>TSH: &lt;0.01 ul/L/mL</td>
</tr>
<tr>
<td>Free T4: 2.50 ng/dL</td>
<td>Free T4: 2.50 ng/dL</td>
</tr>
</tbody>
</table>

**Imaging**

**References**

**Management & Outcome**
Patient was started on propranolol, propylthiouracil (PTU), steroids, apixaban (CHA2DSVasc 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.
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 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

References

A Case Report of Neuro Sarcoidosis

Pavana Sakhamuri, MD; Tristan Dao, MD; Mekha Matthew, DO; Ann Chauffe, DO

Louisiana State University Health Sciences Center at Ochsner University Hospital & Clinics
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.

**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/mielle 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 mainstay 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.

**Imaging**

MRI Brain revealing contrast enhancing lesions

**References**


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.

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
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 HFrefEF, 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 (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.

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

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

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
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 immunosuppression. Bacteria most commonly implicated in retropharyngeal abscesses 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 could not 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.

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.

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.

References

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.

Stool Studies

- Entamoeba Histolytica PCR + positive
- Negative Stool Studies:
  - Ova & Parasites
  - Fecal Leukocytes
  - Cryptosporidium PCR
  - Giardia PCR
  - Dientamoeba frag. PCR
  - Cyclospora PCR
  - Stool Culture

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

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

Stool Culture

- Serum, Urine Studies
  - Serum – all negative
  - Blood, Abx
  - Brucella Abx
  - Coccidioides IgM, IgG
  - Histoplasma Ag
  - Bartonella PCR
  - Echinococcus Abx
  - Strongyloides IgG
  - Hepatitis Panel, T-Spot
  - Blood Culture x2
  - Urine Histo, Blasto- Neg.
  - Abscess Fluid
  - WBC 59,000
  - 51% Neut, 47% Lymph.
  - RBC 43,000
  - Pathology:
    - Sarcoid tissue sample
    - Fungal Cx – neg.
    - Gram Stain
    - AFB Cx – neg.
    - Rare G+ Cocci
    - Aerobic Cx – neg.
    - Aerobic Cx – neg.

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).
- Amoebic 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.

References

Dermatomyositis: The Great Predictor
Chelsea Vaughn MD, Shane Guillory MD
Department of Internal Medicine, Louisiana State University Health Sciences Center, New Orleans, LA

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.

Imaging

• 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 H2O, and further staging imaging was done which revealed bony metastasis.
• He was eventually started on systemic chemotherapy with capecitabine and oxaliplatin.

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.

Hospital Course

• 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 colonoscopy 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 organ 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.

Discussion

https://doi.org/10.21037/atm-20-5215

References
A Case of the Malabsorbed Thyroid Hormone

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Department of Internal Medicine, Louisiana State University Health Sciences Center, New Orleans, LA

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 cholecodocholithiasis 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.

Hospital Course

- Cholangiogram showed cholecodocholithiasis 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 jejunum, ~24% in lower jejunum).
- GI malabsorption etiology (Figure 1):
  - Lactose malabsorption, celiac disease, SIBO, H. pylori, gastric atrophy, biliopancreatic diversion, gastric sleeves, liver cirrhosis, pancreatic steatorrhea.
- Drugs causing malabsorption: PPIs, calcium carbonate, beta blockers, rifaximin, TCAs, bile acid sequestrants.
- Exclusion of pseudomalsobation, 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 (intestinal nonadherence).

<table>
<thead>
<tr>
<th>Table 1: Trend of TSH and FT4 through Hospital Course</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hospital Day 1</td>
</tr>
<tr>
<td>TSH (uIU/mL)</td>
</tr>
<tr>
<td>FT4 (ng/dL)</td>
</tr>
</tbody>
</table>

References


Discussion

- Hypothyroidism is a common disease, easily treated with thyroid hormone replacement therapy (levothyroxine).
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- Exclusion of pseudomalsobation, 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 (intestinal nonadherence).
When Normal Is Not Normal: A Case of Rasburicase-Induced Hemolytic Anemia

Tyler Theriot MD, Robert Thibodaux MD
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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 (Hb 6.5; total bilirubin of 2.7; LDH of 2,383; and an undetectable haaptoglobin.
- 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.

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.

References

Figure 1: Peripheral smear showing Heinz bodies (green) and bite cell (black)
Figure 2: Peripheral Smear showing Heinz bodies (green) and microspherocytes (black)
Figure 3: G6PD Quantitative analysis revealed levels at lower-limit of normal.

RBC
4.14 - 5.80 x10^6/UL
4.15

G6PD Qn
127 - 427 U/10E12
133

RBC
INTRODUCE

• 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.1,4

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 organisms). MRI at our facility demonstrated findings concerning for PRES (Figure 2) prompting transfer to the ICU for IV antihypertensive therapy.5

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.1,4

REFERENCES


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.

Figure 3. MRI Brain

Neurologic Sequela of COVID19 Infection

Alexander Say M.D., Carl Giffin M.D., LSU Department of Internal Medicine

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.
Late Presentation of Rheumatoid Arthritis in Sickle Cell disease: a case report
Tristan Dao, MD; Nicholas Sells, MD
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Department of Internal Medicine- Lafayette, LA

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 sequel 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

Labs on Presentation

<table>
<thead>
<tr>
<th>Lab Value</th>
<th>Reference</th>
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<tr>
<td>ANA Pattern</td>
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<td>ANA Titer</td>
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<tr>
<td>Rheumatoid Factor IgM</td>
<td>$&gt;100$ units</td>
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<tr>
<td>ESR</td>
<td>69/mnt</td>
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<tr>
<td>Cyclic citrullinated peptide IgM</td>
<td>179 units</td>
</tr>
<tr>
<td>C Reactive Peptide</td>
<td>$&gt;52$</td>
</tr>
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<td>Rheumatoid Factor IgM</td>
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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 immunologic 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

References


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.

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

## REFERENCES


1. Th17 cytokine deficiency in patients with Aspergillusskull base osteomyelitis Corine E Delsing et al

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).
- 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 ventilricular dysfunction.
- We present a young otherwise healthy male with no comorbidities who developed fulminant myocarditis requiring a ventilricular 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 electrocardiogram performed revealed an ejection fraction of 15-25% 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 ventilricular and ventilatory support, but was able to be extubated and weaned off ventilicular 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)

- Of note, he had tested positive for COVID-19 approximately 6 weeks after full recovery from a COVID-19 infection.
- Among the 2,116 patients with COVID-19 and myocarditis, 1,895 (89.8%) received a diagnosis of COVID-19 and myocarditis during the same month; the remaining patients received a myocarditis diagnosis 1 month (13.6%) or 2 months (8.3%) after their COVID-19 diagnosis.
- 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 ventilricular assist devices; there is no role for non steroidal anti-inflammatory drugs or corticosteroids in management of viral myocarditis.

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 (89.8%) received a diagnosis of COVID-19 and myocarditis during the same month; the remaining patients received a myocarditis diagnosis 1 month (13.6%) or 2 months (8.3%) after their COVID-19 diagnosis.
- 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 ventilricular assist devices; there is no role for non steroidal anti-inflammatory drugs or corticosteroids in management of viral myocarditis.

References

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

Vishal Busa, MD, Deepi Kantamani, MD, Rameela Mahat, MD, Karthik Reddy, MD

Baton Rouge General Internal Medicine Residency Program, affiliate of Tulane University School of Medicine

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 Guillian-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.

Discussion

• Table showing parameters of the patient during initial course

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Day-1</th>
<th>Day-2</th>
<th>Day-3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Respiratory rate</td>
<td>17</td>
<td>22</td>
<td>32</td>
</tr>
<tr>
<td>Saturation (O2)</td>
<td>94%</td>
<td>92%</td>
<td>86%</td>
</tr>
<tr>
<td>NIF (Negative inspiratory force)</td>
<td>&gt;48</td>
<td>40</td>
<td>30</td>
</tr>
</tbody>
</table>

• 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, ophthalmooplegia.
• 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 correlates 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.

References

Introduction

- Lemierre’s syndrome (also termed postanginal sepsis and necrobacillosis) is a rare condition that usually begins with tonsillitis/pharyngitis and progresses to involve internal jugular vein thrombosis, adjacent soft tissue inflammation, persistent bacteremia, and even septic embolization.¹
- 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

- Though the differential diagnoses can be broad, physicians should consider this condition when patients present with fever, pharyngitis/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.

Conclusion

- Lemierre’s syndrome (also termed postanginal sepsis and necrobacillosis) is a rare condition that usually begins with tonsillitis/pharyngitis and progresses to involve internal jugular vein thrombosis, adjacent soft tissue inflammation, persistent bacteremia, and even septic embolization.¹
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References

Reactive Arthritis: A Joint Effort by Haemophilus and Syphilis?

David Van MD, David Montgomery MD, Jacob Dubuc MD, Hope Oddo-Moise MD

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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 103°F 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.

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 tertiary syphilis; however, these 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.
Introduction

Hypersensitivity pneumonitis is a condition that is often acquired 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 hypcapnic respiratory failure.

The patient was admitted to the ICU for hypoxic hypcapnic respiratory failure presumed due to pulmonary edema 2/2 underlying heart failure.

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 initiation 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.

Bronchial lavage was not performed due to rapid improvement, but it would have suggested the diagnosis if it were to show lymphohcytosis 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 mold 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.

References


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.

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.

REFERENCES
KSHV Inflammatory Cytokine Syndrome (KICS)

S. Sridhar, MD; V. Silver, DO; T. Davidve, MD; N. Patel, MD; L. Engel, MD

Department of Internal Medicine, Louisiana State University Health Sciences Center, New Orleans, LA

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)
- KSHV Inflammatory Cytokine Syndrome (KICS)

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.
- The necessity, 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.

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

S. Sridhar MD, J. Carnicle MD, J. Amoss MD, L. Engel MD
Department of Internal Medicine, Louisiana State University Health Sciences Center, New Orleans, LA

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.

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.

References


Images

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

Figure B: Coronal view
Systemic lupus erythematous as a mimicker of lymphoproliferative disease

Michael P Bank MD, Andrea S Ito MD, Sana A Chaudhary MD, Shane E Sanne DO

Department of Internal Medicine, Louisiana State University Health Sciences Center, New Orleans, LA

Introduction
❖ Systemic lupus erythematos (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.

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].

References

Images

Image 1: CT Chest showing lymphadenopathy

Image 2: CT Abdomen and Pelvis showing splenomegaly
A Rare Etiology of Elevated Anion Gap Metabolic Acidosis: Methanol Toxicity

David Beyer, MD, Chantal Pham, BS, Leon Sanders, MS, Nazary Nebeluk, PhD, David Van, MD, Xing Hou, MD, Shane Sanne, DO, Lee Engel, MD

Department of Internal Medicine
LSU Health Sciences Center, New Orleans, LA

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, Salicylates.
- 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 <4 mg/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.

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.

References

DOI: 10.1056/NEJMA200102083440605
Maternal Outcomes in Subsequent Pregnancies after Peripartum Cardiomyopathy
Keerthish Jaisingh, MD; Pavida Pachariyanon, MD; Hari Bogabathina, MD; Runhua Shi, MD; Sampath Singireddy, MD; Kalgi Modi, MD
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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.

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.

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.

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Figure 1

Mortality

<table>
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<tr>
<th>RG (2/11)</th>
<th>NRG (6/22)</th>
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<td>18%</td>
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0% 5% 10% 15% 20% 25% 30%
A Case of Nontyphoidal Salmonella Bacteremia, Disseminated Histoplasmosis, and Disseminated MAC in an Immunosuppressed Patient

Thuy-Mai Nguyen, MD; Logan Sonier, MD; David Beyer, MD; Brittany Lynn Boudreaux, DO; William Imsais, MD; John Nanfro, MD

Department of Medicine, Section of Infectious Disease, LSUMC-New Orleans, LA

Case Presentation

- 51-year-old man with HIV/AIDS (CD4 count 15), HCV without cirrhosis, nasopharyngeal DLBCL (in remission), cryptococcal meningitis (a/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) 2-3 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: 104.9°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/m²
- Exam: Cachectic advanced HIV/AIDS (CD4 count 15), HCV without cirrhosis, nasopharyngeal DLBCL (in remission), cryptococcal meningitis, oral voriconazole for dual coverage of disseminated Histoplasmosis and cryptococcal meningitis, clarithromycin with ethambutol for disseminated MAC.
- 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 M. avium complex (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 populations such as those with immunosuppression.3,4

References


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 with air bronchograms, multifocal cystic changes, and thickening of the right-sided fissure.
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 1

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.
- 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.3 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.
- 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.5
- 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.

References

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.
- In the case of chronic urinary retention (CUR), however, patients often present without any pain symptoms.2

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.

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.1,3 In our case, our patient had gross abdominal distension and was found to be retaining 8.2L of urine, which appears to be the largest volume of retained urine documented at this time.1
- 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 known 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

References

1 Haltgreaves C, Dorens T. Postobstructive diuresis: pay close attention to urinary retention. Can Fam Physician. 2015;61(2):137-142.
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.

• 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. 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. 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.

• Use of medications, such as steroids, cyclosprine, 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 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%.1

• 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.2

• Symptoms of herpetic colitis are variable, and can include bloody diarrhea, abdominal discomfort, fever, arthralgias, and weight loss.3,4

• 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.

References


Chylothorax Secondary to Nephrectomy and Lymph Node Dissection
V Patel DO, K Hoppen MD, M Modica MD

Introduction
Chyloous 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, chyloous ascites can ascend into the thorax causing pulmonary symptoms. The most common sources of chylothorax secondary to abdominal ascites include pancreatocystectomy and liver cirrhosis; there are few reported cases of chyloous 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
Hyperension, 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, SpO2 98% 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, Pt 211
CMP: Na 137, K 4.0, Cl 101, CO2 30, BUN 16, Cr 196, 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, chyloous ascites has been cited as the cause of chylothorax in 8% of cases. In these cases, pancreatocystectomy and cirrhosis of the liver are the most common cause of chyloous ascites with few reported cases related to nephrectomy. It is important to consider chyloous ascites as the cause of chylothorax in any patient with recent history of abdominal surgery. In these patients, ascites generally resolves with conservative management.

References
• Stevens-Johnson syndrome/toxic epidermal necrosis 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.

Introduction

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, allopurinol, and propranolol. On physical exam, she was most likely experiencing insensible losses due to her mucocutaneous involvement, and skin sloughing over a four day period.

Case Description

The patient progressively became more encephalopathic and hypoxic as a result of her decompensated liver disease. 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 uncomplicated 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.

References


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%.

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

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.

References

One not-so-salty lady
Melanie Wanigatunga, Jerald Taggart, Adrian Baudy IV, Katherine Peacock
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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.

<table>
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<td>Na</td>
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</tr>
<tr>
<td>K</td>
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<td>26</td>
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</tr>
<tr>
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<td>19</td>
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<td>10</td>
</tr>
<tr>
<td>Cr</td>
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</tr>
<tr>
<td>NaU</td>
<td>&lt;12</td>
<td>38</td>
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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, 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 | Urine osms | Urine sodium |
<table>
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<tr>
<td>Low &lt; 20 mM High &gt; 40 mM</td>
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**Pseudo-hyponatremia**
- Hypertriglyceridemia
- High protein (MM, IVlg)
- Hyperglycemia (DKA, HHS)

**Water > solute intake**
- Psychogenic polydipsia
- Beer potomania
- Low-solute diet

**Hypovolemia; non renal**
- GI losses
- Reduced PO intake

**Hypovolemia; renal**
- Renal salt wasting
- Current diuretic use

**Euvolemic hyponatremia**
- SIADH
- Adrenal insufficiency
- Hypothyroidism

**Edematous states**
- Heart failure
- Cirrhosis
- Nephrotic syndrome

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. 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.

**Discussion:**
- 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

**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

**References:**

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

Micrometastasis of breast cancer to bone marrow is not uncommon¹; however, development of symptoms secondary to bone marrow involvement is relatively rare². 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 myelofibrosis⁴. 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.

REFERENCES

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.

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.

References

Ototoxicity is the damage of ear-related structures (auditory nerve or vestibular system) due to a substance, often a medication. 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. Predisposing factors: increasing age, renal dysfunction, co-administration with additional ototoxic agents, prolonged exposure. Symptoms: sensorineural hearing loss, tinnitus, dizziness, vertigo.

**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 intact bilaterally, no signs of effusion.

**Results**

- Following initial amputation, patient was discharged home on vancomycin and cefepime as per culture sensitivities for Acinetobacter baumannii, Pseudomonas aeruginosa, Enterococcus faecalis, and Staphylococcus 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.

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

<table>
<thead>
<tr>
<th></th>
<th>Minimum</th>
<th>Maximum</th>
<th>Mean (Average)</th>
<th>Median</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vancomycin</td>
<td>16.3</td>
<td>28.8</td>
<td>21.2</td>
<td>20.9</td>
</tr>
</tbody>
</table>

**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.

**References**


**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 that 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.

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 CCA 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 cholangiocarcinomas at the single academic institution. Limitations to this study include sample size and examining operators from a single hospital.

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

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Introduction

Growing teratoma syndrome (GTS) is a rare complication of non-seminomatous 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 ifosfamide. 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. Repeat CT imaging revealed worsening retroperitoneal lymphadenopathy measuring 8.6 x 11.5 cm with evidence of compression on the inferior vena cava and vena cava are displaced anteriorly by 2.5cm and 7cm. The right kidney is displaced laterally with moderate hydronephrosis.

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.

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.

References