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Resident Poster Submissions





Vancomycin-Induced Leukopenia

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Introduction

Vancomycin is a commonly used antibiotic for gram-positive infections, especially for methicillin-resistant staphylococcal aureus (MRSA). Although infrequent, vancomycin-induced leukopenia is a serious complication of vancomycin that can occur after a 7-10 days of vancomycin therapy. This rare adverse effect presents a clinical challenge as many patients may require gram-positive antibiotic therapy for weeks to months.

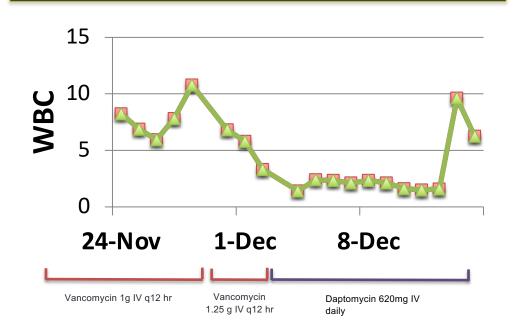
Case Presentation

A 53-year-old man with a past medical history of intravenous drug abuse presented with gradual right-sided weakness progressing over three weeks and was found to have an anterior cervical spine abscess. He reported no medication allergies or intolerances and had minimal other relevant medical history other than benign prostate hyperplasia. On admission, his WBC count was normal at 8.23 K/uL. Given history and presentation, he was empirically started on vancomycin therapy. He underwent debridement of cervical abscess and cultures revealed MRSA with minimum inhibitory concentration of 1. He was initiated on a vancomycin protocol with a goal trough of 15-20 ug/dL.on day of admission. Infectious disease team was consulted and recommended to continue treatment against MRSA for six weeks. After eight days of vancomycin, the patient's WBC count dropped below 5 K/uL to 3.32 K/uL. The WBC count reached its lowest value at 1.42 K/uL with a drop in granulocytes from 72% to 46%. At that time, vancomycin was discontinued and daptomycin initiated at the recommendation of infectious disease. The WBC remained less than 3 K/uL for the following eight days. Additionally, his ANC also decreased Hematology was consulted. Peripheral blood smear revealed some bands, monocytes, but no blasts were identified.

Case Presentation continued

After review, all teams agreed that leukopenia was likely secondary to vancomycin therapy. Following discontinuation of vancomycin, the WBC counts gradually improved to 6.25 K/uL on day of discharge. Consistent with previous data, white blood cells recovered after a week from discontinuation of vancomycin.

Laboratory Review



Discussion

Although rare, vancomycin therapy can contribute to leukopenia. Retrospective data estimate the incidence of vancomycin-induced leukopenia to be 2-8% in hospitalized patients^{1,2}. Analysis of case reports indicates the vancomycin-induced leukopenia occurs 12-21 days after initiation¹. Discontinuation of vancomycin is necessary and improvement in white blood cell counts commonly recover within 7 days of discontinuation without further intervention¹.

In general, the most common cause of leukopenia is decreased marrow production
The mechanism of responsible for the leukopenia is unclear but thought to be antibody mediated destruction of neutrophils².

This is an important side effect to recognize especially in patients who may require weeks of therapy outside of the close monitoring of an inpatient setting. If recognized, therapy adjustments to another agent should be considered.

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A rare case of Pseudo-TMA in a patient with cobalamin deficiency

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Introduction

Pseudo-thrombotic microangiopathy (pseudo-TMA) is a very uncommon, clinical presentation of vitamin B12 deficiency. Patients with severe vitamin B12 deficiency may present with features mimicking TTP such as MAHA, thrombocytopenia and schistocytosis. Primary TMA syndromes include Thrombotic Thrombocytopenic Purpura, Hemolytic Uremic Syndrome, drug induced TMA, and complement-mediated TMA.(1) Pseudo-TMA responds to vitamin B12 replacement whereas Primary TMA requires plasmapheresis or monoclonal antibody therapy. Here, we report a case of pseudo-TMA in a patient with cobalamin deficiency secondary to pernicious anemia.

Case Description

A 55-year-old female presented with 5-day history of sluggishness and profound fatigue. Complete blood count showed macrocytic anemia, thrombocytopenia and leukopenia. Hemoglobin was 5.4 g/dL, MCV was 102FL, platelets were 72,000/MCL and WBC count was 3,820/MCL. Work-up revealed severe vitamin B12 deficiency at <60 pg/mL (193–986 pg/mL) and hemolysis. Indirect bilirubin was increased at 1.3 mg/dL, LDH was increased at >4000 Unit/L (84–246 Unit/L), haptoglobin was decreased at <8 mg/dL (30–200 mg/dL), and negative direct antiglobulin test. Her reticulocyte percent was 1.8% (0.5–2.5%), suggesting suboptimal BM response. Peripheral blood smear showed schistocytes and hyper segmented neutrophils. The patient was afebrile, and her creatinine was slightly elevated at 1.24. Given the presence of hemolysis, thrombocytopenia and schistocytosis, the diagnosis of TTP was a concern and therefore ADAMTS13 activity, was sent out. Vitamin B12 (1000 mcg IM daily) was initiated. Over the next 3 days, the patient's hemoglobin and platelets stabilized. On day 5, ADAMTS13 activity came back normal at 92%. Accordingly, parenteral B12 replacement was continued which resulted in cessation of hemolysis and normalization of hemoglobin and platelets. Finally, the parietal cell autoantibodies came back positive consistent with pernicious anemia

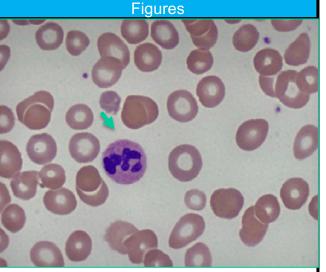
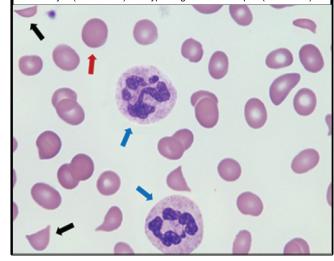


Figure A: Peripheral smear revealing hyper segmented neutrophil(Green Arrow)

Figure B: Peripheral smear revealing macro ovalocytes(Red Arrow), schistocytes(Black arrow) and hyper segmented neutrophils(Blue Arrow)



Discussion

TMA is a well-defined clinicopathological entity. Clinically it is characterized by microangiopathic hypochromic anemia, thrombocytopenia and organ damage. Pathologic characteristics include vascular damage which is manifested as both arteriolar and venous thrombosis.(2)TTP is a primary TMA syndrome with high mortality if unrecognized and untreated. Pseudo-TMA can be experienced in 2.5% of patients with cobalamin deficiency(3). A systematic review on 41 patients with Pseudo TMA highlighted the presence of schistocytes in 76% patients and etiology was thought to be multifactorial(4). However ineffective erythropoiesis and elevated homocysteine levels were thought to be the most common causes(5). Likewise, homocysteine levels were found to be elevated at 200umol/L with suboptimal BM response suggested by reticulocyte percent at 1.8% in our patient. Also, in all these patients LDH and Bilirubin levels were elevated and haptoglobin was decreased. The median LDH reported in this review was 3981units/L while patients with TTP have levels between 1407-1460(17-18), (6)LDH in our patient was reported >4000. ADAMS TS 13 was normal. Parietal cell antibodies came back positive, and she was treated with IM vitamin B12 injections for pernicious anemia with improvement in hemoglobin and platelet counts

Conclusion

Clinical picture of primary TMA syndromes can be similar to Pseudo TMA. When TTP is suspected plasma exchange should be initiated immediately. Pseudo-TMA with cobalamin deficiency does not respond to plasma exchange and require B12 replacement. An early and accurate diagnosis is important to avoid unnecessary, ineffective and potentially harmful therapy.

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Abnormal Presentation of Advanced Gestational Trophoblastic Neoplasm (GTN) with Pulmonary Artery Obstructive Symptoms and Profound Anemia

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Case Presentation

Forty-two year old female with at history of untreated Hep C, Pre-DM, anxiety, and polysubstance abuse presents with the chief complaint of bilateral lower extremity swelling and worsening dyspnea on exertion for 6 days. Initial CXR was consistent with a large R sided pleural effusion. Lab evaluation showed hemoglobin of 5.4, and an RDW of 24.8 with a differential notable for nucleated RBCs of 11%. Further laboratory evaluation showed LDH 946, haptoglobin <1, however her direct antiglobulin test was negative. Interestingly, an HbA1c obtained to workup her history of pre-DM was <2.9 which was thought to be due to an abnormally high RBC turnover. Serum BhCG of 180,000 was detected, which was at the upper limit of normal for a 6-8 week IUP. She reported having a normal menstrual period 1 month prior and denied any sexual interaction within that month at least. Pelvic US without intra-uterine gestation. CT PE evaluation showed that instead of a right sided pleural effusion, there was a large mass incasing the R lung and invading into the mediastinum and abdomen, as well as multiple metastasis in both lungs and diffuse lymphadenopathy. Imaging findings along with such elevated BhCG consistent with a GTN. After consultation with Gyn/Onc specialists she was transferred to a higher level of care with surgical consultation prior to initiating chemotherapy due to concern that after starting chemotherapy she would have massive thoracic or abdominal hemorrhage or develop tumor lysis syndrome and require intubation.

Discussion

GTN refers to a group of malignant neoplasms that consist of abnormal proliferation of trophoblastic tissue, and may follow a hydatidiform mole or a nonmolar pregnancy. Four histologic subtypes include invasive mole and choriocarcinoma (more aggressive, metastasize early, and secret high levels of BhCG) as well as placental site trophoblastic tumor(PTT) and epithelioid trophoblastic tumor(ETT) (less aggressive, generally remain localized, and do not secrete high levels of BhCG). Approximately 50 percent of cases of GTN arise from molar pregnancy, 25 percent from miscarriages or tubal pregnancy, and 25 percent from term or preterm pregnancy. Estimated incidence of GTN after a term pregnancy is 1 per 150,000 and after a spontaneous miscarriage is 1 in 15,000. Tissue diagnosis is not necessary and often avoided due to high risk of hemorrhage. Invasive mole and choriocarcinoma are both highly responsive to chemotherapy agents due to aggressive growth but also likely to develop resistance. PTT and ETT are both less aggressive, more likely to remain in situ, and are less responsive to chemotherapy often requiring surgical excision.

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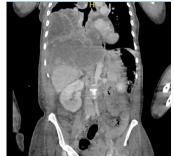
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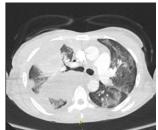
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Upper L: Initial CXR on presentation to the ED. Read as concerning for large volume pleural effusion on right. However, nodular densities in upper lung zones hint at true diagnosis. Upper R: Axial image of thorax showing advanced compression of lung, airways, and vasculature. Mid L: Initial CT AP of patient showing heavy tumor burden of left chest wall with involvement of abdomen and mediastinum.

Mid R: CT AP of patient two months in to therapy. Bottom: Example of choriocarcinoma of uterus(Not from patient)

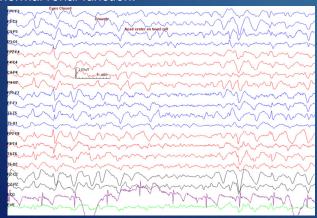


Cefepime Neurotoxicity in the setting of Normal Renal Function

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Introduction

Antibiotics are some of the most prescribed medications in the inpatient setting. Due to the familiarity we often feel with these drugs, it may be easy to overlook potentially iatrogenic side effects they may have on our patients. Although the profound benefits of treating sepsis and infection often outweigh the potential risks of antimicrobial administration, it is important to keep iatrogenic causes in mind when patients develop new problems in the hospital. Beta-lactams such as penicillins and cephalosporins have been known to cause seizures. In particular, neurotoxicity thought to be caused by cefepime has been seen in the setting of decreased glomerular filtration. Here we present a case of potential cefepime neurotoxicity in a patient with normal renal function.



Example of triphasic waves on EEG

Case Description

Patient

81yo male, history of CVA with no residual deficits, ETOH abuse (last drink 2wks ago), and hepatocellular carcinoma admitted for low back pain concerning for osteomyelitis vs metastases. He is alert, oriented to person, place, and situation, able to provide history.

Course

Empirically treated with vancomycin and cefepime (2g q8h). Within 36 hours of antibiotics, still largely at baseline but demonstrated occasional nonsensical speech, which progressed to complete disorientation, paroxysmal grimacing movements with his mouth and repetitively vocalizing the same sound by day 3 of antibiotics.

Diagnostics

Initial workup of encephalopathy only significant for hyponatremia of 131 (had been without good PO intake). Vital signs, other electrolytes, CBC, lactate, ammonia were all within normal limits, as was renal function (GFR >60). CT head showed no convincing evidence of large hemorrhage or midline shift. EEG obtained was negative for seizures, but demonstrated <u>frequent triphasic</u> waves, which is suggestive of metabolic encephalopathy and has been demonstrated in cefepime neurotoxicity.

Resolution

Patient was transitioned from cefepime to ceftriaxone. By 24 hours after his last dose of cefepime, his mental status partially improved and he was oriented to person and place, and by 48 hours, he was completely back to his baseline. The paroxysmal grimacing disappeared.

Discussion

Cefepime neurotoxicity is thought to be due to concentration-dependent GABA receptor antagonism. While most commonly seen among patients with renal impairment, a systemic review suggested cefepime toxicity in the setting of normal renal function may be more likely to occur in patients with pre-existing brain damage, possibly due to increased BBB permeation. Our patient had history of substance abuse and prior stroke. For educational purposes, it may have been interesting to obtain a serum or CSF cefepime concentration before discontinuation of the drug. Threshold cefepime trough levels to cause toxicity have been proposed in literature; one paper proposed therapeutic drug monitoring targeting troughs of <7.5 mg/Lin patients with risk factors for cefepime toxicity. Further research into this topic may be beneficial.

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Subcutaeous Panniculitis Like T-Cell Lymphoma Presenting with Hemophagocytic Lymphohistiocytosis

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Introduction

Subcutaneous panniculitis-like T-cell lymphoma (SPTCL) is a rare peripheral cytotoxic lymphoma that can clinically resemble panniculitis. It is characterized by cytotoxic T cell infiltration of skin tissue. Over the past 2 decades it has come to recognition, accounting for <1% of cutaneous lymphomas. It is a heterogeneous disease, characterized by 2 distinct immune-phenotypical entities: an αβ subtype and a γδ subtype. The αβ subtype is characterized by CD8 positive and CD4 negative neoplastic T cells, while the yδ subtype carries a worse prognosis and is characterized by T cells negative for both CD4 and CD8.

The clinical course of SPTCL varies but patients often report B symptoms, such as fever, weight loss, and night sweats. Hemophagocytic lymphohistiocytosis (HLH) has been reported to be the presenting syndrome in 37% of patients with SPTCL. Co-occurrence of HLH and SPTCL was previously thought to signify a progressive disease course with worse prognosis: however, recently published data brings this into dispute.

Case

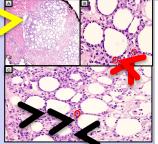
A 34-year-old Asian male with no known past medical history, presented to the emergency department, for evaluation of fever, weight loss, and skin rash of 2 months duration. The patient initially noticed firm, nontender, nonpruritic purple discoloration of his lower back that progressively extended to cover the entire lower back and bilateral flanks (Fig 1A). He also developed fevers, peaking at a maximum of 102 °F, along with fatigue, chills, night sweats, anorexia, and a total weight loss of 16 pounds. He eventually developed similar skin lesions on the right posterior neck (Fig 1B) and in the right groin.

His primary care physician initially suspected an infectious etiology, treating him with ceftriaxone and cefuroxime. Fevers persisted, he received a trial of clindamycin and trimethoprimsulfamethoxazole without improvement. He was not taking any other medications at that time. Computed tomography (CT) scans of the chest, abdomen, and pelvis were negative for lymphadenopathy or masses. Social history included a period of incarceration 2 years prior to his symptom onset, but no recent travel outside of the country. Family history was negative for malignancy and autoimmune disease. He was sexually active with one female partner, and had no previous history of sexually transmitted infections.

Diagnosis & Management



improvement. Patient's lower back before (A and after (C) treatment with 4mg/kg. Additionally patient's posterior neo nodule before (B) and after (B) and after (D) treatment with dexamethason e 12mg daily



lymphocytic infiltrate in a lobular pattern arrow. Rimming of individual adipocytes by atypical lymphoid cells is also exhibited (A,B,C). This is demonstrated b present some

A bone marrow biopsy showed normocellular marrow for age with trilineage hematopoiesis and a full range of maturation. An immunohistochemical CD68 stain performed on the core biopsy highlighted rare cells suspicious for hemophagocytes; however, interpretation was limited by the presence of high background staining. Flow cytometry immunophenotyping was performed on aspirate material and showed no evidence of hematopoietic neoplasia.

A skin biopsy was performed from the lower back and revealed an edematous dermis with an infiltrate of histiocytes and extravasation of erythrocytes concentrated around eccrine coils. There was a concentration of histiocytic and lymphocytic infiltrate in a lobular pattern in the subcutis. Adipocytes in the subcutis were rimmed by mild to moderately atypical, hyperchromatic lymphoid cells, best highlighted by CD8 (Figure 2A-C). These lymphocytes stained positive for CD3 with diminished staining of CD4. CD5 and CD8 stained greater numbers of cells than CD4 with a reversal in ratio of CD4:CD8. CD30, CD20, CD56, and periodic acid-Schiff staining was negative. CD68 highlighted the histiocytes. The delta TCR chain immunohistochemical stain was negative. while β-F1 with appropriate control demonstrated nuclear staining of the atypical lymphoid cells rimming adipocytes, favoring a diagnosis of αβ subtype SPTCL. Alcian blue demonstrated mucin in the dermis, subcutis, and in the histiocytes within the subcutis. The pattern was that of an atypical lymphohistiocytic panniculitis with dermal mucinosis and erythrophagocytosis. Erythrophagocytosis was easily identified in dermal histiocytes. These findings were consistent with a diagnosis of SPTCL.

Based on elevated ferritin, fasting triglycerides, elevated soluble IL-2, persistent fevers >38.5 °F, and the hemophagocytosis seen on skin biopsy, he was diagnosed with hemophagocytic lymphohistiocystosis and was treated with dexamethasone 10 m/m2 daily for 2 weeks. He was started on cyclosporine A 4 mg/kg with significant improvement in skin lesions. At 3-month follow-up, there was complete resolution of symptoms with cyclosporine A treatment. Near resolution of posterior neck lesions was demonstrated (Figure 1D) and improvement in the posterior back lesion (Fig 1C). Laboratory results also improved, with comprehensive metabolic panel demonstrating normalization of aspartate aminotransferase, alanine aminotransferase, and alkaline phosphatase (Table 1). Ferritin remained elevated but was trending downward (Table 1). His activated partial thromboplastin time and complements had normalized. Although his ferritin, triglycerides, and LDH remain elevated, the values were trending toward normal. His uric acid, however, has increased (Table 1).

Discussion

Patients with SPTCL commonly present with erythematous subcutaneous lesions for which the clinical course is varied. Those who present with HLH, elevated LDH levels, leukopenia, or yδ subtype have been reported to have a poorer prognosis. The diagnosis is made via skin biopsy with biopsy revealing dense subcutaneous infiltrates with a pattern of lobular panniculitis, with atypical lymphocytes rimming individual adipocytes.

The most common bone marrow abnormality in SPTCL is hemophagocytosis, as seen in our patient. HLH is a life-threatening syndrome of immune over activation. It is usually triggered by an underlying event, condition, or infection that disrupts normal activity of the immune system. HLH is diagnosed based on clinical presentation and diagnostic criteria from the HLH-2004 trial. HLH is diagnosed based on meeting at least 5 of the 8 criteria, in the setting of a compatible clinical picture. In our patient, HLH was diagnosed as he met criteria with elevated ferritin, persistent fevers, elevated fasting triglycerides, soluble IL-2, and hemophagocytosis visualized on skin biopsy

No standardized approach to the treatment of patients with HLH secondary to underlying SPTCL currently exists. Previously, many patients were treated with cytotoxic chemotherapy regimens (cyclophosphamide, vincristine, doxorubicin, and prednisolone) but several recent cases showed clinical response to treatment with oral steroids alone or a combination of oral steroids with methotrexate or cyclosporine A. This patient's case and other recently published reports demonstrate that HLH does not necessarily predict more aggressive disease course and worse prognosis. Interestingly, the treatment regimen utilized in this case showed rapid and profound clinical improvement in our patient with minimal toxicity.

Variable	Reference Range	Value (Hospitalization)	Value (3 months of treatment)
White blood cell count	4.5 -11 x 10 ³ /µL	5.1 x 10 ³ /µL	6 x 103/µL
Hemoglobin	13.5-17.5 g/dL	10.6 g/dL	11.5 g/dL
Hematocrit	40 to 51%	31.6%	33%
Mean Corpuscular volume	80-100 fL	81.4 fL	85.7 fL
C-reactive protein	<0/9 mg/dL	13.2 mg/dL	<0.05 mg/dL
Erythrocyte sedimentation rate	0-15 mm/h	73 mm/h	47 mm/h
Lactate dehydrogenase	<201 U/L	708 U/L	246 U/L
Ferritin	20-300 ng/mL	10 842 ng/mL	512.2 ng/mL
Activated PTT	24-37 seconds	43.6 seconds	37.5 seconds
Serum IL-2	≤2.0 pg/mL	1538.4 pg/mL	
Serum IL-6	175.3-858.2 pg/mL	7.1 pg/mL	
C2	1.6-4 mg/dL	4.9 mg/dL	2.7 mg/dL
C3	83-180 mg/dL	190 mg/dL	164 mg/dL
C4	18-55 mg/dL	67 mg/dL	51 mg/dL
C5	7-20 mg/dL	23 mg/dL	28 mg/dL
Total complement activity	38.7-89.9 U/mL	>95 U/mL	80.8 U/mL
AST	<45 U/L	351 U/L	24 U/L
ALT	<46 U/L	320 U/L	17 U/L
ALP	20-120 U/L	137 U/L	60 U/L
GGT	<81 U/L	131 U/L	17 U/L
Triglycerides	<150 mg/dL	209 mg/dL	195 mg/dL
Uric Acid	3.5-7.5 mg/dL	3.5 mg/dL	7.9 mg/dL

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Herpes Zoster Opthalmicus: Keep an Eye Out for Encephalitis

VOchsner Health

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Introduction

- Varicella-zoster virus (VZV) can cause primary infection as varicella (chickenpox) which typically manifests as a childhood rash.
- The virus then lays dormant within the nervous system and can later reactivate as herpes zoster (HZ) (shingles) which usually presents as a vesicular rash associated with neuropathic pain along a dermatomal distribution. It is estimated that approximately 1 million cases of HZ occur annually in the United States.
- Herpes zoster ophthalmicus (HZO), defined specifically by reactivation of the virus in the distribution of the ophthalmic branch of the trigeminal nerve (CN V1), is seen in 10-20% of HZ cases.
- Among the rarest complications seen with HZO is encephalitis (less than 0.1% of cases). Most of these cases are reported in patients diagnosed with an immunocompromising condition or those on chronic immunosuppressive therapy.

Initial Presentation

- 78 yo F w/ PMH of HTN and cervical cancer in remission presented to ED w/ acute onset L eye swelling and a periorbital vesicular, burning rash extending to the forehead (fig 1).
- Home medications: amlodipine 10mg daily.
- Afebrile, normal vital signs.
- Physical exam: Decreased visual acuity, normal mental status. otherwise unremarkable.
- · Lab work was not obtained. Imaging was not obtained.
- PO acyclovir administered for HZO & discharged home on PO acyclovir after ophthalmology consult via telephone with plan for close out-patient ophthalmology follow-up.
- · Re-presented to ED 2 days later with AMS, urinary and fecal incontinence, and unresolved left eye pain with worsening vesicular rash (fig 2).
- Pt had not obtained PO acyclovir after initial discharge nor had yet been seen in ophthalmology clinic.
- Admitted for additional workup.
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Hospital Course

- · Hospital Day 1: Ophthalmological exam showed mild ptosis, progressive vesicular rash in the V1 distribution, but negative fluorescein exam and stable visual acuity.
- CBC remarkable for pancytopenia (WBC 2.8, Hgb 10.5, PLT 109) with normal diff. Pt placed on IV acyclovir, PO gabapentin, and gentamicin drops.
- Hospital Day 3: Pt hypothermic (88.6 F), hyponatremic (127), further deterioration in mental status.
 - CXR and non contrast CTH negative. Normal lactate and TSH.
 - Elevated L eye pressures on tonometry (28), +Hutchinson's sign, development of periorbital cellulitis and posterior uveitis w/ scleritis.
 - Pt placed on IV vancomycin and IV levofloxacin for cellulitis (7 days); steroid drops for uveitis and scleritis and timolol-dorzolamide for IOP elevation.
 - Neuro workup revealed progression to acute encephalitis with negative HIV, RPR, and no identified source of immunocompromised state or immune deficiency.
 - MRI brain and orbits non-diagnostic (extracranial and soft tissue swelling in left periorbital region).
- Hospital Day 9: Improvement of mentation and vesicular rash. Worsening scleritis, uveitis, trabeculitis with associated CN VI palsy. No signs of acute retinal necrosis or other retinal pathology.
- · Hospital Day 13: Continued vesicular rash improvement, stable mentation, return of IOP to baseline, and resolution of CN VI palsy. Outpatient ophthalmology follow up arranged. S/p 14 day IV acyclovir course.
- Additional Notable Features of Hospital Course
 - · Hospital course complicated hypothermia and hypoglycemia.
 - Notable normal investigations blood cultures, U/A, CXR, CT AP W, lactate, AM cortisol, lipase, CPK, TSH, MRI brain w/wo contrast (with no hypothalamic CVA), HIV, RPR.
- · Patient was found to have multiple vitamin deficiencies (B6, thiamine, vitamin D) on a background of chronic malnutrition. BG stabilized w/ nutritional optimization.
- Persistence of pancytopenia with neutropenia development during hospital course on background of anemia of chronic disease. Normal folate, LDH, SPEP/UPEP. Peripheral blood smear: occasional droplet cells. Retic Index: hypo-proliferation. Differential diagnosis: acyclovir induced, viral induced, MDS. Outpatient heme-onc consult for bone marrow biopsy was placed on discharge due to neutropenia resolution.

Discussion

- Most HZO cases are reported in patients on chronic immunosuppressive therapy or those diagnosed with an immunocompromising condition.
- Early recognition and treatment of HZO is critical in preventing progressive corneal involvement and possible visual loss.
- Few HZO related encephalitis cases have been reported in immunocompetent patients and even fewer in patients who were already on IV acyclovir.
- · Overall, in terms of treatment, IV acyclovir is recommended for patients with retinitis, immunocompromised state, or those requiring hospitalization for sight-threatening disease.
- Our case supports a low threshold for the use of IV acyclovir vs PO in patients presenting with HZO with potential risk factors for progression to complications such as encephalitis (elderly, malnutrition, pancytopenia of initial unclear etiology).

Learning Objectives

- 1. Recognize encephalitis as a complication of HZO, particularly in the immunocompromised population.
- 2. Identify patient risk factors as indications to consider earlier IV acyclovir initiation in HZO to mitigate opthal/CNS complications.

Clinical Images







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CNS mixed fungal ball in an immunocompetent patient -A rare entity!

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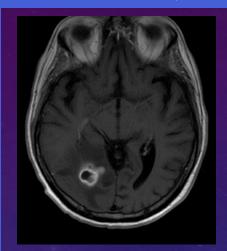


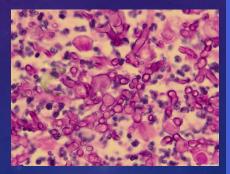
Introduction:

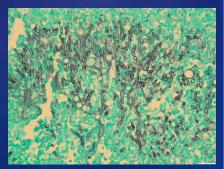
- Aspergillus species are ubiquitous fungi in the environment and are readily acquired through inhalation.
- Immunocompromised individuals are at particularly high risk of progressing to invasive fungal infections following inhalation.
- It can present with deep-seated invasive infections in immunocompromised hosts such as HIV/AIDS and acute leukemias.
- We describe here a case of CNS
 Aspergilloma and Phycomycosis resembling Mucor presenting as homonymous hemianopsia due to an occipital mass in a patient with a history of Acute Myelogenous Leukemia (AML).

Case presentation:

- Our patient is a 64 year old white male with a past medical history (PMH) of acute myeloid leukemia (AML), currently in remission who presented from his ophthalmologist with left sided visual disturbances and facial numbness.
- He was found to have retinal hemorrhages and Left sided hemianopia
- An MRI brain showed an enhancing right occipital mass measuring 2.5x2.1x1.8cm with surrounding vasogenic edema







- The initial differential diagnosis was broad, ranging from infectious, benign to malignant causes.
- Of note, he had a history of invasive pulmonary aspergillosis during his induction phase of chemotherapy for AML which was promptly treated.
- This time, he was empirically started on liposomal amphotericin B while awaiting results of the biopsy (steroids were held). Biopsy showed extensive angio-invasive fungal organisms.
- Some appeared as septate hyphae with 45 angle branching, with features of aspergillosis, while other hyphae were nonseptate with 90 angle branching, demonstrating features of phycomycosis such as Mucorales.
- The mass was completely removed during surgery and the patient was continued on Amphotericin B which was later shifted to Isavuconazole due to renal failure. Post treatment MRI of the brain and aspergillus antigen were negative for residual disease

Discussion:

- CNS aspergilloma is a rare form of invasive aspergillosis associated with high morbidity that can be difficult to diagnose and treat. The gold standard for diagnosis remains to be histopathology as most forms of imaging poorly differentiate between CNS aspergilloma and alternative infections or neoplasms.
- The combination of aspergillosis and mucormycosis in the same fungal element is exceedingly rare with only one other case published. This case shows that both diseases can be treated with surgical excision with Isavuconazole treatment.





A CASE OF COVID-19 ASSOCIATED MULTISYSTEM INFLAMMATORY SYNDROME RESULTING IN NEW ONSET HEART FAILURE IN AN ADULT Marie Bulathsinghala MD PhD¹, Rohan Samson, MD²



Department of Internal Medicine LSU Health Sciences Center¹ and Tulane University², New Orleans, LA,

Introduction

- MIS-C has emerged as a rare but serious manifestation of severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection primarily seen in children
- CDCs definition for MIS-C is (1) < 21 years of age presenting with fever, (2) evidence of inflammation by one or more markers (CRP, ESR, fibrinogen), (3) severe illness requiring hospitalization, with greater than 2 organ systems involved, (4) no other plausible alternative diagnosis, and (5) SARS-CoV-2 infection confirmed by RT-PCR, serology, or antigen testing (or, absent a positive SARS-CoV-2 test, exposure to a suspected or confirmed COVID-19 case within 4 weeks prior to symptom onset).
- The relationship of MIS-C to SARS-CoV-2 infection suggests that the pathogenesis involves post-infectious immune dysregulation.
- There are a limited number of COVID-19 related hyperinflammatory syndrome cases described in adults

Case Presentation

- A 23 year-old man with no pertinent past medical history, presented with fever, fatigue, shortness of breath, exercise intolerance, myalgias, headaches for 1 week.
- The patient had a positive COVID-19 PCR test approximately 5 weeks earlier.
- * The patient was febrile to 102.2, tachycardic, and hypotensive
- Physical exam was notable for bilateral conjunctival irritation and trace lower extremity edema.
- Labs on admit were significant for creatine of 1.67, mild transaminitis, BNP 262, Troponin 0.67, and WBC 12.8.
- Echo performed in the ED demonstrated a decreased LVEF 40-45%, global hypokinesis, and collapsible IVC.

Hospital Course

- Empirically treated with vancomycin and Zosyn for possible sepsis, however no source of infection was ever identified.
- Inflammatory markers were elevated with a LDH 252, D-Dimer 588, Ferritin 1.500, CPK 108, and CRP of 28.
- CT angiogram showed no evidence of acute cardiopulmonary/airspace disease and no pulmonary embolism.
- Patient remained febrile with worsening AKI and inflammatory markers (Fig 1 & 2)
- * Troponins peaked at 1.64 and CRP peaked at 37.
- The patient continued to worsen with no obvious source of infection with a pattern of inflammatory markers consistent with MIS in the setting of recent COVID-19 infection.
- The severity of his presentation prompted treating the patient according to MIS-C guidelines developed for children which included IVIG, IV steroids, and high dose aspirin

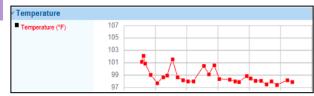


Figure 1: Fever curve

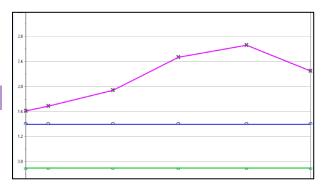


Figure 1: Creatinine

Hospital Course

- Treatment resulted in a quick resolution of his fever and improvement in his cardiac function and end-organ labs and markers.
- Follow up echocardiogram one month later demonstrated a return of normal cardiac function. Subsequent cardiac MRI was also normal.

Discussion

- MIS-C is a post-infectious phenomenon related to IgG antibodymediated enhancement of disease, not the result of acute viral infection
- Pathogenesis of MIS-C. Early infection (phase I) with SARS-CoV-2 is likely to be asymptomatic or mildly symptomatic. The pulmonary phase (phase II) is severe in adults but is mild or absent in many children. The early infection appears to trigger macrophage activation followed by the stimulation of T-helper cells which leads to cytokine release, the stimulation of macrophages, neutrophils, and monocytes, along with B-cell and plasma cell activation with the production of antibodies leading to a hyperimmune response (stage III). (Nakara et al, see figure 3)

Discussion

- Treatment includes a multidisciplinary approach involving cardiology, infectious disease, immunology, hematology, rheumatology, hospital medicine, and critical care, to guide individual patient treatment.
- Glucocorticoids are recommended as a first-line therapy for MIS-C in patients who are hospitalized, along with intravenous immunoalobulin.
- In severe disease, immunomodulatory agents such as the IL-1 inhibitor anakinra or the IL-6 inhibitor tocilizumab are also recommended

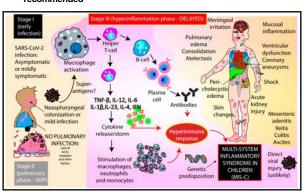


Figure 3: Infographic showing CDC criteria for the diagnosis of MIS-C. A combination of fever, evidence of inflammation, involvement of at least two organ systems, and prior evidence of SARS-CoV-2 infection are required to establish the diagnosis (Nakara et al, 2020)

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HIV-associated plasmablastic lymphoma presenting with spontaneous tumor lysis syndrome

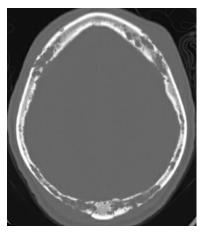
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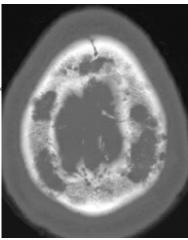
BACKGROUND IMAGES

Plasmablastic lymphoma is a rare form of diffuse, large, B-cell lymphoma (DLBCL) which is often associated with immunosuppression and Epstein-Barr virus (EBV). This disease shares features of both lymphomas and plasma cell dyscrasias. This disease is highly variable in presentation but is most frequently in the GI tract often without lymphadenopathy. Other common sites include: skin, bone, genitourinary tract. Median age of dx ~50 and more common in males (75%)

CASE DESCRIPTION

A 31 year old male with HIV/AIDS presented with severe headache and vision changes for 3 days. He reports that he has had this diffuse throbbing pain throughout his skull associated with nausea, vomiting, and bilateral blurred vision. On arrival, patient afebrile, tachycardic to 104, normotensive. Physical exam notable for exquisitely tender, palpable masses on the skull, otherwise unremarkable exam. Initial labs remarkable for WBC 19.75, Hgb 9.9, Na 122, K 7.1, Cr 4.9, Correct calcium 17.6, Total protein 13.0, albumin 3.4, CD4 count of 11, and LDH 9,195. CT head without contrast revealed diffuse osteolytic lesions throughout the calvarium and skull base with areas of focal intracranial extension concerning for malignancy. Patient admitted to the ICU for further management. Patient continued to have persistently high potassium, calcium, and elevated lactic acid despite continuous IV fluids. Uric acid elevated to 23.9 concerning for tumor lysis syndrome but patient also noted to have G6PD, and so ineligible to receive Rasburicase. Started on allopurinol. On day 3, patient underwent bone marrow biopsy revealing Kappa-Restricted B-cell population with immunehistochemistry concerning for plasmablastic lymphoma, noted to be EBV positive and with MYC/IGH fusion.





Extensive calvarial lytic lesions suspicious for a plasma cell malignancy noted in initial CT head

Patient condition continued to worsen requiring renal replacement therapy. Also begun on ART with emtricitabine-tenofovir + dolutegravir as well as inpatient chemotherapy with CODOX-M/IVAC. Patient received 13 days of therapy but continued to worsen, developing multi-organ failure. Patient made decision to discontinue all treatment and pursue comfort-focused care. He passed away

DISCUSSION

Patients with HIV/AIDS have a significantly elevated risk of developing malignancies, most commonly leukemia and lymphoma. Due its highly variable presentation, aggressive features, and unique cytogenetics, plasmablastic lymphoma can be difficult to diagnose. Treatment regimens are equally difficult to choose from, as there are no established studies due to a paucity of cases and, currently, NCCN does not have recommended treatment. R-CHOP is commonly used, but it is unclear if there is benefit of this regimen over other polychemotherapy such as EPOCH, Hyper-CVAD, or CODOX-M/IVAC.

CONCLUSION

Plasmablastic lymphoma, while rare (only 2% of all HIV-associated lymphoma), is aggressive but often treatable. In this high-risk population, this disease should be considered and identified as early as possible, especially with those who present with classic plasma cell dyscrasia features (hypercalcemia, renal failure, anemia, bone lesions) and/or oral involvement. Initial workup should include bone marrow biopsy with cytogenetics as well as urgent evaluation for chemotherapy.



Heyde Syndrome Complicated by a Dieulafoy Lesion:

A Case Report and Review of Pathophysiology

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Introduction

Hevde syndrome, a triad of aortic stenosis. Von-Willebrand factor deficiency, and gastrointestinal (GI) bleeding from angiodysplasia, is a disease of the elderly1. A Dieulafoy lesion, a specific type of angiodysplasia, is a large, tortuous, submucosal end artery that penetrates through the gastric mucosa and can cause life threatening GI bleeding2. The mortality rate for patients with a Dieulafoy lesion can be as high as 80% but is commonly unrecognized2. We present a case of Heyde syndrome complicated by a Dieulafov lesion in an elderly female and discuss the processes that may contribute to the formation of this type of lesion.

Case Report

72-year-old female with a medical history of AS. diabetes mellitus type 2, chronic kidney disease stage 3, gout, colon cancer treated with resection and colostomy bag placement 18 years prior. hyperlipidemia, hypertension, and morbid obesity presented with GI bleeding evidenced by black tarry stool in her colostomy bag for 7 days. Associated symptoms included fatigue, nausea, and decreased appetite. The patient reported no vomiting, abdominal distension, or abdominal pain. She had not recently taken any nonsteroidal anti-inflammatory drugs. Echocardiography 1 month prior to presentation showed aortic valve area of 1.24 cm2, aortic valve mean gradient of 39 mmHg, and aortic orifice peak velocity of 4.11 m/s, indicative of moderate to severe AS.

The patient was initially seen at a regional hospital where her hemoglobin (Hgb) level was as low as 6.0 g/dL. During her stay at the regional hospital, she underwent push enteroscopy, colonoscopy, tagged red blood cell (RBC) scan, and angiography. The RBC scan showed delayed images for tracer accumulation in the right colon/terminal ileum. She was transferred to our tertiary care center on day 7 of Hospitalization after receiving 5 units of packed RBCs.

On arrival at our hospital, the patient's vital signs were within normal limits. She was alert and oriented. Her colostomy bag was located on the right lower quadrant of the abdomen and contained black tarry stool. Significant abdominal findings included abdominal distension and tenderness at the periumbilical and hypogastric region on deep palpation. A large hernia protruded from the pelvic region. Cardiovascular examination was significant for systolic ejection murmur. III/VI in intensity on the Levine scale, in the aortopulmonary area. Eye examination revealed conjunctival pallor.

On admission, the patient's laboratory results were significant for Hob of 8.3 g/dL and a platelet count of 96 platelets/µL. Video capsule endoscopy showed blood in her proximal small bowel, and double-balloon enteroscopy (DBE) showed jejunal angiodysplasia that was treated with argon plasma coagulation. Repeat DBE on day 3 of hospitalization at our facility showed another small jejunal angiodysplasia that was also treated with argon plasma coagulation. Her Hob remained stable during admission. She was discharged after 3 days of hospitalization with home health. The patient continued to have dark stools after discharge. She was readmitted to the regional hospital 10 days after discharge. Repeat complete blood count showed Hgb of 6.2 g/dL. She was transferred back to our hospital the next day after being transfused another 2 units of packed RBCs. Repeat DBE showed a 1-mm focus of active bleeding in the proximal jejunum consistent with a Dieulafoy lesion (Figure). The lesion was treated with argon plasma at 1 L/min and 25 watts. Two homeostatic clips were placed to prevent further bleeding. India ink 0.3 mL was injected to tattoo the area. The patient's Hqb remained stable at 7.9 g/dL during the postoperative observation period. After 2 days, she was discharged home. At follow-up 1 year later, she reported no GI bleeding symptoms since discharge.





Image 2: Lesion after treatment with argon plasma at 1 L/min and 25 watts. (Photos by Daniel Raines, MD).



Discussion

Our patient had findings characteristic of Heyde syndrome: history of AS, persistently low Hgb. low platelets, and angiodysplasia (the Dieulafov lesion). Our patient's VWF levels were not checked, but VWF deficiency can lead to low platelets. VWF, a high molecular weight multimer, is necessary in maintaining normal coagulation homeostasis, and its role in angiogenesis has been described as well. The sheer stress of AS uncoils VWF multimer to sensitize to ADAMTS13 by exposing the bond between Tyr842 and Met8431. Furthermore VWF adhesive surfaces are exposed which aggregates with platelets causing decreased platelet count. The binding of VWF with platelets because of high shear flow and subsequent sequestration explains our patient's low platelet count3.

The process of angiodysplasia development from VWF deficiency is complex. The vascular endothelial growth factor (VEGF) family and angiopoietin-1 (Ang-1)/Tie2 system play an important role in angiogenesis, While VEGF acts as proliferator, Ang-1 acts as stabilizer.10 Their coexpression leads to the growth and stability of blood vessels. Ang-2 is the antagonistic ligand to the Tie2 receptor. Ang-2 competes with Ang-1 to bind with Tie2. and when bound, promotes vascular destabilization, growth, and inflammation, as opposed to stabilization promoted by Ang-1^{1,4}.

To promote angiogenesis, Ang-2 appears to act synergistically with VEGF. Increased VEGFR2 phosphorylation has been reported in the microvasculature of VWF-deficient mice. Therefore, decreased VWF seems to be associated with overgrown, unstable, and fragile vessels characteristic of angiodysplasia. How VWF modulates VEGFR2 is currently an area of research. One membrane and overlying epithelium, leading to localized ischemia that causes erosion and complete rupture. One hypothesis is that integrin ανβ3, a VWF ligand, binds to VWF to regulate VEGFR2.26 However, the exact signaling that follows this binding is yet to be described^{4,5}.

In vitro studies have shown that VWF-deficient endothelial cells increased the release of Ang-2 from Weibel- Palade bodies, the storage granules of endothelial cells. Increased Ang-2 can lead to unstable angiogenesis. A carbohydrate-binding protein called galectin-3 (Gal-3) plays a role in VWFdependent angiogenesis. Gal-3 can bind to VWF, VEGFR2, and integrin αvβ3, causing a complex process that has a proangiogenic effect.29,30 A Gal-3 inhibitor can reduce angiogenesis. In clinical trials, Gal-3 inhibitors exhibited a good safety profile and may be an additional treatment option for patients with recurrent GI bleed, such as our patient4,5

While the above discussion sheds some light on the process of angiodysplasia, the exact pathophysiological process that results in a Dieulafov lesion, a specific type of angiodysplasia, is unknown6. Unlike a normal arteriole in the GI mucosa, a Dieulafoy lesion tends to maintain its caliber instead of narrowing as a normal arteriole would. Because the lesion fails to taper in caliber, the arterial branch tends to penetrate the outer laver of the mucosal wall. Superficial mucosal erosion is considered the primary event leading to arterial rupture. The pulsatility of the large submucosal artery inflicts local microtrauma and ischemia to the mucosa, causing the lesion to thin over time. Initial rupture can occur from minor resistance in the arterial wall and can cause further rupture of the basement^{6,7,8,9} .

Conclusions

The exact process of how a Dieulafov lesion, a specific type of angiodysplasia, forms is unknown. This lack of knowledge is noteworthy because of the high mortality associated with Dieulafoy lesions. Because of the increasing incidence of Heyde syndrome among the elderly, documentation of potentially life-threatening complications is important. Our case provides evidence that a Dieulafoy lesion is a rare but potential complication of Heyde syndrome.

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A Rare Case of Proximal Intestinal Obstruction From

Superior Mesenteric Artery Syndrome: Anurag Gaddam MD, Jeremy Polman MD, Melissa Bunke MD



Internal Medicine Residency Program

Introduction

Superior mesenteric artery (SMA) syndrome is a rare disorder that is characterized by compression of the third portion of the duodenum due to narrowing of the angle between the SMA and the aorta. Patients typically present with intermittent postprandial epigastric pain, nausea, and bilious vomiting. We report a case of a 25-year-old female who presents with symptoms of intestinal obstruction, diagnostic evaluation revealing SMA syndrome, and successful duodeno-jejunostomy procedure.

Case Description

A 25-year-old female history of cholecystectomy presented with worsening symptoms of nausea, intractable vomiting, postprandial epigastric pain, and twenty-pound weight loss for 2 years. She had multiple hospitalizations and extensive gastrointestinal work-up in the past without any identifiable cause of her symptoms. Physical exam, labs, CT abdomen and pelvis, EGD, colonoscopy, and PillCam endoscopy were benign.

CT angiography of abdomen and pelvis demonstrated a decreased aorta-superior mesenteric artery angle of 9.1 degrees (normal 38 degrees to 65 degrees) with aorta- superior mesenteric distance of 5.1 mm (normal 10 to 28 mm), and compression of the third part of duodenum, confirming the diagnosis of SMA syndrome. She was initially managed conservatively with duodenal and gastric decompression via NG tube, fluid and electrolyte correction, body positioning, proton pump inhibitors, and nutritional support. Unfortunately, the patient had no improvement in her symptoms and subsequently required duodeno-jejunostomy. Upon completion of the procedure, the patient had complete resolution of her presenting symptoms.

SUperior Mesenteric Artery (SMA) Compressed Duodenum Abdominal Aorta Absence of Mesenteric Fat

Discussion

The diagnosis of SMA syndrome requires a high clinical suspicion in order to obtain abdominal angiography in patients with persistent symptoms despite negative endoscopic and conventional radiographic findings. Diagnosis requires an aortomesenteric angle between 6-22 degrees and an aorto-mesenteric distance between 2-8 mm on CT angiography. Patients should initially be managed with conservative therapy. However, if symptoms fail to improve or increase in severity, surgery is the definitive treatment. Surgical options include duodeno-jejunostomy, gastro-jejunostomy, and Strong's procedure. Duodeno-jejunostomy is the procedure of choice, as it carries a 90% success rate.

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A Rare Case of Aerococcus Viridans Endocarditis

Baton Rouge General

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Internal Medicine Residency Program

Introduction

Aerococci Viridans(A. Viridans) is an aerobic, gram positive coccus which is an extremely rare cause of infective endocarditis. We report a rare case of an elederly male diagnosed with Aerococcus viridans Infective Endocarditis.

Case Description

78-year-old male with a history of transcatheter aortic valve replacement (TAVR) in 2018, type 2 Diabetes Mellitus Hypertension presented to the Emergency Department(ED) with a 4-week history of high fevers, chills, rigors, and nausea.

On Initial presentation, the patient was tachycardic, afebrile with normal physical examination findings and no identifiable heart murmurs/skin lesions. Initial blood work revealed normal white cell count of 10,000 and elevated ESR of >120 mm/hr, CRP of 11 mg/l. Preliminary blood cultures grew non-hemolytic *Streptococcus* and empirically started on ceftriaxone.

Final blood cultures revealed *A. Viridans* as the cause of his bacteremia.

A transthoracic echocardiogram (TTE) performed initially was normal. Due to high clinical suspicion of IE in the setting of mechanical aortic valve, a TEE was performed revealing 1.0 cm vegetation on the ventricular side of the mitral valve. Patient was treated with intravenous Ceftriaxone 2gm daily for a duration of six weeks and showed clinical improvement.

Discussion

Aerococcus Viridans is a rare cause of bacteremia and Infective Endocarditis(IE) in humans.

Aerococcal Infective Endocarditis is seen predominantly in people with underlying comorbidities including urologic/prostatic disease, diabetes mellitus, malignancy, immunosuppression and advanced age. The left-sided mitral and aortic valves are the most frequently impacted valves and follow a indolent/subacute course.

Treatment for *A. Viridans* should be started empirically after isolation on blood cultures, and then narrowed based on susceptibility.

Aerococcus is generally sensitive to penicillin and is therefore usually treated with penicillin.
Four to six weeks is the ideal recommended duration of treatment. Delayed diagnosis is a leading cause of poor prognosis and increased morbidity and mortality in these as it results in rapidly enlarging vegetation, emphasizing the need for early detection and initiation of antibiotic therapy.

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A Rare Case of Giant Coronary Artery Aneurysm with Acromegaly

Baton Rouge Genera

Internal Medicine Residency Program

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Internal Medicine Residency Program

Introduction

Giant coronary artery aneurysm is a rare disease with a prevalence of 0.3 to 5% in the USA. We present an unusual case of a giant coronary artery aneurysm believed to be associated with acromegaly

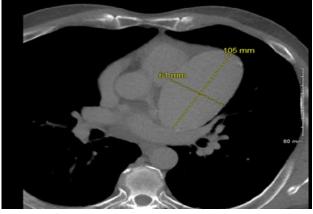
Case Description

We present a 48-year-old Caucasian male with a past medical history of acromegaly, treated by trans-sphenoidal resection of the pituitary gland presented to the emergency department with acute onset of chest pain and shortness of breath. Initial lab work revealed an elevated troponin and the electrocardiogram showed ST-segment elevation in the anterior leads. He underwent an immediate cardiac catheterization which revealed a hugely aneurysmal proximal left anterior descending artery measuring 39 * 76mm. This patient's acute myocardial infarction was attributed to the aneurysmal dilated LAD(left anterior descending) artery and thrombus formation due to sluggish flow. No coronary atherosclerosis was identified, and a vasculitis workup was negative. The patient was not considered a candidate for coronary bypass due to minimal distal LAD target. He was medically managed with Dual antiplatelet therapy and oral anticoagulation.

Follow up echo/CT angiogram of coronaries after 6 years showed an ejection fraction of 40 % and LAD dimensions 60*100mm, large enough to be visible on a Chest X ray.

Figures and Labs

CT angiogram of the coronary circulation showing Giant aneurysm of the left main coronary artery(arrow)



Giant left main coronary artery aneurysm measuring 10.5 cm *6.1cm

Discussion

Acromegaly is a rare endocrine pituitary neoplasm that results in the excess release of Growth hormone. It is usually associated with cerebral and pelvic aneurysms among other cardiovascular complications including hypertension, and cardiomyopathy. As far as we know, this is the only case of coronary aneurysm associated with acromegaly and should be considered as potential complications of this disease. Common causes of coronary artery aneurysms include atherosclerosis, Kawasaki disease, Takayasu arteritis, Marfan's, Ehlers-Danlos, Turner syndrome, and cocaine abuse. Long-term follow-up with echocardiogram and coronary angiograms provides serial size data, but surgical therapeutic options are very limited.

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A Rare Case of Adult Type ALCAPA Syndrome

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Internal Medicine Residency Program

Introduction

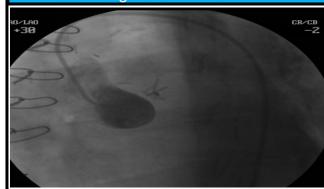
ALCAPA Syndrome (Anomalous left coronary artery from the pulmonary artery) is a very rare congenital heart disease where the left coronary artery (LCA) originates from the pulmonary artery. It affects 1 in every 300,000 live births in the USA and if left untreated, has a 90% mortality within the first year of life. We present a rare case of a 42-year-old woman with ALCAPA Syndrome who presented with Angina at the age of 32 and underwent successful surgical repair.

Case Description

A 32-year-old female with a past medical history of hypertension, non-insulin-dependent type 2 diabetes mellitus, hyperlipidemia, with no family history of heart disease presented with complaints of exertional angina for 2 years and had a normal physical examination. Further workup with coronary angiography showed an anomalous left coronary artery arising from the pulmonary artery. Coronary angiographic findings also revealed a large voluminous right coronary artery (8 mm in diameter) with extensive collaterals to the left coronary artery. Transthoracic Echocardiogram revealed an ejection fraction of 40 % with mild left ventricular hypertrophy. EKG showed nonspecific T wave abnormality in lateral leads. In Extremely rare cases who live to adulthood(adult type) as in our patient, have extensive collateral circulation from the right coronary artery and develop chronic myocardial ischemia resulting in arrhythmias, sudden cardiac death and congestive heart failure.

Our patient underwent surgical repair with Coronary button transfer procedure without any perioperative complications. After 10 years of Post repair, our patient is asymptomatic with Echo findings EF 45 %.

Figures and Labs



Left coronary artery not visualized on left heart catheterization



Right coronary artery(arrow) is large and voluminous with collaterals

supplying the left ventricle before surgical correction of ALCAPA



Left coronary artery -post surgical correction(red arrow)

Discussion

ALCAPA Syndrome is a rare congenital anomaly which develops before birth where the left coronary artery originates from the pulmonary artery. The extent of myocardial ischemia is determined by the development of collaterals between the right and left coronary artery. Diagnosis is usually established with coronary angiography, cardiac MR imaging, multi detector CT angiogram. Surgical correction with Coronary button transfer procedure has excellent long-term results.

References

A rare case of spontaneous diabetic myonecrosis

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Introduction:

Spontaneous diabetic myonecrosis is a rare complication (<270 published reports) of diabetes mellitus.

Case:

A 30 year-old female with a history of type 1 diabetes, hypertension and ESRD reported one day of abdominal pain, nausea, and vomiting, and six days of right thigh pain in the absence of trauma. The patient was diagnosed with diabetic Ketoacidosis which resolved with an insulin drip. She continued to report right thigh pain and weakness. On examination, the patient's right thigh was tender to palpation, along with restricted hip and knee range of motion secondary to pain. An X-ray of the right femur revealed soft tissue swelling without any gas in the tissue, and additional laboratory studies showed the following: CK 165 (20-180), Aldolase 7.8 (<7.7), LDH 263 (110-260), ANA negative, ESR 9, CRP 1.66, RA factor <10, Hb A1C 11.1. TSH, Folate, Vitamin D, Vitamin B12 were normal. Right lower extremity ultrasound showed myositis versus infarction of the muscle without concerns for compartment syndrome or DVT. An MRI showed myonecrosis of the vastus medialis muscle. The patient was diagnosed with spontaneous diabetic myonecrosis and was started on aspirin along with ibuprofen. She had mild subjective improvement of pain after 3 days of NSAIDs and was discharged with outpatient follow-up.

Discussion:

The diagnosis of spontaneous diabetic myonecrosis should be considered when evaluating a diabetic patient with muscle pain. Because it is thought to be related to vascular complications of diabetes, type 1 diabetics present at a younger age than type 2 diabetics. Muscles of the thigh, and posterior calf group are most commonly affected. Physical examination can vary, however, tenderness to palpation, redness, swelling, and weakness are more common findings. Diagnosis is based on exclusion of other causes, including trauma. Though biopsy is the gold standard for diagnosis, it is usually not required. Complications include recurrence and compartment syndrome. Treatment is usually with aspirin and NSAIDs and full recovery can take days to months.

YOchsner Health

Left-Sided Weakness: Not Just a Neural Problem

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Introduction

- Rhabdomyolysis is a clinical entity that results from the breakdown of skeletal muscle resulting in the release of intracellular components²
- The typical presentation includes muscle weakness and pain, pigmenturia, and elevated serum creatinine²
- Muslce weakness typical presents as diffuse or within localized muscle groups³
- At-risk populations include morbidly obese, post-surgical, chronic lipid-lowering agent use, athletes, body builders, crush injuries among others³

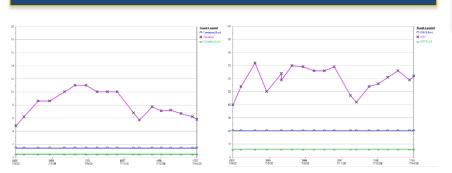
Case Information

- 41-year-old Hispanic male with PMH of anxiety presented to the ED with left-sided weakness
- Last known normal was the night prior to admission
- Physical exam revealed a well-built male with flaccid weakness of left upper and lower extremities with swelling and muscle tenderness
- CTA head and neck were negative for acute pathology
- Initial blood work revealed leukocytosis, polycythemia (Hb 18g/dl), elevated sCr (4.8 mg/dL), elevated BUN (40mg/dL), hyperkalemia (5.8 mEq/L), hyperphosphatemia (5.1 mmol/L, and transaminitis (AST 1,582/ALT 428)
- UA showed 2+ proteinuria, 3+ occult blood, 2RBC/hpf, granular casts
- CPK was >100.000

Clinical Course

- Detailed history revealed anabolic steroid use with Sustanon, Dianabol, Testmax, and Masteron injected into gluteal muscles
- Patient received 1L NS and 500mL LR boluses and started NS at 75mL/hr prior to admission to the medical service
- Maintenance fluids were increased to 150mL/hr with 300mL UOP during first 24 hours
- Nephrology was consulted for AKI/ATN, oliquria, and acidosis
- Initiated on hemodialysis on hospital day #2, Bumex 1mg BID on hospital day #3. IVF changed to NS with bicarbonate on hospital day #4 and oral bicarbonate supplementation was started hospital day #5
- sCr peaked on hospital day #4 and continued to improve. CPK, Hb, AST, ALT trended down with IVF. UOP improved to 3L per day during polyuric phase
- Hemodialysis was discontinued on hospital day #6
- IVF, diuretics, and bicarbonate supplementation were discontinued on hospital day #8
- Muscle weakness and pain improved throughout admission and resolved prior to discharge. MRI could not be completed in-patient due to body habitus
- Patient was discharged on hospital day #9 to follow up with his PCP with a plan to continue monitoring of renal function and outpatient MRI

Results



Serum creatinine trend Reference range included Serum BUN trend
Reference range included

Discussion

- We present an uncommon presentation of rhabdomyolysis with unilateral weakness associated with chronic anabolic steroid use, intended to increase muscle gains and total weight lifted, and requiring hemodialysis
- Rhabdomyolysis was not associated with acute physical activity in this case, unlike previous reports of steroidinduced rhabdomyolysis¹
- This case highlights two important takehome points:
- -Risk of unsupervised anabolic steroid
- Importance of early and accurate rhabdomyolysis diagnosis
- The use of anabolic steroids and the associated rhabdomyolysis caused acute renal injury, a potentially lifethreatening consequence requiring hemodialysis

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Balancing the fine act of anticoagulation in renal vein thrombosis with concomitant ITP : A management challenge !

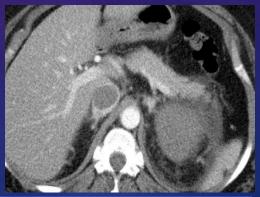
Suma Chennapragada, Shahzeem Bhayani

Department of Internal Medicine, Louisiana State University Health Sciences Center, Shreveport, LAEVEPORT

Introduction:

- The most common causes of renal vein thrombosis include nephrotic syndrome, blunt trauma, renal transplant and underlying renal malignancy.
- Immune Thrombo-cytopenic purpura is a rare cause of renal vein thrombosis.
- Thrombotic complications in ITP are being increasingly recognized (8%) and are proposed to be due to an inherent effect of ITP/ treatment with steroids and IVIG
- We present here a case of ITP with renal vein thrombosis which posed a management challenge to balance anticoagulation in the setting of severe thrombocytopenia





Discussion:

The diagnosis and management of Renal Vein thrombosis poses several clinical challenges particularly for our patient who has a concomitant nephrotic syndrome and chronic thrombocytopenia. A careful review of underlying risk factors for hypercoagulability is quintessential to not only guide management but treat the underlying risk for further thromboembolic events. Careful management of thrombosis with anticoagulation while keeping the platetet counts stable is challenging but crucial and it needs multidisciplinary care.

Case report:

- A 43-year-old female with a Past medical history of HIV, hypertension and ITP (re-fractory to steroids and on eltrombopag)presented with complaints of L sided flank pain.
- Upon admission, her labs showed clumped platelets which appeared to be decreased. Urinalysis showed 3+ proteinuria, 2+ occult blood and 23 RBC, no RBC/WBC casts.
- CT A/P showed extensive thrombosis within the left renal vein extending into the IVC .Patient was deemed to not be a surgical candidate due to borderline platelet counts. Platelet counts were 50,000 on the next day.
- Half dose enoxaparin was started with close monitoring. Counts dropped from 50,000 to 30,000 while on eltrombopag and hence she was started on rituximab. Platelet counts slowly trended up while on rituximab. However after 2 days she developed sudden onset chest pain, tachycardia and shortness of breath. Non contrast CT chest was done as the patient's renal function was compromised and it showed airspace opacities which could be related to pulmonary embolism.
- Patient was transitioned to a heparin drip and IVIG was added which showed appropriate response to > 50,0000 and patient's symptoms improved. Patient was discharged on enoxaparin with a follow up to haematology.



A rare case of Clostridium difficile Perisplenic Abscess after Splenic Embolization.

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1: Department of Internal Medicine, Louisiana State University Health Sciences Center, Shreveport



INTRODUCTION

- · Clostridium difficile is a colonic pathogen known to cause pseudomembranous colitis.
- Extracolonic infection with this organism, particularly perisplenic abscesses are rare with only few reported cases in literature.
- We report here a rare and unusual case of Clostridium difficile perisplenic abscess in a 54-year old female with liver cirrhosis after a splenic artery embolization.
- The patient underwent a Computerized Tomography scan to screen for Hepatocellular Carcinoma after diagnosis of Liver Cirrhosis. She was incidentally found to have a splenic artery aneurysm.
- She underwent a splenic artery aneurysmal embolization along with partial splenic embolization and balloon-occluded retrograde transvenous obliteration of gastric varices.
- A month after the procedure, the patient presented with abdominal pain and was found to have a perisplenic abscess.
- The fluid was aspirated and grew Clostridium difficile on cultures. The patient was subsequently treated with Metronidazole for two weeks until the abscess resolved.
- This case emphasizes the rarity of splenic abscess caused by Clostridium difficile and discusses possible treatment options.

CASE PRESENTATION

- A 54-year-old female patient presented to the Gastroenterology Clinic at a Major Academic Hospital in North-West Louisiana for care of Liver Cirrhosis after recently relocating from Oklahoma.
- The patient was diagnosed with Liver Cirrhosis due to Hepatitis C virus (HCV) infection and alcohol abuse when she was 47 years old. She completed treatment for Hepatitis C genotype 1A in Oklahoma with Ribavirin and Interferon, however she did not respond to treatment.
- She reported that she would drink 12-24 beers a day for over 15 years, however she stopped when she was diagnosed with liver cirrhosis.
- She underwent a Computerized Tomography scan (CT) triple phase of the liver to screen for hepatocellular carcinoma which was consistent with liver cirrhosis with portal hypertension. It also detected a thrombus in the portal vein and a 2 cm splenic artery aneurysm.
- A consultation with an Interventional Radiologist was sought due to presence of the splenic artery aneurysm who recommended embolization.
- The patient underwent the splenic artery aneurysmal embolization along with partial splenic embolization and balloon-occluded retrograde transvenous obliteration of gastric varices.
- She tolerated the procedure well without any immediate complications and was discharged on Amoxicillin/Clavulanic acid 500-125 mg every eight hours for five days as post-procedure antibiotic prophylaxis.
- A month after her procedure, the patient presented to the Emergency Room (ER) with complaints of sharp/stabbing left sided abdominal pain.
- A CT was performed which showed a left sided cystic mass arising from the perisplenic region measuring 16 x 14 x 18 cm which was concerning for a perisplenic abscess versus a hematoma (figure 1).
- Patient's laboratory values (complete blood count, liver and renal function tests) were within normal limits.
- Interventional Radiology performed an aspiration of the fluid cavity which yielded 1500 ml of hemorrhagic fluid. A peritoneal drain was left in place due to large volume of fluid for continued draining.

- The fluid was sent for culture and sensitivity which later grew Clostridium difficile. Blood cultures remained negative. The patient did not have any diarrhea or fevers prior to this admission, and her white blood cell (WBC) count remained normal.
- Infectious Disease consultation was sought and they recommended initiating intravenous (IV) Metronidazole 500 mg every 6 hours for two
 weeks.
- Patient had a Peripherally Inserted Central Catheter (PICC line) placed for IV access and was discharged home to complete the course of IV antibiotics.
- The patient was inconsistently compliant with her IV Metronidazole, and returned to the ER five days after discharge with vomiting and abdominal pain.
- A CT scan was performed which showed interval decrease in size of the splenic fluid collection (figure 2), however showed increase in the
 amount of free peritoneal fluid consistent with ascites.
- She underwent a paracentesis with removal of 3.8 liter of peritoneal fluid. The peritoneal fluid was negative for signs of peritonitis. A sinogram of the peritoneal drain showed that the drain was positioned correctly in the perisplenic fluid cavity and was exchanged and later removed when drainage stopped.
- A blood culture was collected which grew gram positive bacteria, so she was started on Vancomycin. Antibiotic was later de-escalated to Nafcillin after the cultures grew Methicillin-Sensitive Staphylococcus epidermidis (MSSE) in all four blood culture bottles collected. IV Metronidazole was continued for treatment of Clostridium difficile abscess.
- She subsequently developed an acute kidney injury (AKI). A Urine Wright stain showed the presence of eosinophils which confirmed antibiotic related acute interstitial nephritis. Patient's antibiotics were switched to Levofloxacin for MSSE bacteremia and Metronidazole was continued to complete the two-week duration for C. difficile abscess.
- She also received IV fluids and patient's renal function subsequently improved, however the anasarca worsened.
- She was started on treatment for decompensated liver cirrhosis with Furosemide and Spironolactone. She was subsequently discharged when she was medically stable.
- Eleven days after being discharged, the patient returned to the ER, now with worsening ascites, grade 2-3 hepatic encephalopathy, large left sided pleural effusion (deemed to be transudative). The patient went into hypoxic and hypercapnic respiratory failure requiring Bilevel Positive Airway Pressure and later, endotracheal intubation and mechanical ventilation.
- The patient had an episode of hematemesis leading to drop in blood pressure requiring vasopressor support with Norepinephrine. Due to her
 poor clinical status, EGD could not be performed to control the bleeding. She was deemed to in decompensated liver failure with a ChildPugh score of 15 points (Class C) indicating very severe liver disease and 1-to-5-year survival of 50%.
- Overall poor prognosis was discussed with the patient and she elected to go home on Hospice Care. She succumbed to her illness after 21 days.

COMPUTERIZED TOMOGRAPHY IMAGES

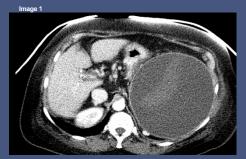




Figure 1: Computerized Tomography image showing hypodense fluid filled cavity in periplenic area measuring 16 x 14 x 18 cm. Figure 2: Computerized Tomography image done ten days after percutaneous drainage of perisplenic fluid collection showing decrease in size of cavity.

DISCUSSION

- Clostridium difficile is one of the most common nosocomial infections causing around 3
 million cases of diarrhea and colitis per year. ⁽¹⁾
- Clinical manifestations of Clostridium difficile infection vary widely. It can cause asymptomatic carriage state, diarrihea (with or without colitis), fulminant colitis, toxic megacolon, recurrent disease (seen in up to 25% of patients). (2)
- Unusual presentations include protein losing enteropathy, extracolonic involvement in the form of appendicitis, small bowel involvement and rarely cellulitis, soft tissue infections, bacteremia, reactive arthritis and abscesses.
- The incidence of extra-intestinal Clostridium difficile infections range anywhere from 0.17%-0.6% of all Clostridium difficile infections. (3)(4)
- As per one retrospective review to identify extra intestinal Clostridium difficile infections, the incidence of abdominopelvic infections was found to be 25%, blood stream infection was 11%, wound infections was 3%, lung infections was 3%, (4)
- The causes of extraintestinal infections include health care exposure (85%), antimicrobial exposure (88%), proton pump inhibitor use (82%), gastrointestinal surgery (96%), intestinal Clostridium difficile infection (45%), malignancy and inflammatory bowel disease. (3)
- The association with previous gastrointestinal surgery postulates that anatomical disruption of the gut barrier leading to bacterial translocation and seeding play a role in Clostridium difficile extraintestinal infections.
- Another reported underlying causative factor was alcohol abuse. (5)
- It hypothesized that dysbacteriosis caused by alcohol use predisposes to the overgrowth of Clostridium difficile in the intestine and thereby leading to extraintestinal scread
- There are three reported cases with similar presentations, the most likely pathological mechanisms of infection have been intestinal perforation after infection or leakage after surgery. (%7/8)
- A compilation of the activity of various drugs against Clostridium difficile showed 100% susceptibility with Metronidazole, vancomycin, ticarcillin, piperacillin-tazobactam and amoxicillin-clavulanate.
- Intravenous versus oral vancomycin for these infections has not been studied very well, however intravenous vancomycin has been used for most cases of Clostridium difficile

bacteremia and abscesses

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A NOT-SO -TYPICAL CRISIS -HEPATIC SEQUESTRATION, A RARE PRESENTATION WITH HIGH MORTALITY IN SICKLE CELL DISEASE!

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Introduction:

- Liver involvement in sickle cell disease can range anywhere from benign hemolytic hyperbilirubinemia to life-threatening hepatic sequestration crisis.
- Management of each of these ends of the disease spectrum varies drastically and hence it is important to recogniZe and distinguish between them.
- We present here a case of sickle cell hepatic crisis presenting as acute direct hyperbilirubinemia with an excellent response to conservative management.

Discussion:

- The incidence of Sickle cell hepatic crisis is around 10% in patients with Sickle Cell disease.
- They usually present with right upper quadrant pain, nausea, vomiting, jaundice and tender hepatomegaly
- Sickle cell hepatic crisis is treated with IV hydration and analgesia.
- In contrast, hepatic sequestration crisis presents with rapidly increasing hepatomegaly and a proportionately falling hematocrit and blood transfusion to prevent symptomatic anemia. Careful clinical monitoring for degree of hemolysis and increase in hepatic size is essential to distinguish between both entities and treat them appropriately

Case presentation:

- A 32 year old AAM with a past medical history of Sickle cell disease (HbSS), seizure disorder presented with right upper quadrant abdominal pain, nausea and vomiting.
- His labs showed leukocytosis with a WBC count of 14.32k/uL, anemia with a Hb of 8.1g/dL, thrombocytosis with PLT of 597k/uL.
- Bilirubin was elevated to 4.9 mg/dL on presentation. Hemolysis labs were elevated and were comparable to baseline. He was started on IV pain medications, IV fluids and incentive spirometry.
- However, within the next 2 days the total bilirubin increased to 25 mg/dL (direct component of 20 mg/dL) with an associated Hb drop from 8.1 to 6.9 g/dL. AST, ALT, ALP also trended unto 3-5 times the upper limit of normal.
- Hepatobiliary work up was ordered which came back negative. Ultrasound abdomen showed gallbladder sludging, MRCP was negative for biliary obstruction. Patient was continued on conservative management after which his pain improved and liver chemistries trended down. He was discharged in stable condition with a HemOnc follow up for periodic partial exchange transfusions.

LUNG NODULES EXPLAINED BY A REMOTE HISTORY OF DRY EYES!

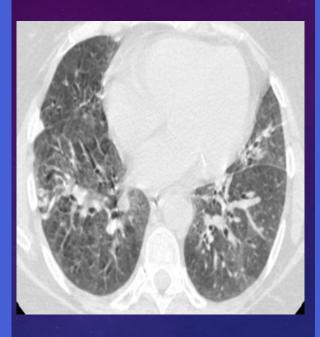
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Introduction:

- Lung nodules are a rare presentation of Sjogren's syndrome.
- Ruling out malignancy and other infectious causes is of prime importance in recognizing autoimmune causes of lung nodules.
- We present here a rare case of pulmonary nodules which were thought to be related to malignancy; however, a remote history of dry eyes revealed the cause to be related to Sjogren's syndrome.



Discussion:

- The prevalence of pulmonary involvement with Sjogren's syndrome ranges from 10-20%.
- Interstitial lung disease patterns which are usually seen include non-specific and usual interstitial pneumonia, nodular lymphoid hyperplasia, and nodular amyloidosis.
- Nodular lesions can mimic metastatic malignant lesions and thus a thorough exploration of the patient's history and review of scans is needed to reach the diagnosis.

<u>Case presentation:</u>. 57-year-old female with past medical history of cirrhosis of the liver, HCV (completed treatment), pancytopenia presented to the hospital due to shortness of breath, productive cough, and chest pain. Imaging showed bilateral lung nodules which were present since the past 2 years. They were cystic, ground glass in nature, with the largest nodule measuring up to 13 mm. The lesions were characterized as infectious/inflammatory/malignant. Patient was being followed by pulmonology and was awaiting a lung biopsy .She had two admissions over the past year for obstructive pneumonia.

Upon admission, patient's respiratory status was stabilized, and workup was started for an acute infectious process. Repeat CT chest showed worsening of the bilateral lesions and interval compression fractures of T7 and T8 raising our concerns for malignancy. Tuberculosis was ruled out and patient was started on broad spectrum anti-biotics. Her sputum cultures grew methicillin-resistant staphylococcus aureus (MRSA) and pseudomonas aeruginosa. Bronchoscopy showed reactive bronchial cells with macrophages with no evidence of malignancy. Detailed medical chart re-view noted treatment for dry eyes in the past suggesting possible autoimmune etiologies. In the meantime, labs were positive for ANA, SSA, and SSB antibodies.

Rheumatology was consulted and agreed that findings could be related to Sjogren's syndrome; hence, she was started on steroids with dramatic improvement .



Blame It on the Virus: A Rare Case of Epstein-Barr Virus Induced Autoimmune

Hepatitis in a Young Adult Chad Kaplan, MD; Jeremy Polman, DO



Internal Medicine Residency Program Baton Rouge General Internal Medicine Residency Program Internal Medicine Residency Program

Introduction

The Epstein-Barr virus (EBV) has been cited as one of the many potential triggers of autoimmune related diseases. Although rare, there have been documented cases of acute EBV infection in relation to autoimmune hepatitis (AIH). Here, we present one such case.

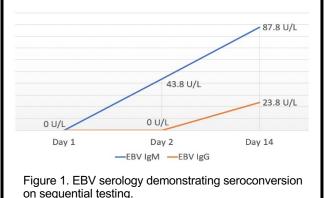
Case Description

A 19-year-old Caucasian female with a remote history of infectious mononucleosis presented to the emergency department with nausea, poor appetite, and myalgia for the past week. She denied sore throat, rash, or weight loss. Physical exam revealed scleral icterus and splenomegaly without lymphadenopathy. Laboratory studies revealed AST 1014 U/L, ALT 1027 U/L, ALP 243 U/L, and total bilirubin 2.3 mg/dL. CT abdomen with IV contrast demonstrated an enlarged spleen (21.5cm) without liver abnormality. Initial extensive hepatic evaluation (see Table 1) was unrevealing. EBV IgM and IgG titers were undetectable. Repeat EBV IgM on Day 2 was slightly elevated at 43.8 U/ml while IgG remained undetectable. After receiving supportive care for five days, liver enzymes gradually improved and the patient was discharged to home. Two weeks later, the patient presented with similar symptoms and transaminitis. EBV IgM titer was 87.8 U/mL and EBV IgG was now detectable at 23.2 U/mL (see Figure 1). A liver biopsy showed lobular hepatitis with bridging necrosis and abundant plasma cells suggestive of autoimmune hepatitis. EBV was notably absent from biopsy. Prednisone 40mg daily was started empirically with gradual improvement in transaminitis. The patient was evaluated one month after discharge, at which point the liver enzymes had returned to normal. Azathioprine 50mg PO twice daily was started with concomitant Prednisone taper.

Figures and Labs

Hepatitis A Virus IgM	Non-reactive
Hepatitis B Virus Surface Antigen	Non-reactive
Hepatitis B Virus Core IgM Antibody	Non-reactive
Hepatitis C Virus	Non-reactive
Ferritin	9.1 ng/mL
Ceruloplasmin	23.3 mg/dL
Alpha 1 Antitrypsin	102 mg/dL
Anti-Smooth Muscle Antibody	Negative
Mitochondrial M2 Antibody	Negative
HSV 1, 2	Negative
C-ANCA	Negative
P-ANCA	Negative
Atypical P-ANCA	Negative
HIV	Negative
Parvo IgG, IgM	Negative
Liver Kidney Microsomal Antibody Type 1	3.9 mg/ml
Direct Coombs Test	Negative
ANA	Negative
TTG IgA, IgG	Negative
Antigliadin IgA, IgG Antibody	Negative

Table 1. Extensive work-up for transaminitis.



Discussion

As demonstrated by this case, the history of EBV was a major consideration which prompted serial testing. Although the patient had a clinical history of mononucleosis, initial serological evaluation for EBV IgG was undetectable. Ultimately, the patient seroconverted from acute infection with EBV IgM positivity followed by EBV IgG positivity two weeks later. The pathology findings on liver biopsy were consistent with AIH, and it is suspected that acute EBV infection triggered AIH in this patient. A particular viral trigger for AIH has not been established, but there may be an antigen that provokes autoimmune dysfunction in genetically susceptible individuals. Serial testing of EBV titers may also be valuable in patients that are seronegative in the initial evaluation. Ultimately, the patient was diagnosed with AIH which was confirmed by successful immunosuppressive treatment and return of transaminases to normal level.

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A Case of Acute Pancreatitis Caused by COVID-19 in an ESRD Patient

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Introduction

Coronavirus disease 2019 (COVID-19) has created a pandemic which is ravaging the world. Its symptoms range from asymptomatic to severe and possibly death. While the primary organ system affected is the lungs, there are studies of systemic effects including manifestations in the gastrointestinal tract. We present a case of COVID-19 induced pancreatitis in an end-stage renal disease patient.

Case

A 32-year-old African American male with a past medical history of ESRD, HIV/AIDS, and hypertension presented with epigastric abdominal pain, vomiting, and diarrhea for 2 days. His lipase was found to be elevated and was diagnosed with pancreatitis. Patient was also incidentally found to be COVID-19 positive but did not require oxygen.

His medications included Tenofovir, Ritonavir, and Rilpivirine of which Rilpivirine was recently added. Infectious diseases was consulted who stated that the antiretroviral medications were unlikely to be the etiology. He was treated with aggressive intravenous fluids and pain control and the epigastric pain resolved.

Discussion

One of the more commonly affected extrapulmonary systems for COVID-19 is the gastrointestinal system but symptoms are usually nausea, vomiting, and diarrhea. Our patient presented similarly but also had pancreatitis. The patient did not have any alcohol use, hypertriglyceridemia, or gallstones. There are a few case reports of COVID-19 induced pancreatitis but not in ESRD patients. [1][2][3] In this scenario, the patient was on antiretroviral therapy however infectious diseases did not believe those medications to be the cause of pancreatitis. Drugs causing pancreatitis are split into classes (I-IV), based on the evidence of causing pancreatitis, with most cases of pancreatitis caused by classes I/II. However, the only medication the patient was on that is included in these classes was Ritonavir which is class IV. Ultimately, the treatment for COVID-19 pancreatitis was the same as other etiologies of pancreatitis and consisted of intravenous fluid, bowel rest, and pain control. De-Madaria et al postulated that the there should be an increase in idiopathic cases of acute pancreatitis if it is indeed caused by COVID-19, The following support that.[3] According to a retrospective cohort study analyzing 11,883 hospitalized patients with COVID-19 from 12 hospitals in the USA, there were 32 cases of acute pancreatitis, yielding a point prevalence of 0.27%, 69% of them idiopathic. [4] A retrospective study involving more than 63,000 patients with COVID-19 from 50 Spanish emergency rooms before hospitalization reported a frequency of acute pancreatitis of 0.07%. [5] Refer to Table 1 for common typical and atypical symptoms of COVID. We recommend a low threshold for diagnosing extrapulmonary manifestations of COVID-19.

Table 1: Typical and Atypical Clinical Features of COVID-19 referenced from					
Typical Features	Occurrence (%)	Atypical Features	Occurrence (%)		
Fever	88	Neurologic	50		
Fatigue	70	Muscle pain	29		
Dry cough	68	Skin rash	20		
Anorexia	40	Sore throat	17		
Myalgia	35	Psychiatric	14		
Dyspnea	31	Headache	12		
Sputum production	27	Rhinorrhea	<7		
Anosmia and dysgeusia	19-34	Chest pain	5-50		
Diarrhea	10	Pseudochilblain	<5		
Abdominal pain	9	Conjunctivitis	<3		
Nausea and vomiting	4	Guillain-Barré syndrome	NA*		

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Introduction

- Ascites is the pathological accumulation of fluid in the abdominal cavity.
- It is multifactorial
- Low serum ascites albumin gradient (SAAG) (< 1.1 g/dL) ascites is seen with tuberculosis, peritoneal malignancy, pancreatic ascites, nephrotic syndrome or protein-losing enteropathy.
- A high SAAG (= 1.1 g/dL) suggests portal hypertension due to liver cirrhosis, heart failure or Budd-Chiari syndrome.
- Ascitic fluid in heart failure is typically clear in appearance and characterized by high SAAG and total protein in ascitic fluid (TPAF).
- We present a unique case of hemorrhagic ascites caused by heart failure, where the patient's SAAG was < 1.1 g/dl.

Case Description

- A 62-year-old African American male presented with complaints of worsening shortness of breath, abdominal distension, fatigue and scrotal swelling for 6 months.
- PMH: Severe pulmonary hypertension, congestive heart failure and end stage renal disease on hemodialysis.
- Laboratory workup was normal. Abdominal ultrasound showed large volume free intra-abdominal fluid.
- He was dialyzed with fluid removal for three consecutive days with no symptomatic improvement.
- Paracentesis obtained hemorrhagic fluid with fluid studies were significant for SAAG 0.9 g/dl and TPAF 4.6 g/dl.
- Cytology on two separate samples reported no malignant cells.
- CT abdomen and pelvis obtained after the procedure was unremarkable.
- Patient's hemoglobin stayed stable at 11.2 g/dL post procedure (11.4 pre-procedure).
- Symptoms significantly improved after fluid removal and he was discharged in stable condition.

Congestive Heart Failure Masquerading <u>As Hemorrhagic Ascites</u>

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Discussion

- Hemorrhagic ascites in the absence of an identifiable cause often presents as a clinical and therapeutic dilemma.
- We ruled out a traumatic tap as our patient produced homogenous red fluid throughout in his fluid samples without clearance of the red
 color.
- CT findings ruled out intraperitoneal bleed, intra-abdominal masses and pancreatitis.
- Cytology of the ascitic fluid was negative for any malignant cells in two separate samples, with a sensitivity of 62% [1-2].
- The ascitic fluid ADA was 3.1 IU/L, ruling out peritoneal tuberculosis with 100% sensitivity and 97.2% specificity [3].
- The high ascitic protein LDH of 117 U/dL was consistent with the results of Runyon et al who showed that heart failure patients had a higher ascitic LDH compared to patients with liver cirrhosis (110 +/- 54 vs 54 +/- 95, p < 0.02). Similarly, TPAF was typically > 2.5 g/dL in heart failure patients and < 2.5 mg/dL in liver cirrhosis [4], consistent with our findings.
- The patient's ascites was attributed to heart failure secondary to severe pulmonary hypertension, despite his low SAAG.



Conclusion

- Ascites due to heart failure is usually represented by a SAAG of > 1.1 g/dL, however our patient's ascites had a SAAG of 0.9.
- It is important to work up patients properly to determine cause of ascites.
- This is a unique case of hemorrhagic ascites due to congestive cardiac failure with a SAAG < 1.1 g/dL.

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Look What the Cat Dragged In: A case of neuroretinitis

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Introduction

Neuroretinitis is the constellation of acute unilateral vision loss secondary to an edematous optic nerve associated with star-shaped macular exudates. Though neuroretinitis carries a wide differential including sarcoid papillitis, syphilitic perineuritis, and Lyme disease- Cat-scratch disease is amongst the most common infectious culprits of neuroretinitis. However, only 1-2% of patients with CSD develop neuroretinitis. CSD is caused by the gram-negative intracellular bacillus Bartonella henselae. Humans typically acquire CSD from a cat bite, scratch, or exposure to fleas. We present a case of CSD neuroretinitis in a

Case

The patient is a 27-year-old female with PMHx of migraines, HCV, and T2DM who presented with 5 days of migraine, left monocular vision loss, ophthalmalgia, and left-sided numbness. On exam, patient had complete OS vision loss with IOP of 19 mmHg, macular star, as well as diminished sensation to the left face, left upper and lower extremities.

Patient was admitted for suspected optic neuritis secondary to multiple sclerosis and started on IV steroids. MRI brain and orbits were unremarkable. Extensive workup ensued with labs significant for elevated ESR, and positive ANA. LP revealed normal opening pressure, no oligoclonal bands, glucose 80, protein 58, WBC 14 with lymphocytic predominance, albumin 38, and elevated IgG. Despite the patient's denial of recent cat exposure, Bartonella IgG and IgM titers were positive. She was subsequently treated with rifampin and doxycycline. Though the patient had left-eye blindness at discharge, by her three-week follow-up visual

acuity in the affected eye improved significan **Discussion**

Although literature remains controversial, corticosteroids and antimicrobials are often used to treat neuroretinitis. A retrospective cohort study demonstrated improvement in ocular symptoms in patients receiving antimicrobials plus corticosteroids in contrast to patients receiving antimicrobials alone. However, the optimal corticosteroid regimen remains unknown

In the aforementioned study, patients received oral prednisone 40 to 80 mg/day tapered over 6 weeks. Other studies recommend using 1mg/kg for two weeks then tapered over four weeks. This patient received two doses of methylprednisolone and showed visual improvement after 3 weeks. This case emphasizes the necessity for more rigorous studies evaluating the appropriate treatment regimen for CSD neuroretinitis

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ADULT STILL'S DISEASE: A CLASSIC PRESENTATION OF A RARE ILLNESS

YOchsner Health

Koyenum Obi, MD; Harith Baldawi, MD

Introduction

Adult Still's disease is a rare illness and an adult version of juvenile idiopathic arthritis that causes high fevers, rash and joint pain. It primarily affects young adults, predisposing them to chronic arthritis and when severe, symptoms can be debilitating. We present a patient with a classic presentation of this rare illness.

Case Presentation

A 23 year old woman with no medical history presented with a 10 day history of diffuse rash, polyarthralgia, polymyalgia and intermittent fevers. Her symptoms initially began with a "butterfly rash" on her face that spread to her back, thighs, and chest wall associated with dry eyes and rings surrounding her eyes. Her rash was worse with onset of fevers. Physical exam revealed elevated temperatures up to 102 degrees; malar, periocular erythema and edema; a diffuse, patchy, erythematous rash involving the chest, abdomen and all four limbs. She was also tender to touch in her ankles, wrists and metacarpophalangeal joints.

Labs included negative autoimmune work up (SSA, ANA, RF, immunoglobulin and complement levels), Ferritin of 4,564, WBC 19, CRP of 233, ESR of 73. CT abdomen revealed splenomegaly, iliac chain and periaortic lymphadenopathy. A skin biopsy showed neutrophilic urticarial dermatosis with rare epidermal dyskeratosis consistent with adult-onset still's disease. Rheumatology was consulted and prednisone was initiated but discontinued due to worsening rash. The patient was started on naproxen 500mg twice daily which improved her symptoms. Due to intolerance of steroids, the patient was treated with Anakinra outpatient as recommended by rheumatology.

Conclusions

Adult Still's disease is very uncommon with an estimated annual incidence of 0.16 cases per 100,000 people with equal distribution between the sexes. Being a diagnosis of exclusion, a thorough history and physical examination is highly important. The clinical presentation is heterogenous, but typical features include daily fevers, cutaneous rash worse during febrile hours, arthritis/arthralgia, leukocytosis and marked elevation in serum ferritin, as presented in this case. Treatment include NSAIDs,

Treatment include NSAIDs, steroids, DMARDS or biologic therapy.

Disclosures

The authors have no financial disclosures or conflicts of interest.

EDWARDSIELLA TARDA: A PISCINE CULPRIT OF ACUTE CHOLANGITIS

YOchsner Health

Koyenum Obi, MD; H. Harvak Hajebian

An 82-year-old male who was admitted for NSTEMI s/p

percutaneous coronary intervention was incidentally found

Introduction

Edwardsiella tarda, a member of Enterobacteriaceae family, is a gram negative, facultative anaerobe found in the digestive tracts of eels, flounder, and most notably, catfish. Although rare in humans, E. tarda bacteremia have been reported in Japan, from consumption of infected wild-caught eel and catfish. Consumers of wild-caught catfish, a cultural past-time and culinary delicacy to the Louisiana region may be at high risk. This case reviews a typical presentation of cholangitis.

Case Presentation

to have leukocytosis and bacteremia. The patient complained of abdominal pain, fevers, chills and decreased appetite. Past medical history was noted for Type II DM and cholecystectomy. Social history is pertinent for a fishing trip 2 weeks prior to presentation along with frequent catfish consumption. The patient was hemodynamically stable and physical examination was unremarkable.

Significant labs included a WBC of 18,000, Alkaline Phosphatase of 950, AST 95, ALT 65, GGT 1450. Blood cultures were positive for *Edwardsiella tarda*. An abdominal ultrasound revealed mild prominence of the common bile duct (CBD) and intrahepatic biliary duct dilatation. An MRCP revealed intra and extrahepatic biliary dilatation. The patient underwent an ERCP with a biliary sphincterotomy and removal of a stone from the CBD. The patient was initiated

on aztreonam, de-escalated to oral amoxicillin-clavulanate for a total of 14 days. Follow up liver enzymes weeks after

the procedure had markedly improved.

Discussion

Cases of E. tarda bacteremia are limited, however several studies include a link to consumption of raw, undercooked, or wild-caught fish, with evidence of a fatal water and food-borne infection. Our patient's history of frequent fishing and catfish consumption, in combination with a history of gallstone disease, likely reflects the etiology of the acute cholangitis. E. tarda is considered pan-sensitive to antimicrobial therapy, with natural resistance to benzylpenicillin, polymyxin B, colistin and reports of mixed sensitivity to tetracyclines.

Disclosures

The authors have no financial disclosures or conflicts of interest.



SPLENOSIS MIMICKING AS MESENTERIC METASTATIC DEPOSITS

Koyenum Obi, MD; Ghady Moafa, MD; Nicholas Cappadona, MD

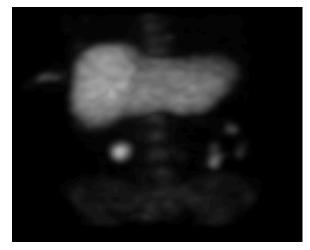


Introduction

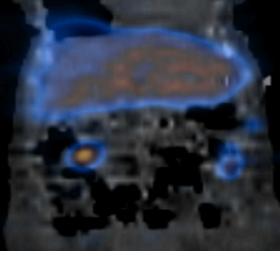
Splenosis, known as ectopic spleen tissue is a benign condition caused by auto-transplantation of splenic tissue. It is usually found in the abdominopelvic cavity and rarely intrathoracic, intracranial cavities, and occurs as a result of splenic trauma or splenectomy.

Case

We present a cale of the cale of the present a cale of the present a cale of the present history of splenectomy due to hereditary spherocytosis who was admitted for altered mental status. History was unable to be obtained on initial presentation, however physical examination was pertinent for abdominal tenderness. Urinalysis was positive for pyuria. Further work up including A CT renal stone was obtained to evaluate patency of an existing ureteral stent. However, incidental CT findings revealed multiple soft tissue density lesions scattered throughout the mesentery measuring up to 3.4 cm in size and many additional soft tissue density lesions tracking along the left pericolic gutter. Findings were concerning for malignancy. The initial decision was to pursue a biopsy of one of the lesions for definitive diagnosis. Considering the patient's prior history of splenectomy, imaging of the lesions were further reviewed which appeared well circumscribed resembling splenic tissue. A Tc-99m sulfur colloid scan was obtained that showed increased uptake in the aforementioned soft tissue lesions on CT scan with findings consistent with splenosis. The scheduled biopsy was cancelled. The patient was treated for pyelonephritis and her symptoms resolved.



A. Tc-99m scan- coronal image showing uptake in anterior hemiabdomen and posterior left upper quadrant



B. Tc-99m scan- coronal fusion view showing uptake in anterior hemiabdomen and posterior left upper quadrant

Discussion

Splenosis, while rare, is incidentally identified on abdominal imaging and unfortunately commonly misdiagnosed as malignancy, often leading to biopsy for tissue sampling. Splenosis should be highly considered as a differential in patients with prior splenic trauma or splenectomy, with newly acquired intra-abdominal soft tissue lesions. Further measures such as a Tc-99m sulfur colloid scan or Tc-99m-tagged damaged RBC scan is useful in diagnosing this benign condition. This knowledge prevents extensive workup, unnecessary invasive interventions and avoids associated risks such as intra-abdominal hemorrhage during biopsy of a highly vascularized splenic tissue. When confirmed, surgical removal is not indicated unless symptomatic.

Disclosures

The Authors have nothing to disclose.

Viral Prodromes and Young Patients: A Usual but Cautionary Tale of Avoidable Pitfalls



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Case Presentation

A 25y old woman with no known health condition was admitted with 6 days of progressively worsening diffuse myalgia and subjective fever. Patient also noted fatigue, headache, arthralgia, nausea and poor appetite. She had tenderness to palpation in the right upper quadrant of abdomen. Work-up revealed leukopenia (1640/uL), thrombocytopenia (45000/uL) and elevated liver transaminase levels with an AST 53 U/L and ALT 44 U/L. A peripheral smear revealed reactive atypical lymphocytes with 17% band cells. Vit B12, folate, and copper levels were found to be normal. Serologic testing for CMV, EBV and viral hepatitis were non-reactive. Initial diagnostic screen by HIV 1/2 antigen/antibody immunoassay (4th generation IA) was repeatedly reactive but the HIV 1/2 supplemental antibody differentiation assay was nonreactive for both HIV 1 and 2. Due to a high clinical suspicion, a HIV 1 DNA and RNA Qualitative PCR testing was subsequently performed which revealed HIV genetic material and thus, the patient was diagnosed with Acute Retroviral Syndrome.

Learning Objectives

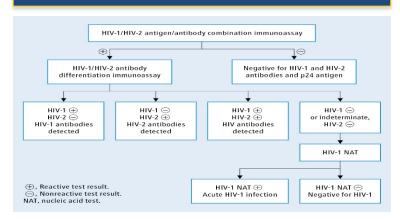
- 1. Recognize acute HIV as an uncommon but important cause of cytopenia
- 2. Describe the influence of HIV on hematopoiesis and the pathophysiology of cytopenia
- 3. Outline the diagnostic tests for acute HIV and the importance of confirmatory testing.

Discussion

Myalgia and fever are commonly encountered by physicians in daily practice. In combination with peripheral cytopenia, these symptoms should raise the suspicion for acute viral infections and HIV should be an important part Acute retroviral syndrome is the of the work-up. constellation of symptoms in acute HIV infection that commonly occur between 2-6 weeks after infection. This can comprise of fever, fatigue, rash, pharyngitis, myalgias, diarrhea, headache and lymphadenopathy. These common symptoms may generate a low suspicion of HIV and lead to a missed diagnosis— as many as 70-75% patients reported in studies. The symptoms correlate with peak viremia and with seeding of lymphoid organs during the seroconversion period of acute HIV when the immune system mounts an early but insufficient response against the virus reducing the viral load. Patients are highly infectious during acute infection and can result in substantial transmissions. Additionally, earlier initiation of ART in this period leads to enhanced normalization of CD4+/CD8+ ratio and reduced rates of serious illness at 3 years compared to initiation later in the course or based on the CD4+ count.

Regardless of the disease stage, HIV has been proposed to infect the auxiliary cells of bone marrow (macrophages and microvascular endothelial cells) leading to a regenerative marrow failure by reducing the ability of hematopoietic stroma cells to respond to regulatory signals that normally increase blood cell production ondemand.

Diagnostic Algorithm



Diagnostic testing for HIV should begin with a 4th generation immunoassay (IA) which can detect both antibodies (IgG and IgM) to HIV-1 and 2 as well as the p-24 antigen. A reactive initial IA needs to be followed up with a supplemental differentiation assay that differentiates between antibodies to HIV-1 and 2. A discordance between a reactive screening IA and negative supplemental assay should not be erroneously interpreted as negative HIV as the P-24 antigen can be detected as early as 2-4 weeks before an antibody assay becomes positive. Thus, a positive antigen test but a false negative/indeterminate supplemental assay can result in a missed HIV diagnosis early in the course of infection. Instead, a confirmatory nucleic acid amplification test (NAAT) should always be performed when results from both initial tests are discordant. Confirmatory NAAT testing has been shown to prevent false negative conclusions in as many as 9 - 32% patients reported in studies.



Correlation of Gastrointestinal Symptoms at Initial Presentation with Clinical Outcomes in Hospitalized COVID-19 Patients: Results from a Large Health System in Southern United States

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Background

We aimed to understand the association of gastrointestinal (GI) symptoms at initial presentation with clinical outcomes during COVID-19 hospitalization

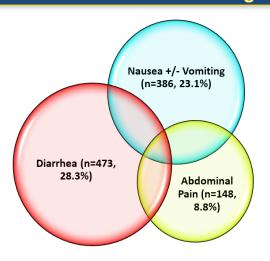
Methods

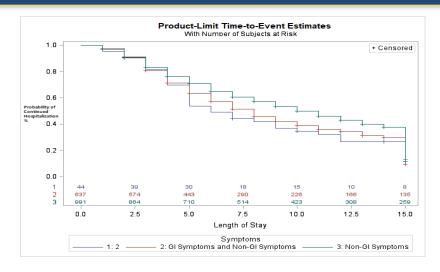
This retrospective, multicenter cohort study included consecutive hospitalized COVID-19 patients between March 1st and April 21st 2020 from a single, large health system. The presence of GI symptoms was assessed at initial presentation and included one or more of the following: nausea, vomiting, diarrhea and abdominal pain. Patients were divided into three cohorts: Only GI symptoms, GI and non-GI symptoms, and only non-GI symptoms. Hospital outcomes were assessed till 12 weeks of hospitalization or discharge. The primary outcome was the association of GI symptoms with mortality. Secondary outcomes included (1) time-to-event analysis for probability of survival and continued hospitalization, (2) prevalence of GI symptoms, and (3) associations of GI symptoms with other clinical manifestations, patient characteristics and clinical events during hospitalization.

Results

1672 COVID-19 patients were hospitalized (mean age: 63 +/- 15.8 years, females: 50.4%) in our system during the 7-week period. 40.7% patients had at least one GI symptom and 2.6% patients had only GI symptoms at initial presentation. Patients presenting with GI symptoms (with or without non-GI symptoms) had a lower mortality rate compared to patients presenting with only non-GI symptoms (20% vs. 26%; p<0.05). The time from hospitalization to being discharged was less for patients presenting with only GI symptoms (7.4 days vs >9 days, p<0.0014).

Figures and Comclusion





		All Patients (N=1672)	Cohort A: Only GI Symptoms (N=44)	Cohort B: GI + Non-GI Symptoms (N=637)	Cohort C: Non-GI Symptoms (N=991)	p- Value
ICU N (%)		625(37.38)	13(29.55)	231(36.58)	372(38.24)	0.4398
	Days to ICU from admission (Mean,±SD)	2.39±3.59	4.31±5.31	2.88±4.11	2.02±3.10	0.0023
	Number of Day in ICU (Mean,±SD)	9.91±8.12	7.77±7.33	9.61±7.73	10.17±8.38	.4487
In-hospital All-Cause N (%)	e Mortality	399(23.86)	9(20.45)	128(20.09)	262(26.44)	0.0118
Length of Stay (Mean,±SD)		10.20±9.64	7.43±6.38	<i>9.61±9.58</i>	10.77±9.76	0.0229

COVID-19 patients presenting with GI symptoms have improved hospitalization outcomes, including a lower in-hospital mortality rate and shorter hospitalization. Presence of GI symptoms appear to be a positive prognosticator in COVID-19 patients



Acute Hepatitis Secondary to Intravenous Methamphetamine Use

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Case Presentation

A 37-year-old white male with a past medical history of untreated Hepatitis C (diagnosed in 2017) presented with sudden onset pleuritic, sub-sternal chest pain, as well as multiple episodes of non-bloody emesis and generalized weakness starting 2-3 days prior to admission. Social history included ongoing intravenous drug use (methamphetamine).

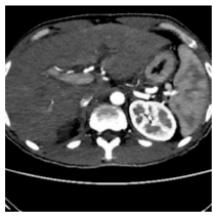
At presentation he was afebrile with stable vital signs. Physical exam was significant for scleral icterus, jaundice, and right upper quadrant tenderness. Initial laboratory data revealed ALT 2,370, AST 2,002, ALP 428, total bilirubin 3.1, INR 1.3, D-dimer 2,168, LDH 419, and Troponin <0.02. A toxicology screen was positive for THC and methamphetamine. Acetaminophen level was within normal limits. Hepatitis panel was positive only for Hepatitis C antibody with RNA log of 6. Anti-nuclear antibody and anti-SSB antibody were positive with the remainder of the extractable nuclear antigen panel returning negative.

CT chest, abdomen/pelvis, and RUQ US with doppler were ordered to rule out thromboembolism, specifically Budd-Chiari syndrome in the setting of elevated D-dimer and hepatic transaminases.

Imaging revealed diffuse periportal edema and patent vasculature with no filling defects to suggest thrombosis.

Following admission, his liver enzymes continued to improve with supportive care and the patient was discharged to a rehab center with close follow up scheduled.

Imaging









Discussion

- Methamphetamine is a highly addictive, recreational drug that acts as a sympathomimetic.
- The clinical presentation of acute intoxication is highly variable but typically includes agitation, diaphoresis, tachycardia, hypertension, hyperthermia, or possible psychosis.
- Multiple mechanisms of methamphetamine-induced hepatocellular damage have been proposed.
- The most prominent theories include:
- methamphetamine-induced hyperthermia
 - ischemia
 - -cytotoxic metabolites
- -hepatocyte mitochondrial dysfunction due to reactive oxygen species.
- Additionally, recent in vitro research suggests methamphetamine may have immunos progressive actions, which could potentiate viral replication in hepatocytes.

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GASTRIC METASTASIS FROM RENAL CELL CARCINOMA

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Case Presentation

A 72 year old white man with a history of renal cell carcinoma treated with left nephrectomy in 2007 but not chemo-radiation, end stage renal disease on hemodialysis, and remote history of peptic ulcer disease (>30 years) presented to the Emergency Department following multiple episodes of acute onset melena with associated light-headedness and normocytic anemia. The night prior to presentation, he began to experience epigastric abdominal pain, bloating, and generalized weakness followed by multiple bowel movements with small volume, black, tarry stool. The patient was hemodynamically stable upon arrival and his hemoglobin down trended from 8.4 to 7.6 with multiple episodes of melena the morning following admission.

EGD revealed three cratered, malignant appearing, friable, nonbleeding gastric body ulcers. The ulcer on the lesser curvature had a visible vessel and the ulcer base was injected with epinephrine and treated with bipolar cautery. A separate ulcer was biopsied for histology. His gastric mucosa was very friable and with scattered nodularity. Random gastric biopsies were taken to evaluate for H. pylori. Additionally, the duodenal bulb had two superficial, clean-based, nonbleeding ulcers approximately 3-5mm in diameter with associated duodenitis.

Pathology results of ulcer revealed oxyntic gastric mucosa with atypical clear cell proliferation consistent with metastatic renal cell carcinoma. The neoplastic cells showed strong expression of PAX 8 and patchy expression of epithelial membrane antigen (EMA). Giemsa stain for Helicobacter-like organisms was negative. After diagnosis, CT images were ordered for staging and close follow up was scheduled with his oncologist.

Endoscopic Findings

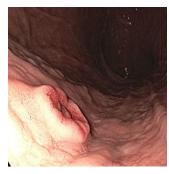


Figure 1: Gastric body



Figure 3: Gastric ulcer with visible vessel



Figure 2: Gastric body ulcer



Figure 4: Duodenitis

Discussion

- Gastric metastatic disease is an infrequent finding with a highly variable clinical presentation and endoscopic appearance.
- The most common primary malignancies associated with metastases to the stomach:
 - Breast cancer (27.9%)
 - Lung cancer (23.8%)
 - Esophageal cancer (19.1%)
 - Renal cell carcinoma (7.6%)
 - Malignant melanoma (7.0%)
- Gastric metastasis are most commonly a single lesion, found in the gastric body.
- Late metastasis is typical with some cases found up to 20 years following initial primary cancer diagnosis.
- Clinical history and presentation may include nonspecific symptoms such as fatigue, weakness, weight loss, abdominal pain, anemia, and signs of gastrointestinal hemorrhage, as in this case.

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Figure 1-4: Esophagogastroduodenoscopy performed by Lilia Stefanwisky, MD and John Hutchings, MD



Acute Adult T-Cell Leukemia: A Consequence of HTLV-1 Infection

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Background



- Overall USA prevalence 0.025% [1]
- Louisiana, Texas, and Florida have highest US prevalence
- Select communities in Africa, Australia, and the Caribbean Islands have high prevalence up to 25%

Case Description

НРІ	45-year-old African American women with three weeks of fatigue, diffuse arthralgia, urinary incontinence, and bilateral upper and lower extremity pruritic rash.		
Physical Exam	Papulonodular rash of on all extremities Diffuse lymphadenopathy		
Labs	Bone marrow biopsy:	DCD2, D5, D25 CD8, dT O% D3+ mature Cells rates	
Management	da-EPOCH (5 cycles) followed by CVAD/MTX-Cytarabine (1 cycle) Allo-HSCT with Fludarabine/total birradiation She has achieved complete response	ody	

Peripheral Smear

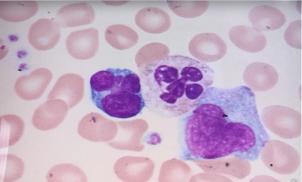


Figure 1: Clover Leaf appearance of the Leukemic T-Cells seen in our

Skin Biopsy

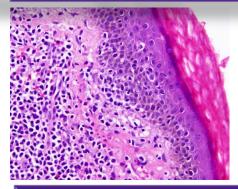


Figure2: Epidermotropis m and microabscess formation [3]

HTLV-1 Oncogenesis [4]

Tax (Grow)	•	Activates protooncogenes (NFkB, CDK) Inhibits tumor suppressors (P53) Inhibits DNA repair and spindle assembly
HBZ (Hide)	•	Inhibits Tax expression that is highly immunogenic
(Illue)	•	Helps virus evade cell mediated immunity

- 3-5% of HTLV-1 infections progress to ATL [5]
- 30–50 year incubation period necessary for oncogenesis

Diagnosis – Shimoyama Classification [6]

					_
Туре	Abnormal T- lymphocytes in peripheral blood	Lymphocyte count	Calcium	LDH	Additional tissue involvement
Smoldering	Around 5%	normal	normal	Up to 1.5x normal limit	May have cutaneous involvement
Chronic	> 5%	lymphocytosis	normal	<2x normal limit	None
Lymphomat ous	<1% circulating cells	Lymphocytosis	normal	<2x normal limit	Diffuse lymphadenopathy (LAD) presence of visceral lesions
Acute	>5% (high levels)	lymphocytosis	elevated	>2x normal limit	LAD, visceral lesions, cutaneous lesions

Management

Chemotherapy

- EPOCH
- VCAP-AMC-VECP
- Zidovudine/Interferon-alpha

Chemotherapy + Allogeneic HSCT

• May prolong median survival (6.7 vs 14 months) [7]

Conclusion

- Median overall survival 6-10 months despite aggressive chemotherapy
- Salvage regimens include Mogamulizumab, an anti-CCR4 monoclonal antibody based on the MIMOGA study with OS of 16 months

- 1 and 2 infections among first-time blood donors-United States, 2000-2009. In: Journal of Infectious Diseases. Vol 209.; 2014:523-531. doi:10.1093/infdis/jit497
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BACKGROUND

Spondyloarthropathy: common extraintestinal manifestation of Crohn's Disease[1] Seen in around 30% of patients[1]

Anti-TNF alpha therapies, such as Infliximab can also induce polyarthritis.

Usually after multiple doses, but rarely as in our patient may occur after a single dose.

CASE DESCRIPTION

HPI	•	57-year-old male with known history of Crohn's
		disease and stage 1 colon cancer admitted for diffuse
		joint pain and stiffness Began 1 day prior to
		admission Joints affected: bilateral
		wrists, ankles, knees, and jaw.
	•	Pain aggravated on movement with mild relief with NSAIDs
	•	Joint stiffness causing
		difficulty gripping objects,
		inability to articulate, chew food, and ambulate leading to falls.
	•	Denied trauma, fever, chills,
		night sweats, abdominal pain, constipation, diarrhea, hematochezia, and melena
	•	Patient recently started on
		Infliximab for maintenance of Crohn's disease and receive his first dose 10
		days prior to admission.

Physical Exam

B/L wrists and ankles with flexion deformity, tight clenching of jaw.

Tenderness, localized warmth, decreased range of movements of bilateral shoulders, knees, and interphalangeal joints of the hands and feet.

Muffled speech and trismus noted



Figure 1 and 2: Revealing the flexion contracture and stiffness of patient's interphalangeal joints of the feet and the hands on the day of admission

LABS	9/17/19	8/15/2020	9/13/2020 (Day of Admission)	9/14/2020
WBC (K/uL)			13.59	
CRP (mg/dL)		0.38	3.69	
ESR (mm/hr)		12	23	
Anti-Infliximab Antibody (Normal <50 U/mL)	<0.22		339	416
Infliximab Drug Level (normal >5 mcg/mL)	0.4		2.7	1.1

Imaging	•	Radiography of bilateral wrists, ankles, knees, cervical spine, sacroiliac joint, thoracolumbar spine - unremarkable
Treatment		IV ketorolac, IV methylprednisone 40mg, and oral azathioprine 150mg Infliximab discontinued; outpatient Vedolizumab was initiated No further episodes of polyarthritis reported
DICCHECION		1

Pathophysiology of paradoxical events in Anti-TNF Alpha Therapy

Mechanism		Description
Cell lysis		Immune system exposed to cell fragments and DNA triggering non-specific antibody formation Downregulation of CRP, causing decreased clearance of apoptotic cells
Immune Dysregulation	•	T-cell induced B-cell activation potentiating antibody formation [2]
Autoimmune Phenomena	•	Usually under the presence of high levels of ANA and anti-dsDNA [2]

Infliximab monotherapy or combination therapy with azathioprine?

SONIC trial: Fewer antibody production in combination therapy vs Infliximab monotherapy [3]

Importance of Testing for Anti-Infliximab antibody levels prior to First infusion

- Antibody concentration of >8.0 g/mL predicted shorter duration of response and higher risk of infusion reactions, relative risk of 2.4 (p <.001) [4]
- Increased anti-Infliximab antibodies in patients who underwent prior colectomy [5]
- Infliximab concentrations significantly lower in patients with infusion reactions [4]

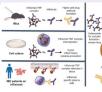


Figure 3: Bar-Yoseph et al concluded in a mice study that there was an increase in antibodies w dose of infliximab given and concentration of infliximab-TNF complexes detected in blood. [8]

Gastroenterolo

Managing Paradoxical Arthralgia in TNF

- Changing the dose of drug or frequency has not ben effective
- Initiation of steroids and restarting immunomodulators effective in few patients [6]
- Premedication with 3 days of corticosteroids [7]
- Switching to Ustekinumab or vedolizumab [1]

CONCLUSION

- Further studies regarding pathophysiological mechanisms, dose and duration correlation to symptoms need to be performed
- Effective management of paradoxical arthralgia still hasn't
 have getablished.
- We hope more avenues to research open and these adverse effects are accounted for prior to initiation of anti-TNF

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Clinical Consequences of ZRSR2 Spliceosome Mutation



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Background Case Presentation Discussion • Spliceosome is a multiprotein complex that 28-year-old white male with a Spliceosomes and the Immune System myriad of autoimmune diseases induces alternative splicing HPI presents for acute flair of • Greatly increases epigenetic diversity Spliceosomes are key to diverse antigen autoimmune gastroenteropathy due • Plays a key role in development and function of recognition of T and B cells to subtherapeutic tacrolimus levels antigen specific lymphocytes [1] Abnormal U2 leads to immune Aplastic anemia due to primary bone marrow failure dysregulation Common Variable Poor antigen Immunodeficiency Disease recognition Autoimmune Gastroenteropathy **PMH** Autoimmune Atopic dermatitis Autoimmune gastroenteropathy diseases Inflammatory polyarthropathy Myelodysplastic Syndrome (MDS) Adenocarcinoma of the colon status Leukemia [4] post total colectomy Immune dysfunction (-) HIV, CMV, hepatitis, HPV6, Autoimmune diseases Parvovirus, ANA screen Immune deficiencies PT/INR, PTT, d-dimer, ferritin, iron Exons ligated profile were normal Adenocarcinoma (35 cases) [5] (-) Direct, Indirect, Supercomb's test Figure 1: Mechanisms of splicing via spliceosomes [2] (-) Fanconi and telomerase gene Novel Therapeutic Targets ZRSR2 Gene panel, Cincinnati inherited children's BMT panel Splicing modulator compounds Flow Cytometry: No abnormal clone • Zinc-finger-RNA-binding-motif-and- Sudemycin Bone marrow biopsy Work Up serine/arginine-rich-2 (ZRSR2) Trilineage • H3B-8800 X-linked hypocellularity · Homozygous mutation more likely to cause :1% blasts Conclusion Normal clinical findings cytogenetics, • Codes for U2 splicesome protein We believe genetic landscaping paves a way for immunohistoch • Mutations are clinically implicated in emistry, and modern medicine, providing newer hopes for cure myelodysplastic syndrome and leukemia FISH with targeted interventions and new (-) MDS and pathophysiologic mechanisms for disease process. PNH References **Next Generation** Homozygous ZRSR2 gene mutation Sequencing X chromosome (human)[1] myelodysplasia. Nature. 2011;478:64-69. 10.1038/nature10496 Anti-thymocyte antiglobulin, cyclosporine, prednisone in the past Currently - Tacrolimus and **Treatment** eltrombopag Unable to find hematopoietic stem



The Traveling Tick: Neurological Manifestations in Late Tick-Borne Illness Devulapalli P, MD, Michael C, MD, & Alawale O, MD

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Introduction

Infections caused by Borrelia burgdorferi, predominantly seen in the Northeast, are categorized into three main stages: early localized, early disseminated, and late. Depending on the stage, various organ involvements are seen, including the skin, joint, cardiac, and nervous systems. Specifically, Lyme meningitis can manifest as headaches, neck stiffness, and visual impairment. The following case illustrates a patient with serology and CSF proven Lyme meningitis.

Case

A 45 year old female with a past medical history of type II diabetes mellitus and hypertension initially presented for evaluation by Ophthalmology, given complaints of progressively worsening blurry vision for the past two years. Patient noted that over the past few months, she had now developed bilateral peripheral vision loss, intermittent headaches, lightheadedness, and photophobia, which prompted her to come in for examination. MRI Head-Orbits was obtained to investigate her painless vision loss, and showed scattered punctate nonspecific T2 hyperintensities of the bifrontal subcortical white matter. Given the patient's age this was less likely to be microvascular ischemic changes, and the distribution of the lesions was not typical of demyelinating disease. It was recommended that vasculitides and Lyme disease be ruled out, for which the workup included: ANA, ANCA, CBC, ESR, CRP, CMP, FTA- Abs, RPR, Echo, Carotid doppler, LP, and Lyme antibodies.

Imaging

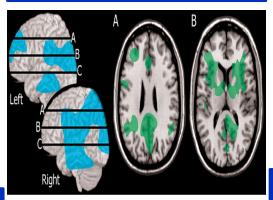


Figure 1- Data from imaging studies have reported decreased cerebral blood flow or metabolism in patients with Neuroborneliosis. One study reported decreased perfusion in the white matter with normal perfusion of gray matter; displayed here on a surface rendering of the brain in blue. Another study reported decreased perfusion in medial temporal, lateral and medial cortices, fronto-temporal white matter, and basal ganglia; approximations of which are represented by axial sectional images in green (Hurley & Taber, 2008).

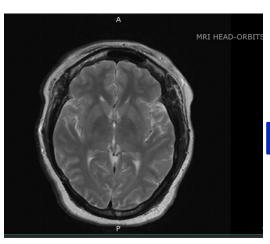


Figure 2- Patient's MRI Head-Orbits W/WO Contrast. Scattered punctate nonspecific T2 hyperintensities of the bifrontal subcortical white matter may reflect minimal microvascular ischemic change, however in light of the patient's young age other considerations including Lyme disease, demyelinating disease (although distribution not typical for demyelinating disease), vasculitis/vasculitides, remain a possibility.

Though the vasculitis workup was negative, the Lyme IgM western blot was positive. On referral to Infectious Disease, the patient admitted to a tick bite in 2016 while living in rural Shreveport, Louisiana, and was uncertain of treatment. Of note, physical findings were negative for Brudzinski and Kernig signs. Subsequently, the LP showed CSF with 2 WBC, 225 RBC, 47% segmented neutrophils, 40% lymphocytes, glucose of 109, and normal protein, consistent with Lyme meningitis- CSF pleocytosis. Patient was admitted to initiate her four week treatment with IV Ceftriaxone.

Discussion

Despite Lyme disease being a rare condition in the Southern states, this case emphasizes the importance of keeping Lyme meningitis as a differential. Given the majority of patients develop skin manifestations, rarely does Lyme meningitis present solely as a neurological manifestation. While symptoms can be very nonspecific, CSF findings can be just as variable, prompting focus on the less abrupt onset of symptoms and findings of pleocytosis in CSF.

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Too Scared to Eat: An Unusual Case of Acquired Angioedema Cynthia Michael, MD & Hailey Danielson, MD Department of Internal Medicine, Leonard J. Chabert Medical Center, Houma, LA



Introduction

Angioedema can be broadly categorized as hereditary or acquired, with distinguishing between the two considered necessary when determining etiology and management. Additionally, acquired angioedema is associated with malignancy, warranting an efficient work up. The following case is of a young woman with recurrent episodes of acquired angioedema.

Case

A 39 year old female, with no past medical history, presented with recurrent episodes of angioedema, without urticaria or pruritus, secondary to an unknown trigger. Since moving to Louisiana two months ago, the patient has had numerous ED visits for sudden onset shortness of breath and angioedema, all of which resolved with administration of epinephrine. The patient provided no new exposures, and noted she had consumed nothing but crackers, bananas, and water for 2 weeks, out of fear of having another reaction. On her current admission. the patient reported her symptoms were significantly worse from prior episodes, and her home epi pen no longer provided relief. Physical exam was notable for severe angioedema, stridor, and respiratory distress. The patient was stabilized on an Epi drip, and admitted for evaluation of acquired angioedema. During admission, the patient was started on a clear liquid diet with plan to slowly advance. However, almost immediately developed an anaphylactic reaction, more severe than any previous episode. Her symptoms began to improve after Epi x3, Benadryl, Solumedrol, Famotidine, Ativan, and Racemic Epi nebulizer treatments were provided.

Consider Hereditary Angioschems (HACE) Recurrent ephandes of abdominat pain and vomiting Required female and a special continuation of a special continuation of a special continuation of the speci

Fig 1- Hereditary Angioedema Diagnostic Algorithm (Bowen et al., 2010).



Fig 2- Soft tissue swelling of the neck during recurrent angioedema attacks. Lateral radiograph of the neck shows thickening of the retropharyngeal soft tissue (asterisk), and thickening of the aryepiglottic folds (arrow) (Ishigami et al., 2014).

The patient was then kept NPO until the following afternoon, when she was started on a tablespoon of Boost every hour. By the third tablespoon, the patient developed abdominal pain and throat swelling that resolved with PRN meds. Labs were significant for normal C4, low-normal C1q, and normal C1INH. Her work-up was negative for SLE, MGUS, and NHL, however her H. pylori stool antigen was positive. The patient started quadruple therapy, and on follow up evaluation reported significant improvement in her episodes, with resolution by the end of treatment.

Discussion

Though acquired angioedema commonly presents with a low C4, C1q, and C1INH, cases with a normal C1q and C1INH have been reported. In light of this, factors such as absent urticaria and pruritus, no family history of angioedema, and an age group closer to their 40's, should be favored in its diagnosis. Lastly, acquired angioedema warrants the work-up of commonly associated diseases such as: MGUS, SLE, NHL, and H. pylori, as their treatment can lead to cessation of the angioedema.

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Not All That "Troponins" Is ACS

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Introduction

The field of cardio-oncology grows in importance as cancer nips at the heels of cardiovascular disease as the leading cause of death in the United States. Previously concerned with the effects of conventional chemotherapeutic regimens and radiation, the growing application of immunotherapy adds a new dimension to this burgeoning area of study. Immune checkpoint inhibitor (ICI) cardiotoxicity is underrecognized and may easily be confused for cardiac decompensation secondary to an acute coronary syndrome (ACS)

Case Report

An 88-year-old man with metastatic hepatocellular carcinoma, atrial fibrillation, chronic kidney disease, and previous tobacco use presented with two days of progressive dyspnea. Workup was notable for an elevated BNP and a significantly elevated troponin of 18 ng/mL. ECG showed a new left bundle branch block and ORS morphology concerning for electrical alternans. Echocardiogram revealed a low-normal EF, a moderate pericardial effusion without tamponade physiology, and no focal wall motion abnormalities. Cardiology evaluated the patient and offered him cardiac catheterization, but he elected to defer this given his multiple comorbidities in favor of medical management. His troponin level fluctuated throughout the admission, peaking at slightly greater than 20 ng/mL. His dyspnea improved gradually without specific treatment after several days. Although ACS topped the differential early on, when the patient's troponin remained elevated for several days, focus shifted to an autoimmune-like myocarditis. After discussion with cardiology and oncology, the patient's presentation was attributed to ICIassociated cardiotoxicity related to nivolumab that was started approximately one week prior to admission

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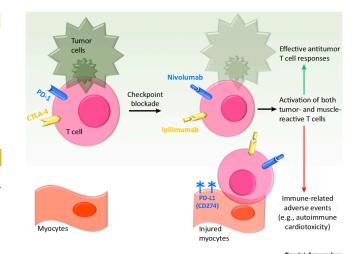


Figure 1: Combination Immune Checkpoint Blockade Immunotherapy Enhances T Cell Response Against Both Tumor Cells and Myocytes

* Borrowed from Autoimmune Cardiotoxicity of Cancer Immunotherapy³



Figure 2: Pericardial Effusion from Nivolumab-Induced Pericarditis

Discussion

- ICIs enhance T cells' ability to recognize foreign cells and facilitate immunemediated cell death. Similar to prescribing other chemotherapeutic agents, the art of medicine must be used to balance the enhanced recognition of oncologic cells with the destruction of cardiac myocytes via molecular mimicry
- While ICIs more commonly cause gastrointestinal, kidney, and endocrine toxicities, a small subset of patients will experience cardiotoxicity
- Cardiotoxicity usually occurs within the first three weeks of treatment and is more common in dual-ICI strategies
- Myocarditis and conduction abnormalities are the most common entities associated with ICI cardiotoxicity
- ICI-related cardiac injury is likely under-reported given that most elevations in troponin are attributed to myocardial oxygen demand-supply mismatch in the setting of a provoked inflammatory state with concurrent toxic chemotherapy
- Troponin may become elevated in a variety of cardiac insults other than ACS, including heart failure, myocarditis, cardiomyopathy, valvular dysfunction, arrythmias, and pulmonary embolism
- It has been proposed that oncologists trend weekly troponins to more easily detect the development of subclinical myocarditis
- The most successful approach to treating ICI-associated cardiotoxicity has proven to be a prolonged steroid taper while optimizing cardiac function with angiotensin-converting enzyme inhibitors, beta blockers, and diuretics
- However, symptomatic ICI-associated cardiotoxicity is associated with a very poor prognosis and is rapidly fatal
- This case illustrates the presentation and natural history of a rare clinical entity and serves as a reminder to consider a broad differential when presented with elevated cardiac biomarkers, even in patients with classic risk factors for coronary disease





Breathless: A Striking Presentation of Squamous Cell Carcinoma of the Lung

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Introduction

Airway obstruction complicates nearly 20-30% of cases of lung cancer. Whether intraluminal from primary lung malignancy or extraluminal compression from adjacent structures, central airway obstruction (CAO) is a dramatic presentation.[1] Here, our authors share a rare case of lung cancer initially presenting as bilateral CAO.

Case Description

A 74 year old Caucasian male with longstanding alcohol and tobacco use saw his primary care physician for dry cough. He was diagnosed with and treated for bronchitis for 4 months, until he had sudden worsening of dyspnea and pleuritic chest discomfort. He then presented to an outside hospital emergency department, where he was hypoxic on room air. Non-invasive positive pressure ventilation was attempted, but' the patient remained hypoxic and was ultimately intubated. Chest x-ray (CXR) obtained at that time demonstrated left mainstem bronchus (LMB) obstruction (Figure 1), prompting transfer for higher level of care. Upon arrival to our institution, medics relay that the patient's oxygen saturation (SaO2) remained 30% – 50% since intubation.

Once transferred, the patient suffered two episodes of cardiopulmonary arrest and was re-intubated to confirm tube placement. Persistent hypoxia led to subsequent position change into the right lateral decubitus position which increased SaO2 to 70%. He was moved to the intensive care unit where emergent bronchoscopy revealed fungating lesions in bilateral mainstem bronchi. The LMB was 75% occluded by the lesion while the remainder of the lumen diameter was filled with blood clot (Figure 3). Right mainstem bronchus (RMB) had a 50% occlusion (Figure 4). After suctioning away clot in the LMB, his SaO2 improved to 100%. A subsequent repeat bronchoscopy on hospital day 2 included balloon bronchoplasty, Argon plasma coagulation tumor destruction, snare electrocautery, and endobronchial biopsies. The pathology report resulted with poorly differentiated squamous cell carcinoma with satellite lesions in the trachea. The patient was extubated on hospital day 3 and began further treatment shortly after discharge.

Figures





Figure 1. (Left) CXR shortly after re-intubation confirming tube placement and with near complete left lung atelectasis. Evidence of tracheal deviation is also noted. Figure 2. (Right) CXR following initial bronchoscopy





Single-lung mechanical ventilation goals include adequate oxygenation while avoiding lung injury from overventilation. Studies have shown that lung-protective ventilation strategies (LPVS) used with acute respiratory distress syndrome (ARDS) are most useful here. While an in-depth discussion of LPVS is outside the scope of this discussion; it is worth noting that this technique is useful due to the pathophysiologically similar changes seen in single-lung ventilation and ARDS.[4]

Conclusion

Malignant CAO often presents in a late stage and carries a very poor prognosis if left untreated. It should be considered if clinical suspicion is high. In this case, immediate stabilization focused on a secure airway allowed time for the healthcare team to consider further measures. In this case, prompt repositioning and bronchoscopic intervention were able to identify and relieve obstruction. A dedicated team of specialists can assist in swift intervention to provide rapid symptom relief, even in the acutely ill patient.

Discussion

Acute obstruction of the central airways by malignant tumor can be a frightening presentation and is almost always associated with a poor prognosis. In an unstable patient with a unilateral obstructing lesion emphasis lies on swift identification, a skilled airway team, and enabling gas exchange in the non-obstructed lung. [2] In lateral decubitus positioning, there is beneficial shift in blood flow in the dependent lung by approximately 25% when compared to supine. [3] With the threezone model of pulmonary circulation and the understanding that blood flow tends to favor the dependent areas of the lung; shifting a patient into a lateral decubitus position allowed for favorable gas exchange. Wieslander et al explain that in a lateral position, there is a sizeable shift in vessel distension and subsequently, the blood flow in the dependent lung. Which is likely due in part to the lack of smooth muscle within the venous walls. [3] Further supplementing this pathophysiological change is the hypoxic pulmonary vasoconstriction and relative right-to-left shunt generated by the non-ventilated lung.[4]

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Chronic Intermittent Hypoglycemia in a Non-Diabetic Patient



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BACKGROUND

Insulinomas were historically believed to be neoplastic proliferations of beta-islet cells of the endocrine pancreas. New studies suggest that these tumors are more commonly neoplastic proliferations of acinar cells of the exocrine pancreas with pluripotent cells capable of endocrine functionality. ¹² Clinical manifestations include intermittent episodes of neuroglycopenic symptoms, associated with both fasting hypoglycemia and post-prandial hypoglycemia. The mechanism of high-levels insulin secretion is not well understood. A recently identified mRNA splice variant in the noncoding region of neoplastic beta-cells increases translation of slice variant mRNA over native insulin mRNA.

CASE DESCRIPTION

A 51-year-old woman with HIV on HAART (CD4 1000 cells/uL, viral load undetected), ESRD on hemodialysis, antiphospholipid syndrome on warfarin, and diverticulosis presented to the hospital with early satiety, diffuse non-specific abdominal pain, lethargy, and several episodes of non-bloody, non-bilious emesis. A week prior to admission she had been diagnosed with calciphylaxisis, with large violaceous wounds with eschar on her right medial thigh that had ruptured and were oozing purulent discharge. Physical exam was notable for hypotension, tachycardia, lethargy and diaphoresis. Leukocyte count was 17.34K/uL. Initial blood glucose level was 47 mg/dL which improved to 130 mg/dL after urgent administration of dextrose 25g intravenously.

She was diagnosed with sepsis due to cellulitis from her thigh wound. Wound cultures results with *E. coli* and *Pseudomonas aeruginosa*. She underwent surgical debridement and received a 10-day course of antimicrobials, with marked clinical improvement. Her hypoglycemia on presentation was attributed to sepsis physiology.

Throughout the course of hospitalization, she continued to experience persistent postprandial hypoglycemia (less than 50mg/dL), associated with nausea, confusion, and lethargy. She underwent a 72-hour fasting lab protocol, ordered to obtain serial lab draws for C-peptide, proinsulin, insulin, and secretagogues while the patient's blood glucose dropped to less than 50mg/dL before urgent correction with IV dextrose.

72 hour fasting lab results were: c-peptide elevated at 6.19ng/ml (0.78-5.19 ng/mL), pro-insulin elevated at 124 pmol/L (3.6-22 pmol/L), insulin 5.4 uU/mL (within normal limits)

A computed tomography angiogram (CTA) of the abdomen and pelvis revealed a dilated distal pancreatic duct with possible obstruction in the pancreatic body with an ill-defined non-enhancing serpiginous low-density mass in the pancreatic tail duct (Fig 1). Endoscopic ultrasound (EUS) with FNA was performed and found an irregular mass in the pancreatic body that measured 13mm X 9mm (Fig 2), pathology was negative for insulinoma. Due to her multiple medical comorbidities, the patient was deemed a nonsurgical candidate. She was treated with octreotide 50 mcg subcutaneous three times daily (TID), and then transitioned to diazoxide 50mg/ml TID with good response.

The sensitivity of EUS and biopsy is approximately 75% for insulinoma, so the patient was scheduled for close follow up for re-evaluation at the Neuroendocrine Tumor Clinic to plan further imaging such as DOTATATE PET to identify localized lesions. The patient was discharged to a long-term acute care hospital for rehabilitation. Shortly after discharge, she developed septic shock and expired.

IMAGING

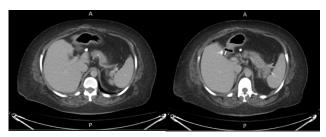


Figure 1: Computed Tomography Angiogram (CTA) Abdomen/Pelvis in series showing dilated distal pancreatic duct with an ill-defined non-enhancing serpiginous low-density structure in the pancreatic tail duct

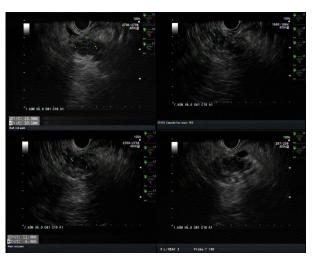


Figure 2: Endoscopic ultrasound (EUS) showing a hypoechoic irregular mass in the pancreatic body measuring 13 mm X 9 mm at its largest in cross-sectional diameter.

The authors have no financial disclosures or conflicts of interests

DISCUSSION AND OUTCOMES

This case of recurrent postprandial hypoglycemia, CNS dysfunction associated with hypoglycemia, and dramatic resolution of symptoms with dextrose resuscitation is consistent with insulinoma. The patient's biochemical laboratory findings of elevated c-peptide and proinsulin, as well as imaging concerning for a pancreatic mass, additionally make the diagnosis of insulinoma highly likely.

Insulinomas are rare tumors that represent the most common etiology of hyper-insulinemic hypoglycemia in adults. § Frequency is approximately one per million and represent 2% of all pancreatic neoplasms. § 1.3 Greater than 90% of insulinomas are benign, solitary, and < 2 cm in diameter and are often equally distributed throughout the gland. Mean age at presentation is 45 years with a female:male ratio of 3:2.9 Fewer than 10% are associated with the multiple endocrine neoplasia type 1 (MEN1) syndrome, and 5–10% of tumors are malignant. ^{2,9,10} Multiple insulinomas can be seen in 2–3% of sporadic cases, usually seen in MEN1. ^{3,5,9}

The episodic nature of pulsing insulin secretion by insulinomas leads to periodic hypoglycemic attacks. §13 Diagnosis and accurate laboratory assay can be challenging. Clinical signs include episodic tremor, palpitations, lethargy, behavioral changes, diaphoresis, and other neurologic changes including encephalopathy. Symptoms can mimic cardiac arrythmia, psychiatric behavioral disturbances, a variety of neurogenic etiologies. §38

The **Whipple's Triad** of symptoms is used to help focus the screening process for endogenous hyperinsulinemia etiologies:⁸

- 1. Hypoglycemia (plasma glucose < 50 mg/dL)
- 2. Neuroglycopenic symptoms
- 3. Prompt relief of symptoms following the administration of glucose

With high clinical suspicion for insulinoma, biochemical markers have nearly 100% sensitivity and specificity to rule out an endogenous cause from hyperinsulinemia. A 72-hour fast test is the gold standard for the diagnosis of endogenous hyperinsulinism. C-peptide levels > 0.2 nmol/l also suggest insulinoma. With imaging, sensitivity and specificity, as well as detection of extrapancreatic extension, are generally superior with MRI.

Endoscopic ultrasound is the best procedure to localize suspected insulinomas and for planning surgical strategy. ⁴ Surgical excision is the treatment of choice with rate of cure for benign insulinomas above 98%. ¹⁴ Only 5-12% of insulinomas are malignant and usually metastasize to the liver with a median survival period of 2 years. ¹⁵ Palliative therapies may include surgery, chemotherapy, embolization, radiofrequency ablation, and somatostatin analogs. ¹⁵ This patient was treated with somatostatin analogues and an alpha-glucoside inhibitor, due to her multiple comorbidities leading to her poor surgical candidacy. Diazoxide may lead to profound fluid retention, which may complicate volume status for hemodialysis-dependent patients.

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Mistaken Acute Coronary Syndrome for Myocarditis in a Young Patient with COVID-19



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BACKGROUND

- · COVID-19 has affected more than 5 million people globally.
- Cardiovascular complications such as tachyarrhythmias, heart failure, myocarditis, and acute coronary syndromes have all been linked to COVID-19³

CASE DESCRIPTION

- A 39-year-old female with type 2 diabetes and tobacco abuse presented with
 worsening dyspnea on exertion after caring for her COVID-19 positive mother
 at home. She was afebrile, hemodynamically stable with leukocytosis of 20K,
 elevated D-dimer 638 ug/L, lactate 23.4 (normal range 4.5-19.8). She had
 markedly elevated troponin I markers >98 ng/mL (normal range less than 0.04)
 and CK-MB 247.1. Rapid test was positive for COVID-19.
- Her electrocardiogram (ECG) showed a new right bundle branch block (RBBB) with Q waves in the anterior septal leads. Computed tomography angiogram (CTA) was negative for pulmonary embolism but showed bilateral ground glass patchy opacities consistent with SARS-CoV-2 pneumonia. A transthoracic echocardiogram (TTE) showed reduced ejection fraction (EF) of 25-30%, LVEDD 5.1 cm, hypokinesis of mid-distal antero-septal wall and apical akinesis.
- On the grounds of the patient's age, hemodynamic stability, lack of typical chest pain symptoms and recent positive test of COVID-19 she was presumed COVID-19 induced myocarditis as the initial primary differential.

MANAGEMENT

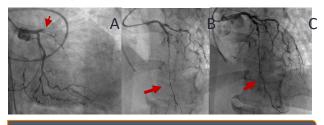
- Over the first 24 hour of her hospital admission, the patient became hemodynamically unstable (worsening lactic acidosis, shortness of breath, with coolness to the lower extremities, and pitting edema). Additional inflammatory biomarkers seen elevated with myocarditis were negative. This led to suspicion of ACS. Protocol was initiated with 325 mg of aspirin, 600 mg of clopidogrel and heparin infusion and patient was taken to the cath lab.
- Left heart and right heart catheterizations were preformed with findings of Proximal LAD lesion, 100% stenosed and Mid RCA complete total occlusion, with collateral supply from Left circumflex artery. PCWP of 33 mmHg, CO 4.2 L/min, CI of 2.1 L/min/m²LVEDP (Pre): 32 mmHg on dobutamine gtt.
- Successful percutaneous coronary intervention (PCI) of LAD was done with drugeluting stent (2.25x30mm Onyx) placed proximal to mid LAD lesion. Intra-aortic balloon pump (IABP) support (1:1) was required for mechanical support of her cardiogenic shock which was removed after 2 days upon clinical improvement of physical exam and hemodynamics to CO= 5.8 L/min, CI = 2.74 L/min/m², off of inotropic support and IABP weaned to (1:2)
- The patient stabilized over the next several days with removal of IABP and she was discharged on goal directed medical therapy with cardiac rehab

IMAGING

Figure 1: EKG showing Q-waves present in the anteroseptal leads



Figure 2: (A) RAO CAU view showing acute thrombotic occlusion of the proximal LAD (B) LAO view of the LAD with wire in place before stenting (C) LAO after percutaneous intervention and stenting



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DISCUSSION

- The incidence of ACS with COVID-19 patients is unknown. A meta-analysis of 10 case—control studies conducted by Barnes et al. demonstrated a two-fold increased risk of acute MI in patients with recent influenza infection or respiratory tract infection.
- COVID-19 induced myocarditis can mimic acute coronary symptoms with symptoms
 of dyspnea, chest pain, elevated cardiac biomarkers (troponin I, etc.) and ischemic
 ECG findings². Therefore, the significance of elevated troponins requires careful
 consideration, as it is a nonspecific marker of myocardial injury. Additionally, ECG
 findings such as T-wave inversions and ST-segment elevations can be found in both
 ACS and myocarditis.
- Therefore, in COVID-19 positive cases where myocarditis is the primary differential, ACS should still be ruled. If the diagnosis is unclear, findings such as leukocytosis, elevated CRP, and pericardial effusion on echo are more indicative of myopericarditis/ myocarditis.
- Multiple mechanisms have been proposed as the pathogenesis of COVID-19 related myocardial injury. Elevated proinflammatory cytokines commonly observed in COVID-19 infections, may trigger an exaggerated response from the immune system leading to myocardial injury.^{3,4}. Another possible mechanism is high stress states associated with an acute illness (like COVID-19), causing a catecholamine surge with systemic inflammation leading to plaque destabilization, rupture, and ACS.⁴

CONCLUSION

- We present a case of acute coronary syndrome in the setting of COVID -19 with no chest pain that was mistaken initially for myocarditis.
- High suspicion of ACS is required in these young patients as well in the setting of
 COVID-19 because acute MI patients with concomitant viral respiratory
 infections are less likely to receive cardiac catheterization across all age groups
 when compared with patients with acute MI's alone⁵. Therefore, clinicians should
 be careful when initially assessing COVID 19 infected patients with concomitant
 heart disease as ACS may be missed leading to higher rates of mortality.

FOLLOW UP

Patient clinically improved after 4 days of hospitalization and was
medically optimized on goal directed medical therapy upon discharge. Her
ejection fraction did not recover and she follows in Heart Failure/
Transplant Clinic outpatient for management of her NYHA I-II ACC
Stage D heart failure. She is not a transplant candidate due to her BMI >
35. She has not had additional admissions for acute decompensated heart
failure and is scheduled to have an ICD placed with electrophysiology for
primary VT prevention.



Case Report: A Delayed Onset of Posterior Reversible Encephalopathy Syndrome

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Introduction

Posterior reversible encephalopathy syndrome (PRES) is a central nervous system disorder of cerebral vasogenic edema that typically includes a heterogeneous mix of headache, decreased level of consciousness, seizures, vision changes, and posterior cerebral edema on head imaging.

Case Presentation

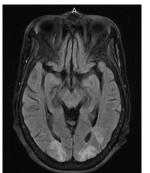
We present a 54-year old African American female with a known history of systemic lupus erythematosus, hypertension, and roux-en-Y gastric bypass correction of 8-month-prior initial sleeve gastrectomy who presented to the emergency department with altered mentation and a JP drain along with IV antibiotics secondary to post-surgical intra-abdominal abscess. Computed tomography of the head two days apart. Leflunomide and azathioprine for lupus were held on admission. Diagnostic paracentesis and drain aspiration of post-surgical fluid collections near the gastrojejunal anastomosis were significant for SBP and several candida species, respectively. Appropriate antimicrobials were continued. Blood cultures from three separate days were negative to completion.

Case Presentation continued

Due to the patient not taking anything by mouth with her continued declining mentation but without needs for the ICU, she was only being treated with as needed anti-hypertensives, with systolic pressures ranging from 130s-160s and diastolic pressures ranging from 80s-low-100s. On the 3rd day of as needed hypertensive control, she had a generalized tonic-clonic seizure with residual new focal neurological deficits, without vision changes. The seizure resolved with intravenous benzodiazepines. Computed tomography of the head and magnetic resonance imaging of the brain at the time were significant for frontal, parietal, and occipital lobe cortical and subcortical edema (Figures 1 and 2).

The patient was stepped up to a critical care setting, begun on an IV nicardipine drip, and loaded with levetiracetam. After rapid improvement in the next 30 hours, the patient was back on tight blood pressure control with oral medications and without further need for anti-epileptics. Patient was stepped down and subsequently discharged to inpatient rehabilitation several days later without residual neurological deficits.

Figure 1. MRI Axial T2 with FLAIR of the brain without contrast demonstrating cortical and subcortical edema in the occipital lobe of the brain. This edema was present diffusely in the cortical regions of the parietal and frontal lobes, as well.



Discussion

The onset of PRES here was in the setting of lupus with immunomodulatory therapies, as well as sepsis of suspected intra-abdominal source—all of which have been documented as precipitants of PRES—but in this case with delayed presentation of classic imaging findings of PRES with good response to usual treatments.

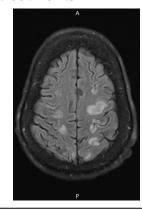


Figure 2. Axial T2 with FLAIR of the brain without contrast with a more cranial view than in Figure 1. This view demonstrates subcortical edema diffusely throughout the occipital and parietal lobes, bilaterally.



A Peculiar Encephalopathy: A Rare Case of Pneumatosis Intestinalis Secondary to Cytomegalovirus

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INTRODUCTION

Pneumatosis intestinalis (PI) is characterized by the formation of gas within the walls of the small or large intestines. It has a poorly understood pathophysiology that may be attributed to bacterial, biochemical, and/or mechanical abnormalities related to the mucosal barrier in the gut walls.

CASE DESCRIPTION

A 74-year-old male kidney transplant patient presented with a two-month duration of decreased appetite and progressively worsening altered mental status associated with new short-term memory deficits and intermittent confusion.

Exam: Disorientation without other neurological deficits, but with an unremarkable abdominal exam.

Labs: Initial laboratory work-up showed an elevated creatinine (5.7) with normal anion gap metabolic acidosis, hypoalbuminemia (2.5), normal urine analysis, normal thyroid function, and ammonia levels. He tested negative for BK virus, hepatitis A, B, and C, HIV, and syphilis. Blood cultures were negative. CSF studies were normal with negative testing for CMV, cryptococcus, enterovirus, HSV, VZV, TB, toxoplasma, and West Nile.

Imaging:

- CT head and MRI brain were unremarkable.
- Chest x-ray ordered to rule out infectious etiology of encephalopathy incidentally detected air within the wall of the colon at the hepatic flexure (figure 1).
- Follow-up CT of the abdomen confirmed air within the walls of the ascending colon and proximal transverse colon consistent with PI (figure 2).

IMAGING

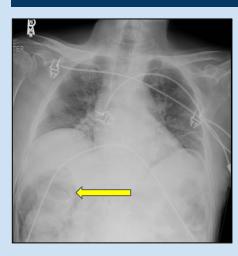


Figure 1: X-ray showing air at hepatic flexure.

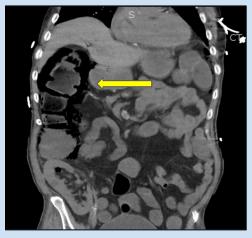


Figure 2: CT showing air within walls of ascending colon.

TREATMENT COURSE

The patient underwent right sided hemicolectomy and ileostomy creation. Pathology of resected colon was negative for malignancy but ultimately revealed evidence of cytomegalovirus. In addition, blood cultures later revealed disseminated Candida albicans felt secondary to possible bowel microperforation. Mental status and kidney function improved following a two-week course of Ganciclovir and a prolonged course of Micafungin.

DISCUSSION

Case reports vary in associated symptoms from asymptomatic to development of abdominal pain and distension, watery diarrhea, and weight loss. Diagnosis is usually by abdominal radiograph or CT.

Case reports of disease in kidney transplant recipients are sparse. There are limited reports of the disease process contributing to encephalopathy.

This case is unique in the subtlety in which it contributed to the patient's symptoms and neurological decline, especially considering the benign abdominal exam and absent gastrointestinal symptoms compared to the majority of reported cases. It's no surprise the diagnosis was an incidental finding within the margins of a chest x-ray.

There are a lack of strong recommendations regarding suggested treatment. The general consensus is that the underlying etiology should be treated and that any signs of developing peritonitis or perforation should warrant surgery. However, it is difficult to discern at what point surgery should be offered to the asymptomatic or patients whose symptoms fail medical management. Surgery was chosen for this patient given degree of air within colon walls and concern for high risk of perforation and adverse outcomes with immunocompromised status.

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Worm-Tales: a rare presentation of Strongyloidiasis in an immunocompromised patient

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Introduction

Infections due to Strongyloides stercoralis are prevalent in rural areas of tropical and subtropical regions, and cases in the United States are more common in the Southeast. Immunocompromised hosts with HTLV-1, HIV/AIDS, Malignancy, Congenital Immunodeficiency, or Alcoholism are at an increased risk of developing disseminated disease and are more prone to atypical presentations. The following case describes a patient with serology proven Strongyloidiasis.

Case

A 44 year old male with HIV presented to the hospital with arthralgias, myalgias, and fevers. He reported pain in his knuckles and toes that started one day after re-initiating antiretroviral therapy (Dolutegravir/Lamivudine), and concomitantly noted a cough and sore throat. CT chest showed focal consolidations in bilateral lobes, mediastinal lymphadenopathy, and splenomegaly. Studies were negative for Legionella, Streptococcal antigens, and Mycoplasma antibodies. The patient was treated for community acquired pneumonia, and his antiretroviral medication was held. As he continued to have polyarthralgia: fingers, wrists, elbows, ankles, spine, and primarily the neck, despite holding Dovato, the medication was resumed. Two weeks post hospital discharge, and four days after resuming Dovato, the patient developed fever, chills, fatigue, and a productive cough with brown-grey sputum. Labs were significant for: ESR 42, CRP 86.1, and a positive ANA screen; however, the ANA profile/pattern/titers, Rheumatoid factor, Fungal immunodiffusion test, Quantiferon Gold, and AFB sputum cultures were negative.



Figure 1: CXR AP Portable showing multiple consolidations

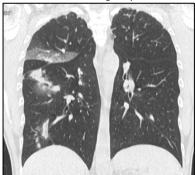


Figure 2: CT chest without contrast showing multiple focal areas of consolidation, ground-glass opacification, and regions of focal bronchiectasis involving the right upper,

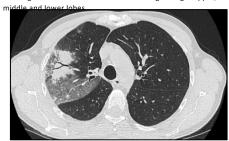


Figure 3: CT chest without contrast showing right upper lobe consolation with air bronchograms and focal areas of bronchiectasis

The patient was transitioned from Dovato to Biktarvy and was referred to Pulmonology. It was then that the patient was noted to have peripheral eosinophilia. Workup for infectious process, inflammatory conditions, and eosinophilia included: CCP, ANCA, Aspergillus IgE, and stool O&P, which were negative. Bronchoscopy showed a Staph aureus infection, but despite appropriate treatment, his cough and debilitating arthralgias persisted. The last study done was Strongyloides IgG antibody, and the patient tested positive. Treatment with Ivermectin for disseminated strongyloidiasis was initiated. Within four weeks, the patient's symptoms resolved, eosinophilia trended down, and Chest CT markedly improved.

Discussion

With a mortality rate of 70-100%, higher rates being in disseminated disease, Strongyloidiasis is not to be overlooked. While eosinophilia can be observed, it is often absent in disseminated disease. Patients often present with Gastrointestinal, Pulmonary, and Cutaneous manifestations. Less commonly, Nephrotic syndrome, GI bleeding, Ascites, Hepatic lesions, and Arthritis can be seen. Early treatment ensures the highest chances for survival.

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A Gut Feeling: An Obstructing Pulmonary Mass Related to Inflammatory Bowel Disease

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Introduction

The least common extra-intestinal manifestation (EIM) of inflammatory bowel disease (IBD) is considered to me pulmonary [1]. While this has been described for decades, the exact mechanism leading to pulmonary involvement is still uncertain, as these symptoms do not seem to favor one single pattern of initiation or presentation. Pulmonary involvement typically manifests as a chronic productive cough and may include dyspnea, abnormal chest imaging, and even airway stenosis. Here, the authors present one such case.

Case Description

A 35 year old female with a history of UC, in remission and previously on mesalamine, presented to her primary care provider with persistent cough and fever x 3 weeks. She' was diagnosed with and treated for community acquired pneumonia with antibiotics and steroids. Initially, she did show some improvement but refractory symptoms led to Pulmonary referral and additional antibiotic therapy. Further clinical decline prompted hospital admission where lab work showed eosinophilia of 9, erythrocyte sedimentation rate 87, and c-reactive protein 12 which was consistent with inflammatory process. Bilateral upper lobe consolidations were noted on chest x-ray (CXR) and non-contrast chest CT (Figure 1, 2). Interestingly, CT also displayed a 12 milimeter (mm) x 13mm x 6mm soft tissue structure at the bifurcation of the upper and lower lobe bronchi, obstructing the left mainstem bronchus (LMB).

During elective bronchoscopy, the LMB demonstrated circumferential airway thickening causing 50% luminal diameter narrowing, obvious cobble-stoning, and areas of white eschar. Biopsies revealed eosinophils and pulmonary alveolar macrophages with few neutrophils which was consistent with moderate acute and chronic inflammation, but no granulomatous inflammation or malignant cells. All infectious panels were negative. She was discharged with a 3 month prednisone taper starting at 60mg/day. Repeat bronchoscopy 6 weeks later showed improved but persistent LMB narrowing. At 6-month follow up bronchoscopy, only mild residual hyperemia and slight focal luminal narrowing remained

Figure 2. CT chest showing soft issue

Discussion

With the rising incidence of IBD worldwide, there has been increased focus on EIMs, especially the respiratory system [1, 2]. These EIMs are theorized to be secondary to chronic, systemic inflammation. While integumentary EIMs are the most commonly diagnosed, pulmonary manifestations have traditionally been documented as the most rare. However, there are some studies that challenge this idea. It had been documented that almost 40-60% of IBD patients may have some degree of subclinical lung involvement evidenced through alterations in pulmonary function tests (PFTs) or high resolution tomographic imaging [5].

Respiratory involvement may range from subclinical and only seen on PFTs, to severe and easily diagnosed by symptoms or imaging. While bronchiectasis is the most-frequently reported pulmonary EIM of IBD; it is followed closely by chronic bronchitis in a non-smoking population [3, 4, 6]. It is worth noting that inflammatory lesions and even airway rupture or enterocutaneous fistulas have been reported in those with IBD [7]. The pattern of symptom onset is quite puzzling and highly variable within all systems. Some EIMs are closely linked to disease activity and relative flare-ups while others are sometimes present even after surgical resection of diseased colon. Respiratory manifestations do not seem to have a clear relationship with the current stage of bowel disease severity or flare up [2, 3, 4].

Imaging findings include CXR, which can be normal or show narrowing of large airways. CT imaging often includes findings consistent with bronchiectasis with dilated airways, patchy areas of perfusion, air trapping, "tree in bud" opacities, or ground glass appearances [7]. BAL is largely non-specific and non-diagnostic but is paramount for discovering chronic inflammation and can show alveolar neutrophils and macrophages.

Treatment of IBD is largely based on medication regimens that include sulfasalazine and mesalamine (5-ASA) which are known for their intrinsic ability to cause pulmonary side effects [4]. In our case discussed above, the patient was previously on 5-ASA therapy but was not at the time of this episode. Treatment of the pulmonary EIMs is driven almost exclusively by steroids in which the data for use is empiric, at best. In any case, delineation between systemic vs inhaled is largely determined by the severity of inflammation and pattern of involvement. Inhaled steroids appear to be more effective and better tolerated than their oral counterparts [8].

Conclusion

The relationship between IBD and associated respiratory EIMs is quite surprising. Considering that respiratory manifestations do not seem to have a clear relationship with the stage of bowel disease severity or activity; this case demonstrates the importance of considering EIMs of IBD in patients with clinical remission.

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A Little DILI For Your Spider Bite

Health Sciences Center

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Case Presentation

A 45-year-old woman with a history of gastric bypass surgery, recently treated hepatitis C, alcohol and cocaine abuse presented to the Emergency Department with two weeks of epigastric abdominal pain associated with fevers, chills, diffuse myalgias, fatigue, early satiety, nausea, and dark-colored urine. Further review of systems was notable for a recent spider bite which was treated with 3 days of amoxicillin, followed by 6 days of oral Trimethoprim-sulfamethoxazole (TMP-SMX) 40-200mg/5mL suspension BID about 3 weeks. She was concurrently on a course of doxycycline for presumed endometritis. On physical exam, she had a positive Murphy's sign, jaundice and scleral icterus. Laboratory studies showed AST 137, ALT 136, ALP 688, total bilirubin 4.6, and GGT 306. RUQ ultrasound was significant for gallbladder wall thickening and increased vascularity in the wall of the gallbladder. CT abdomen showed no evidence of gallbladder obstruction or cholecystitis. MRCP confirmed no biliary obstruction. Work up for infectious, obstructive, autoimmune, and genetic etiologies was unremarkable. All potentially hepatotoxic medications were stopped but worsening of liver function continued, stabilized, and then began to decline over the course of her hospital stay. Liver biopsy revealed granulomatous hepatitis with irregular peri-granulomatous and background large-droplet steatosis that, in the setting of negative laboratory work-up, led to the diagnosis of TMP-SMX-induced granulomatous hepatitis with predominant cholestasis. She was started on ursodiol and discharged with outpatient follow-up.

Discussion

TMP-SMX-induced liver injury can range from mild liver enzyme elevations to acute liver injury. Most cases resolve quickly, within 2-8 weeks, but can last for months in cases of severe cholestasis. Some documented cases have resulted in the need for a liver transplant. In most cases sulfonamide is the causative agent, but trimethoprim also has associated cases of hepatotoxicity, so it is best to avoid switching to trimethoprim alone and identify the patient as allergic to TMP-SMX. Liver injury with the use of TMP-SMX is rare relative to the widespread use of this drug, and this case illustrates that even short courses may put a patient at risk.

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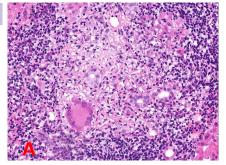
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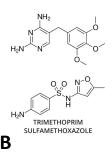


Table 3. Most Frequent Causes of Idiosyncratic Prescription Drug-Induced Liver Injury.*					
Rank	Agent	Year of FDA Approval	No. (%)†	Major Phenotypes	
1	Amoxicillin-clavulanate	1984	91 (10.1)	Cholestatic or mixed hepatitis	
2	Isoniazid	1952	48 (5.3)	Acute hepatocellular hepatitis	
3	Nitrofurantoin	1953	42 (4.7)	Acute or chronic hepatocellular hepatitis	
4	TMP-SMZ	1973	31 (3.4)	Mixed hepatitis	
5	Minocycline	1971	28 (3.1)	Acute or chronic hepatocellular hepatitis	
6	Cefazolin	1973	20 (2.2)	Cholestatic hepatitis	
7	Azithromycin	1991	18 (2.0)	Hepatocellular, mixed, or cholestatic hepatitis	
8	Ciprofloxacin	1987	16 (1.8)	Hepatocellular, mixed, or cholestatic hepatitis	
9	Levofloxacin	1996	13 (1.4)	Hepatocellular, mixed, or cholestatic hepatitis	
10	Diclofenac	1988	12 (1.3)	Acute or chronic hepatocellular hepatitis	
11	Phenytoin	1946	12 (1.3)	Hepatocellular or mixed hepatitis	
12	Methyldopa	1962	11 (1.2)	Hepatocellular or mixed hepatitis	
13	Azathioprine	1968	10 (1.1)	Cholestatic hepatitis	

* Data are from Chalasani et al. ¹³ The listed agents are those most frequently implicated in a total of 1257 cases of druginduced liver injury reported between 2004 and 2013; agents were classified as definite, highly likely, or probable causes (in 899 cases). Agents that ranked from 14th to 25th in frequency were hydralazine, lamortigine, and mercaptopurine (9 cases each); atorvastatin and moxifloxacin (8 cases each); and allopurinol, amoxicillin, duloxetine, rosuvastatin, tellthromycin, terbinafine, and valproic acid (7 cases each). FDA denotes Food and Drug Administration. † The percentages have been calculated on the basis of a total of 899 cases of drug-induced liver injury.

 $\hbox{A. Example of granulomatous hepatitis}\\$

nistopatholog

B. Organic Formula of TMP-SMX



HYPOGLYCEMIA UNAWARENESS AND CO-OCCURRENCE OF THYROID AUTOIMMUNITY AS FEATURES TO HELP DISTINGUISH BETWEEN TYPE 1 AND TYPE 2 DIABETES IN AN ADULT PATIENT

Health NEW ORLEANS

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Introduction

- Type 1 Diabetes Mellitus (T1DM) and Type 2 Diabetes Mellitus (T2DM) can share many presenting symptoms including polyuria, polydipsia, and polyphagia
- Adult age at diagnosis is associated with T2DM, but T1DM can also occur in adults
- Initial labs in both disease states reflect hyperglycemia and may reflect ketosis
- A correct diagnosis is critical to maximize patient outcomes, to ensure effective medication dosing, and to limit dangerous adverse events such as hypoglycemia

Case Presentation

- A 49 year-old man with a history of T2DM (diagnosed at age 31) and suspected acquired hypothyroidism s/p neck irradiation to treat
- T-cell lymphoma endorsed episodes of tremors and sweating x 3 months
- During these episodes, self-monitored blood glucose would range between 40-60 mg/dL
- For management of presumed T2DM, the patient was on a regimen of insulin glargine 30 units nightly and Metformin 1,000 mg BID
- Previous labs were positive for insulin antibodies though the significance was unclear in the setting of long-term insulin exposure
- He was referred to Endocrinology; additional labs revealed elevated anti-Glutamic Acid Decarboxylase antibody and low C-peptide levels. confirming T1DM
- There was no prior lab evaluation of his hypothyroidism, however anti-thyroid peroxidase antibody was elevated to 88.8 IU/mL, confirming a diagnosis of concomitant Hashimoto's thyroiditis
- Metformin was discontinued from his medications; his basal insulin dose was lowered and prandial insulin was added
- Levothyroxine was also added
- Discussions were started for a possible continuous glucose monitor to aid recognition of hypoglycemia

Pathophysiology of T1DM and T2DM

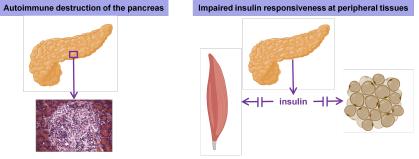


Figure 1. Schematic illustrating mechanisms of T1DM (left) and T2DM (right). Portions of this figure were created using BioRender.com. Histological image from Greaves 2007.

	Type 1 Diabetes Mellitus	Type 2 Diabetes Mellitus
Onset	Sudden	Gradual
Typical age at onset	Primarily childhood or adolescence	Predominantly adults
Underlying Pathophysiology	Autoimmune destruction of pancreatic β-cells	Insulin resistance, relative insulin deficiency
Body habitus	Thin or normal weight	Often obese
Insulin deficiency	Absolute deficiency	Relative deficiency
C-peptide levels	Low	Normal to elevated
Prone to ketoacidosis	More frequently	Less frequently
Prone to hypoglycemia	More common	Less common
Autoantibodies	Usually present	Usually absent
Treatments	Lifelong insulin administration	Diet, exercise, weight loss; possible progression to insulin administration
Concordance Studies	Identical twins <50% concordance	Identical twins 70-90% concordance
HLA associations	Yes, usually HLA-DR genes	No
Other autoimmune diseases	More often, often involving thyroid	Less often

Table 1. Comparison of characteristics between T1DM and T2DM.

Discussion

- While overlap exists between symptoms and lab findings in T1DM and T2DM, correction of underlying metabolic disturbance is critical for treatment and prognosis
- T1DM is caused by autoimmune destruction of insulin-producing pancreatic β-cells leading to absolute insulin deficiency with resulting hyperglycemia and requirement for exogenous insulin to correct hyperglycemia
- T2DM is caused by impaired insulinresponsiveness of insulin-sensitive peripheral tissues (skeletal muscle, adipose tissue, pancreas, and liver) and relative insulin deficiency, leading to hyperglycemia
- In T2DM, there is no destruction of insulinproducing pancreatic β-cells, leaving insulin production and secretion preserved initially; initial T2DM pharmacotherapies are aimed at increasing insulin sensitivity and secretion
- Insulin sensitization of peripheral tissues is relatively preserved in T1DM, leading patients to be more susceptible to hypoglycemia
- There is association and co-occurrence between T1DM and other autoimmune diseases, particularly autoimmune thyroid disease
- Obesity and development of T2DM are also strongly correlated
- Practitioners should consider a missed T1DM diagnosis in the setting of a thin patient with preserved insulin sensitivity and/or other autoimmune disease

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Figure 1. Histological image courtesy of Greaves, P. (2007). Liver and pancreas. Histopathology of Preclinical Toxicity Studies, 457-



A Case of Necrotizing Myositis and Rhabdomyolysis Secondary to HMG-coA Reductase Inhibitor



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Introduction

- HMG-coA reductase inhibitors are essential medications for lowering cholesterol and reducing mortality from cardiovascular disease
- HMG-coA reductase inhibitors have wellestablished side effects of myositis and rhabdomyolysis
- Skeletal muscle injury results in release of intracellular contents, including creatine kinase and myoglobin, into the bloodstream
- Myoglobin accumulation can cause severe nephrotoxicity, such as acute tubular necrosis
- Prompt identification of offending agent, discontinuation, and intravenous fluid resuscitation are important for resolution

Case Presentation

- A 78 year-old female with history of CAD s/p PCI, HTN, T2DM, Hypothyroidism presented with chief complaint of progressive muscle weakness for one month
- Physical exam: 2/5 strength of hips with flexion but preserved strength at knees & ankles; exam revealed restricted active ROM of upper extremities with inability to abduct shoulders above 90°
- Initial labs: elevated high-sensitivity CRP at 14.79 mg/L (nl <3.19 mg/L) and ESR at 40 mm/hr (nl 0-20 mm/hr)
- Additional labs: elevated creatine kinase (CK) of 13,519 U/L (nl 20-180 U/L), elevated AST of 397 U/L (nl 10-40 U/L) and elevated ALT of 396 U/L (nl 10-44 U/L) without elevated alkaline phosphatase or total bilirubin
- Dipstick UA: 3+ occult blood but microscopic UA with only 3 RBC/hpf
- Serum and urine myoglobin elevated at 2,186 mcg/L (nl <90 mcg/L) and 663 mcg/L (nl <65 mcg/L); aldolase high at 59.7 (nl 1.2-7.6 U/L)
- Home medications reviewed, pt taking Atorvastatin 80 mg daily
- Anti-HMG-coA reductase antibody level ordered; elevated at 131 units (nl 0-19 units)
- Pt administered IV fluids over next five days to dilute myoglobin and to prevent renal toxicity, worked with PT/OT, and was transferred to a SNF once CK levels were stable

Pathophysiology of HMG-coA Reductase Inhibitor Myositis

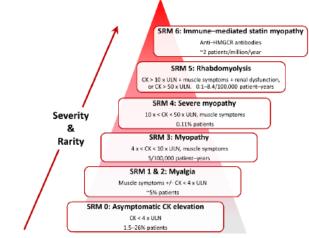


Figure 1. Schematic depicting classifications of Statin-Related Myotoxicities (SRM) according to severity and rarity. Obtained from Turner, R. M., & Pirmohamed, M. (2020). Statin-related myotoxicity: A comprehensive review of pharmacokinetic, pharmacogenomic and muscle components. *Journal of clinical medicine*, 9(1), 22.

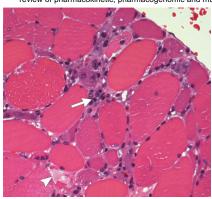


Figure 2. Skeletal muscle fiber necrosis (arrowhead) and macrophage infiltration (arrow) in muscle specimen collected from patient with positive HMG-coA reductase antibodies. Image obtained from Mammen, A. L. (2016). Statinassociated autoimmune myopathy. New England Journal of Medicine. 374(7): 664-669

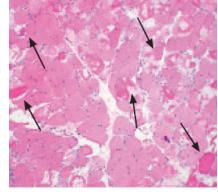


Figure 3. Significant skeletal muscle necrosis (arrows) in skeletal muscle specimen collected from patient experiencing HMG-coA reductase inhibitor rhabdomyolysis. Image obtained from Bosch, X., Poch, E., & Grau, J. M. (2009). Rhabdomyolysis and acute kidney injury. New England Journal of Medicine, 361(1), 62-72.

Discussion

- Increasing incidence of hyperlipidemia and evidence-based mortality benefit from HMGcoA reductase inhibitors have increased the number of patients on these medications
- Statin-related myotoxicities (SRM) are the most frequently reported adverse effect, estimated 66% of all adverse events
- Effects can include skeletal muscle pain, stiffness, heaviness, and weakness
- SRMs classified according to symptoms and lab values (CK level, anti-HMG-coA antibody presence) with seven distinct classifications ranging in severity from myalgia to myositis
- The most severe classification, SRM 6 or immune-mediated statin myopathy, characterized by presence of anti-HMG-coA antibodies
- SRMs most often occur during 1st year of therapy (median onset at 1 month after starting therapy) but can happen at any time
- Several putative mechanisms currently being investigated as factors to contribute to myocyte dysfunction in SRMs, including mitochondrial impairment, coenzyme Q10 depletion, calcium signaling disruption
- Management of myopathy or rhabdomyolysis includes monitoring serial CK values with IV fluid repletion to prevent renal toxicities
- Doctor-physician discussion frequently in weighing risks and benefits of HMG-coA reductase inhibitor therapy when deciding to continue, suspend, or discontinue therapy

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Catheter Directed tPA for treatment of acute PE in setting of Sickle Cell Disease

Austin Tutor MD, Justin Price MD, Jose Tafur MD

BACKGROUND

- Hemoglobin SC disease is the second most common type of sickle cell disease (SCD).
 Venous thromboembolism is a well-known complication of SCD and can present as pulmonary emboli.
- Mortality rate of acute pulmonary embolism has been estimated to be up to 30%.

CASE DESCRIPTION

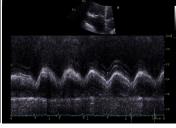
- 37-year-old male presented with abdominal pain and shortness of breath. Laboratory evaluation on admission was significant for elevated liver enzymes and bilirubinuria. A CT angiogram was unable to be performed secondary to acute renal failure. A VQ scan showed a high probability for PE.
- Transthoracic echo was notable for severe right ventricular (RV) failure with RVEDD
 6.28cm and TAPSE 1.1cm, flattening of the interventricular septum, and severe pulmonary artery systolic pressure (PASP) elevation to 82mmHg.
- Additionally, he was found to have multiple DVTs and small arterial thrombi in his distal lower extremities.

INTERVENTION

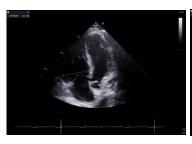
 Due to his RV and multisystem organ failure despite treatment with continuous IV heparin, we proceeded with a catheter directed tPA continuous infusion for 12 hours at 1mg/hr in addition to dobutamine.



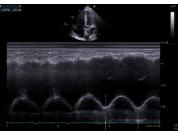
RVEDD before tPA = 6.3 cm



TAPSE before tPA = 1.1



RVEDD after tPA = 5.8 cm



TAPSE after tPA = 2.8

OUTCOME

- After 5 days of treatment, PASP decreased to 34mmHg and his liver and renal function returned to normal. He was able to be weaned off dobutamine and transitioned from intravenous heparin to apixaban.
- He was discharged with no cardiac symptoms (NYHA Class I).

DISCUSSION & CONCLUSION

- Hypercoagulability in SCD is well established; however, these patients are also at a higher risk of hemorrhage, including ICH, than the normal population. This may be in part due to hemorrhagic conversion of undiagnosed small vessel ischemic stroke and years of progressive central nervous system vascular inflammation. These features of SCD present a unique challenge when choosing treatment of acute PEs.
- In this case, the benefits of tPA outweighed the risk of hemorrhage; therefore, catheter directed tPA was chosen as rescue therapy for our hemodynamically stable intermediatehigh risk sub-massive PE patient (Class Ila, European Society of Cardiology).



Respiratory Distress after an Overdue Apartment Cleaning

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Introduction

Allergic bronchopulmonary aspergillosis (ABPA) is a lung disease found primarily in people with asthma or with cystic fibrosis that results from an IgE-mediated hypersensitivity reaction to antigens of Aspergillus fumigatus.

Case Presentation

- 26 year old African American woman with a history of mild intermittent asthma and ectopic pregnancy.
- Presented to the hospital with 2 weeks of nasal congestion, non-productive cough, shortness of breath, and wheezing
- Her apartment was recently cleaned due to mold growth.
- Use of an albuterol inhaler failed to relive her symptoms.

Hospital Course

- Initial CBC showed a WBC count of 7x10° cells per liter with eosinophilia (6.2%), and chest X-ray demonstrated a dense right supra-hilar focal opacity concerning for a mucus plug with dense atelectasis.
- * Aspergillus IgE testing resulted positive at 7.64.
- Treatment with albuterol, ipratropium, prednisone, and chest physiotherapy resulted in significant symptomatic improvement and near-complete resolution radiographically in one day.

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3 Marion, DW. Clinical manifestations and diagnosis of allergic bronchopulmonary aspergillosis. In: UpToDate, Post, TW (Ed), UpToDate, Waltham, MA, 2021

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Fig. 1: Initial chest X-ray demonstrating a dense right supra-hilar focal opacity concerning for a mucus plug with dense atelectasis.



Fig. 2: Follow up chest X-ray after treatment demonstrating near-complete resolution radiographically in one day.

Discussion

- Aspergillus molds are present ubiquitously in the environment, especially in organic matter.
- Patients can present with bronchospasm, pulmonary infiltrates (usually mucoid impaction), bronchiectasis, fibrosis, and eosinophilia.
- Diagnostic testing includes Aspergillus skin test, elevated total serum IgE, elevated IgE specific to A. fumigatus, peripheral blood eosinophilia, and radiographic evidence of parenchymal infiltrates, bronchiectasis, dilated bronchi, and mucoid impaction.
- Treatment involves long-term steroids (weeks to months depending on activity and severity of the disease).
- Anti-fungals may help decrease exacerbations and act as a steroid-sparing agent.
- Antibiotics are required in the presence of superimposed bacterial infections.
- Other treatments include omalizumab in severe cases, and supportive therapy such as salbutamol nebulizers with hypertonic saline, mucus clearance valves or

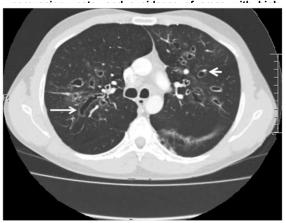


Fig. 3: Computed Tomography Scan of Thorax Showing Central Bronchiectasis in Case of ABPA

This is a computed tomography (CT) scan of the thorax showing central bronchiectasis. It is identified by the 'signet ring' (short, thick arrow) and 'string of pearls' (long, thin arrow) appearances. Mucoid impaction and dilated bronchi are also present.



Cyclical Fever after Afro Nation Ghana Music Festival

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Introduction

- Malaria should be suspected in patients with travel to endemic regions.
- Symptoms of malaria include fever and flu-like illness, including shaking chills, headaches, muscle aches, fatigue, nausea, vomiting, diarrhea, and jaundice.

Case Presentation

- 30 year old African American woman presented with a 3 day history of fever (as high as 103°F), malaise, fatigue, headache, intermittent, generalized abdominal cramping, nausea, and diarrhea.
- Two weeks prior, she traveled to Ghana for a music festival where she used bug spray but not mosquito nets, drank bottled water but used tap water to brush her teeth, did not get pretravel vaccines, took onceweekly unknown malaria pills obtained in Ghana, and had contact with a fellow traveler who was sick with malaria.

Hospital Course

- Labs showed a WBC of 3.9x10° cells per liter, 23.5% monocytes, hemoglobin 11.7 g/dl, LDH 315 units/L, CRP 17 mg/L, AST 51 units/L, ALT 49 units/L, T. Bili 2.3 mg/dl
- Serum positive for Malaria antigen.
- Blood smear positive for Plasmodium falciparum antigen.
- A 3 day course of atovaquone-proguanil provided significant improvement of her symptoms.



Malaria transmission is not known to occur

Malaria transmission occurs throughou

Malaria transmission occurs in some place

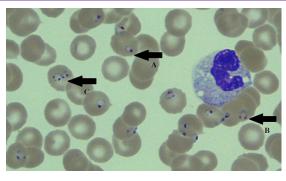


Fig. 1: Giemsa-stained peripheral blood smear. Arrow A showing a classic, ring-shaped trophozoite of *Plasmodium falciparum*. Arrow B showing a classic, headphone-shaped trophozoite of *P. falciparum*. Arrow C showing two trophozoites of *P. falciparum* within the same red blood cell.

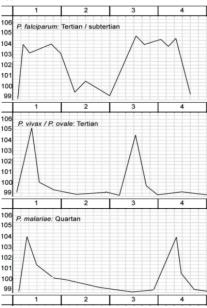


Fig. 2: Graph of the typical fever curve for common types of Plasmodium species

Discussion

- Malaria is transmitted through a female Anopheles mosquito infected with the parasite Plasmodium (most commonly P. falciparum, P. vivax, P. malariae, and P. ovale).
- Following a 2 week incubation period, clinical manifestations include fever, malaise, diaphoresis, nausea, vomiting, abdominal pain, headache, and cough.
- Symptoms are caused by both parasitized and nonparasitized RBCs adhering to small blood vessels, resulting in small infarcts, capillary leakage, and organ dysfunction which includes impaired consciousness +/seizures, ARDS, metabolic acidosis, renal failure, hepatic failure, coagulopathy +/- DIC, severe anemia, massive intravascular hemolysis, and hypoglycemia.
- Uncomplicated malaria should be suspected in any febrile patient with travel to an endemic region, including sub-Saharan Africa and parts of Oceania such as Papua New Guinea.
- Diagnosis is made with light microscopy of blood smears and rapid diagnostic tests.
- Treatment of uncomplicated malaria in areas with chloroquine resistance includes artemisinin combination therapy, atovaquone-proguanil, quininebased regimens, and mefloquine.
- Mosquito bite prevention and chemoprophylaxis for high-risk areas with atovaquone-proguanil, mefloquine, doxycycline, and tafenoquine reduce risk of infection.

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- 3 Classic image: peripheral blood smear in a case of *Plasmodium falciparum* cerebral malaria
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The Curious Case of the Confused Lady

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



Introduction

- Wernicke's encephalopathy, characterized by a classic triad of nystagmus, ataxia, and confusion, is a neurological condition resulting from thiamine deficiency.
- . If not properly recognized and treated early, Wernicke's encephalopathy can to debilitating and permanent neurological damage.

Case Presentation

- * A 38-year-old woman with a history of hyperthyroidism presented to a rural hospital with lower extremity weakness, shortness of breath, and subjective fevers with diaphoresis.
- At that time she appeared alert, interactive, and oriented. She was notably tachycardic and labs demonstrated a TSH of less than 0.01 with a an elevated free T4 of 4.35. Prior to transfer, she received beta blockers for her tachycardia and methimazole for presumed thyroid storm.
- Upon arrival to our hospital, she was febrile to 100.5° F and tachycardic to 162 beats per minute. She was disoriented and hallucinating. Exam revealed horizontal nystagmus. Labs again demonstrated an elevated free T4 but now showed a lactic acidosis.
- * She received propranolol and PTU for her thyrotoxicosis.
- * After hospital day 2, the patient was no longer hallucinating and most of her symptoms had resolved
- * However, over the course of the next several days, she remained disoriented in addition to a persistent lactic acidosis despite her approaching euthyroid levels and adequate fluid resuscitation.
- * The persistence of her altered mental status despite treatment of the thyrotoxicosis was concerning.
- EEG and MRI brain were both unrevealing.
- During her prolonged hospitalization, it became clear that the patient's altered mental status was consistent with confabulation which paired with her nystagmus was concerning for potential Wernicke's encephalopathy.
- High dose thiamine was started and serum levels were collected.
- * Her mental status, nystagmus, and lactic acidosis improved slowly with supplementation.
- * Suspicion for Wernicke's was confirmed when her thiamine level returned low at 29 nmoles/L.

Pentose Phosphate Pathway **Glycolysis** Increased Glucose TPP oxidative ransketolase stress Ribose 5-phosphate NADPH Lactate **±** Pyruvate TPP Lactic Dehydrogenase Acidosis - Acetyl-CoA ---> Myelir TCA Cycle Figure 1. Thiamine forms the crucial component for the α-ketoglutarate production of Thiamine Succinyl-CoA Pyrophosphate (TPP) which works as an essentia coenzyme in many critical biochemical reactions Dehydrogenase

- Encephalopathy
- Oculomotor dysfunction
- Nystagmus, lateral rectus palsy, and conjugate Gait ataxia
- Vestibular dysfunction
- without hearing loss · Peripheral neuropathy
- Hypothermia Amnesia (anterograde
- retrograde, fixation) Confabulation Minimal content in
- · Lack of insight

Apathy

- Behavioral/Personality
- Psychosis, agitation, depression, anxiety, irritability, emotional lability, and insomnia

Cognitive

- Impaired concentration. confusion, poor orientation and immediate recall amnesia, and constructional difficulties

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ONTROPORTURATION

Incidence



Figure 2. Neurologic Manifestations of Thyrotoxicosis vs W-K Syndrome

Figure 3. Epidemiology of WE. (a) Incidence. (b) Ratio of WE in men to women in alcohol and non-alcohol-related causes.3

Discussion

- Wenicke's encephalopathy has an incidence of 0.6 to 2.2 % of the general population and 12.5-35.0% of alcohol dependent patients and is thus frequently overlooked in the nonalcoholic patients presenting with altered mentation.3
- * Thyrotoxicosis artificially increases metabolic resulting in induction of the Krebs cycle and utilization of thiamine stores. 1
- Once thiamine stores have ben fully exhausted, the body shifts to anaerobic metabolism due to the lack of Thiamine Pyrophosphate (TPP) availability to act as a cofactor in two key, ratelimiting steps of the Krebs cycle.1
- * This global increase in the utilization of anaerobic metabolism results in the production of lactic acid which can be persistent despite fluid resuscitation as seen in this patient. 1
- * Wernicke's encephalopathy, a severe neurologic condition caused by the exhaustion of thiamine reserves, can result in debilitating neurological complications and death if not properly recognized and treated early. 2
- Wernicke's encephalopathy is an uncommon complication of thyrotoxicosis, but it's early recognition and treatment in these patient can prevent severe complications of Wernicke's.3
- * Thiamine should be given empirically as soon as the suspicion for Wernicke's encephalopathy is established and treat should be continued until there is no more progression of neurological symptoms.4

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IS MY PATIENT ALLERGIC TO NAFCILLIN OR CEFAZOLIN?

NEW ORLEANS
School of Medicine

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- □ A 66-year-old man with history of left fifth toe osteomyelitis and subsequently ray amputation in 2017, well controlled type two diabetes, and hypertension, presented with fever and infected left diabetic foot wound.
 □ Initially, the patient was treated with empiric vancomycin and cefepime.
- □ He was found to have methicillin sensitive staphylococcus aureus (MSSA) bacteremia and left foot (third and fourth metatarsi) osteomyelitis with pathologic fractures. Empiric antibiotics were de-escalated to nafcillin and metronidazole.
- □ About 48 hours of initiating nafcillin, the patient developed a non-palpable, non-blanching, morbilliform rash. Skin biopsy suggested leukocytoclastic vasculitis.
- He also had mildly elevated creatinine that was resolved with fluid resuscitation. Nafcillin was switched to cefazolin. The patient was subsequently discharged with home cefazolin therapy.
- ☐ After two weeks of cefazolin, the patient presented to clinic for follow up with worsening rash and kidney function with nephrotic range proteinuria requiring intermittent hemodialysis. Cefazolin was switched to vancomycin.
- ☐ Kidney biopsy was suggestive of focal crescent glomerulonephritis with dominant mesangial IgA deposition. The remainder of the workup including ANA, cANCA, complement levels (C3 and C4), rheumatoid factor (RF) was unremarkable.
- ☐ The patient's kidney function recovered with steroid therapy. He completed intravenous antibiotics and was discharged home with two more weeks of doxycycline. His rash resolved, and his kidney function returned to his baseline without any further need for dialysis.

Microbiology

June 2016: Left fifth toe osteomyelitis

- Wound swab with Enterobacter cloacae complex, diphtheroid mixed species, and Peptostreptococcus.
- Bone biopsy culture with no growth.
- Initially treated with vancomycin and piperacillin-tazobactam. Completed piperacillin-tazobactam for eight weeks, followed by oral ciprofloxacin and metronidazole.

January 2017: Left fifth toe acute on chronic osteomyelitis

- Wound swab with mixed Gram positive flora.
- Biopsy culture with no growth.
- Initially treated with vancomycin and piperacillin-tazobactam. Patient had ray amputation of fifth metatarsus. Discharged with ciprofloxacin and clindamycin.

June 2020: Left third and fourth MSSA osteomyelitis

Discussion

- ☐ Penicillins are the most common cause of drug allergy. Previous literature suggested cefazolin may be safe for use in patients with non-IgE mediated hypersensitivity reaction to nafcillin.
- ☐ While leukocytoclastic vasculitis was undoubtedly associated with nafcillin, the onset of glomerulonephritis could be related to cefazolin.
- Our case is similar to a previous case report that described a patient developing crescentic glomerulonephritis after receiving cefazolin for MSSA post-laminectomy wound infection.

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Trippin': A Case of Paraneoplastic Myasthenia Gravis **Secondary to Squamous Cell Carcinoma**

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Introduction

- Myasthenia Gravis (MG) is an autoimmune disease in which acetylcholine receptor antibodies attack acetylcholine receptors at the neuromuscular junction. This results in progressive voluntary muscle weakness with repetitive use.
- Paraneoplastic syndromes are signs or symptoms that occur remote from the site of the primary tumor or metastasis. They are seen in approximately 10% of patients with lung cancer.

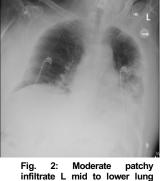
Case Presentation

- * A seventy-five year old man with a history of unresectable squamous cell carcinoma of the lung was transferred from an outside medical facility for investigation of a left lung consolidation.
- In the emergency department he reported shortness of breath for 1 week and progressive generalized weakness to the point that he was unable to rise from a chair. He reported receiving multiple radiation and chemotherapy treatments 6 months previously.
- . On physical exam his vital signs were within normal limits with O2 sat 96% on room air. He had significant kyphosis with inability to lift his head, droopy eyelids, bibasilar crackles L>R, impaired balance, and gait instability.
- Labs on admission were significant for pancytopenia, CRP 137.7. ESR 100. ferritin 1224.
- Radiographs were obtained, as shown to the right.

CXR and chest CT 2 months apart



basilar atelectasis/scarring, small L pleural effusion.



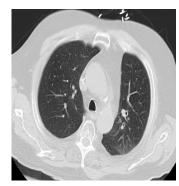


Fig. 3: Mild L basilar atelectatic



Fig. 4: Mixed multifocal parenchymal consolidation and ground-glass opacity with septal interlobular thickening involving the left upper lobe and lingula

Hospital Course

- He was started on ceftriaxone and azithromycin for potential community acquired pneumonia.
- During admission, outside records were retrieved that showed he had started following with a neurologist for management of myasthenia gravis symptoms one month after his diagnosis of lung cancer. Records indicated he was initially treated with pyridostigmine 5 months previously, but this was discontinued due to diarrhea. He had an admission 2 months prior where he was treated with plasmapheresis and IVIG. 1 month prior he was started on mycophenolate mofetil. A serologic test confirmed ACh receptor antibodies 28.3nmol/L (0-0.24).
- Ultimately, the pulmonary opacities were deemed to be organizing pneumonia as a consequence of previous radiotherapy. Antibiotics were stopped and prednisone was initiated. Physical therapy provided functional improvement. He never required supplemental O2 during his stay. He was discharged with oral prednisone, trimethoprim-sulfamethoxazole and provided outpatient follow up.

Discussion

- This patient did not have the typically associated paraneoplastic syndromes of lung cancer. Hypercalcemia can be seen with squamous cell carcinoma, and SIADH or Lambert-Eaton calcium channel antibodies can be seen with small cell
- Also, myasthenia gravis has an associated thymoma 15-20% of the time, but there was no evidence of that in this patient.
- Based on the timeline of events, our team ultimately concluded that his myasthenia gravis was a paraneoplastic syndrome of his squamous cell carcinoma.

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MULTIFACTORIAL CAUSES OF DIARRHEA IN AIDS

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Introduction

HIV/AIDS infection is a viral illness acquired through intercourse or exposure to blood of infected blood. Patients in the later stages of their illness can develop opportunistic infections and risk is increased after CD4 count falls below 200. Therapy for AIDS includes antiretroviral therapy in addition to prophylactic medications for opportunistic infections.

Case Presentation

A 54 year old African American man with a past medical history of HIV/AIDS presented with a chief complaint of non-bloody, non-bilious diarrhea, up to 10 episodes a day for 7 months. The patient also reported frequent night sweats, chills and intermittent fevers in addition to 50 pound weight loss despite compliance with his antiretroviral therapy, which was restarted a month prior to the onset of his symptoms. During the admission, the patient was found to be profoundly hyperkalemic and hyperchloremic in addition to having acute kidney injury secondary to volume depletion from frequent bowel movements. Abdominal CT sacn demonstrated pericolic fat stranding and pancolitis. The patient was initially started on broad spectrum antibiotics and volume resuscitated. His stool was antigen positive for campylobacter and giardia, in addition to being positive for norovirus, campylobacter, cryptosporidium and giardia on Biofire panel. He was continued on his ART therapy, started on azithromycin and metronidazole, and he was aggressively fluid resuscitated.

Discussion

Patients infected with HIV who have high viral loads and low CD4 counts are at risk for opportunistic infections. The diagnoses can be especially challenging for patient's with advanced stages HIV illness. In our patient's case, suspicion was high for opportunistic infection and treatment was initiated prior to test results. For giardia, he was treated with metronidazole; for campylobacter he was treated with azithromycin. For cryptosporidium there is no targeted therapy and he was maintained on antiretroviral therapy. He received supportive care for norovirus. On discharge his bowel movements were improved, although not completely resolved. Further improvement was expected as his immune system reconstitutes.

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doi:10.1001/jama.282.23.2220.

Figure A: Case courtsey of M. Melvin PHiL rlD 3740
Figure B: Case courtsey of RE Weaver r PHiL rlD 6654
Figure C: Case courtsey of CDC

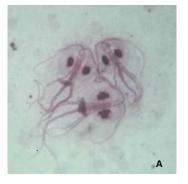


Figure A: Giardia trophozoites in Giemsa stain.



Figure B: Photomicrograph of Campylobacter Jejuni with characteristic S-shaped forms**A**

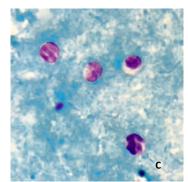


Figure C: Cryptosporidium oocytes seen on acid-fast stain



B12 Deficiency Masquerading as Thrombotic Microangiopathic Anemia Priscilla Soto Justiniano, MD¹, Asma Samreen, MD¹, Ernest Quintin, MD² Department of Internal Medicine¹, Department of Hematology-Oncology², Leonard J. Chabert Medical Center, Houma, LA¹,²

Introduction

A rare manifestation of cobalamin deficiency is Pseudo-Thrombotic Microangiopathic Anemia (TMA) which is characterized by MAHA, thrombocytopenia and presence of schistocytes. This presentation often prompts clinicians to start plasma exchange (PEX) therapy empirically for suspicion for a TMA such as

Thrombotic Thrombocytonenic Purpura

Case

A 54-year-old male with schizophrenia, diabetes, hypertension, cirrhosis and history of hepatitis C and stroke presented with fatigue, nausea, vomiting and muddy-brown urine for one week. Pertinent physical exam findings were icteric sclera, dry oral mucosa, and right sided residual weakness from a past stroke. On presentation his hemoglobin was 3.9g/dL, hematocrit 12%, MCV 89, WBC 4.01K/uL and platelets 29K/uL. Peripheral smear showed the presence of schistocytes, spherocytes, anisocytosis and poikilocytosis. His total bilirubin was 7.5, LDH 9536 and Haptoglobin <10. Renal function was normal. Blood was transfused and plasmapheresis was initiated for concern for acquired TTP.

Detected levels of ADAMTS13 were 49%, not suggestive of TTP and PEX was discontinued. Additionally, his WBC decreased, which prompted further workup for pancytopenia. He had B12 deficiency with levels <146 and normal folate. Repletion was initiated parenterally. Further workup for B12 deficiency revealed positive anti-parietal antibodies suggestive of pernicious anemia. Within 5 days of discontinuation of plasmapheresis and concomitant B12 repletion his WBC count and platelets normalized, his hemoglobin steadily improved and total bilirubin was back to baseline. B12 injections were scheduled to

continue outpatient thereafter Images

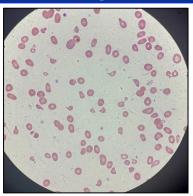


Figure 1: Peripheral smear showing, anisopoikilocytosis, schistocytes and thrombocytopenia. (Wright's stain, x1000)

Discussion

Clinicians must initiate plasmapheresis when there is high clinical suspicion for TTP, before confirmatory tests have resulted, because of the rapidly fatal nature of the disease.

However, other differentials for MAHA should also be considered. Pseudo-TMA, is one important entity to recognize and to initiate appropriate therapy. Repletion of cobalamin typically results in resolution of this clinical manifestation. This case illustrates a new diagnosis of pernicious anemia presenting as a rare manifestation of severe B12 deficiency, which in turn was a mimic for a rare hematologic emergency.

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A CASE OF THROMBOTIC THROMBOCYTOPENIC PURPURA IN A JEHOVAH'S WITNESS PATIENT



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Introduction

TTP is a rare medical emergency that is highly fatal without prompt treatment. It is caused by thrombotic microangiopathy from reduced levels of ADAMTS13, leading to thrombocytopenia and microangiopathic hemolytic anemia (MAHA). Standard treatment is with plasma exchange (PEX) and immunosuppressive agents. We present a fatal case of acquired TTP without PEX in a Jehova's Witness patient.

Case

A 47-year-old African-American female presented to the hospital with chest pain, dyspnea, dark urine, and bleeding from her gums for three days. Physical exam revealed tachypnea, blood in her mouth and petechiae in bilateral lower extremities.

She was profoundly thrombocytopenic and anemic, had increased reticulocyte count and total bilirubin, and low haptoglobin. Renal function tests were normal. Peripheral smear showed schistocytes. Presentation was consistent with MAHA with high clinical suspicion for TTP.

She declined any blood products, including plasma. She received high-dose steroids, Rituximab infusion and attempts were made to expedite the delivery of Caplacizumab. On the 3rd day, ADAMTS13 level resulted as <1%. Her urine turned black, she developed headaches and worsening chest pain. Electrocardiogram was normal. On the 5thday, Caplacizumab arrived at the treating facility, however before it was administered, she was found unresponsive and without a pulse. Unfortunately, the patient passed despite resuscitative efforts.

Images

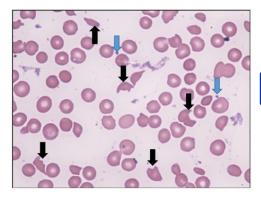


Figure 1: Illustration of a peripheral smear with schistocytes black arrows) and low platelets (blue arrows). Hashmi, H. R., Diaz-Fuentes, G., Jadhay, P., & Khaia, M. (2015).

Discussion

Treatment options for TTP without PEX are limited. Caplacizumab, a direct monoclonal antibody against the von Willebrand factor, was approved in 2019 for the treatment of TTP and showed promising results. Limited medical literature is available discussing the efficacy of Caplacizumab without PEX. This case would have potentially presented additional information on the treatment of TTP in patients who are unable to receive blood products. Unsuccessful cases of treatment in Jehovah's Witnesses are underreported. This case further illustrates the rapidly fatal progression of this disease. Salient clinical features were headaches as neurologic manifestations, chest pain and sudden cardiac death as an ultimate cardiac sequela of TTP.

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Improperly treated anxiety and mood disorders results in untreated inflammation

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Introduction

It is well documented that patients with inflammatory bowel disease (IBD) are more likely to have anxiety and mood disorder (AMD) comorbidity. Specifically, IBD patient populations will have a depression and anxiety rate of 21.2% and 19.1% respectively, which is increased compared to the general population of 13.4% and 9.6%. Psychological distress can be exacerbated by the activity and severity of a patient's IBD, as is a decrease in overall life satisfaction and quality of life. Unfortunately, AMD can have direct impact on patients maintaining adherence to any treatment plan. Several factors have been found to have negative impacts on adherence in patients with AMD including anxiety of medical therapy. anhedonia, distrust with the medical professional. and overall psychological distress. Knowing this. the authors assessed the local outpatient population to evaluate for patients with AMD not receiving IBD therapy to reengage these patients by approaching primary care providers in hopes of providing appropriate therapy.

Method

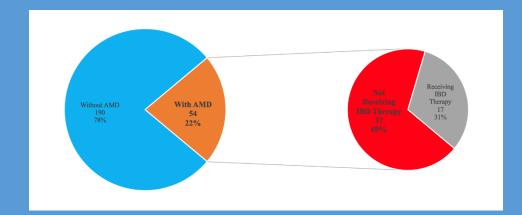
An electronic medical record search for patients with IBD within a local outpatient population, resulting in a starting patient population of 545. Exclusion criteria were applied to the starting patient population, resulting in the research study population of 244. The exclusion criteria were patients younger than 20 years of age, older than 80 years of age, and those currently being treated by an IBD specialist. The research study population was then assessed for patients receiving IBD therapy and for patients with AMD.

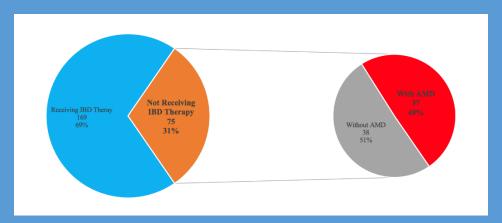


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Patients with improperly treated anxiety and mood disorders are more likely to have untreated inflammatory bowel disease.





Results

Of the 244 patients in the research pool, 190/244 (78%) of patients did not have AMD diagnosis while 54/244 (22%) had AMD comorbidity [Figure 1].

Of the 54 patients with AMD comorbidity, an alarming number of patients 37/54 (69%) were not receiving IBD therapy while only 17/54 (31%) of patients with AMD comorbidity were receiving IBD therapy [Figure 1].

Of the 244 patients in the research study pool, 169/244 (69%) were receiving IBD therapy while 75/244 (31%) were not receiving IBD therapy [Figure 2].

Of the 169 patients receiving IBD therapy, 17/169 (10%) patients had AMD comorbidity. Alarmingly, of the 75 patients not receiving IBD therapy, 37/75 (49%) had AMD comorbidity [Figure 2].

Of the 190 patients without AMD comorbidity, 38/190 (20%) were not receiving IBD therapy [Figure 2].

Discussion

The retrospective chart analysis demonstrates that of the 54 patients with AMD, 37 (69%) were not receiving IBD therapy. Out of the 75 patients not receiving IBD therapy, 37 (49%) of the patients had AMD. This is an alarming revelation given that previous research demonstrates a 20% prevalence of AMD in the general population. This increased rate of AMD is strongly related. In our chart review. patient's refused IBD therapy due to apathy toward their diagnosis while others simply were not interested in receiving treatment. In order to combat the negative impacts of mental illness on adherence, we recommend using a multifaceted approach that blends both medical and psychological interventions. The authors also recommend utilizing screening tools to gauge the presence of mood disorders in this patient population in order to adequately diagnose and treat this comorbidity. Psychological approaches to treating patients who are dealing with IBD can include Cognitive Behavioral Therapy (CBT). Acceptance and Commitment Therapy (ACT), and even some evidence backing the use of Gut-Directed Hypnotherapy. Many of these approaches target individual's thought and behavior patterns. as well as integration of mindfulness and acceptance. Our study proves this as AMD affects patients willingness and adherence to IBD therapy. This reinforces the paramount importance of behavioral health being embedded within the treatment of IBD.



A Strange Diagnosis of Cytomegalovirus Colitis in an Immunocompetent Patient 🧥

endola MD

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Internal Medicine Residency Program

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Introduction

- Infection of the colon with cytomegalovirus (CMV) will lead to a disease state known as CMV colitis. CMV colitis typically presents with symptoms of fever, abdominal pain, and diarrhea with intermittent hematochezia.
- This disease is typically seen in patients with underlying immunocompromising illness such as AIDS [1] or in patients taking immunosuppressive medication, such as transplant recipients [2].
- However, there have been few rare reports of CMV colitis in immunocompetent patients [3].

Case Description

- This is a 59-year-old Caucasian female with a past medical history of essential hypertension who presented to the emergency department complaining of bright red blood per rectum.
- She has noticed intermittent bright red blood on toilet paper after having bowel movements, intermittent and crampy abdominal pain, increasing weakness, and increasing fatigue over the last 6 months. She is currently unable to perform her activities of daily living secondary to these symptoms.
- She had never had an esophagogastroduodenoscopy (EGD) or colonoscopy. She denies gastrointestinal diseases, including inflammatory bowel disease.
- At time of presentation, the patient was noted to be hypotensive, have bright red blood per rectum, and hemoglobin of 6 g/dL. She received 2 units of packed red blood cells, was initiated on intravenous pantoprazole, and was scheduled for EGD and colonoscopy.
- EGD was non-revealing and demonstrated normal mucosa in the esophagus, stomach, and proximal duodenum.
- Colonoscopy revealed many large, non-bleeding ulcerations located at the ileocecal valve, ascending colon, transverse colon, and descending colon, which can be seen in Figure 1 and 2.
- Biopsies of the right and left colon were obtained, which demonstrated focal active colitis with ulceration with fibrinopurulent exudate. Immunostaining for cytomegalovirus (CMV) was positive for suspected viral inclusions compatible with CMV related colitis, which can be seen in Figure 3. Biopsies were negative for dysplasia or malignancy.
- The patient was initiated on oral valganciclovir 900mg orally twice a day.
- Further evaluation of the patient did not reveal immunocompromising states, including HIV, or immunosuppressive medication use. Patient was discharged on hospital stay day four after adamantly requesting to return home.
 The patient unfortunately did not present for her follow up appointment with her primary care provider or qastroenterologist.

Figures





Figure 1 and 2: Image of ileocecal valve and descending colon ulceration obtained during colonoscopy.

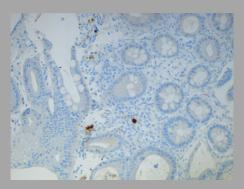


Figure 3: Immunohistochemical stain of colon biopsy demonstrating cytomegalovirus inclusion bodies viewed at 20x objective.

Discussion

- Confirmation of CMV colitis requires identification of cytomegalic viral inclusions on colon biopsies. Antibody testing against CMV antigens can be completed as well. Once diagnosis is established, prompt treatment with antiviral agents, including ganciclovir and valganciclovir, should be initiated [4].
- Theoretically, early initiation of treatment may decrease the risk of mortality; however, further studies need to be completed to fully evaluate risk of mortality in the immunocompetent patient after initiation of antiviral treatment.
- Immunocompetent individuals with CMV colitis typically present with severe, life threatening infection or complications [5]. One meta-analysis of outcome in CMV colitis infections in the immunocompetent patient demonstrated death in about 31.8% of patients greater than the age of 55 [6].
- Another meta-analysis demonstrates the in-hospital mortality rate of CMV colitis in the immunocompetent as 71.4% if symptoms of shock and lower gastrointestinal bleeding are severe enough to require intensive care unit admission.
- Along with immunocompromised states, there have been documented reports of other clinical associations that may aid the physician in establishing CMV colitis on the differential. One study demonstrates that elderly patients with diabetes mellitus, chronic renal failure, or ischemic heart disease may be at greater risk of developing CMV colitis when compared to a young, healthy patient [7].
- Another infrequently documented comorbidity that may increase the risk of CMV colitis in the immunocompetent is inflammatory bowel disease (IBD), specifically ulcerative colitis (UC) [7]. One source documents cytomegalovirus as a potential exacerbating factor for those with UC [8].

Conclusion

Although rare, this case demonstrates the importance of considering CMV colitis as a differential in an immunocompetent patient with mild symptoms of hematochezia, diarrhea, and abdominal pain.

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Resolution of Metastatic Crohn's Disease after Treatment with Certolizumab

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Introduction

Crohn's disease (CD) is a chronic inflammatory bowel disease (IBD) which may have distinctive mucocutaneous manifestations. Up to 33% of IBD patients have at least one extraintestinal disease manifestation. This is a case of Metastatic Crohn's disease (MCD) on the upper cutaneous lip that significantly resolved with only two injections of certolizumab after multiple failed therapies. This highlights the importance of a thorough H&P and histopathological confirmation. To our knowledge, this is the first case to describe success in treating MCD with certolizumab alone.

Clinical History

A 63-year-old African American woman with a previous history of Crohn's disease presented to the dermatology clinic for evaluation of a rash located on her face, especially on the right upper lip. She reported that the rash appeared two years prior. It was associated with pruritus and moderate swelling. Patient had tried Humira, Imuran, Remicade, Plaquenil, Minocycline, and intralesional Kenalog with minimal improvements. Her prescription medications include monthly vitamin B12 injections, prednisone, Norco, and tizanidine.

- Review of Systems: No systemic complaints. Denies fevers, chills, or arthralgias. No recent infections. No blisters or changes in personal care products.
- Pertinent past medical history: Flexible sigmoidoscopy in 3/2015 showed ostomy stricture dilation, cellulitis, and severe Crohn's left colon → referred for colectomy and end ileostomy in 4/2015

Physical Examination

Infiltrative orange-yellow papules were distributed on the right upper cutaneous lip and right superior medical buccal cheek. Patient with noted right-sided asymmetric facial swelling primarily located peri orally.

Histopathology

A 4.0 mm punch biopsy was performed. Histopathological findings revealed multiple nodular granulomatous infiltrates extending from the superficial reticular to deep reticular dermis. The granulomas were composed of numerous histiocytes as well as a moderately dense inflammatory infiltrate made up predominantly of lymphocytes with scattered plasma cells and eosinophils.

- S-100 staining was negative.
- PAS and Fite stains were negative for infectious organisms.



BEFORE Certolizumab injections (picture taken 5/26/2016)



AFTER Certolizumab injections (picture taken 11/14/2016)

Discussion

Metastatic Crohn's disease is a rare, non-contiguous cutaneous manifestation of primary CD, being the least common presentation of extra-intestinal CD.¹ Most cases of MCD occur on the lower extremities, intertriginous areas, face, and genital area.² No clear correlation between the development of MCD and luminal disease activity has been established. This patient had a metastatic cutaneous presentation of Crohn's disease present for two years that ultimately responded to the induction dose of 400 mg certolizumab (2 injections of 200 mg).

Certolizumab (certolizumab pegol) is a PEGylated antitumor necrosis factor biologic therapy approved for use in Crohn's disease, psoriatic arthritis, rheumatoid arthritis, and ankylosing spondylitis. A case report by Kiuru et al. described a patient with MCD on the left calf that completely resolved after six weeks of treatment with certolizumab in combination with methotrexate. Adalimumab, infliximab, antibiotics, systemic & topical corticosteroids, methotrexate, and azathioprine have demonstrated benefit.^{4,5}

This patient was prescribed a maintenance dose of certolizumab 400 mg every other week and will require longterm follow up.

Conclusion

- Metastatic Crohn's disease is a rare manifestation of primary CD and has no clear association with luminal disease activity.
- Dermatologists & Internists should include this entity as a differential diagnosis when treating a persistent plaque or nodule in a patient with a history of CD.
- More controlled trials and case reports are needed when considering treatment options.

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Challenging the status quo in acute pancreatitis

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Tulane University
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Learning Objectives

- Recognize anchoring and premature closure as cognitive biases in clinical reasoning
- 2. Contrast imaging modalities commonly used to diagnose acute pancreatitis

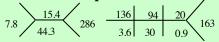
Clinical History

A 65-year-old man with a history of chronic pancreatitis and alcohol use disorder presented with 3 days of sharp epigastric abdominal pain that radiated to his back. There was associated nausea and vomiting. He reported increased alcohol intake.

Physical Exam

He was afebrile and normotensive. There was mild epigastric tenderness to palpation without guarding or rebound. Normal bowel sounds throughout.

Initial Workup



Total bilirubin: 0.6 mg/dL AST/ALT: 19/15 IU/L Alk phos: 76 IU/L Lipase: 333 IU/L

CT abdomen/pelvis: Edema in head of the pancreas, with adjacent new infiltration of fat in the anterior pararenal space, chronic dilatation of the main pancreatic duct, and punctate calcifications consistent with acute on chronic pancreatitis



Figure 1: CT visualization of main pancreatic duct dilation and punctate parenchymal calcifications (chronic) along with new infiltration of fat and edema (acute). Gallbladder and biliary tree show no calcified gallstones. No intrahepatic or extrahepatic biliary ductal dilation identified.

Hospital Course

Despite adequate fluid resuscitation and analgesia for 24 hours, the patient's abdominal pain was unimproved, requiring reassessment.

Repeat labs demonstrated AST/ALT of 71/39 IU/L, alk phos of 245 IU/L, total bilirubin of 2.7 mg/dL with a direct bilirubin of 1.2 mg/dL.

A RUQ ultrasound revealed a dilated bile duct, main pancreatic intrahepatic biliary ductal dilatation, and dilated gallbladder with internal echogenic sludge without obstructing calculi.

Patient was then transferred same-day for definitive treatment with ERCP.

Subsequent Workup

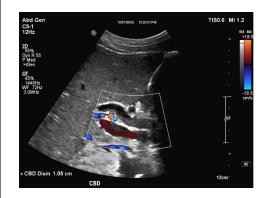


Figure 2: RUQ ultrasound demonstrates dilated extrahepatic common bile duct at the porta hepatis, measuring 1.05 cm.

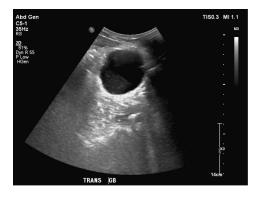


Figure 3: Gallbladder sludge identified within distended gallbladder (14.5 cm longitudinally) without wall thickening, pericholecystic fluid, or sonographic Murphy's sign.

Discussion

Anchoring is the tendency of clinicians to fixate on an initial diagnosis, despite the introduction of new information warranting reevaluation. Its cause is multifactorial, including elements of confirmation bias, failure to reassess during a patient's hospital course, and attending to findings that had been specifically sought after. Anchoring is further exacerbated by premature closure, a failure to consider alternative diagnoses once an initial diagnosis is made.

Commonly, a CT scan with contrast allows for pancreatic parenchymal visualization to identify pancreatitis. Biliary ductal dilation is best visualized through RUQ ultrasound, however given there was an alternative cause of pancreatitis in this case (alcohol use), no LFT elevations concerning for obstruction, and no obvious ductal dilation on CT scan, an ultrasound was not ordered at the time of presentation.

When symptoms worsened after initial clinical improvement, this prompted the team to consider an alternative explanation and workup was expanded. At that point in the hospital course, new LFT elevations were discovered and ultrasound was obtained and revealed new ductal dilation and evidence of obstruction.

Conclusion

- Anchoring is a common cognitive bias in medicine. The internist can minimize the chance of anchoring by not overlooking findings inconsistent with the leading diagnosis.
- Ultrasound is the test of choice to identify biliary ductal dilatation.

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Angiotensin-converting enzyme inhibitor, a rare cause of small-bowel Angioedema. A case report

Ghady Moafa, MD, David Muscat, DO, Nicholas Cappadona, MD, Koyenum obi,



BACKGROUND

An angiotensin-converting enzyme inhibitor is widely used to treat hypertension. Angioedema is a recognized side effect of ACEI, caused by a reduction in bradykinin degradation.

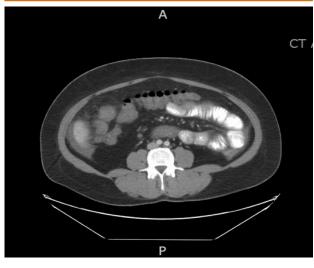
Clinical manifestations consist of edema of face, lips, tongue, uvula and upper airways, sometimes requiring intubation or tracheotomy in severe cases. Here in our case, we present a rare case of ACEI induce small-bowel Angioedema.

CASE DESCRIPTION

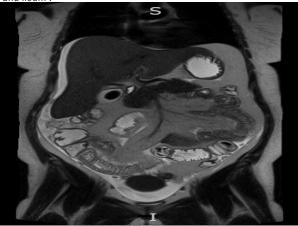
Our case presents a 34 years old female with a past medical history of hypertension who presented with generalized abdominal pain, nausea and diarrhea for ten days. After an extensive work up which was largely unrevealing, she was discharged home for outpatient follow-up. Shortly after discharge, she experienced worsening symptoms, prompting another ED visit. CT abdomen was performed which was consistent with findings of inflammatory bowel disease. She underwent further testing with EGD and Colonoscopy, which were significant for gastritis.

On the second presentation, she was vitally stable. Laboratory workup was significant for leukocytosis of 31. Repeated CT abdomen showed inflammation of the entire jejunum and ileum consistent with enteritis. Magnetic Resonance Enterography was done later, which showed a Long segment of submucosal edematous small bowel wall thickening involving most of the ileum and jejunum—finding highly suggestive of ACEI induced small-bowl Angioedema. ACEI discontinued

IMAGES



A: CT A/P showed continued inflammation of the entire jejunum and ileum .



B: MRI Enterorrhaphy: showed Long segment of submucosal edematous small bowel wall thickening involving majority of the ileum and jejunum.

DISSCUSSION

Angiotensin-converting enzyme inhibitors are commonly prescribed to manage HTN, HF, and diabetic nephropathy. One of the most common side effects of ACEI therapy is angioedema along with dry cough and hyperkalemia. Angioedema is characterized by localized temporary swelling, which can affect all layer of the skin or the walls of hollow viscera such as the gastrointestinal tract. . It can be present as early as four weeks after starting treatment and symptoms can last up to 9 years. Patients can present with multiple non-specific complaints, including abdominal pain, diarrhea and weight loss. The mechanism of ACEI induced angioedema is still not fully understood. It is often a diagnosis of exclusion, although imaging modalities may help establish the diagnosis, like the MRE that led to a diagnosis in our case. Treatment include discontinue the ACEI and supportive care.

CONCLUSION

ACEI induce small-bowel angioedema is a diagnosis of exclusion; clinicians must be aware of this rare and potentially fatal side effect which can present as unexplained abdominal pain shortly after initiation of therapy. These patient will often undergo unnecessary invasive workup. Most importantly, failure to diagnose Angioedema can lead to life-threatening conditions.

 The authors have no financial disclosures or conflicts of interests



Mycobacterium Avium Complex Infection Cofounding Treatment of Hodgkin Lymphoma

NEW ORLEANS

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Introduction

- · When staging Hodgkin lymphoma, a positron emission tomography-computed tomography (PET/CT) scan used with fludeoxyglucose F18 (FDG) is the imaging modality of choice
- · FDG acts as an analog of glucose tagged with fluorine-18 and it is metabolized in a similar manner
- Tissues with a higher rate of glycolysis show a greater uptake of FDG on PET imaging. Higher rates of glycolysis in tissues are seen in malignancy, infection, and inflammation

Case Presentation

- · A 46-year-old woman with a history of mycobacterium avium complex (MAC) infection who began treatment one year prior and was currently receiving ethambutol, rifampin, and azithromycin. Upon presentation, patient reports two-month history to have progressive weight loss, night sweats, fatique, and weakness
- Excisional lymph node biopsy was performed which revealed classic Hodgkin lymphoma with mixed cellularity & also positive for acid-fast bacilli
- Initial FDG PET/CT revealed a Deauville score of 5 & the patient was initially stage IVb
- Received two cycles of adriamycin, bleomycin, vinblastine. and dacarbazine (ABVD)
- Repeat PET/CT demonstrated improvement in overall lymphadenopathy but still with an elevated Deauville score of 5 due hypermetabolic activity in neck, mediastinum and abdomen
- Chemotherapy regimen was escalated to four cycles of bleomycin, etoposide, doxorubicin, cyclophosphamide, vincristine, procarbazine, and prednisone (BEACOPP)
- Further FDG PET/CT scan also revealed a Deauville score of 5. Decreased metabolic activity in the spleen and in multiple cervical and pelvic lymph nodes. Increased activity right paratracheal lymph nodes and cervical level 4 lymph nodes
- Repeat exicsional lymph node biopsy favored MAC infection and was negative for lymphoma
- Ethambutol and azithromycin were continued for the MAC
- The patient was determined to be remission from her Hodakin lymphoma and was continued she remained on surveillance for Hodgkin lymphoma

Figures

Deauville score (DS)

- Score 1 no uptake
- □ Score 2 uptake ≤ mediastinum
- □ Score 3 uptake > mediastinum but ≤ liver
- □ Score 4: moderately ↑uptake > liver
- □ Score 5 markedly ↑uptake > liver and/or new sites of disease

Figure 1: The Deauville score is used in the initial staging and treatment response of Hodgkin lymphoma when using PET/CT

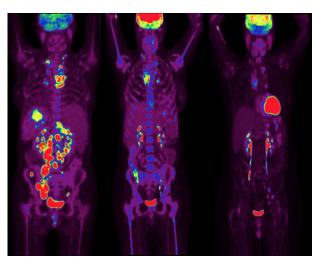


Figure 2: Pictured are three different images of maximum intensity projection nuclear medicine PET/CT using FDG. The far-left image is first PET/CT before chemotherapy, the middle image is after two cycles of ABVD, and the third image is three months after four cycles of BEACOPP.

Discussion

- · This case highlights the importance of viewing the entire clinical context when evaluating PET/CT scans
- FDG uptake by inflammatory and infectious processes can be mistaken for malignancy
- Common conditions that can cofound FDG PET/CT include infections, recent procedures, and recent radiation therapy
- The standard up take value (SUV) cannot discern between inflammation. infection, or malignancy
- Hodgkin lymphoma is a chemotherapy sensitive neoplasm. ABVD therapy cures around 70% of advanced-stage Hodgkin lymphoma patients. When given initially BEACOPP cures 85% to 90% of Hodgkin lymphoma patients
- Treatment success rate of pulmonary mycobacterium avium complex infections ranges from 32% to 65%
- · HL & MAC infections symptoms overlap
- · Recognizing these issues can aid in avoiding iatrogenic injury from chemotherapy toxicities, radiation therapy, and unneeded biopsies

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A Rare Case of Pericardial Abscess

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Introduction

Suppurative Pericarditis is an infection confined to the pericardial sac and is characterized by the presence of gross pus or microscopic purulence. (1) It a high mortality rate and prompt diagnosis and treatment are essential. (2,3) A rare complication of suppurative pericarditis is a pericardial abscess.

A review of the current literature yielded only a few cases of Staphylococcus Aureus pericardial abscess with only one other documented case in an

We present an unfortunate case of Staphylococcus Aureus pericardial abscess with tamponade physiology complicated by concurrent mycotic aneurysm of the ascending aorta, endocarditis, and cardioembolic strokes.

Case

A 48 y.o. male with a past medical history of Bipolar disorder, schizophrenia, and IVDU with recent MRSA bacteremia and suppurative pericarditis presented to the ED with shortness of breath for 4 days. He was admitted 7 weeks prior with MRSA suppurative pericarditis requiring pericardial drain placement for tamponade physiology but he left against medical advice. He had two subsequent admissions with recurrent pericardial drain placement but again left against medical advice. He then began to have progressively worsening shortness of breath a few days prior to admission and was found to be in respiratory distress upon EMS activation. On presentation, he was febrile to 103.9 and tachycardic. Physical exam was significant for an uncomfortable appearing diaphoretic middle-aged male with tenderness to palpation of the anterior chest wall, and increased JVP. Significant labs included a LA of 7.2, Cr of 1.4, BNP of 245, platelet count of 415, and a WBC count of 8.8. Toxicology was positive for opiates and cocaine. Blood cultures were positive for MRSA. The CXR showed cardiomegaly. His EKG demonstrated sinus tachycardia with right axis deviation and inverted T waves in the anterior and lateral leads. He was initiated on vancomycin and piperacillin-tazobactam and IVF. Shortly after admission, the patient had an acute change in mental status with concern for seizure like activity. A stat CT head was negative for acute intracranial abnormality. He was moved in the ICU and intubated for persistent tachypnea as well as airway protection. A stat echocardiogram revealed a large heterogeneous mass compressing the right ventricle as well as a dilated aortic root measure 4.2 cm. An MRI brain showed multiple scattered focal areas of restricted diffusion consistent with both acute and sub-acute infarcts in both cerebral and cerebellar hemispheres. CT chest with contrast confirmed an 11.8 x 6.6 cm pericardial fluid collection as well as a saccular aortic aneurysm suspicious for a mycotic aneurysm. He was not surgical candidate for aneurysm repair due to his poor clinical status and active infection. Drainage of the abscess was deferred due to overall poor prognosis. After a goals of care discussion with family, his status was changed to DNR and he was transitioned to comfort measures only.

Discussion

Bacterial suppurative pericardial effusions are rare causes of pericardial disease, estimated to account for less than 1% of pericarditis cases.(3) MRSA suppurative pericarditis is exceedingly rare in the modern antibiotic era, especially in the absence of previous surgical intervention with er than 10 cases noted in the current literature. (3)

The process of infecting the pericardium is thought to occur via one of a few different mechanisms including penetrating trauma (including surgery). direct extension from a neighboring intrathoraccic process, contiguous spread from an existing cardiac infection, or hematogenous spread^(3,8). Our ient was septic and also had co-existing endocarditis.

The diagnosis of suppurative perricaditis is primarily made with echocardiography and if needed, CT scan. (9)

One potential complication of MRSA suppurative pericarditis is pericardial abscess. These pericardial fluid collections have a high mortality rate and require prompt diagnosis in order to quickly initiate antibiotics therapy and arrange for drainage. One of the biggest contributors to mortality is tamponade physiology as was the case in this patient. (3,10)

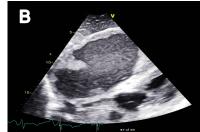
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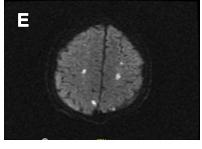
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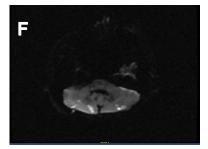












- A. Echocardiogram 4 chamber view with pericardial abscess compressing right ventrical
- B. Echocardiogram heterogenous pericardial abscess
- C. CT chest with contrast depicting ascending aortic aneurysm
- D. CT chest with contrast with large anterior pericardial abscess
- E. CT head without contrast DWI with bilateral cerebral infarcts
- F. CT head without contrast DWI with bilateral cerebellar infarcts



A rare case of refractory thrombocytopenia as a predominant presentation of early onset lupus, partially responsive to Eltrombopag

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INTRODUCTION

Mild thrombocytopenia is one of the common manifestations of SLE being present in 25 to 50 percent of patients with lupus. [1]. ITP is the most common cause of severe thrombocytopenia in SLE, with immune-mediated destruction of platelets and megakaryocytes as the pre-dominant mechanism.

CASE

A 14-year-old girl presented to ED with petechial rash, bruising and severe epistaxis which was treated with mechanical interventions such as tongue depressor alligator clip followed by nasal packing. Extensive work-up including bone marrow evaluations were negative for malignancy. She was presumptively diagnosed with ITP and treated with steroids and IVIG with no improvement. Rheumatologic workup done at that time was positive for hypocomplementemia, +ANA, dsDNA, anti-Smith, and RNP with evidence of schistocytes and low haptoglobin suggesting combined ITP and autoimmune hemolytic anemia. She continued to have worsening of symptoms despite being on Rituximab and long-term steroid taper and therefore started on Eltrombopag with mild improvement platelet count but clinically significant symptomatic improvement.

Overt bleeding and signs of thrombocytopenia have been controlled over past 3 years, despite having platelet count between 30-50 thousand. Patient is currently 18 years old living a normal life participating actively in her college basketball team.

DISCUSSION

Eltrombopag is a Non-peptide TPO receptor agonist approved by FDA for the management of ITP. It is highly effective with average platelet count ranging above 50,000. [2] Our patient did not have significant improvement in her platelet count, however had improvement in quality of life and reduction in clinically significant bleeding.

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A Cardio-Destructive Consequence of Cupid's Disease

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INTRODUCTION

Aortic dissection is a rare complication of syphilis in the antibiotic era which makes diagnostic assumption even more difficult.

Thoracic aortic aneurysm is a known complication of late syphilis with large aneurysms causing symptoms with a masseffect on neighboring mediastinal structures.

Risk factors for aortic dissection include uncontrolled hypertension, atherosclerosis, pre-existing aortic aneurysm, aortic valve defect, genetic disorders, infectious causes, connective tissue disorders.

This case describes a 46-year-old female who presented with left arm numbness and pain.

CASE DESCRIPTION

A 46-year-old African-American female with a history of hypertension, Crohn's disease and chronic hepatitis B presented with complaints of acute onset left arm numbness, pain and weakness when walking her dog. She initially presented 5 days before her current admission for Crohn's exacerbation and was adequately treated for the same. During her current presentation, she was significantly hypertensive with systolics in the 220s.

Physical examination was significant for decreased distal pulses in left upper extremity (LUE). Labs were only significant for mild leukocytosis. LUE arterial ultrasound showed distal subclavian vs proximal axillary artery thrombus with high bifurcation of the brachial artery and minimal distal ulnar artery flow. CT angiography of LUE confirmed the findings. Heparin drip was initiated and vascular surgery were consulted following which she underwent open embolectomy of the left distal axillary artery.

A CT angiography chest pulmonary embolism protocol was also ordered initially but was done after the embolectomy. It revealed filling defect within the distal descending aortic arch with early dissection suspected. The patient did not have any prior CT scans of the thorax.

CASE DESCRIPTION CONT.

Trans thoracic echocardiogram showed mild left ventricular hypertrophy and left ventricular ejection fraction of 60%. She was started on Heparin and Clevidipine infusion and was transferred to an outlying facility where she underwent thoracic endovascular aortic repair with successful stent placement. She was discharged on chronic anticoagulation.

Incidentally, during her initial admission for her Crohn's exacerbation, she was found to have a positive syphilis antibody and diagnosed as latent syphilis of unknown duration. Infectious disease was consulted at that time and recommended 2.4 million units benzathine penicillin G weekly for 3 weeks. She received her first dose at initial diagnosis.

RADIOLOGY



Image 1. CT Angio Thorax - Axial view



Image 2. CT Angio Thorax – Sagittal view

CONCLUSION

Reports of aortic dissection secondary to syphilis is uncommon in literature. In untreated syphilis, aortitis/aneurysm may manifest after 15-30 years from initial infection.

Management of aortic dissection depends on whether it is an ascending [Type A] aortic dissection or descending [Type B] aortic dissection.

Acute Type A aortic dissection is a surgical emergency. Type B dissection is generally managed medically initially, with surgical intervention reserved for those who develop complications related to dissection [dissection extension, malperfusion]

Endovascular repair is preferred over open repair for Type B aortic dissection. Open surgical repair may be needed for those whose anatomy will not support an endovascular stent–graft and for patients with high risk genetically mediated conditions.

This case highlights the potential complication of untreated syphilis and raises a question of whether we need to consider screening ultrasound of aorta or CT thorax in patients with latent untreated syphilis.

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Adenosquamous Carcinoma: An Abnormal Presentation of a Rare Malignancy

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Introduction

- Colorectal cancer is the 3rd most common cancer affecting Americans today. It is influenced by both environmental and genetic factors.
- Colon cancer is uncommon before the age of 40 and incidence increases significantly with each successive decade. Although the absolute incidence is highest in those age 50 and over, there has been an overall downtrend in rates observed among the screened compared to those age 20-39 who have had an increase in incidence.
- Interestingly, prevalence of signet cell histology was significantly greater in those under 40 years of age. Signet cell histology was also significantly associated with a more advanced stage at presentation, poorly differentiated tumor grade, and worse prognosis compared with rectal adenocarcinoma.

Case Description

A 22 y/o Hispanic male presented with RUQ abdominal pain and hematochezia for 2 months. Patient reported associated anorexia with 30 lbs weight loss.

On PE, the patient was noted to have RUQ tenderness to deep palpation with left supraclavicular and cervical lymphadenopathy. CT A/P w/contrast revealed thickening of colon splenic flexure suspicious for infiltrative neoplasm, bilobar hepatic metastasis, and diffuse abdominal and plevic lymphadenopathy.

Colonoscopy revealed large, infiltrative, friable mass of proximal descending colon and splenic flexure with 50% involvement of luminal circumference. Biopsies taken were consistent with poorly differentiated carcinoma, consistent with adenosquamous carcinoma (Ad-SCC) of the intestine.

Imaging



Image 1. CT Abdomen/Pelvis With Contrast



Image 2. Abdominal Ultrasound

Labs

LAB TEST	LAB RESULT
AST	52
ALT	79
ALP	197
Hb	11.8
Bilirubin	Normal

Discussion

Ad-SCC is a rare and aggressive cancer associated with a worse prognosis compared to adenocarcinoma. These are rare tumors, accounting for about 0.05-0.2% of all colorectal malignancies and are associated with higher overall colorectal specific mortality compared to adenocarcinoma.

The patient's young age and distant metastasis at presentation makes this an exceedingly rare presentation. However, recent studies have shown incidence of colorectal cancer has increased in patients under 40 and they are more likely to have signet cell histology when compared to those age 50 and above.

Given the worse outcomes associated with signet cell histology, thorough evaluation of young patients with rectal symptoms is necessary. Changing guidelines for earlier screening may be warranted.

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Cryoglobulinemia and Systemic Manifestation of Untreated HCV

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Introduction

Painful purpuric lesions, ulcers, arthralgias, peripheral neuropathy and glomerular nephritis should raise concern for cryoglobulinemia.

Additionally, A history of viral illness, particularly treatment naïve HCV in the settings of these clinical findings should raise suspicion.

Case Description

- 55 y/o female presented with complaints of abdominal pain and prolapsed ostomy s/p diverting colostomy secondary to Fournier's gangrene.
- PMH notable for recurrent skin infections requiring surgical amputation/debridements, treatment naïve HCV with cirrhosis.
- During admission, patient also complained of pain in hands with discolored digits with purpuric lesions.
- Abdominal US revealed splenomegaly.
 CT thorax revealed patchy consolidative changes of RLL, mediastinal and right hilar adenopathy.
- Rheumatology and ID were consulted.
- Typically treatment consists of prednisone and Rituxan. However, given concern of infection and poor healing in setting of planned ostomy revision, these treatments were held and colchicine BID was started. Patient was also started on EpIcusa, Ribaviran and Viread for prevention of HBV reactivation.

LABS

LAB TEST	LAB RESULT
CRP	9
ESR	40
C3	71
C4	<2.9
Cryoglobulin Ab	(+)
HepBs Ab	(+)
HepBsAg	(-)

Images



Left upper Extremity Digits (Nov 2020)



Left upper Extremity Digits (Dec 2020)

Conclusion

- Cryoglobulinemia should be considered when evaluating a patient with HCV as symptoms vary.
- Typical presentation includes LE palpable purupura (upper extremity in this patient).
- However, it is imperative to consider and rule out other diagnoses (Vasculitis, Buerger's, Systemic Sclerosis, APLA).
- Treatment goal is eradication of HCV since cryoglobulins are a systemic response to the virus.
- Cryoglobulinemia often presents with Meltzer's triad (purpura, arthralgia, weakness).
- If suspected, it is important to differentiate between types of cryoglobulinema when patient's PMH is unknown.
- Type I manifests with vascular occlusion such as digital ischemia and skin necrosis related to hematologic malignancy.
- Mixed type is typically associated with nonspecific symptoms (Meltzer's triad) and commonly associated with persistent viral infections or autoimmune diseases. Our patient showed additional subclinical pulmonary manifestations (small airway disease, dyspnea, organizing pneumonia) splenomegaly and lymphadenopathy consistent with mixed type.
- Recognizing cryoglobulinemia in patients with untreated HCV is important as it requires treatment of underlying disease for eradication of the virus and improvement of cutaneous and systemic manifestations.

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Stabilization of Cushing's Disease with Etomidate

Austin Tutor MD, Harmonjot Khaira MD, Cathy Wentowski MD

BACKGROUND

- Cushing's disease specifically refers to a pituitary adenoma which produces excessive ACTH leading to hypercortisolism. The incidence of Cushing's disease is approximately 1-2 per million per year.
- Classic cushingoid features include obesity, diabetes mellitus, striae, hypertension, weakness, headaches, fluid retention, moon facies, buffalo hump, and electrolyte abnormalities (hyponatremia, hyperkalemia).

CASE DESCRIPTION

- 56-year-old male presented due to recurrent anasarca with severe respiratory distress and hyperkalemia.
- CT imaging showed pulmonary nodules and adrenal hyperplasia. He was subsequently found to have elevated <u>ACTH of 317 and cortisol of 60.</u> CT head showed a large pituitary mass.
- Pulmonary nodules were ruled out with a PET scan. Given his severe symptoms, we elected to stabilize his cortisol levels prior to surgical intervention.

INTERVENTION

 Hypercortisolism was first treated unsuccessfully with ketoconazole; therefore, more aggressive management was pursued with an etomidate protocol developed by University of Wisconsin. <u>He was given a 5 mg bolus</u> <u>followed by infusion at 0.02mg/kg/hr, titrating by 0.02</u> <u>mg/kg/hr q6hr until goal cortisol (5-25mcg/dl) levels</u> were achieved.



OUTCOME

- After five days, his cortisol decreased <20 and he had symptomatically improved. He underwent transsphenoidal hypophysectomy which confirmed a strongly ACTH positive corticotrophin adenoma.
- Postoperatively, he developed adrenal insufficiency requiring stress dose steroids and was briefly treated for diabetes insipidus with DDAVP. He was discharged to rehab and is doing well today.

DISCUSSION & CONCLUSION

- Cushing's sources are classically pituitary, adrenal, or ectopic.
- ACTH levels and lung PET scan ruled out adrenal and lung sources, respectively. We elected against petrosal sinus sampling due to concerns for etomidate interfering with the results.
- Pituitary adenoma was the most likely source, so we decided to proceed with resection for both diagnosis and cure.
- The patient initially had severe metabolic disturbances and volume overload; therefore, etomidate, a known cortisol synthesis inhibitor, was crucial in reducing his perioperative risk.
- Overall, this case highlights a novel approach to stabilizing the effects of Cushing's disease prior to intervention.



Medullary Infarct in the Setting of Granulomatosis with Polyangiitis

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Introduction

Granulomatosis with Polyangiitis is a vasculitis that typically presents with upper respiratory, pulmonary, and renal symptoms, but can also affect the central nervous system increasing the risk of stroke. Medullary infarctions are rare but can present with a variety of symptoms, including vertigo, hemiparesis, blurred vision, loss of sensation, dysphagia, and respiratory dysfunction.

GPA is thought to be due to increased inflammation in cerebral vessels, as well as from granuloma formation from antineutrophil cytoplasmic antibodies (C-ANCA) against proteinase 3 (PR-3) and myeloperoxidase.

We present a case of a medullary infarct in the context of granulomatosis with polyangiitis.

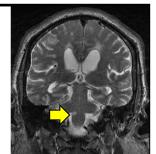
Case Description

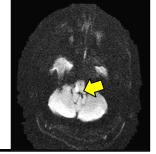
A 58-year-old male was admitted from the ED with hypertensive emergency, AKI, dysphagia, and sinusitis. His BUN/Cr was elevated to 52 g/dL and 3.6 mg/dL, respectively. His past medical history included CKD, hypertension, and type 2 diabetes mellitus. On the day after admission, the patient developed left arm numbness, weakness, right eye blurry vision, and a short-stepped gait.

Barium swallow study indicated a small hiatal hernia. A CT head without contrast did not reveal any acute changes. MRI of the brain revealed an acute infarction in the right medulla. The patient was started on Plavix and Neurology was consulted. A carotid U/S was unremarkable. Ophthalmology was consulted for the right eye blurry vision, but did not report any significant findings and recommended high dose solumedrol.

The patient's ESR and CRP were elevated at 95 mm/hr and 11 mg/L, respectively. Autoimmune workup came back positive for ANA and PR3, and high-dose steroids were initiated. A kidney biopsy was consistent with diabetic nephropathy. His blurry vision, strength, and dysphagia improved throughout the hospital stay. The patient remained admitted for 16 days and was discharged home with prednisone taper and Plavix.

Imaging





ANA lgG and

PR3

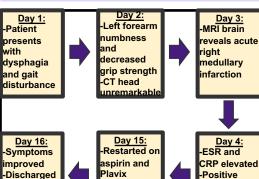
Image 1. T2 weighted MRI indicating infarct in right medulla

Image 2. Diffusion weighted MRI indicating infarct in right medulla

prednisone

taper

Timeline of Events



Conclusion

The presentation of acute cerebral infarction in the setting of granulomatosis with polyangiitis is uncommon, only occurring in approximately 2-8% of cases. The patient in this case presented with dysphagia, gait change, and abdominal symptoms initially. Symptoms could have been due to his chronic conditions including diabetes, CKD, and hypertension, but the new-onset arm weakness and blurry vision warranted further investigation. Medullary infarction in this setting is rarely seen and presentation is nonspecific, but due to the hypercoagulable state of the disease it should remain on the differential.

Medullary infarction can be secondary to several etiologies, but physicians should always consider the whole clinical picture. The associated symptoms in addition to his neurological deficits raised suspicion for widespread vessel involvement. This led to further diagnostic testing and an accurate diagnosis, allowing for appropriate treatment and follow up.

Current literature on presentation of granulomatosis with polyangiitis primarily addresses PNS involvement such as cranial nerve deficits, but previous reports exist of patients with CNS involvement including cerebral ischemia, hemorrhage, meningitis, encephalitis, and seizures. Prognosis depends on previous risk factors as well as the timing and severity of the infarction, but early detection and treatment proves vital in reducing disease progression. MRI should be a standard test ordered for patients with previous history of vasculitis, presenting with symptoms suggesting cerebral involvement.

In patients with underlying vasculitis, one must have a high index of suspicion for neurologic involvement as early recognition is vital to prevent increased risk of mortality associated with the disease. This can reduce diagnostic errors in patients with vasculitis as symptoms can be nonspecific. Early diagnosis is critical to improving patient survival and quality of life.

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Optimization of Guideline Directed Medical Therapy in **Outpatient Management of Heart Failure**

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Introduction

Heart failure accounts for a significant portion of emergency department (ED) visits and hospitalizations. Despite literature showing a significant mortality benefit in heart failure with reduced ejection fraction (HFrEF), many evidence-based strategies for treatment, particularly aggressive titration of dosages, have been underutilized.

In our Internal Medicine clinic, although most Physicians are very familiar with HFrEF and its medications, there are still inconsistencies in systematic optimization of quideline directed medical therapy (GDMT).

Study Aim

This QI project is directed towards Residents to achieve fulfillment of GDMT. We hope to observe clinical improvement in Internal Medicine clinic patients with HFrEF as evidenced by decreased ED visits and hospital admissions related to heart failure. Our intervention is a Resident education program primary aimed at raising awareness in proper implementation of GDMT.

Methodology

- After a literature review of established recommendations on improving mortality in HFrEF, patients of the LSUHSC-Lafayette Internal Medicine Residency outpatient clinic program were evaluated.
- A total of 26 adult patients with an EF of 40% or less were identified and followed for 6
- Fulfillment of GDMT was evaluated prior to any intervention, with attention to dosages as well as cardiology follow up.
- An increase in aggressive titration of HF medications was expected as illustrated in the GDMT trials.
- These included the COPERNICUS-HF, MERIT-HF. COMET. SOLVD trial. ATLAS trial. and PARADIGM-HF trials.
- The above trials were reviewed and a Resident education intervention was provided.

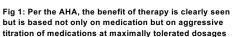
Results

- Preliminary results showed a modest increase in fulfillment of GDMT.
- Patients on suboptimal doses of either beta blockers or ACE inhibitors/angiotensin receptor blockers/neprilysin inhibitors were evaluated.
- Need for dosage increases were defined as patients not on maximally tolerated dosages of GDMT medications, and need for addition of spironolactone for ejection fractions below 35% per AHA guidelines.
- Prior to the intervention, 22/70 (31.4%) of GDMT parameters were met, compared to 4 months after the intervention in which 31/70 (44%) of the parameters were met.

Medical Therapy for Stage C HFrEF: Magnitude of Benefit Demonstrated in RCTs

GDMT	RR Reduction in Mortality	NNT for Mortality Reduction (Standardized to 36 mo)	RR Reduction in HF Hospitalizations
ACE inhibitor or ARB	17%	26	31%
Beta blocker	34%	9	41%
Aldosterone antagonist	30%	6	35%
Hydralazine/nitrate	43%	7	33%





American Heart Association

Discussion

Despite clear evidence of benefit of GDMT therapy, most patients do not reach their maximally tolerated dosages. Although data is preliminary, a modest improvement was seen with Resident education and awareness.



Conclusion

Although abundant literature exists on treatment of HFrEF and methodology of medical management, implementation proves to be difficult within the outpatient setting. A multidisciplinary approach would likely be more beneficial in reaching target dosages and ultimately showing the benefit illustrated in literature.

Resident education alone resulted in a relative increase of 13% fulfillment of GDMT within the setting of an Internal Medicine Resident clinic.

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The Neuropsychiatric Inferno of Lupus

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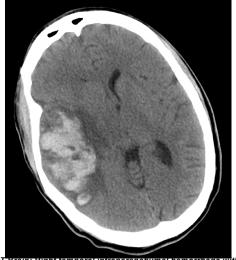
Introduction

- SLE is a common inflammatory autoimmune disease characterized by a heterogeneous clinical presentation.
- Among various life threatening clinical presentations of SLE, Lupus cerebritis is an umbrella term that describes the neuropsychiatric manifestations of lupus.
- Lupus Cerebritis can manifest with depression, anxiety, headaches, dizziness, memory deficits. More severe symptoms may include florid psychosis, seizures, stroke, and coma.
- Although LC poses a diagnostic challenge, early diagnosis is essential to decrease mortality and morbidity of patient.

Case Description

- 23-year-old African American female presented with a 5 month history of depressed mood and intermittent confusion.
- Ten days prior to admission she was evaluated in an emergency department (ED) for debilitating arthralgias.
- At that time, she was given 125mg of intravenous methylprednisolone and her symptoms resolved.
- Two days prior to admission, she was noted to be disoriented with expressive aphasia and illogical thinking.
- On arrival to our ED, she was noted to have worsening confusion and somnolence after a generalized tonic-clonic seizure.
- Autoimmune work-up ensued and was notable for positive Anti nuclear antibody(ANA), dsDNA, anti-Smith antibody, SSA antibody, RF IgG, IgM. C3 and C4 were also low.
- Cerebrospinal fluid analysis revealed leukocyte count of 42/mL with 63% polymorphs,31% lymphocytes, an erythrocyte count of 3,000/mL, glucose of 37 mg/dL, and protein of 230.9 mg/dL.

Imaging



CT Brain: Right temporal intraparenchymal nemorrhage with right-to-left midline shift. Mass effect with Sulcal effacement can also be noted.

Conclusion

- Lupus Cerebritis carries a poor prognosis and up to nine-fold increase in mortality when diagnosed late in the disease course.
- Early diagnosis and treatment is essential to reducing morbidity and mortality.
- Spontaneous ICH, which may result from thrombocytopenia, hypertension and hyperlipidemia, is a serious comorbidity of patients with SLE.
- Our patient presented with a spectrum of mood changes, confusion, seizures, and eventual intracranial hemorrhage.
- It was later confirmed that her disease course was due to Lupus Cerebritis.
- As physicians we should be more cautious when a SLE patient presents with spontaneous ICH for its greater instability compared with the general population.
- In such cases, besides removing the hematoma, decompressive craniectomy may be considered earlier, even when initial intracranial pressure is not high, to alleviate the possibility of multiple spontaneous ICHs and cerebral swelling.

Case Description cont.

- Magnetic Resonance Imaging of the brain revealed bitemporal cortical-based signal abnormality with petechial hemorrhages.
- There was a concern for antiphospholipid antibody syndrome and the patient was fully anticoagulated.
- On the third day of hospitalization, the patient was noted to have acute loss of motor function and sensation of her left side.
- Subsequent computed tomography scan of the head revealed a 7.5 cm right temporal intraparenchymal hemorrhage with a 7mm midline shift.
- She was emergently transferred to a tertiary care center for craniotomy.

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New Changes or Old News? How The Medical History Can Change a Diagnosis

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Learning Objectives

- 1. Review the clinical presentation of post-stroke neuropsychiatric disorders
- 2. Recognize the value of obtaining an accurate history

Case Report

A 68-year-old man presented with confusion and bizarre behavior for one month. He had a history of CVA approximately 5 weeks prior, ESRD, and poorly controlled diabetes mellitus. No psychiatric history was reported.

He was afebrile and hypertensive. Neurologic exam revealed residual expressive aphasia and impaired concentration and memory. No strength deficit was appreciated.

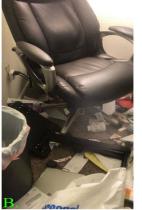
Psychiatric examination revealed disinhibition and inappropriate behavior with expansive affect characterized by grandiosity, mood lability, and flight of ideas.

Labs

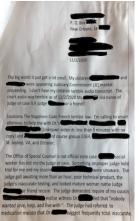
Glucose: 120 mg/dL	RPR:
Calcium: 8.9 mg/dL	nonreactive
Sodium: 141 mEq/L	pH: 7.39
TSH: 3 mU/L	PaCO2: 40
Hemoglobin: 8 g/dL	mmHg

Vitamin B_{12:} >1000 pg/mL **PaO2:** 80 mmHg









Images of the patient's apartment obtained from collateral contacts (A, B). Cognitive assessment documentation (C). Documentation from the judicial commitment hearing (D). Images of the patient's apartment were taken 1 month prior to the patient's CVA.

(A) A knife and shattered glass covering the kitchen floor. (B) Office which collateral contact described as a "walking hazard." (C) Montreal Cognitive Assessment completed day 7 of hospitalization. (D) Page 1 of 3 of the patient's self-drafted letter to the judge presiding over the judicial commitment hearing.

Discussion

Neuropsychiatric disorders are common after stroke, with depression and anxiety presenting in 35% and 25% of stroke survivors, respectively. Neuroplasticity changes post-CVA can also lead to behavioral changes that include

- Catastrophic Reactions
 - disruptive emotional outbursts involving agitation or aggressive behavior
- Pathologic Affect
 - mood and emotional lability often expressed as pathologic laughing and crying

Primary psychiatric disorders, however, may be mistaken for post-CVA neurocognitive disorders, especially when accurate baseline function is difficult to obtain.

An accurate history is of utmost importance when assessing baseline function in stroke survivors who subsequently present with

- 1) neurocognitive symptoms that do not correspond with the location of infarction
- 2) manic symptoms
- 3) communication difficulties that hinder accurate self-reporting.

It is critical for the internist to obtain collateral information that contextualizes patient baseline behavior, personality, and functionality.

Intervention and Result

Mental health consultation was obtained and due to lack of history of longstanding symptoms, the patient's clinical picture corresponded with a neurodegenerative post-CVA sequelae. The patient was treated with Quetiapine and Divalproex Sodium which improved manic symptoms.

Take home points

- An accurate history is the primary tool to distinguish comorbid psychiatric disease from post-CVA neurocognitive disorder
- Distinguishing primary psychiatric disease from post-CVA neurocognitive sequelae can be consequential to a patient's hospital course and disposition.

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Ticagrelor Induced Diffuse Alveolar Hemorrhage after PCI Intervention

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Introduction

Diffuse alveolar hemorrhage (DAH) is a medical emergency that must be immediately recognized and treated. It is characterized by bilateral pulmonary infiltrates, hypoxia, hemoptysis, and/or falling blood count.

Treatment ranges from supportive measures, to withdrawal of offending agents, steroid administration, plasmapheresis, and/or direct immunosuppression.

Here we will discuss a case of Ticagrelor (Brilinta) induced DAH. Ticagrelor is an orally administered direct-acting P2Y₁₂-receptor antagonist that bind reversibly and noncompetitively

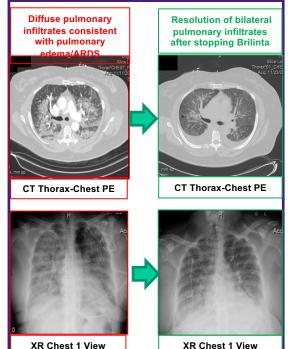
Case Description

- A 56 -year-old African-American female with a history of hypertension, hyperlipidemia, diabetes, and tobacco abuse presents with one-week of episodic, sharp, pressure-like, substernal chest pain that worsened with activity.
- EKG at admission revealed sinus tachycardia with ST elevations in the inferolateral leads and elevated troponin-l.
- She was given Aspirin, started on a heparin drip, and underwent emergent PCI with DES x 2 to the RCA and Circumflex artery.
- Post-procedure she was bolused with tirofiban, loaded with ticagrelor $180~\mathrm{mg}$, and transferred to the ICU.
- In the morning she was started on dual antiplatelet therapy (DAPT) with Aspirin 81mg and Brilinta 90mg, Atorvastatin 80mg, and Lopressor 25 mg BID
- Meanwhile, overnight she developed SOB and hemoptysis.

Physical Exam/Laboratory Data

- Vitals were concerning for fever, tachycardia, and tachypnea.
- Additional physical exam findings were significant for bilateral rhonchi and coarse breath sounds.
- Labs revealed hyponatremia, elevated BNP, leukocytosis and a hemoglobin decrease from 13.5 to 11.5.
- CT Angiography revealed diffuse bilateral infiltrates and CXR revealed worsening bilateral opacities concerning for DAH.

Diagnostic Imaging



Clinical Course

- Brilinta was immediately discontinued and replaced with Plavix. Broad spectrum antibiotics were started.
- Ultimately, she was intubated for acute hypoxemic respiratory failure, DAPT was discontinued, and methylprednisolone administered.
- She was extubated after a bronchoalveolar lavage, which revealed blood-tinged aspirate without signs of active bleeding.
- Aspirin and Plavix were restarted once her hemoptysis resolved. Her shortness of breath improved as well as her lab and imaging findings.
- She was discharged home with home oxygen with close pulmonology and cardiology follow-up

Discussion

- This case highlights the need for immediate recognition of Ticagrelor induced DAH and discontinuation of therapy in preventing life threatening progression to acute hypoxic respiratory failure.
- This can be recognized via clinical symptoms/signs, imaging, and/or worsening respiratory status on ABG

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What's Causing that Fever?

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Introduction

Here we present a case of a young female with persistent fever of unknown origin.

Case Description

A 23-year-old African-American female with no past medical history presented to the ED with fever for 7 days.

Pain in the right knee and ankle associated with swelling and soreness had improved with activity 1 week prior.

The patient ultimately visited Urgent Care 1 day later where she was found to be febrile and prescribed Amoxicillin.

Joint pains resolved but she continued with fevers up to 103 F. She also began to notice an erythematous rash on her arms and chest associated with the fever.

In the ED, she was started on broad spectrum antibiotics and infectious workup was underway. WBC rose to 19,000 on day 1 with her inflammatory markers significantly elevated and remained febrile.

CT abdomen/pelvis performed that day revealed abdominopelvic lymphadenopathy as well as bilateral axillary lymphadenopathy.

Infectious workup was negative; however, ferritin was >13,000. Rheumatology was consulted at that time.

IV Solu-Medrol was started in which the patient responded well. Fevers abated on day 2 of admission.

Labs

TEST	RESULT
Sodium	136
Potassium	3.4
Chloride	104
CO2	23
Calcium	8.2
Glucose	101
BUN	6
Creatinine	1.00
GFR	88
Total Bilirubin	0.3
Direct Bilirubin	0.2
AST	67
ALT	38
Alk Phos	50
LDH	503
Ferritin	13,375
CRP	8
ESR	71
WBC	19.4
Hemoglobin	11.3
Hematocrit	35.6
Platelets	312
C3	24
C4	110
Globulin	26
Procalcitonin	0.64
IgG	1711
IgA	463
IgM	236
Urine protein/creatinine	202
ANA titer	1:80
ANA	Positive
dsDNA	Negative
RNP	Negative
MPO	Negative
PR-3	Negative
ANCA	Negative
SSA	Negative
SSB	Negative
Smith Antibody	Negative

Imaging

IMPRESSION

- No definite CT evidence of focal abnormality or other suspicious process to suggest source of patient's fever.
- Enlarged bilateral axillary and abdominopelvic lymph nodes are nonspecific and could represent element of reactive adenopathy. However, more sinister cause for lymphoproliferative disease is not entirely excluded and close monitoring recommended to ensure resolution.
- Additional chronic secondary details discussed above.

Conclusion

- The patient requested to be discharged and was sent home with prednisone 60 mg with a steroid taper, OI prophylaxis with Bactrim DS 3 times weekly, and Protonix for GI prophylaxis per Rheumatology.
- Patient has since been seen by Rheumatology as well as our medicine clinic and has done well on prednisone 60 mg/day with steroid taper. She is currently no longer on prednisone and has had no recurrence of fever or rash.
- Adult onset Still's disease is a rare autoimmune disease on the spectrum of systemic onset juvenile arthritis causing high spiking quotidian fevers, arthritis, rash, and lymphadenopathy.
- It is generally seen in patients aged 15-25 and 36-45 years. Some common associated lab abnormalities include neutrophilic leukocytosis, abnormal liver function, and elevated acute phase reactants (ESR, CRP, and ferritin).
- We present this case to remind Physicians to always keep this disease on your mind when a patient presents with fever of unknown origin with negative infectious workup.



Classic Myopericarditis in a Young Male

Austin Tutor MD, Caley McIntyre

BACKGROUND

- The pericardium consists of two layers, a serous visceral layer and a fibrinous parietal layer, which create a potential space, the pericardial cavity.
- Acute inflammation of the pericardium is referred to as pericarditis. Conversely, myocarditis is inflammation of the muscle tissue.

CASE DESCRIPTION

- 41-year-old male with no significant past medical history who presented due to chest pain with associated shortness of breath following a 2-week history of fever, cough, and myalgias.
- The pain was worse with lying flat and improved with sitting upright/leaning forward.
- On arrival, patient was hemodynamically stable and febrile. He was noted to have an elevated troponin of 8 and <u>EKG with diffuse 2mm ST</u> <u>elevations</u>. Additional labs showed elevated CRP and ESR.
- Subsequent TTE revealed ejection fraction of 55%, no wall motion abnormalities, normal diastolic function, and no pericardial effusion.
 <u>Troponin eventually peaked at 24</u>. He was diagnosed with viral myopericarditis.

INTERVENTION & OUTCOME

- He was started on ibuprofen and colchicine with improvement of his symptoms and down trending of his cardiac biomarkers.
- He was discharged home with cardiology follow up. Upon outpatient visit he had no cardiac symptoms or complications.

DISCUSSION & CONCLUSION

- Classically, pericarditis presents as positional and pleuritic chest pain.
- Physical exam findings can include a friction rub, signs of pericardial tamponade (pulsus paradoxus, hemodynamic instability), or signs of constrictive pericarditis (Kussmaul sign).
- Labs usually show elevated inflammatory makers. EKG typically reveals diffuse ST elevations and PR depression.
- If the myocardium is involved, patients can also present with elevated cardiac biomarkers, arrhythmias, or depressed cardiac function with global or regional wall motion abnormalities. TTE is crucial for evaluating evidence of tamponade and cardiac function. If regional wall motion is present, patients should undergo further evaluation with a left heart catheterization to rule out



A Case of Critical and Acute Decline in IgA Nephropathy

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Introduction

IgA Nephropathy (IgAN) is considered one of the most common primary glomerular diseases worldwide and is especially prominent in the Asian and Caucasian community's around the world (1). Clinically, this disease can present at any age though with peak incidence in the second/third decades of life (2). Historically, the prevalence in the USA ranges in 16th percentile while the prevalence increases as you travel east (2). While the mechanism of damage is clearly understood the cause of activation of IgA deposition remains unclear.

Case Description

- 34-year-old Vietnamese male with complaints of abdominal pain, constipation, nausea/vomiting, indigestion and weakness for approximately 1 week.
- Patient noted to be jaundice, tachycardic, tachypneic and hypoxic.
- BiPAP initiated and transferred to the ICU.
- CXR: diffuse ground-glass airspace disease.
- His condition rapidly decompensated; eventually requiring mechanical ventilation.
- Emergent dialysis, plasma exchange, high-dose steroids x3 days.
- Pulmonary-renal syndrome was suspected at that time. Rheumatologic and nephrotic disease specific markers: positive HBcAb and a suppressed IgG while all other testing (including C3 and C4 levels) returned negative or WNL.
- Percutaneous Renal Biopsy: Sclerosing IgAN with severe interstitial fibrosis and tubular atrophy (Figures 1-3)

Pathology Images



Figure 1. Percutaneous Renal Biopsy

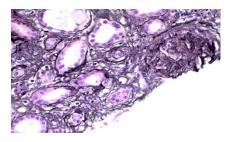


Figure 2. Percutaneous Renal Biopsy

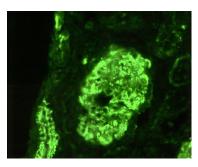


Figure 3. Percutaneous Renal Biopsy

Laboratory Findings

Chemistry:

Na 130 K 5.2

CO2 14

BUN/Cr 183/30

Lipase 811 Lactate 3.3

Hematology:

WBC 13.8

H&H 3.5/10.8, Retic Count 5.2

ESR >140 CRP 7.6

COVID-19: Negative

Peripheral Smear: Anisopoikilocytosis, reactive neutrophils consistent with chronic disease and acute inflammatory process

Urine Chem: Prot/Creat 4,778

Compliment (C3/C4): WNL

Conclusion

IgAN is a particularly fascinating disease that manifests with regularity in the Asian population. This presentation of IgAN occurring concomitantly with a pulmonary-renal syndrome picture was an interestingly cryptic and suggestive manifestation of the disease that we felt was worth noting.

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A Puzzling Case of Optic Neuritis

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Introduction

Neuromyelitis Optica (NMO), also known as Devic's disease, is a demyelinating autoimmune disease affecting the central nervous system. It has been historically considered a variant of multiple sclerosis (MS).

Patients typically present with attacks of optic neuritis and acute transient myelitis. A definitive diagnosis is made by the detection of autoantibodies against Aquaporin-4 (AQP4-IgG).

Case Description

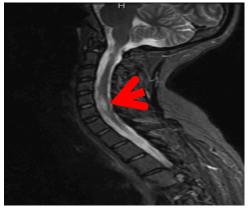
- 35-year-old right-handed female.
- No significant past medical history. No recent trauma.
- 4-day history of sudden pain/numbness affecting left arm and right lower extremity causing significant difficulty with ambulation.
- She endorsed visual hallucinations, retro-orbital pain with lateral and upward gaze, and decreased visual acuity. She denied any changes in color.
- She was admitted for seizure-like activity (tonic spasms) and further workup.

Imaging

CT Head

•No acute intracranial hemorrhage or edema MRI Brain

No active intracranial lesions.



MRI cervical spine:

·Signal changes on the left C5-6 suggesting

aemyeiination

Laboratory Findings

Lumbar puncture results:

- Positive for myelin basic protein (12.3)
 - · Positive for oligoclonal bands.
 - Positive for protein (23.1)
 WBC < 3. no RBC
 - VDRL non-reactive

HIV testing: Negative Autoimmune workup:

- RNP, MPO, ANCA, ANA, Anti-Smith, dsDNA, SSa, SSb: negative
 - Serum NMO-Ab level positive (226.9)
- The patient did not meet clinical McDonald criteria for multiple sclerosis.
- Solumedrol was started at that time due to high suspicion of an autoimmune process, which improved her symptoms.

Conclusion

The high probability of a misdiagnosis of NMO for other autoimmune disease processes is what leads us to present this case. Due to the relative rarity and lack of literature for NMO, we felt a discussion of our patient's presentation was necessary to expand on such sparse information.

Greater consideration for the testing of AQP4-IgG in cases where the diagnosis may be unclear could lead to increased detection. However, we must note that multiple autoantibodies have been indicated in this disease noting that approximately 10% of patients with NMO are seronegative for AQP4-IgG.

As with any disease, the goal is always to find a cure which starts with a better understanding of prevalence of the disease.

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Treatment Resistant Prolactinoma: Reasons Beyond Non-Compliance

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Introduction

- Prolactinomas are the most frequently occurring pituitary tumor.
- Hypersecretion of prolactin from these adenomas suppresses gonadotropin function leading to deficiencies in sex hormones.
- Larger tumors can suppress TSH and ACTH by mass effect.
- In most cases, Cabergoline therapy leads to normoprolactinemia and significant tumor shrinkage.
- Treatment-resistant prolactinomas require more invasive approaches likes surgery and radiation which greatly increase morbidity and mortality.
- The Ki-67 index is a burgeoning metric that could be used to identify prolactinomas which are more likely to resist treatment and recur.

Case Presentation

- A 21-year-old man with one-year history of seizure and prolactinoma presented with a new seizure.
- His first and only seizure occurred one year prior, and brain MRI discovered a large 5.7 x 4.6 x 6.2 cm macroadenoma filling his pituitary fossa.
- Prolactin at that time was >2,000 ng/mL, and additional labs included TSH of 0.329 uIU/mL, total testosterone of 86 pg/mL, free testosterone of 10.5 pg/mL and LH of 6.81 mIU/mL
- He was diagnosed with prolactinoma and discharged on Cabergoline, Keppra and Synthroid with regular follow-up scheduled with Endocrinology.
- Over the past year the patient remained seizure-free on his stable dose of Keppra.
- Prolactin levels continued to be >2,000 ng/mL despite compliance with maximum dose Cabergoline therapy of 1 mg twice weekly.

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Diagnostics

- A new brain MRI revealed a 5.7 x 5.7 x 6.3 cm adenoma increased in size from the previous study.
- The mass filled the pituitary fossa and now invaded the clivus, the left cavernous sinus and the left orbital apex.
- It also extended into the medial aspect of the left temporal fossa and caused mass effect and leftward displacement of the left temporal lobe.
- Prolactin was still elevated at >2,000 ng/mL and TSH, T4 and 8:00 am Cortisol were within normal limits.



Management

- Neurosurgery was consulted and the decision was made to proceed with endoscopic transsphenoidal resection of approximately 1/3 of the tumor mass.
- The patient received 100 mg of Solu-Cortef and 1,000 mg of Keppra perioperatively.
- A tissue biopsy was collected from the prolactinoma and sent to the pathology lab which revealed a Ki-67 proliferative index of 15%.
- His IV steroids were transitioned to PO Cortef 20 mg BID during hospital stay on which he was discharged.
- While in hospital, his urine specific gravity and urine osmolality were monitored for the development of post-operative Diabetes Insipidus.
- They remained within normal range and were 1.013 and 658 respectively at discharge.
- Neurosurgery plans to see patient again in 4-6 weeks to perform a left middle fossa craniotomy to remove remaining 2/3 of the tumor.

Discussion

- The first-line treatment for macroprolactinomas (size >1 cm) is with dopamine agonist therapy.
- Cabergoline is preferred as the initial agent due to better toleration of side effects.
- Research on the success of Cabergoline therapy includes a prospective study achieving normal prolactin levels in 81% of patients with macroprolactinomas and significant tumor shrinkage in another 92%.
- Another study showed normoprolactinemia reached in 77% of patients with macroprolactinomas.
- Dopamine agonist therapy should be escalated to the maximum dose before surgical intervention is considered.
- Dopamine agonist resistance is defined as the failure to achieve normal prolactin levels or reduce tumor size by 50% while receiving the maximum dosage.
- Transsphenoidal surgery is the next step to treat macroprolactinomas in patients who could not tolerate dopamine agonists or did not respond to therapy.
- A retrospective study observed a 60% remission rate in patients with non-invasive macroprolactinomas treated with transsphenoidal resection.
- Diabetes Insipidus is a common complication of transsphenoidal surgery for all pituitary adenomas including prolactinomas, with transient DI presenting in 10-30% of patients and permanent DI in 2-7%.
- Ki-67 is a protein marker associated with proliferative activity and invasiveness of tumors.
- The average Ki-67 proliferation index for prolactinsecreting adenomas was measured to be 3.37% +/-1.8%.
- Research has shown that invasive pituitary adenomas have elevated Ki-67 indices when compared to non-invasive ones, and a high Ki-67 index with MRI evidence of cavernous sinus invasion has been associated with a high likelihood of posttreatment recurrence.
- This patient's markedly elevated Ki-67 proliferation index of 15% might explain why his adenoma resisted maximum dose Cabergoline therapy, and the need for close monitoring for recurrence after surgical resection.



Bacteremia due to an Atypical Source of Endocarditis

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Introduction

- Infective endocarditis (IE) is an infection of the endocardial surface of the heart. Most often, it describes the infection of one or more heart valves.
- Between 2000 and 2011, IE was noted to have an incidence in the United States of about 15 per 100,000 population.
- İE continues to be characterized by increased morbidity and mortality and is now the third or fourth most common life-threatening infection syndrome, after sepsis, pneumonia, and intra-abdominal abscess.
- Typically, nonvalvular endocardial infection is secondary and is associated with valvular vegetations, myocardial abscesses, cardiac structural abnormalities, mural thrombi, cardiac tumors, or prosthetic materials.
- Primary mural endocarditis is an extremely rare infection in which nonvalvular endocardial involvemen is seen without any cardiac structural abnormalities such as ventricular septal defects.

Case Description

- A 53 year-old male with a history of HIV disease and intravenous drug use (IVDU) presented with shortness of breath and chest pain associated with syncopal episodes at home and difficulty with ADLs.
- On arrival he had fever, tachycardia, tachypnea and hypotension. Workup was significant for acute renal failure and lactic acidosis.
- He was then admitted to ICU for septic shock and started on norepinephrine. Cultures were drawn and broad spectrum antibiotics with vancomycin and meropenem were initiated.
- He required hemodialysis shortly after admission due the development of oliguria despite aggressive fluid resuscitation.
- Blood, urinary and respiratory cultures grew methicillin resistant staph aureus (MRSA). Antibiotics were deescalated to vancomycin.
- A transthoracic echocardiogram (TTE) revealed a new reduced ejection fraction of 20% and an apical echogeni mass. Blood cultures persistently grew MRSA.
- The patient then went into acute respiratory failure requiring intubation. A CT thorax was done and revealed septic emboli. Once the patient was stabilized, a transesophageal echocardiogram (TEE) was performed which confirmed an echodensity at the apex of the right ventricle with features suggestive of mural endocarditis.
- Because of his renal failure and inability to achieve appropriate vancomycin trough levels, his antibiotic regimen was changed to Linezolid for anti-MRSA and antitoxigenic coverage in addition to Ceftaroline to provide a bactericidal antistaphylococcal agent.
 Daptomycin was deemed an inadequate antibacterial choice given the infection's pulmonary involvement.
- Due to his continually positive blood cultures, he was then transferred to another facility for CV surgical intervention.
- Ultimately he was able to clear his cultures, made clinica improvement and was stepped down to the floor.

Imaging

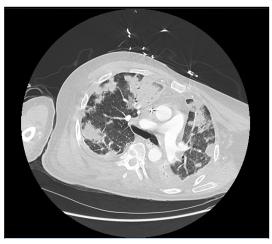


Image 1. CT Thorax with Contrast showing numerous scattered areas of infiltrate which are each focal and have signs of early central cavitation which are findings consistent with septic emboli

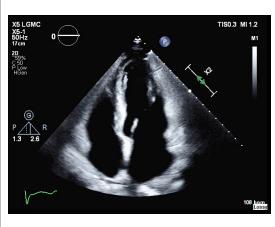


Image 2. Transthoracic Echocardiogram demonstrating apical echodensity concerning for mural endocarditis

Conclusion

- This case demonstrates the range of complications that can occur due to an infected mural endocarditis in the setting of an immunocompromised patient with a history of IVDU.
- The choices made in antibiotic therapy required careful consideration given the patients new onset renal failure in addition to the location of his endocarditis.
- Infective endocarditis (IE) can evolve into a complicated case of mural endocarditis that has a high risk of mortality. IE is defined by the microbial colonization of an endocardial surface.
- The most common pathogens noted in mural endocarditis are staphylococci, viridans streptococci, Enterococcus species, Salmonella species, Bacteroides fragilis, Candida species, and Asperdillus species
- Differentials can include thrombus, cardiac tumors, angiosarcomas, or myocardial abscesses.
- Mural endocarditis is rare and can develop in any of the cardiac chambers, particularly in a critically ill patient who has myocardial abscesses. It is difficult to establish a diagnosis.
 The use of TTE and TEE is necessary not only for diagnosis, but for identification of the size and location of the lesions as well.
 Evaluation for surgical intervention is a key component to management.
- In the 19 cases reported in the literature, vegetations were located in the left ventricle in 13 patients (68%), in the right ventricle in 4 patients (21%), in the left atrium in 1 patient (5%), and in both ventricles in 1 patient (5%). In the patients who had left ventricular mural vegetations, these were located at the apex in 4 patients (29%), at the posteromedial papillary muscle in 3 patients (21%), in the outflow tract in 2 patients (14%), on the intraventricular septum in 2 patients (14%), at the anterolateral papillary muscle in 1 patient (7%), on the lateral wall in 1 patient (7%), and at both the apex and posteromedial papillary muscle in 1 patient (7%).
- Mural Endocarditis is typically noted with predisposing factors such as congenital anomalies, devices, and/or systemic immunosuppression as was in this case. Without risk factors, it is highly unlikely to develop a hematogenous infection of this kind.

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Avoiding TEE and Invasive Intervention in Low Risk PFO

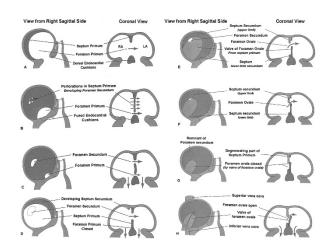
A Case of Paradoxical Stroke

Brandon Bunol, MD. Kyler Kingston, MD. Osaid Saqqa, MD. Hanyuan Shi, MD.

Tulane University, New Orleans, LA

BACKGROUND

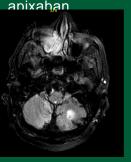
Patent foramen ovales (PFO) can be incidentally found as part of a stroke work-up on a transthoracic echocardiogram (TTE) with bubble study. Transesophageal echocardiogram (TEE) is the standard for elucidating the size of the defect, however, is an invasive procedure with risk and cost. This case describes using PFO scoring systems and TTE to avoid overutilization.

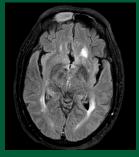


CASE

A 65-year-old man with hypertension initially presented after being found down at home. EKG showed sinus rhythm with PACs. MRI / MRA brain (figure) showed new infarction of superior left basal ganglia and posterior left hippocampus with old lacunar infarcts (Figure). A TTE showed EF > 55%, with positive bubbles crossing the septal wall after 4-5 cycles concerning for PFO or from bubbles passing the lungs. Venous doppler showed left femoral and brachial thrombus. There was a concern for paradoxical embolus from the PFO and possible need for surgical closure.

The ROPE (Risk of Paradoxical Embolization) score was calculated to be low at 3 (age, hypertension, previous stroke on imaging). Although cryptogenic stroke was in two different territories, telemetry did not show any arrythmias, and the decision was made to treat with apixaban (for DVTs) and aspirin for secondary prevention. TEE was not performed after discussion with cardiology as CVA subcortical and less likely secondary to embolic phenomena. Patient was discharged with aspirin and







MRI brain with contrast showing tiny ischemic infarction involving superior aspect of left basal ganglia and posterior aspect of left hippocampus. MRA H/N shows no further stenosis, aneurysmal dilatation

DISCUSSION

PFO associated with cryptogenic stroke, in 20-25% of all adults, but in 40% adults with cryptogenic stroke. The RoPE score was developed from a study of 3,023 patients from 12 databases and a predictive model was developed to stratify

Age (y)	
18–29	+5
30-39	+4
40–49	+3
50-59	+2
69–69	+1
≥ 70	+0
Total RoPE score	0-10

Patient Characteristic	Points
No history of hypertension	+1
No history of diabetes	+1
No history of stroke or TIA	+1
Nonsmoker	+1
Cortical infarct on imaging	+1

ROPE SCORE	PFO PREVALENCE (%)	PFO ATTRIBUTABLE FRACTION (%)
0-3	23	0
4	35	38
5	34	34
6	47	62
7	54	72
8	67	84
9-10	73	88

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Diagnostic Difficulties of Pleural Tuberculosis

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Introduction

Case Description cont.

Conclusion

Mycobacterium tuberculosis primarily infects the lung parenchyma but also spreads to the host's blood stream causing extrapulmonary disease. One of the most common forms of extrapulmonary tuberculosis (TB) is pleural tuberculosis, which presents as an exudative lymphocyte rich effusion.

Tuberculous pleural effusion usually presents acutely with fever, cough, and pleuritic chest pain often in an immunocompetent host. Diagnosing pleural tuberculosis is diagnostically challenging.

Sputum acid-fast bacilli (AFB) cultures were collected. The patient underwent thoracoscopy and a biopsy of the parietal pleura was taken.

The patient was discharged to complete RIPE therapy.

At week five, sputum cultures from the pleural tissue was positive for acid-fast bacilli, more specifically, Mycobacterium tuberculosis.

- The gold standard for diagnosis remains detection of mycobacterium tuberculosis in pleural fluid, pleural biopsy specimen, or histologic demonstration of caseating granulomas in the pleura along with acid fast bacteria.
- Unfortunately, as seen in our case, TB culture limits immediate utility and PCR sensitivity can be insufficient.
- The easiest way to diagnose pleural tuberculosis, in areas of high prevalence of disease, is with elevated ADA. This test is only 92% sensitive with a cut off of 40 U/L.
- Our case illustrates that even when the adenosine deaminase is well below the range of abnormal, tuberculosis should remain high on the differential in high-risk populations.
- Multiple tests are available to aid in the diagnosis of tuberculosis, nevertheless the disease can still be elusive.
- Diagnostic procedures for tuberculosis should not be abandoned in the context of a negative ADA.

Case Description

21-year-old male immigrant from Mexico who presented with fever, nonproductive cough, dyspnea, chest pain, fatigue, and weight loss

Left pleural effusion seen on X-ray

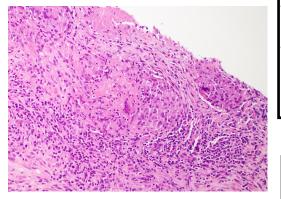
Thoracentesis revealed lymphocyte rich exudate (97%)

Pleural fluid revealed:
Low pleural fluid adenosine
deaminase (ADA).
Negative PCR of pleural fluid.
Negative T-spot tests.

Clinical suspicion of tuberculosis remained high.

RIPE therapy was initiatied.

Pathology



Parietal pleura biopsy revealing several necrotizing granulomas

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Renal Medullary Carcinoma: A Tragic Diagnosis

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Introduction

Renal medullary carcinoma (RMC) is a rare aggressive malignancy found almost exclusively in African Americans with sickle cell trait. Males are more commonly affected in a 2:1 ratio. The hallmark clinical symptoms are hematuria, flank pain and palpable mass, with hematuria often associated with thrombosis and renal ischemia. Due to its rarity, only one observational study has been done describing treatment and outcomes of patients with RMC and evidence based treatment quidelines are not currently delineated.

Review of Literature

Renal medullary carcinoma (RMC) is a non-clear cell carcinoma that makes up <0.5 % of all renal cell carcinomas (5). Typical presentation of RMC is a young African American male with sickle cell trait or less commonly hemoglobin SC disease (3). Primary presenting symptom is gross hematuria with or without flank pain and 75% of these cases have right sided kidney origin.

There are distinct immunohistochemical markers associated with RMC. It often exhibits expression of cytokeratin AE1 and 3 which correlates with a epithelial classification as does most RCC (2). One distinctive staining technique demonstrated with RMC is the loss of nuclear expression of INI1 which is key in differentiating RMC from collecting duct carcinomas and is associated with more aggressive forms of RMC (4). SMARCB1 (SWI mating—type switching/SNF related matrix-associated, actin-dependent regulator of chromatin, subfamily B, member 1) is a tumor suppressor gene associated with sickle cell trait and loss of staining is often demonstrated in RMC (2).

Treatment options are largely unstandardized and there are few options found to be effective in RMC. For localized disease, surgical nephrectomy shows some survival benefit (median survival of 16-18 months)(5). With advanced disease (median survival time of 4-5 months), patients often receives regiments with MVAC (methotrexate, vinblastine, adramycin and cisplatin) or gemcitabine, palcitaxel and bevacizumab.

Case Presentation

A 22 year old African American male presented to a tertiary referral center with a 2 week history of progressive shortness of breath and back pain exacerbated by movement. Additionally, he'd reported worsening rib pain, emesis, dry cough, night sweats, fever and unintentional weight loss. He denied any recent or remote history of overt hematuria, testicular, or inguinal swelling. Patient's past medical history was only notable for sickle cell trait. Physical examination was notable for cervical lymphadenopathy on palpation as well as tachycardia and wheezing with auscultation. There was palpable hepatomegaly and abdominal tenderness noted. A small right sided hydrocele was present. Initial routine metabolic studies noted an isolated creatinine of 1.6 and an AST/ALT of 64/70.

Computed tomography of the abdomen and pelvis was completed which demonstrated bilateral hilar adenopathy, sub-centimeter pulmonary nodules (Figure 1), extensive retroperitoneal lymphadenopathy, multiple hyperdense liver lesion and a right sided renal vein thrombus (Figure 2). Additional tumor related laboratory studies were sent for lymphoma and testicular cancer. LDH was elevated at 430. The tumor markers α -fetoprotein and β -HCG were not significantly elevated. He was then taken for ultrasound guided liver biopsy which demonstrated poorly differentiated carcinoma with generalized discohesive cell-cell attachments with focal microcystic architecture. Immunophenotyping demonstrated positive staining for AE1/AE3, EMA, and loss of INI1 staining along with CD45, MART1, CD30, PLAP, and CEA most consistent with a diagnosis of metastatic medullary carcinoma in this patient with known sickle cell trait. Due to diffuse spread, significant tumor burden, and increasing oxygen demand, the patient was initiated on paclitaxel and carboplatin for one cycle and diischarged on home oxygen with close follow-up.

Patient presented back to hospital approximately 5 days later with worsening back pain, tachypnea, tachycardia and worsening respiratory status and was found to have a new onset pericardial effusion and extensive bony metastasis to his lumbar and thoracic spine on MRI. He underwent pericardial drain placement and was seen by radiation oncology for palliative radiation. The patient received one session and was discharged with follow up the next day at a specialized oncological institute with active clinical trials. Regrettably, despite maximal medical therapy, the patient passed away less than 3 months after diagnosis.

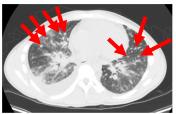


Figure 1 (Above): Axial CT of the chest
demonstrating innumerable sub-centimeter nodules
throughout the lungs (red arrows)

Figure 2 (Left):
Coronal CT of the chest demonstrating retrocaval lymphadenopathy (green arrow), renal artery and venous stenosis and associated thrombosis (blue arrow), and multifocal hypoechoic liver densities (red arrow) suspicious for metastatic disease



Discussion

Unfortunately, RMC has a tendency to present late (92% stage 3/4) with metastatic disease being the most common presentation (5). This often heralds a rapid clinical decline and terminal prognosis. Common sites of metastases include the regional lymph nodes, lung, liver, bone and inferior vena cava (5).

Despite a strong association between sickle cell trait and disease, screening has not been included as part of guidelines owing to the extreme rarity of the malignancy and unclear benefit. Given the propensity for sickle cell patients to develop nephropathy, routine urinalysis may be a cheap, clinically beneficial screening tool in sickle cell trait and disease which.

Conclusions

We present a case of a 22 year old African American with metastatic renal medullary carcinoma of the kidney. RMC is an extremely rare malignancy found exclusively in patients with sickle cell disease and trait. The disease presents late owing to it being asymptomatic until advanced. The prognosis is grim with median survival of 4-5 months. No medical therapy has been found to be consistently effective however multiple clinical trials are currently in various stages of development. No tools for early detection have been shown to have utility owing to the rarity of the cancer.

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Acknowledgement: The authors of this poster would like to specially thank Dr. Fady Farag of specially thank

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Diffuse Alveolar Hemorrhage in Mixed Connective Tissue Disease

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Introduction

Diffuse alveolar hemorrhage (DAH) is a potentially fatal complication seen with inflammatory and autoimmune conditions, among other etiologies. DAH can be associated with multiple different autoimmune conditions. Here, we present a case of DAH secondary to mixed connective tissue disease (MCTD).

Case Description

A 42-year-old male with a history of CAD, DVT, hypertension and PE requiring IVC filter in 2014, presented with nonproductive cough, myalgias, and pleuritic chest pain. CTPE revealed bilateral ground glass opacities. Sars-CoV-2 and influenza testing were negative. Broad spectrum antibiotics were initiated, and he was discharged after 4 days.

He returned 2 weeks later with the same symptoms. On hospital day 4 he developed progressive shortness of breath with eventual hemoptysis. CT thorax revealed diffuse bilateral ground glass opacities concerning for DAH.

Autoimmune workup revealed ANA (1:2560), RNP 807, and IgG 2568 with negative DsDNA, SSA, SSB, Jo, and Smith antibodies. Pulse doses of methylprednisolone were started. However, his oxygenation requirements increased requiring intubation. Following three courses of plasmapheresis, his respiratory function improved allowing extubation. Cyclophosphamide was added, but worsening respiratory compromise led to reintubation and shortly thereafter, asystole. ACLS was initiated but ROSC could not be achieved, and the patient expired.

Imaging

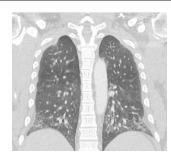


Figure 1- CT thorax with PE protocol from

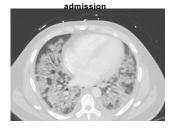


Figure 2- CT thorax with PE protocol 4 days later



Figure 3- Chest X-ray from day 7 hospitalization

Lab Results: Autoimmune Workup

Antibody	Titer/Value
ANA	>1:2560 (speckled pattern)
RNP	807
ANCA	Negative
MPO	Negative
Smith	Negative
DsDNA	Negative

Conclusion

- While the prevalence of MCTD is low and extreme presentations such as this one are rare, it should be on the differential when considering the etiology of DAH.
- MCTD is an autoimmune condition which combines clinical features of systemic lupus erythematous, scleroderma, and polymyositis/dermatomyositis.
- Usually present with high ANA titer with positive RNP antibodies
- MCTD has a particular effect on the pulmonary vasculature, causing intimal proliferation of the pulmonary arterioles resulting in pulmonary hypertension.
- Only a minority of patients with DAH have hemoptysis.
- Diffuse ground glass opacities on CT are a strong indicator of DAH.
- Plasmapheresis, cyclophosphamide, and high dose steroids are the foundation of therapy for severe DAH resulting from autoimmune etiologies.

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Utilization of POCUS by Resident Physicians in the Early Evaluation and Diagnosis of Cardiac Dysfunction

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Introduction

Heart failure (HF) affects approximately 6.2 million adults in the US alone and is a leading cause of hospitalization (Virani et al). The correct and swift identification of patients with HF promises to not only provide clinically beneficial data, but may also have implications in reducing inpatient hospital length of stay as well as maximizing hospital revenue.

In Southwest Louisiana, we rely heavily on the complete echocardiogram. This diagnostic procedure performed by trained technicians provides us with information that guides our course of action in clinical medicine. This can introduce large time gaps in the appropriate treatment of some patients.

The utilization of point of care ultrasound (POCUS) in the early evaluation and diagnosis of cardiac dysfunction and its causes may aid Physicians in making critical clinical decisions. This in turn may ultimately facilitate enhancements in real-time patient care and cost-effective management.

Project Aim

Project Aim:

To train Resident Physicians to accurately utilize POCUS in diagnosing ejection fraction changes, right heart strain, cardiac tamponade and the presence of thromboembolic disease.

Application of POCUS



IM PGY-2 Resident David Heintze, MD demonstrates the use of POCUS at the bedside.

Project Methodology/Outcome

Project Methodology:

- Formal training of IM Residents in the use of POCUS.
- Equipping IM Residents with portable ultrasound equipment.
- Reviewing IM Resident-read ultrasounds with official TTE within a 48hr period.

Expected Outcome:

To increase the accuracy of diagnoses by 75% through interpretation of POCUS on approximately 50 patients within a 6 month period

Conclusion

In addition to assessing the Residents' POCUS accuracy, comparisons will also be used to reflect on the amount of "over/under" estimation. This will later help with the implementation of necessary changes to minimize unintended harm associated the use or misuse of POCUS information.

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COVID Induced Thyroid Storm

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Introduction

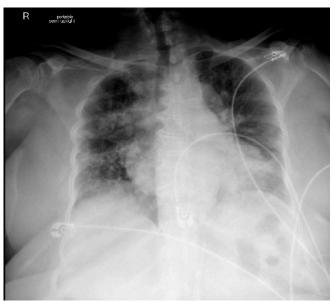
Thyroid storm is a rare condition most commonly caused by a secondary insult to underlying Graves' disease although other thyroid diseases are also implicated. The most common insult is infection. Thyroid storm is the severe end of the spectrum of thyrotoxicosis and is characterized by end organ compromise. It is important to recognize this condition because it carries a high mortality rate. In this case the most likely trigger was COVID-19.

Case Presentation

A 71 year old woman with left leg sciatica, HTN and subclinical hyperthyroidism presented with myalgias, confusion, abdominal pain, and decreased appetite for one week. Upon arrival, she was found to be febrile to 101.5 degrees Fahrenheit, in atrial fibrillation with rapid ventricular response (RVR) and had oxygen saturations in the 80s. Her chest x-ray demonstrated bilateral opacities and she tested positive for COVID-19. Her Labs were significant for TSH 0.01, free T4 1.25, and total T3 92. She was initially given metoprolol and amiodarone, which resolved the atrial fibrillation with RVR, and was started on propylthiouracil and hydrocortisone for thyroid storm. Propanolol was held due to low blood pressures. During the hospitalization, the patient was intubated for worsening respiratory status. She did receive azithromycin, ceftriaxone and hydroxychloroquine for COVID. Additionally her hospital course was complicated by acute kidney injury which resolved spontaneously. Eventually she was able to extubated and weaned to room air. The patient was discharged home with methimazole, metoprolol and apixaban.

Discussion

Thyroid storm is a clinical diagnosis based on an exaggerated response to elevated thyroid hormone levels. In patients with diagnosed or undiagnosed hyperthyroidism many precipitating factors like acute illness can induce thyroid storm. In the case of our patient, COVID-19 most likely triggered her presentation. While lab results typically show elevated free T4 and total T3, treatment for thyroid storm should be based on high clinical suspicion. The Burch-Wartofosky Point Scale can be used to assess the likelihood of thyroid storm. This scale includes factors such as temperature, CNS dysfunction, tachycardia, atrial fibrillation, heart failure, GI dysfunction and presence of a precipitating factor. Initial management involves increasing adrenergic tone with beta-blockers and reducing peripheral conversion of T4 to T3 with propylthiouracil.



Chest x-ray from admission showing multifocal pneumonia

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The Renal Realm of COVID-19

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Introduction

Coronavirus Disease-19 (COVID-19) has not only wreaked havoc in the fabric of our societies, it has managed to create distinct pathophysiological blueprints i every organ system.

One of the primary physiological manifestations seen in nearly 23% of all cases is direct acute kidney injury (AKI), unveiling unique diagnoses proven via biopsies.

There exists very meager data and workup documenting the renal manifestations of COVID-19 from a histological and pathological standpoint.

Data from October 2020 in *medRxiv* noted a 23% incidence of AKI in 85 individuals, of which 60% were considered to be high-risk. The autopsy reports of six deceased patients revealed "severe acute tubular necrosis with lymphocyte and macrophage infilitration." [1]

There are autopsies that have revealed the presence or viral particles in renal tubular epithelium. [2] Additionally some autopsy reports did not reveal direct COVID-19 viral particles in the kidney and hinted that COVID-19 may behave as other viral nephropathies in directly infecting the kidney tubules and generating tubular inclusions in the cytoplasm. [1]

Case Description

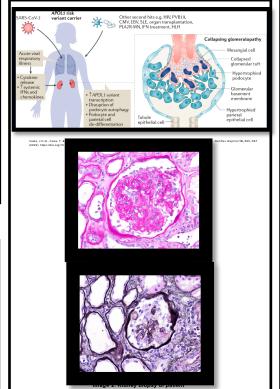
A 49 year old female with a history of uncontrolled type 2 diabetes (recent A1c greater than 13.0), chronic kidney disease (CKD) stage 2, and hypertension presented with nausea, vomiting, and overall weakness for the past four days.

She was admitted for hyperosmolar hyperglycemic state, COVID-19 infection, and new onset AKI on pre-existing CKD. Months prior her baseline creatinine level was 1.00 and glomerular filtration rate (GFR) was 76. On admission creatinine was 3.30 and GFR 19. Despite aggressive fluid resuscitation renal function only nominally improved to serum creatinine of 2.60 and Nephrology was consulted.

Urine studies revealed nephrotic range proteinuria with urine protein to creatinine ratio elevated at 28,280 mg/gm and microalbuminuria of 6470 mg/L.

With proteinuria of this magnitude the initial etiology of renal failure was attributed to diabetic nephropathy, but the patient had been diagnosed with diabetes only four years prior at age forty-five.

Images



Case Description cont.

Of note, early diabetic nephropathy does not cause this degree of renal failure within a matter of just four years; therefore further evaluation was warranted.

Pertinent negatives included the following antibodies: anti-nuclear, anti-Smith, anti-nuclear cytoplasmic; HIV and hepatitis panels; complement levels normal; and serum and urine protein electrophoreses for M spike.

With laboratory assessment fairly inconclusive, she ultimately received percutaneous kidney biopsy which did reveal collapsing glomerulopathy.

Discussion

- Collapsing glomerulopathy has been known to affect patients of African descent with presence of APOL1 risk variant especially in the setting of HIV. In the renal realm of COVID-19, collapsing glomerulopathy, amongst other nephrotic syndromes, is not uncommon and is a rather important and emerging association.
- The emergence of AKI in patients infected with COVID-19 is linked to a worse prognosis and death in hospitalized patients. [2]
- The mechanism of renal injury in COVID-19 is the through the ACE-2 receptor found in higher concentrations in the kidney versus the lung. This receptor is more prevalent in the apical brush border of renal tubules rather than the glomerulus or endothelial cells. [1] [2]
- Patients with pre-existing diabetic kidney disease already have an increased chance of developing AKI due to upregulation of the ACE receptor, which at baseline is primed for inflammation even before COVID-19 infection. [1]
- Although AKI may be attributed to acute tubular necrosis from septic shock, COVID-19 infection demands further investigation due its unique pathophysiological fingerprint in the kidney. Additional research would allow for the development of targeted treatments to prevent worsening outcomes in the renal realm and improve overall wellbeing of patients.

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Treating Thyroid Storm When You Don't Have The Guts

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Introduction

Thyroid storm is an emergent, often-fatal thyrotoxic state associated with severe clinical manifestations, such as hemodynamic collapse, hyperpyrexia, and altered mentation. It often has precipitating factors such as surgery, sepsis, iodine

Diagnosis is clinical but can be supported by Burch and Wartofsky Point Scale

Treatment involves supportive measures. various forms of thyrotoxicosis-directed treatment. and addressing any precipitating factors.

As demonstrated in the following case, it is mportant to be aware of the profound cardiovascular dysfunction in thyroid storm and to be cognizant of various treatment modalities available to treat complicated patients, such as when the GI tract is compromised.

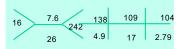
Initial Presentation

Patient is a 38-year-old female with PMH of a goiter who underwent emergent ex-lap and Graham patch repair for a perforated duodenal ulcer at an outside facility, likely secondary to toxic indestion. She was found to have hyperthyroidism and developed thyroid storm post operatively. Patient developed ventricular fibrillation (V-fib) with ROSC and was transferred to our hospital for further care.

- T 99.3 F, HR 156, RR 20
- BP 96/60-MAP 72
- SpO2 100% on ventilator FiO2

40% Labs

- TSH < 0.008
- Free T4 > 5
- AST/ALT 1.788/426
- Alk Ph 179
- · Billi-Total 2.1; Billi-Direct 1.8
- PT/INR 37.3/3.8
- Troponin 0.48



Application of Quantitative Diagnostic Tool on Initial Presentation

Burch Warofsky Scale	
Criteria	Points
Temperature 99-99.9 F	5
HR > 140	25
Atrial Fibrillation Present	10
CHF Absent	(
GI-Hepatic Dysfunction Moderate	10
CNS Disturbance absent	(
Precipitant Present (surgery, sepsis)	10
Total	60
Score > 45 - Thyroid Storm is likely	

Management & Course

Norepinephrine, esmolol drip, dexamethasone, and piperacillintazobactam were started for septic shock and thyroid storm. Given recent duodenal perforation, all enteral medications, including oral thioamides, were initially avoided. IV methimazole (MMI) was not available at our facility.

Over the next 24 hours the patient had 16 episodes of V-fib, obtaining ROSC each time with one round of ACLS. During these 24 hours, one round of plasmapheresis was performed; Lidocaine drip was started to help terminate Vfib; and Esmolol was switched to IV propranolol, after which V-fib resolved. Patient developed Afib with RVR which corrected with continuation of B-Blocker. The next day she received two plasmapheresis treatments and PTU via retention enema x5 doses (400mg/60ml-Fleet enema; 60ml g4h).

Patient was subsequently transitioned to PTU, Lugol's solution, and dexamethasone all via NG tube. By day 5 the patient was weaned off pressors, extubated, and transitioned from IV propranolol to oral carvedilol. On day 12, PTU was transitioned to MMI. For the remainder of her hospital stay, thyroid hormones and hemodynamic status were well-controlled.

Overview of Management

- Dexamethasone, Zosyn, Pressors, Lidocaine drip
- Plasmapheresis x3, Enteral Lugol's solution (NG
- Rectal PTU → PTU (NG tube) → MMI (NG tube)

Patient's Cardiovascular Complications

- V-Fib x16
- Afib w/ RVR

Conclusion

The above case not only reinforces knowledge of thyroid storm's profound cardiovascular effects but also highlights the need to know alternate strategies of treating thyroid storm in patients with GI compromise. Thyroid storm is typically treated with beta-blockers, thioamides, iodine, and steroids; and any underlying, causative condition must be treated. In extreme cases cholestyramine and plasmapheresis can also be used. GI compromise poses challenges to administration of thioamides

Acutely, PTU is preferred over MMI due to PTU's inhibition of peripheral thyroid hormone conversion. PTU and MMI can be given orally or through an NG tube. Past case reports also show it is possible to prepare rectal suppositories, retention enemas, or even IV formulations from PTU or MMI tablets. A standard IV MMI preparation is available, but not in the USA. The following tables are compiled from limited data based on prior case reports from 1981-Present:

Methimazole-Suppository

Dissolve 1200 mg MMI in 12 ml of water and add to 52 ml cocoa butter containing two drops of polysorbate (Span) 80. Sit mixture to form an ulsion and pour into 2.6 ml suppository mold to cool

Propylthiouracil

Dissolve 200mg of propylthiouracil in a polyethylene glycol base and put into suppository tablets

Dissolve eight to twelve 50mg tablets of PTU in 90ml of sterile water

Dissolve eight 50 mg tablets of propylthiouracil in 60 ml of mineral oil nema (eg. Fleet mineral oil) or in 60 ml of sodium phosphates enema solution (eg, fleet enema phoshpo soda)

Methimazole IV

Dissolve 500mg MMI powder in 50ml pH-neutral isotonic saline and filtering through a 0.22 micrometer filter. Administer to patient over 2 minutes

Dissolving tablets in isotonic saline made alkaline (pH 9.25)

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Unexpected Culprit of HIV-Associated Dysphagia

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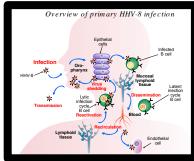
Introduction

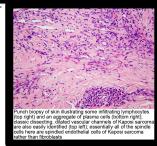
- When generating a differential for odynophagia or dysphagia in HIV patients, candida, cytomegalovirus, and herpes simplex virus are high on the list of etiologies. However a more occult and often misdiagnosed etiology is Kaposi's sarcoma.
- Kaposi's sarcoma is an endothelial cell malignancy initially transmitted via saliva caused by human herpesvirus-8 that is considered an AIDS-defining
- Cutaneous involvement of Kaposi's sarcoma (KS) is well regarded but gastrointestinal involvement of KS remains underdiagnosed in HIV patients.
- Nearly 40-50% of patients with cutaneous KS have concurrent visceral involvement, the most common site being gastrointestinal.
- Notably, it is important to remember that visceral involvement can exist without cutaneous involvement. Additionally, Kaposi's sarcoma can occur even in individuals with well-controlled HIV

Case Description

- We present a case of a 43-year-old female presenting with dyspnea upon rest, tense anasarca, hematemesis, dysphagia, and recent onset of multiple violaceous maculopapular lesions on the abdomen. face, and lower extremities. Of note, she had innumerable tattoos and was monogamous with only one male partner.
- Initial workup was remarkable for new diagnosis of advanced HIV-1 with CD4 count of 65 and viral load of 227,000. Skin biopsy was positive for Kaposi's sarcoma.
- Patient was initiated on highly active antiretroviral therapy (HAART) with Tivicay and Descovy; prophylaxis with Doxycycline and Rifampin: and Diflucan and Nystatin for esophageal candidiasis. There we plans to initiate liposomal doxorubicin versus paclitaxel for malignancy but with worsening dysphagia, she required multiple readjustments to HAART to decrease pill burden.
- The patient received an upper endoscopy resulting in biopsy-proven presence of visceral KS in a gastric nodule, small bowel, and distal esophageal polypoid mass.

Images & Clinical Presentation





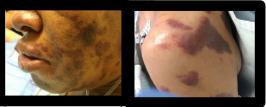


Image 1. Kaposi's sarcoma

Image 2. Kaposi's sarcom

In the following days, her respiratory status began to decline considerably requiring thoracenteses. She developed new onset acute renal failure. Shortly afterwards she was upgraded to ICU for shock and intubated due to iatrogenic pneumothorax versus respiratory

Discussion

- Visceral KS requires a high index of suspicion in HIV patients with even subtle gastrotintestinal symptoms and warrants urgent endoscopy for biopsyproven diagnosis and management.
- Screening endoscopies for visceral KS remains a hot topic for debate among clinicians. In Japan screening endoscopies are performed in even asymptomatic HIV patients that meet the following criteria: CD4 less than 100, men who have sex with men (MSM), and pre-existing cutaneous KS.
- Initiation of (HAART) in KS is key and beneficial in disease regression. With the onset of even subtle gastrointestinal symptoms additional therapy with chemotherapeutics is necessary.
- Chemotherapy treatment consists of pegylated liposomal doxorubicin or paclitaxel. The liposome component of liposomal doxorubicin reduces overall pharmacological toxicity but also allows for better penetration into KS lesions.
- Despite combined HAART and systemic chemotherapy patients still have a 13% chance of relapsing after one year of treatment compliance.

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Case Description cont.

acidosis. Unfortunately, the patient expired due to multi-organ dysfunction during the hospital



Unsuspected Causes of Hypertension

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Introduction

Pheochromocytomas (PC) are catecholamine-secreting tumors that arise from the chromaffin cells of the adrenal medulla and other extra-adrenal sources. The annual incidence of PC is approximately 0.8 per 100,000 personyears, however this is thought to be an underestimation as a substantial portion of PC's were diagnosed at autopsy (Beard et al). With the relative rarity of this disease coupled with its oftencomplicated presentation, it is not uncommon to see this disease misdiagnosed.

Case Description

68-year-old Caucasian female presented after a fall at home.

PMH: Chronic tobacco use, hypertension, hypothyroidism secondary to partial thyroidectomy, coronary artery disease, paroxysmal atrial fibrillation, periodic headaches, back pain and dizziness

Elevated troponin with normal EKG led to treatment for NSTEMI

CT head: WNL

LHC: LVEF of 35-40% with 60% mid-LAD

Coreg and Ramipril were started and patient discharged.

A few weeks later...

CC: chest pain radiating between her shoulder blades

CTA Chest: 1.7cm soft tissue density in the right lower lung lobe and a 1.6cm right adrenal nodule.

PET/CT: peak SUV of 1.6 in the lung nodule and a peak SUV of 2 in the adrenal gland nodule. Biopsy of lung nodule via bronchoscopy

unattainable due to its small size and anatomic location.

CT-guided needle biopsy of right adrenal mass: Pheochromocytoma.

CT Surgery performs lobectomy of right lung with lymph node biopsies: Adenocarcinoma of likely lung primary origin.

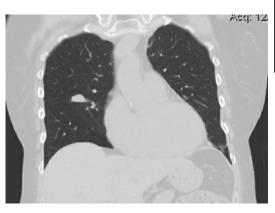
Laboratory Findings

U24 Normetaneph-LC	* 349
U24 Metanephrine-LC	* H 615
U24 Creatinine-LC	* L 718

Radiology Images



Image 1.



lmage 2.

Conclusion

It is estimated that approximately 0.2-0.6% of patients with hypertension have a concurrent PC (Omura et al). In fact, approximately 15-30% of the population of hypertensive patients have secondary hypertension and resistant hypertension was noted in approximately 34% of hypertensive patients in the ALLHAT trial (Koch et al). This patient's presentation attests to the semi-cryptic nature of PC.

This case also demonstrates that, despite a suspected cardiac etiology and incidental pulmonary malignant process, searching for the overall causation of the patients presenting symptoms may be more difficult than it first appears. After completing a secondary hypertension workup (as well as dietary and lifestyle changes) with unremarkable results it is important to consider endocrinopathies as an underlying cause, despite their rarity.

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A Unique Case of Dalbavancin Associated Drug-induced Immune Thrombocytopenic Purpura (D-ITP)

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Introduction

Dalbavancin is a semisynthetic glycopeptide with higher activity against grampositive bacteria (including MRSA) compared to its natural counterpart, Vancomycin [1].

It is given as a two-dose regimen; intravenous loading dose followed by 2nd dose one week later.

Adverse reactions reported include IgE mediated hypersensitivity and skin reactions, rapid infusion related red-man syndrome, transaminitis. D-ITP typically manifests within 2 weeks of initial exposure to offending drug [2].

Case Description

A 39-year-old Hispanic male presented with one day history of purpuric rash that began over his feet and progressively spread to the torso.

Patient was admitted 3 weeks prior for right great toe osteomyelitis. He was treated with 7 days of inpatient Vancomycin and Meropenem and was discharged home on outpatient IV Dalbavancin and oral Ciprofloxacin for 42 days.

Review of systems negative for fevers, sick contacts/travel. Physical examination revealed non-palpable purpura over torso and bilateral lower extremities.

Hematology labs revealed a substantial drop in platelets from 330,000/mm3 to 3,000/mm3 over an 8-day period.

Patient was immediately started on high dose Solumedrol and platelet transfusion.

Extensive workup negative for infectious/ rheumatological etiologies, HIT, DIC, TTP and microangiopathic hemolysis. He received intravenous immunoglobulin for D-ITP.

Clinical Presentation/Drug MOA



Figure 1: Non-palpable purpura over left lower extremity of the

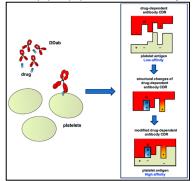


Figure 2: Schematic representation of the binding of a drugdependent antibody (DDab) to a platelet glycoprotein. CDR; complementary determining region [3].

Case Description (continued)

- After extensive review of medications and lab work, we deduced that his D-ITP was associated with Dalbavancin because significant thrombocytopenia was observed 8 days following 2nd dose of Dalbavancin.
- Over the hospital stay, his thrombocytopenia and purpuric rash improved. He was discharged on Prednisone 40mg twice daily for 5 days for D-ITP and Ciprofloxacin for osteomyelitis.
- During outpatient follow up visit, his platelet count improved to 167,000/mm3.

Conclusion

- DDab results in the drastic thrombocytopenia seen in D-ITP by 2 major mechanisms. Firstly by DDab induced platelet destruction in the reticuloendothelial system by macrophages. Secondly by DDab induced inhibition of GPIb/IIb/IIIa glycoproteins expressed on Megakaryocytes resulting in megakaryocytopoiesis suppression [3].
- This case illustrates the importance of recognizing Dalbavancin associated D-ITP to aid in prompt initiation of treatment. The crucial first step in management is to remove the offending drug.
- While there are not many reports on thrombocytopenia from Dalbavancin, we propose that it would be beneficial to obtain follow up labs after Dalbavancin's 2-dose regimen is completed as surveillance for D-ITP.

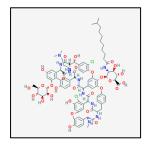


Figure 3: Chemical structure of Dalbavancin

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Ascites as a Sequela of Myxedema Coma

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Introduction

Ascites, the accumulation of fluid in the peritoneal cavity, is a common chief complaint that can be diagnosed via physical exam and confirmed via ultrasound

In 85% of patients, cirrhosis is the etiology of the fluid buildup However, the etiology is multifactorial, requiring analysis of the fluid and use of the serum ascites albumin gradient formula

Other common causes include viral hepatitis, malignancy, congestive heart failure and veno-occlusive disease

Less common (roughly 4% of cases), hypothyroidism can be the etiology

Here, we present present a patient with classic ascites in the setting of myxedema coma

Patient Presentation

38-year-old African American male presents with 6 months of progressively worsening fatigue associated with intermittent abdominal pain and distention

History of hypothyroidism with noncompliance with Synthroid for the last 6 months

On examination, patient was noted to be somnolent but intermittently arousable to questions

Admission Vitals: T: 36.4 HR: 57 RR: 18 BP: 112/70 SpO2:

Diagnosis of myxedema coma was made based on a score of 60 on the Myxedema Coma scoring system

Imaging



Figure 1: Ultrasound of the liver: Normal homogenous pattern with no evidence of liver

Labs

Labs on Admission	Value	Normal Value
Sodium	137	135-145
Potassium	3.7	3.5-4.9
Chloride	99	98-107
CO2	28	22-28
Calcium	8.8	8.5-10.1
Glucose	115	76-106
TSH	51.83	0.3-3.76
Free T4	0.54	0.76-1.46

Ascitic fluid lab value	9/7/2020	12/19/2017	8/6/2017
Serum Albumin	3.4	3.4	3.6
Ascitic Albumin	0.8	2.5	2.5
SAAG score	2.6	0.9	1.1
Total Protein	1.4	4.7	4.2

Table 1: A comparison of the SAAG and total protein values for the patient on three different visits to the hospital.

Case Discussion

- On admission, patient was emergently started on thyroid replacement, with a loading dose of 200 mg IV Levothyroxine followed by 125 mcg po daily. He received 5 mcg of Liothyronine daily.
- Workup for other etiologies of ascites were all negative. This included echocardiogram (EF 60% with no diastolic dysfunction), ANA and anti-smooth muscle antibody and CT abdomen/pelvis showing no evidence of malignancy.
- Patient eloped before completing his treatment. Prior to leaving, patient was noted to have significant reduction in distention and tenderness of abdomen. TSH was 36.27 and T4 was 0.54.

Conclusion

- This case discusses an uncommon cause of ascites.
 While most cases will be secondary to cirrhosis, there are other possibilities
- Because it is rare, ascites secondary to hypothyroidism is not regularly included in SAAG score charts and the expected value of the SAAG and total protein are unknown
- In previous similar case studies, it has been seen that the SAAG score is < 1.1 and that there is a high total protein.
- While our patient's fluid study values only fit this clinical picture once, we still have a high suspicion that his hypothyroidism was driving his ascites
- We were able to rule out other etiologies of his ascites, including the most common ones like cirrhosis and malignancy.
- The patient's abdominal distention and pain resolved dramatically despite only being treated for his hypothyroidism
- Though a paracentesis was done, less than 100 cc of fluid was removed as this was diagnostic. It is unlikely that this volume made a huge difference
- Our case demonstrates that hypothyroidism should be on the differential for a patient with new-onset ascites when the common etiologies do not appear relevant.

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Atypical Cardiac Tamponade as Sequelae of Rheumatoid Arthritis

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Introduction

Rheumatoid Arthritis (RA) is a chronic multisystem disease which affects approximately 1% of the population.

Typical findings are persistent inflammatory synovitis which usually involves peripheral joints in a symmetric distribution.

Extra-articular findings are common in RA, especially cardiac, roughly 30-50% of patients develop pericardial effusions.

Less than 10% of pericardial effusions in RA are of clinical significance

Case Description

A 60-year-old female presented to the emergency department after experiencing multiple falls and generalized weakness for 2 weeks duration.

Previous medical history of breast cancer, hepatitis B, type II diabetes mellitus, COPD and RA were documented.

Patient had an echocardiogram completed as an outpatient, 1 month prior to presentation, which showed a moderate pericardial effusion, however repeat echocardiogram before admission showed resolution of the effusion without intervention.

Physical Exam

T: 36.6°C HR:78 RR: 22 BP: 124/77 S pO2: 99%

Case Description cont.

Physical exam was unremarkable, including the absence of a friction rub and JVD. Lung auscultation was positive for bilateral mild wheezing.

On admission, patient became acutely hypoxic requiring 2L of nasal cannula. CT angiogram was showed a large pericardial effusion. A stat echocardiogram was ordered which revealed a large pericardial effusion (largely posterior) with right atrial collapse consistent with early tamponade physiology.

Heart rate and blood pressure remained stable with the patient remaining on 2L nasal cannula. A repeat EKG showed low voltage with no evidence of electrical alternans. Patient was transferred to an outside center for a pericardial window.

Pericardial window was preformed and drained 426cc of pericardial fluid. Pathological analysis showed evidence of acute fibrinous pericarditis without signs of malignancy or infection.

Imaging

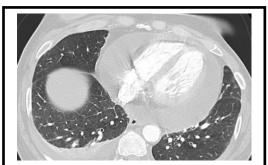


Fig 1: CT angiogram thorax: Large pericardial effusion and small right & trace left pleural effusions. Negative for pulmonary thromboembolic disease. Emphysema and similar slight ground-glass attenuation of the lung parenchyma compared to the prior CT.

Labs

Labs on Admission	Value	Normal Range
Sodium	123	135-145
Chloride	87	96-106
AST	75	5-40
ALT	254	7-56
NT Pro BNP	3791	<125
WBC	16.4	4.5-11
Hgb/Hct	8.8/27.4	12-16/35-46
ESR	36	0-20
CPR	13.99	0-0.5

Conclusion

- This case discusses the finding of pericardial effusion, a sequelae of rheumatoid arthritis.
- Pericardial effusions can be seen in up to 30-50% of patient with RA, however, less than 10% of cases are of clinical significant.
- This case demonstrates a clinically significant cardiac tamponade and fibrinous pericarditis in the context of RA.
- This patient presented twice hemodynamically stable and without typical exam findings before current admission and diagnosis of recurrent pericardial effusion with tamponade physiology.
- Patients with RA may have dangerous extra-articular findings which need to be considered during hospital admission. Although pericardial effusions of clinical significance in RA are uncommon, this sequelae should be considered in patients with abnormal or unstable vital signs.

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Cocaine-induced Asthma Exacerbation

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Introduction

Asthma is an inflammatory disorder characterized by cough, wheezing, dyspnea and airflow obstruction

Pathogenesis revolves around bronchial hyperreactivity, usually due to allergens such as mites and pollens

Cocaine is known to cause several lung pathologies, including ischemia, barotrauma and hemorrhaging

The drug stimulates the sympathetic nervous system, leading to bronchoconstriction and narrowing of airways, possible causing complete obstruction

Here, we present a patient who presented in an acute asthma exacerbation episode following inhalation of cocaine

Patient Presentation

- 43-year-old male presented post cardiac arrest in respiratory failure following the use of cocaine. ROSC and intubation were attained at scene.
- History of unquantified asthma but with PRN albuterol use

Vitals:

T: 37.3 HR: 99 RR: 27 BP: 109/72 SpO2: 94%

Pertinent Admission Labs:

WBC: 16.2Lactic Acid: 14.3

UDS: (+) cocaine
U/A: Leukocyte esterase and nitrite
negative, trace blood, bacteria

Images



Image 1: Chest x-ray on admission: Hyperexpanded lungs but no evidence of pneumothorax, infiltrate or



Image 2: CT Thorax on day 2 of admission: No evidence of pulmonary embolism, hemorrhaging, ground-glass opacities or ravitation

Case Description (labs/tables, etc.)

In	Initial ABG:		
Blood Gas	11/12/2020 (02:35)		
pH	7.12		
pCO2	59		
PO2	69		
НСО3	20.3		

Progression of ABG:

Blood Gas	11/12/2020 (11:37)	11/13/2020	11/14/2020	11/15/2020
pH	7.34	7.45	7.38	7.48
pCO2	44	40	55	48
pO2	76	68	89	91
нсоз	23.7	27.8	32.5	35.7

Hospital Course

- Continuous albuterol treatments, magnesium sulfate, methyl prednisone and antibiotics were started in the ED. Antibiotics were discontinued on day 2 due to lack of evidence of an infection
- During admission, a diagnostic workup was completed to rule out other etiologies of the patient's respiratory failure. This included:
- Bronchoscopy that was only significant for mucus occluding the superior segment of the right lower lobe
- Despite improvement in respiratory status, the patient was encephalopathic on arrival and his neurological status progressively declined. Patient was declared brain dead on day 5 of admission

Conclusion

- This case brings light to the possibility of cocaine-use as a cause of exacerbation of asthma symptoms, though this is a diagnosis of exclusion
- Though our patient had no documented pulmonary function test, his history of albuterol use with symptomatic improvement and chest x-ray findings of chronic obstructive changes in the setting of no significant tobacco use makes asthma likely
- Additionally, the patient's diagnostic workup revealed no other pathological cause of the patient's respiratory
- Finally, the patient's respiratory status improved on typical asthma exacerbation management
- Our patient's case correlates well with a previous case report written by Zhou et al. about several patients who were treated for asthma exacerbation and their only known inciting factor was cocaine use (1)

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Concurrent Multiple Sclerosis and Human Immunodeficiency Virus

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Introduction

Both multiple sclerosis and HIV have been well studied throughout medical literature. There is a presumed negative association between acquiring both MS and HIV simultaneously. However, there have been some rare case reports of patients having both MS and HIV. Occurrence of MS in some instances were noted specifically during acute HIV infection or during HIV seroconversion¹.

This is a case of concurrent MS and HIV despite being well-controlled with antiretroviral therapy.

Case Description

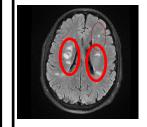
A 54-year-old African American male with HIV presented with urinary incontinence, weakness, frequent falls, and unsteady gait for approximately 1 month. Patient is a poor historian, but per his chart, these symptoms had been present much longer.

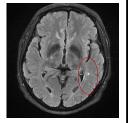
In 2015, he presented to the ED with a 3-day history of fluctuating slurred speech, right-sided facial numbness, dysphagia, and blurred vision. MRI brain was suspicious for demyelinating plaques. At that time Neurology diagnosed him with a "possible lacunar stroke".

For the next 2 years, he had worsening generalized weakness and significant gait and balance disturbances.

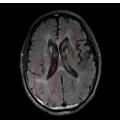
Imaging

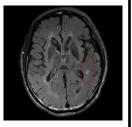
MRI Brain T2-FLAIR: 2015





MRI Brain T2-FLAIR: 2020





Case Description cont.

MRI in 2018 showed FLAIR hyperintensities throughout the periventricular
white matter with lesions in the cerebrum
and cerebellar hemisphere. LP showed CSF
containing protein - 61.7 and IgG Index of
0.80. Neurology suggested that symptoms
were secondary to uncontrolled
comorbidities. Throughout his hospital
course, he was afebrile, blood and urine
cultures remained negative, and HAART
was resumed.

2020 MRI of the brain showed stable T2/FLAIR hyper-intensities compared to those in 2018.

Conclusion

Neurology was again consulted due to suspicion of demyelinating disease. LP was deferred given previous LP findings that met McDonald's Criteria. Patient's symptoms improved and he was discharged with suspected MS, likely presenting in 2015. Immunomodulators and/or steroids were not started at this time pending outpatient Neurology follow up and workup to rule out other demyelinating disorders.

Evidence shows that HIV patients can acquire a wide spectrum of autoimmune diseases during acute HIV infection or during phases of increasing low CD4 counts while not on HAART. Although there is low incidence of MS seen in patients with well-controlled HIV on HAART, those with non-progressive HIV may be equally susceptible to precipitating MS.

Diagnostic guidelines for MS recommend excluding chronic infections before concluding MS diagnosis—potentially accounting for underreported and underrecognized MS-HIV cases.¹ Therefore, the possibility of concurrent MS-HIV should never be excluded.

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Cytopenias to Polycythemia Vera: An Unusual Case

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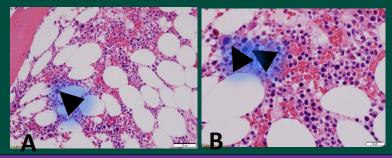
PRESENTER: Shaukat, Rimsha rshauk@lsuhsc.edu

INTRODUCTION

- -Hematological malignancies can take years to develop and are usually preceded by clonal mutations.
- -Premalignant lesions i.e. idiopathic cytopenia of undetermined significance (ICUS), Clonal cytopenia of undetermined significance can evolve into myeloid neoplasm.

CASE DESCRIPTION

- -81 y/o female presented with leukopenia, WBC of 2600/uL with ANC 620/uL. Hemoglobin 14.9 g/dL and platelets 156,000/uL
- -Bone marrow biopsy showed mild dysplasia with normal cytogenetics. Testing for clonal mutations were not performed at that time. Treated with G-CSF along with darbepoetin alfa for a decade.
- -Patient did well until mid-2019 when she started to develop thrombocytosis and erythrocytosis with WBC of 11.7 x103/uL, Hb 9.5 g/dL, and PLT 187,000/uL. Subsequent workup revealed JAK2 V617F mutation. She was initiated on phlebotomy, hydroxyurea and later switched to Ruxolitinib due to grade 3-mucositis.
- -Over the next few months she developed a leucoerythroblastic picture. Flow cytometry of blood showed increased myeloblasts of 6.5% of total cells. BMB showed hypercellular marrow with atypical megakaryocytic hyperplasia, erythroid hypoplasia with moderate to severe reticulin fibrosis, consistent with secondary myelofibrosis given history of polycythemia vera. Cytogenetics showed normal karyotype. Next generation sequencing showed ASXL1 mutation along with JAK2 mutation.
- -Given that the blast count was less than 10%. decision made to continue Ruxolitinib with the plan of initiating a hypomethylating agent if disease progression is noticed.



Findings on bone marrow core biopsy of 2006:

(A) Orderly myeloid maturation, normoblastic erythroid maturation, and focal dysmorphic megakaryocytes (arrows). Which is better observed at higher magnification (B) (H&E, 20x and 40x, respectively).

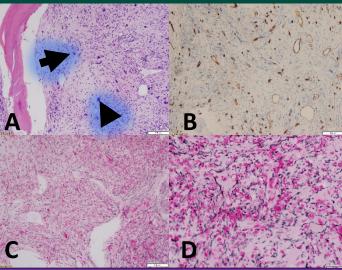


Figure 2. Finding on bone marrow core biopsy of 2020.

(A) Bone marrow core biopsy demonstrates hypercellular for the patient's age marrow with triliniage hematopoiesis, left shifted granulopoiesis, and erythroid hypoplasia. Also, we can observe bone marrow fibrosis and clustering of megakaryocytes with abnormal lobation and nuclear hyperchromasia (arrows) (H&E, 10x), CD34 stain (B) highlights vascular structures and highly increased number of blast cells (CD34 immunostaining, 10x). Reticulin stain (C, D) shows moderate to severe fibrosis (Reticulin immunostain, 10x and 40x, respectively).

DISCUSSION

- -This is an unusual case where a patient with cytopenias, either idiopathic cytopenia of undetermined significance or clonal cytopenia of undetermined significance requiring growth factor support, acquired a JAK2 mutation. Hence, required reversal of the treatment goal to phlebotomy, cytoreduction and eventually developed secondary myelofibrosis.
- -A causal relationship has not been reported in the literature between between G-CSF and JAK2 mutation. However, it is known that Philedelphia negative MPN (myeloproliferative neoplasms) are associated with a mutation in JAK2 V617F, DNMT3A or TET2.

CONCLUSION

It is unknown whether the JAK2 mutation occurred rather by chance or if the use of G-CSF contributed to this. Further research is required to investigate if there is any association or causation between the use of G-CSF and JAK2 mutation.

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Cytokine Storm in a Patient with Chronic Kidney Disease Stage IV

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Introduction

The main cause of death in patients with coronavirus disease 2019 (COVID-19) is acute respiratory distress syndrome (ARDS) with cytokine storms (1). Higher levels of inflammatory cytokines and chemokines are associated with lethal complications and poorer outcomes (1). IL-6 can mediate positive feedback loops (IL-6 amplifier) to induce cytokine storm (1).

Patients with chronic kidney disease (CKD) have dysregulated host immunity, persistent systemic lowgrade inflammation, increased cytokine production, and significant comorbidities (2); and are at increased risk for infection-related mortality (3).

Case Description

An 85-vear-old Caucasian woman with a past medical history significant for HFrEF (30%, moderate to severe MR/AR; 7/21/2020), atrial fibrillation on Eliquis, CKD stage IV, mild to moderate CAD (LHC, 4/1/2016), and depression presented to the emergency department in August 2020 for dyspnea on exertion, worsening orthopnea and chills. CT thorax revealed patchy peripheral reticular opacification of the bilateral lungs, mild bilateral pleural effusions and cardiomegaly. SARS-CoV-2 PCR was positive. Initial laboratory findings included : Cr 1.9mg/dL, BUN 34mg/dL, eGFR 27mL/min, NTproBNP 14,258, LDH 2052. Ferritin 380.7. CRP 8.6. ESR 22. WBC 5.4, D-dimer 0.41.

Although her hospital course was mainly uneventful, renal indices, inflammatory markers, and respiratory status continually worsened. On day 8, she was transferred to inpatient hospice on BPAP. Laboratory findings revealed: Cr 3.6mg/dL, BUN 102mg/dL, eGFR 13mL/min, NT proBNP 93,037, LDH 1458, Ferritin 10,041.4, CRP 22.0, ESR 51, WBC 7.3, D-dimer 2.75.

Results

Da	y	Cr	BUN	eGFR	NT proBNP	LDH	Ferritin	CRP	ESR	WBC	D- dimer
1		1.9	34	27	14258	252	380.7	0.6	22	5.4	0.41
2		1.8	37	28						3.7	
3		2.1	48	24		383	641	0.8	13	9.2	0.42
4		1.8	54	28						11	
5		1.8	54	28	37597	313	807.3	2.9	18	9.7	0.6
6		2.1	54	24	66999	516	1027.1	19	42	11.6	0.82
7		2.2	60	23	97300	747	1993.9	26	59	8.7	1.29
8		2.5	67	19	93037	820	2896.2	28	67	8	1.48
9		3.1	83	15	•					7.4	
10)	3.6	102	13	·	1458	10041	22	51	7.3	2.75

Table 1: Significant laboratory findings during hospital course.



Figure 1: Hospital Day vs Serum Creatinine (mg/dL)

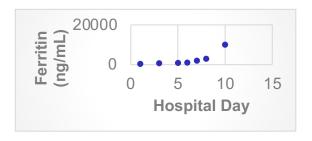


Figure 2: Hospital Day vs Ferritin Level (ng/mL)

Discussion

Patients with CKD have dysregulated immune systems and greater comorbidities, which are risk factors for severe COVID-19 disease (3). They are in states of chronic inflammation, and those on HD have 8-10 times higher cytokine levels compared to healthy individuals (2). CKD patients infected with COVID-19 are at greater risk for hospitalization and death due to the infection (5).

COVID-19 severity is associated with increased levels of inflammatory mediators (cytokines, chemokine) such as IL-1, II-7, IL-10, and TNF (1). Activation of IL-6 amplifier via NF-kB, STAT3 and non-immune cells can induce a cytokine storm, which leads to dysregulated inflammation, multiorgan failure and lethal complications of COVID-19 (1). Interestingly, filtration membranes used in hemodialysis could be designed to facilitate the clearance of cytokines and chemokines (4). However, it must be noted that activation of inflammation in hemodialysis can occur from bioincompatible dialysis membranes and exposure to contaminated dialysate (2).

Conclusion

Individuals with CKD who are hospitalized for COVID-19 have higher levels of cytokines and are at greater risk for lethal complications and death (3). Dysregulated immunity and chronic inflammation in chronic kidney disease may exacerbate inflammatory cascades that trigger IL-6 amplifiers to initiate cytokine storm. Further research is needed to risk stratify CKD and ESRD patients with COVID-19.

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Extracorporeal Membrane Oxygenation (ECMO) in Refractory Asthma

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Introduction

Asthma is defined by airflow limitation, underlying inflammation, and bronchial hyperresponsiveness to variable stimuli.

It is prevalent in roughly 8% of Americans.

Common symptoms include shortness of breath, coughing, and wheezing.

Case Description

The patient is a 36-year-old African-American female with history of hypertension and mild persistent asthma who presented to the hospital with worsening shortness of breath, wheezing, and chest tightness over 24 hours.

She was given methylprednisolone, albuterol-ipratropium, and oxygen en route via EMS.

On arrival, she was tachycardic, tachypneic, hypertensive, and required 4 liters of nasal cannula.

On exam, she was moderately distressed, had diffuse bilateral wheezing, and displayed use of her accessory muscles.

She was given magnesium sulfate, normal saline bolus, methylprednisolone, and continuous albuterol.

Initial lab work and chest x-ray were

An arterial blood gas (ABG) revealed hypoxemia and she was admitted to the hospitalist service.

On reevaluation, she had continued diffuse wheezing, tachypnea, and sternal retractions and was placed on BiPAP.

Unfortunately, she developed worsening respiratory distress, lethargy, and fatigue and repeat ABG showed respiratory acidosis and hypercapnia.

She was upgraded to the intensive care unit for intubation, mechanical ventilation, and sedation

She was started on sodium bicarbonate, magnesium sulfate, paralytics, and nebulized racemic epinephrine.

Ketamine infusion and epinephrine drip were added for bronchodilatory effects.

Case Description (continued)

Due to continued bilateral wheezing and poor air movement, Heliox (helium-oxygen) was initiated.

Ultimately, she deteriorated and developed worsening acidosis and hemodynamic instability and ECMO was pursued for venovenous CO2 removal.

After initiation of ECMO, her hemodynamics and airway pressures stabilized, acidosis resolved, and bronchospasm improved.

Respiratory status remained stable and ECMO was decannulated after 72 hours.

Soon thereafter, she was extubated and downgraded to the hospitalist service and weaned from nasal cannula as tolerated

She was discharged with close Pulmonology follow-up

Laboratory Data

PFTs from 2019: FEV1/FVC ratio: 66%, FEV1 80%

Sample ABG	art	art	art	arterial	art	arterial	art
Treatment	3L nc	bipap	vent	vent	vent/ecmo	vent/ecmo	vent
Site Art	Radial Lt	Radial Lt	Radial Lt	Radial Lt	Art Line	Art Line	Art Line
pH Art	7.370	* C 7.220	* C 7.160	* C 7.020	H 7.500	H 7.510	H 7.510
POC pH							
CO2 Art	35.0	* C 54.0	* C 97.0	>150	*C 51.0	42.0	42.0
POC pCO2							
pO2 Art	L 68.0	H 259.0	L 76.0	H 102.0	89.0	81.0	88.0
POC pO2							
HCO3 Art	L 20.2	22.1	H 34.6	incalculable	H 39.8	H 33.5	H 33.5
POC HCO3							
CO2 Totl Art	L 21.3	23.8	H 37.6	incalculable	H 41.4	H 34.8	H 34.8
POC TCO2							
02 Sat Art	L 92.7	H 99.8	L 91.0	incalculable	H 98.0	97.0	H 98.0
POC sO2							
D base	L -4.4	L -6.1	H 3.1	incalculable	H 14.5	H 9.5	H 9.5
POC BE							
THB ABG	13.3	13.3		12.7			
CO Hgb	* 1.9	* 1.5		* 1.7			
Met Hgb Art	1.2	0.9		1.2			
O2 Hgb	L 92.9	H 97.5		94.7			
CaO2	17.4	18.8		17.0			
Ionized Calcium	L 1.10	1.15	L 1.08	1.21	1.15	1.17	L 1.11
Sodium RT	L 133.0	L 134.0	137.0	139.0	139.0	L 136.0	L 136.0
Potassium RT	3.70	4.00	H 5.70	4.90	L 3.00	4.00	4.00
Allens	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Setting 1 ABG		bipap initiate	prvc/ac	ac 36	(c) prvc a/c 6x300	ac 12 vt 430	AC/PRVC
Setting 2 ABG		15/+5	420x30	pc@40	p+5 35%	p+5 30%	12 x 430
Setting 3 ABG		100%	p+5	p+5	(c) ECMO 3L 100%	ecmo 100%@2L	+6 25%
Setting 4 ABG			40%	55%		sweep 3600	

Images



Image 1: ECMO device in use at patient's



Image 2: Portable CXR post ECMO cannulation

Conclusion

Successful management of status asthmaticus is dependent on early intervention.

When conventional therapies have been exhausted without satisfactory improvement, the use of ECMO allows the opportunity for bronchial inflammation to subside

In this case, use of ECMO in the setting of refractory hypercapnia was effective in preventing mortality.

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Overuse of Telemetry in Medicine Wards - A Quality Improvement Project

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Introduction

According to Chaline et al. the overuse of telemetry monitoring causes a host of issues including increased cost of patient stay, risk of delirium, and alarm fatigue. They utilized a SoIVE method for quality improvement and were able to reduce their telemetry use by 23.1%.[1]

Stoltzfus et al. used a PDSA method to improve appropriate use of TM. Their project provided an 11.1% absolute reduction in the use of TM. [2,3]

Project Aims

Telemetry monitoring (TM) is widely used at Ochsner University Hospital & Clinics. It is often used inappropriately which leads to a shortages of monitors, inappropriate work-ups, alarm fatigue, and excessive waking of patients at night.

Systematically review TM use on the med-surg units to evaluate the percentage of patients being placed on TM and the length of use.

Utilize the PDSA method to improve the appropriateness of TM use.

Provide education based on the American Heart Association (AHA) 2017 Telemetry Monitoring Practice Guidelines.

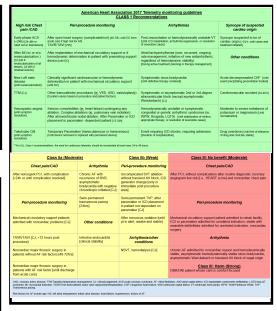
Methodology

Review and obtain data on the current use of TM on med-surg units.

Internal Medicine (IM) Residents and Family Medicine Residents (FM) will be educated on the appropriate use of TM.

An informative lecture will be given to IM residents during their clinic lecture to allow for 100% education of IM residents.

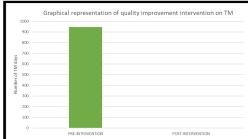
TM AHA Guideline Education Card



Methodology (continued)

- FM residents will be educated during their weekly program didactic time. Faculty will also be informed of the project during their monthly Faculty meting.
- Education will be in the form of PowerPoint presentations that review the AHA guidelines for appropriate TM.
- Color-coded, laminated, pocket sized educational cards (as seen above) summarizing the AHA 2017 TM guidelines will be made available and distributed to all Residents and Faculty. This will aid Residents by providing best practice recommendations for those placing inpatient orders for telemetry monitoring.
- Once all Residents and Faculty have been educated, we will prospectively analyze the use of TM over a 1 month period.

Conclusion



- The graph above represents the pre-intervention TM days admitted to IM and FM services during the month of January 2021. We are awaiting the post-intervention data which will be collected for the month of March 2021.
- In the upcoming month our goal is to analyze the post- intervention TM days. We hope to see, at minimum, a 10 percent reduction in telemetry monitoring.
- If this intervention is successful, we propose expanding this education to our affiliated hospitals in the health system. In the setting of a pandemic it is very crucial that resources are conserved and measures are taken to avoid waste. We are hopeful this project will help us achieve this goal.

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A CASE OF TRANSFUSION RELATED BACTERIAL INFECTION

Health NEW ORLEANS

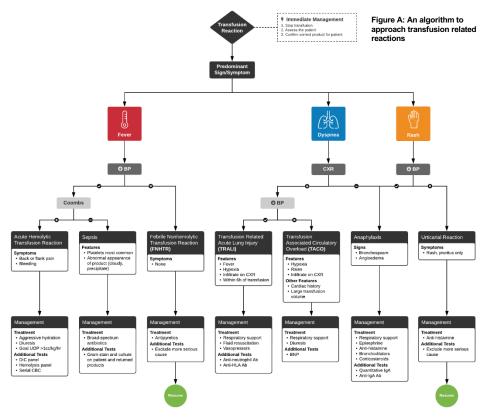
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Introduction

Transfusion of blood products are a common therapy given in the hospital. While usually very well tolerated, blood product related transfusion reaction can vary greatly from very common and mild to life threatening and severe.

Case Presentation

A 32 year old woman with a history of myelodysplastic syndrome with multiple transfusions presented with a 7 day course of increasing shortness of breath, fatigue, dyspnea on exertion, pallor and chest pain. The patient was alert and cooperative, tired appearing, had generalize pallor with pale conjunctiva. She was also noted to be mildly tachycardic. Labs were positive for a hemoglobin of 4.2 and a hematocrit of 11. She was started on an infusion of packed red blood cells. About 20 minutes into the infusion, she began to complain of increased SOB and chest pain and felt ill, having multiple episodes of emesis and diarrhea. She then developed rigors and spiked a fever of 105.1, became profoundly tachycardic and hypotensive and was given epinephrine, diphenhydramine and solumedrol. Repeat labs included CBC, LDH, haptoglovin, d-diner, fribringgen, Direct Antiglobulin Test, and procalcitonin. She had a hemoglobin of 3.9. WBC count of 0.4 with absolute neutrophil count of 300, and pro-cal of 524. She was started on epinephrine infusion, then switched to levophed and started on antibiotics. The transfused blood was tested again for compatibility and it was thought that patient could safely resume transfusions and tolerated them well. Gram stain on the transfused blood demonstrated Gramnegative rods in addition to Gram-positive cocci, and cultures of the transfused blood grew Rahnella aquatilis and Pseudomonas fluroesence The natient's blood cultures.



Discussion

Transfusion reactions are a relatively common occurrence; they can range from mild to severe and life threatening. Transfusion emergencies include TACO and TRALI, as well as hemolytic reactions. Transfusion related sepsis is a rare but documented adverse reaction (~1:5 million). In immunocompetent patients, *Rahnella aquatilis* is a relatively weak pathogen found in fresh water. There are, however, other documented cases of patients who become septic from this microorganism. *Pseudomonas fluroesence* is also a relatively weak pathogen, but case reports illustrate that this organism can be a source of bacteremia in patients who received contaminated infusions.



Figure B: Rahnella Aquatilis grown on an ALD agar culture plate



Figure C: Pseudomonas Fluroesence grown on sheeps agar culture plate

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Figure A: Image courtsey of Eric Madden Figure B: Case courtsey of CDC PHiL rID 6617 Figure C: Case courtsey of Pete Seidel rID 12457



Diagnosis and Management of Cardiac Hemochromatosis

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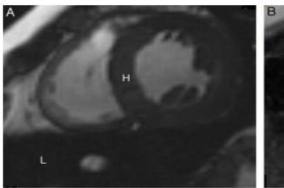
Introduction

Cardiac hemochromatosis is an important and preventable cause of heart failure. It often starts in the middle ages with hidden progression to cardiac dysfunction. It is characterized by dilated cardiomyopathy with dilated ventricles, and reduced ejection fraction. Unfortunately, once heart failure develops, there is a rapid deterioration. Therapeutic phlebotomy is the treatment of choice in nonanemic patients with cardiac hemochromatosis.

Case

A 51 year-old-male was admitted to our hospital with acute decompensated heart failure and new onset atrial fibrillation. He had a history of hereditary hemochromatosis, however, was noncompliant with medical follow up and with phlebotomy. On examination he appeared to have bronze skin and yellow eyes along with significant peripheral edema. Iron metabolism markers were obtained and were highly elevated. Echocardiography was significant for dilated ventricles, reduced ejection fraction, and reduced fractional shortening. Cardiac magnetic resonance (CMR) was consistent with diagnosis of myocardial siderosis. Standard HF treatment along with chelation therapy and regular phlebotomies were initiated

Results



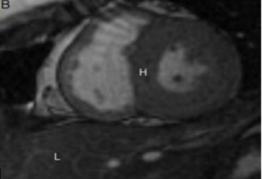


Figure 1. Cardiovascular magnetic resonance T2* images showing the heart (H) and liver (L) from two different patients at the same echo time. (A) Dark signal indicating severe myocardial and liver siderosis. (B) Normal myocardial and liver signal suggesting mild iron deposition.

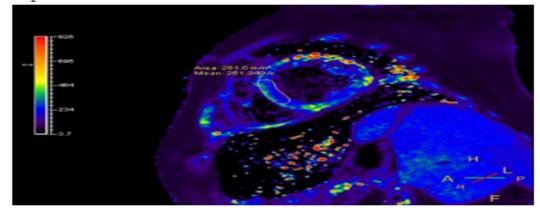


Figure 2. Evidence of severe myocardial and mild hepatic iron overload on T2*/R2* mapping:

Conclusions

In conclusion cardiac

hemochromatosis should be considered in any patient with new onset heart failure and treatment should be started in a timely manner.

Diagnostic confirmatory testing is typically pursued with cardiac MRI. Once diagnosed, treatment focuses on the reduction of the total iron load. Therapeutic phlebotomy is typically pursued, except for individuals with concurrent anemia or severe congestive heart failure (NYHA class IV). In those patients, treatment of choice is iron chelation therapy-including agents such as deferoxamine.

if left untreated the average survival is less than a year in those with severe cardiac impairment. However, if diagnosed and treated early, the survival rate can approach that of the regular heart failure population.

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The Dreaded Complication of Necrotizing Anterior Scleritis

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Introduction

Non-infectious anterior scleritis (Image 1) is associated with significant ocular comorbidity and reduced quality of life¹. Specifically, necrotizing scleritis patients have a high incidence of permanent visual loss².

In this case we wish to discuss the importance of early diagnosis and treatment of scleritis and the potential consequences of delayed intervention, including scleral melting, necrotizing scleritis, and dreaded globe perforation³

Case Presentation

- This was a 29-year-old African American male with past medical history of hydrocephalus with shunt, seizure disorder, and hypertension who developed granulomatous necrotizing scleritis with uveitis that eventually progressed to perforation.
- He initially presented to an ER with complaints of severe left eye pain for two weeks. Examination revealed left eye conjunctival injection. He was diagnosed with bacterial conjunctivitis and sent home with antibiotic eye drops.
- One month later, he re-presented with non-traumatic left eye pain and decreased visual acuity for several weeks.
 Examination revealed diffuse hyperemia of the left globe with scleral thinning and uveal prolapse supranasally.
- He was treated with methylprednisone 1g/day for 5 days followed by prednisone taper to 20mg/day and started on Methotrexate.
- Due to unresolving inflammation, he was then started on Humira with successful cessation of active inflammation.
- He is currently scheduled for a scleral patch and is only able to perceive light.

Case Images

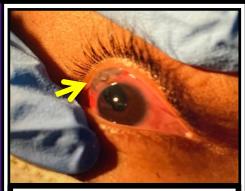


Image 1. Necrotizing scleritis with active inflammation on second presentation





Images 2A, 2B, & 2C: Scleromalacia of left eye while on Prednisone, MTX, and Humira, awaiting scleral patch graft

Conclusion

- Anterior scleritis (AS) is a difficult diagnosis to make because it can be confused with other more common pathologies such as conjunctivitis and episcleritis.
- AS usually presents with photophobia, severe dull, boring pain that is often worse at night and with eye movements.
- The vessels in AS are often largely dilated and have a violet-bluish hue, in contrast to conjunctivitis which has no photophobia, pain with extraocular movements and minimal to no eye pain^{1,4}.
- About 40-50% of non-infectious scleritis patients have an underlying systemic autoimmune condition, such as RA, GPA, microscopic polyangiitis, SLE and sarcoidosis^{5,6}.
- Potential complications include keratitis, cataract formation, uveitis, and scleral thinning which can result in globe perforation, which was evidenced in our patient⁷.

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TTP Associated with COVID Pneumonitis

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Introduction

The SARS-CoV-2 Virus has been associated with immune complications, thrombotic microangiopathy syndromes in addition to pulmonary infection (1).

We are reporting a case of TTP associated with COVID-19.

Case Description

- A 71 year old woman with an extensive medical history including atrial fibrillation, diastolic heart failure, diabetes mellitus, hypertension, and coronary artery disease was brought in by her family with complaints of increased confusion, physical deconditioning, generalized weakness and difficulty ambulating for 1 day.
- A day prior she had been discharged home after a hospital course during which she was treated for COVID pneumonia.
- She had come in with a three week history of nonproductive cough and had demonstrated bilateral alveolar and interstitial infiltrates with bihilar congestion on chest xray.
- The patient received Ceftriaxone, Azithromycin, Remdesivir and also Dexamethasone. On her second presentation she was noted to be confused and lethargic.

Case Description cont.

- Given her altered mentation, hemolytic anemia, thrombocytopenia, and acute kidney injury, the following were ordered due to suspicion of TTP vs autoimmune thrombotic microangiopathy: Haptoglobin, Fibrinogen, ADAMTS13, C3/C4, Reticulocytes and peripheral smear.
- Nephrology was consulted for plasmapheresis given suspicion of the patient having TTP secondary to COVID Pneumonia.
- She was initiated on plasmapheresis and ADAMTS13 level came back as 54 with Haptoglobin of 19. C3/C4 levels of 37/<2.9 and peripheral smear showed schistocytes confirming diagnosis of AI TTP. She was then initiated on Plama Exchange.

Lab Results at 2nd Admission (Post COVID-19 Diagnosis)

Lab Test	Result		
BUN/Creatinine	53/1.60		
Total Bilirubin	3.0		
LDH	584		
Ferritin	333		
ESR/CRP	9/0.5		
PT/PTT	7.7/87.6		
WBC	15.5		
Hb/HCT	10.5/37.3		
Platelets	85 (156 at 1 st Admission)		

Discussion

COVID-19 has been associated with several reported cases of TTP which could be due to ADAMTS13 inhibitors in addition to the cytokine storm and diffuse endothelial inflammation attributed to it (2). Interestingly, a similar case of relapsing autoimmune TTP associated with COVID 19 was reported by Capecchi, M. et al. however unlike our case, the patient had experienced a prior bout of TTP secondary to bacterial pneumonia (3). GBS, APS, AI Hemolytic Anemia and ITP have also been associated with COVID-19 indicating the importance of considering these disease processes as part of the differential when treating COVID pneumonia (4).

Conclusion

This case presents an instance of TTP developing in a patient who had just been treated for COVID Pneumonitis with the typical findings of microangiopathic hemolytic anemia, consumptive thrombocytopenia, renal insufficiency and altered mentation.

It provides insight into the variable complications COVID can lead to and calls for further research into diagnosing and treating them earlier given the limited number of cases of COVID-associated TTP that have been reported.

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