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**Effectiveness of Vagal Maneuvers in Supraventricular Tachycardia:
A Network Meta-Analysis**
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[Judges Award: Best Poster]

Introduction

Vagal maneuvers are recommended as first-line treatment for paroxysmal supraventricular tachycardia (PSVT) before medical intervention to terminate PSVT. We evaluated the effectiveness of different types of vagal maneuvers in treatment of PSVT, specifically comparing modified Valsalva versus standard Valsalva maneuvers.

Methods

Two existing meta-analyses were updated with a search for newer trials using a mix of methods as described at openMetaAnalysis (<https://openmetaanalysis.github.io/Supraventricular-tachycardia-treatment-with-vagal-maneuvers/>). Randomized controlled trials were added that included the Valsalva maneuver for treating supraventricular tachycardia. Studies were abstracted into standardized tables of PICO attributes, Cochrane risk of bias, and results. The strength of evidence was qualified with a Grading of Recommendations Assessment, Development and Evaluation (GRADE) Profile.

Results

Results are online at <https://openmetaanalysis.github.io/svt/>. Five trials were included: three trials from before 2000 that compared the standard Valsalva to carotid sinus massage (CSM) and two recent trials that compared the modified and standard Valsalva maneuvers. When the two newer trials were pooled together, the modified Valsalva maneuver is favored with an RR of 2.60 (95% confidence interval: 1.90-3.58) and heterogeneity of $I^2 = 0\%$. Network meta-analysis indicated that the modified Valsalva had a 93% probability of being the most effective vagal maneuver and will convert almost 50% of episodes. The next most effective maneuver was the supine Valsalva; least effective maneuvers were the standing Valsalva and CSM. Using the GRADE framework, the quality of evidence was moderate due to small number of patients studied. In addition, more than 25% of participants were from studies with unclear quality due to lack of trial registration.

Conclusions

The modified Valsalva was the most effective vagal maneuver for achieving normal sinus rhythm. Although the evidence is of moderate quality, we encourage trial of the modified Valsalva maneuver due to its safety and low cost. Execution of the maneuver can be standardized by simply blowing into a 10 cc syringe with sufficient force to move the plunger. If the equipment is not available, a supine Valsalva maneuver should be tried for hemodynamically stable patients.

The Daily Complete Blood Count: Do We Get It When It Really Counts?
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Introduction

Routine laboratory tests are a common source of medical overuse and have been targeted by the ABIM's Choosing Wisely campaign as a means of eliminating medical waste. This quality improvement project aimed to decrease the amount of daily Complete Blood Count (CBC) lab tests obtained on an inpatient academic general medicine team by 10% by utilizing multiple interventions.

Methods

The QI project was conducted at KUMC February-May 2017 with an academic general medicine team. The intervention was primarily carried out via education of the physician team regarding costs, consequences and indications for a CBC including monitoring for bleeding, new anticoagulation start, anemia, thrombocytopenia and sepsis. Other interventions included placing reminder fliers in team areas, incorporating a labs section into physician EMR notes, addressing daily labs with nursing during multidisciplinary rounds and sending reminder e-mails to involved physicians. The team's patients were initially chart reviewed for 30 days pre- and post-intervention to determine the percentage of patients with a CBC each day before extending the project to nearly two months duration. Pre-post analyses were then performed using both data ranges.

Results

For the 30-day pre-post analysis, the project resulted in a decrease in the percentage of patients with a daily CBC of 0.056 (6.2%), from 89.8% pre-intervention (n = 30) to 84.2% post-intervention (n = 30). This reduction did not meet our original goal of a 10% decrease in lab frequency but did produce a statistically significant result ($p < 0.05$). With extension of the project, however, the percentage of patients with a daily CBC decreased by only 0.025 (2.8%), from 90.8% pre-intervention (n = 59) to 88.3% post-intervention (n = 52), not meeting our initial goal nor statistical significance ($p = 0.19$).

Conclusions

A multifaceted QI project using education and several avenues of physician reminders can result in a small, but significant, decrease in daily CBCs at a major academic medical center; however, extension of the project demonstrated difficulty sustaining consistent results long-term. This project suggests that decreasing lab frequency can be an effective means of reducing medical waste, but success likely hinges on the resolve of the involved staff and implementation of more interactive and persistent interventions. Additional studies are warranted regarding further expansion of this approach to include routine labs for all inpatient general medicine teams, investigation of patient outcomes and development of more effective means for achieving long-term sustainability.

Early Rehabilitation in Mechanically Ventilated Patients: A Meta-Analysis
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Introduction

Critical illness weakness is a commonly acquired condition noted amongst patients in the intensive care units. Critically ill patients often are placed on bedrest to allow for minimal activity and stimulation while acute medical problems are being treated. Barriers to mobilization, such as concern for endotracheal tube displacement or agitation of patients, often increase time to ambulation. As a result, prolonged immobilization can lead to myopathies and neuropathies. Early therapy and ambulation in patients often lead to a decrease the length of stay in the intensive care units (ICU). This meta-analysis examined the association between early mobilization and length of intensive care unit stay on mechanically ventilated patients.

Methods

An electronic search of PubMed was conducted using the following terms: “mechanically ventilated ambulation,” which resulted in 47 articles. Studies were included if they were randomized controlled trials. The patient population was limited to only mechanically ventilated patients in the intensive care unit. The main intervention examined was early rehabilitation, which included bed-bound exercises and ambulation. Studies were utilized if they included length of stay in the intensive care unit as the primary or secondary outcome.

Results

A total of 656 patients (336 in the intervention arm) were identified across four randomized clinical trials. Three studies reported outcomes as the median and inter-quartile range. The mean and standard deviation were estimated from the median and inter-quartile range. Owing to significant heterogeneity across the studies ($\tau^2=8.33$, $P\text{-val}<0.0001$), a random effects model was used to examine the effect of early rehabilitation on the length of ICU stay. Mean difference was used to examine this effect. Mechanically ventilated patients receiving early rehabilitation were estimated to spend on average about three fewer days in the ICU compared to patients receiving routine care (95% CI: -5.87 to 0.31, $P = 0.08$).

Conclusions

Early rehabilitation practices may not lead to a significantly lower duration of stay in the ICU for critically ill patients, although the results trended toward it. Our findings are in agreement with a larger study by Tipping et al. However, our analysis included fewer studies, which contributed to significant amount of heterogeneity. Median and inter-quartile range reported in most of the studies suggested that the data may not be normally distributed; thus the normality assumption for the meta-analysis is dubious. Based on this meta-analysis, early rehabilitation may reduce the average length of ICU stay, although not significantly.

Colorectal Cancer Screening: A Shift Toward a “No Preference” Strategy
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Introduction

Current guidelines for colorectal cancer (CRC) screening offer conflicting recommendations regarding the preference of one screening strategy over another, specifically fecal immunochemical test (FIT) and optical endoscopy (OE). Given that colonoscopy is the most accurate tool for CRC detection, many guidelines recommend a sequential approach for CRC screening starting with colonoscopy before moving to other strategies. However, more recent guidelines are moving towards a “no preference” strategy. Our aim was to perform a systematic review and meta-analysis to evaluate adherence and detection rate of CRC by FIT compared to OE in effectiveness trials. For OE, we included studies of either sigmoidoscopy or colonoscopy.

Methods

We included randomized trials comparing the effectiveness of FIT to OE for CRC detection. We searched Medline, Google Scholar, and Cochrane through May 2017. Effectiveness trials are defined as studies that measured the performance of an intervention under “real world” conditions. Eligible studies were searched for variables of interest including attendance rate and CRC detection. All statistical analyses were performed using the R Programming Language. Outcomes were reported as Relative risks (RR) with 95% confidence interval and heterogeneity was reported as I² statistics.

Results

A total of seven randomized controlled trials were eligible and included in the analysis. There were a total of 59912 subjects in the FIT group and 63100 in the OE group. Attendance rate was significantly lower for OE compared to FIT (RR= 0.62, 0.49-0.78; I² = 99%). CRC detection was comparable between the two groups regardless of OE technique used (RR= 1.25, 0.61-2.57; I² = 44%). Restricting the analysis to subjects who complied with testing, no statistically significant benefit was found for colonoscopy or sigmoidoscopy over FIT for CRC detection. However, a trend towards significance was noted when both OE techniques were combined (RR=1.69, 1.00-2.85; I² = 74%).

Conclusions

While colonoscopy is the most accurate screening test for CRC in efficacy trials, FIT is as effective as colonoscopy for CRC detection under real world circumstances. The effectiveness of screening depends not only on the sensitivity for neoplasia but also on population attendance and other factors that are undetected in efficacy trials. This supports the shift of more recent guidelines towards offering multiple screening options for the patient, specifically FIT and colonoscopy rather than a sequential approach. However, given the heterogeneity of the studies and the absence of long-term randomized trials assessing mortality of FIT vs colonoscopy testing, preference of one strategy over another is controversial and further studies are needed.

Treating More, but Making a Difference?
Assessing Guideline Recommended De-Escalation of PPI Therapy for Upper-GI Bleeds
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Introduction

Gastrointestinal hemorrhage is a common reason for inpatient hospitalization with over 500,000 admissions in 2012 at a cost of nearly 5 billion dollars. The current American College of Gastroenterology (ACG) guidelines delineate appropriate therapy for suspected and endoscopically-confirmed ulcer and other upper gastrointestinal (GI) bleeding, however adherence to guidelines including appropriate de-escalation of therapy in low-risk individuals has not been evaluated. In this review we analyze the adherence to ACG guidelines with regards to de-escalation of therapy post-endoscopy in low-risk patients from IV to oral proton pump inhibitor (PPI) therapy.

Methods

An IRB-approved search was made using the HERON data repository for patients admitted during 2016 to KU Medical Center for GI bleeding who underwent upper endoscopy. In total, 897 patients were identified, and 100 charts were randomly selected for analysis by a team of reviewers using a pre-generated template for data collection. Pre-endoscopy PPI therapy, endoscopic findings and consultative recommendations, and post-endoscopic PPI therapy route were recorded and a separate reviewer compiled results for analysis.

Results

Of the 897 patients screened, 100 randomized charts were reviewed and 67 were found to meet the criteria of admission for suspected upper GI bleed with endoscopy performed during admission with results available for review. For empiric therapy, 100% of patients were started on intravenous PPI drip or intermittent IV therapy pre-endoscopy, consistent with guidelines. Of the inpatient admissions, 57% (38 patients) were found to have low-risk endoscopic findings, defined as flat or clean-based ulcers with no active signs of bleeding. For these low-risk patients 32% (12) were found to not have therapy de-escalation post endoscopy, with continued previous PPI drip being the main variance.

Conclusions

While guidelines are well-established for PPI therapy in upper GI bleed and suspected bleed, a lack of adherence to de-escalation in treatment regimens may lead to overtreatment and prolonged hospital stay. The ACG guidelines outline that if no high risk sequelae are found then therapy can be de-escalated to oral and the patient discharged the same day. With nearly 900 admissions annually for GI bleed and over 3,000 patient days, the tendency of overtreatment with IV medications may increase hospital days an estimated 200 days annually, at a cost of nearly 500,000 dollars per year. Further study is needed in this arena to determine both estimated cost-savings for adherence to guidelines as well as difference in patient outcome in groups in guideline-adherent versus guideline non-adherent groups.

**Primary Mycobacterium Avium-Intracellulare Septic Arthritis:
A Case Review and Systematic Review of Literature
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KU School of Medicine-Wichita**

Introduction

Mycobacterium avium-intracellulare (MAI) is a well-recognized opportunistic organism implicated in pulmonary infection in an immuno-compromised patient. Primary septic MAI arthritis, however, is uncommon with few cases described in the literature. This systematic literature review compiled and assessed available evidence regarding the a) average time to diagnosis from first symptom, b) relationship to previous trauma/procedure on the joint, c) immune status, d) mode of diagnosis, e) treatment strategy, and f) outcomes of primary MAI septic arthritis.

Our case involved a 69-year-old male with polymyositis on chronic low disease steroids who developed spontaneous left shoulder pain and swelling thought to be streptococcal septic arthritis and treated with empiric antibiotics with no resolution. MRI showed glenohumeral damage and inflammation. Synovial aspiration, incision and drainage, and surgical debridement were essential to obtain cultures and ascertain pathological diagnosis of MAI arthritis 5 months after the onset. There was a high degree of resistance to first line ethambutol and the patient was treated with azithromycin and rifabutin with good improvement.

Methods

A systematic database search according to the PRISMA statement was conducted. Reference lists were searched for cases of primary MAI septic arthritis. Our case was included in the analysis of outcomes.

Results

The primary search yielded 28 articles. Two articles were excluded since they were not cases of “intra-articular” infections. Forty-two cases were reported from the 26 articles plus our case was added for analysis. Data were extracted by two reviewers and analyzed by a third independent reviewer.

Average time to diagnosis from the onset of first symptom ranged from 1 - 156 weeks (mean: 27 weeks). Spontaneous onset was noted in 17 (40%) cases; while recent or remote history of trauma was described in 7 (16%) cases. Four (9%) cases reported some form of procedure prior to the onset of articular symptoms. Nineteen (44%) patients were immune-compromised. Among those 19, 6 (31%) had HIV-AIDS related disease and 10 (53%) had an autoimmune condition and were on long-term immunosuppressive treatment. Thirty-two (75%) cases required extensive incision drainage, surgery, culture, and histopathology of the debrided synovia to establish the diagnosis. Resistance was described to first line anti-mycobacterial infections in 7 (16%) cases. Treatment lasted for 12 - 18 months with 28 cases (65%) reporting good outcomes with no sequelae. Five patients (12%) had severe immune-compromised states or extensive resistance patterns to drugs and poorer outcomes including death.

Conclusions

Spontaneous infection with atypical bacteria including MAI should be considered early when dealing with patients who are immuno-compromised. Synovial biopsy, MRI, and AFB cultures from incision, drainage and debridement of the joint may be required to make the diagnosis. It is imperative to get susceptibilities of the isolate and treat accordingly for at least 12 - 18 months for good results.

**Are Antibiotics Beneficial After Incision and Drainage of Skin Abscesses?
A Network Meta-Analysis
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KU School of Medicine-Wichita**

Introduction

The Infectious Diseases Society of America (IDSA) 2014 Guidelines for the Diagnosis and Management of Skin Infections only recommend antibiotics after drainage of skin abscesses when systemic inflammatory response syndrome (SIRS) is present. Regarding choice of antibiotics, “an antibiotic active against MRSA is recommended for patients ... who have failed initial antibiotic treatment or have markedly impaired host defenses or in patients with SIRS and hypotension.” Since 2014, conflicting trials have been since published. We meta-analyzed all trials since the emergence of community MRSA infections (1990s) of antibiotics versus placebo in patients after incision and drainage and the antibiotic impact on clinical cure versus placebo.

Methods

We used methods of openMetaAnalysis which combine features of scoping, rapid, and living reviews. We searched for trials in prior meta-analyses with a mix of methods including ClinicalTrials.gov and a PubMed vector search (“See Related Articles”). Quality of trials was assessed using the Cochrane Risk of Bias Tool. Meta-analysis used a random effects model with Hartung-Knapp approach. Network meta-analysis used a Bayesian framework and arm-based study results. Analyses were done with the R Programming Language.

Results

Results are online at <https://openMetaAnalysis.github.io/ssti>. Restricting the analysis to trials using antibiotics active against the prevailing staphylococcus aureus, the benefit was significant (RR 0.67, 95% CI 0.53 to 0.84, I² = 0%). Network meta-analysis shows the relative risks of failure from adding antibiotics: trimethoprim-sulfamethoxazole (TMP-SMX) 0.60 (95% CI: 0.36 to 0.84); clindamycin 0.49 (0.23 to 1.00); cephalexin 1.8 (0.56 to 6.4). In the only placebo controlled trial of children, the relative risk of TMP-SMX was 0.78 (0.17 to 3.61). In addition, the failure rate among children was much lower (5.3%) than among adults 26.4 (range: 9.5 to 31.1). The trials were of high quality as 85% of subjects used in the principal analysis were at low risk of bias.

Conclusions

In adults undergoing drainage of skin abscesses, antibiotics that target MRSA improved clinical cure rates. In children, the risk reduction may be similar, but insufficient research precludes certainty and the low rate of failure among untreated children reduces absolute benefit.

Acute Myeloid Leukemia after Treatment for Pancreatic Neuroendocrine Tumor with Temozolomide and Thalidomide

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Introduction

With the acceleration in new drug developments, patients have more treatment options for improved survival and quality of life. However, adverse effects that manifest later in life, including secondary malignancies caused by chemotherapy and radiation, may limit drug use. Temozolomide is an alkylating agent that is FDA-approved for the treatment of glioblastoma multiforme and refractory anaplastic astrocytoma. Unfortunately, a number of cases have reported the development of hematologic malignancy after temozolomide therapy in patients with gliomas. Though not FDA-approved, the National Comprehensive Cancer Network (NCCN) includes temozolomide as an option for treating metastatic neuroendocrine tumors (NET). Unlike gliomas, there are no published reports of leukemogenic potential in the treatment of NET. This is the first case presentation showing the development of Acute Myeloid Leukemia (AML) after temozolomide treatment in a patient with metastatic pancreatic NET.

Case Presentation

A 29-year-old female presented to endocrinology for diabetes and hypercalcemia in April 2005. Past medical history included episodic palpitations, hypertension, hypothyroidism, anxiety, and obesity. Abdominal imaging revealed a 10.7 x 8.8 x 9.5 cm lobulated mass within the pancreatic tail and multiple liver masses. Pancreatectomy and debulking of the liver masses confirmed the diagnosis of moderately differentiated NET in the pancreas with liver metastasis. Workup for Multiple Endocrine Neoplasia 1 and 2 was negative. In addition to octreotide therapy, she underwent a number of transhepatic arterial chemoembolizations to control extensive liver-dominant disease. Despite these measures, CT showed progressive disease. Systemic therapy with bevacizumab was started in April 2007 which improved her hypercalcemia, flushing, and diarrhea.

A few months later, she experienced upper GI bleeding from a friable, gastric mass. Bevacizumab was discontinued. Radiation therapy resulted in good palliation of the bleed. However, she continued to show clinical, biochemical, and radiographic evidence of rapidly progressive disease. In March 2008, she started a treatment of 150 mg/m² temozolomide daily for 7 days, every 14 days and daily thalidomide with intermittent treatment breaks. This provided tumor shrinkage and symptomatic relief.

In March 2011, she complained of worsening fatigue. Labs revealed macrocytic anemia and thrombocytosis. Bone marrow evaluation and cytogenetics showed presence of infiltrating monomorphic myeloid blasts and presence of inversion 3q with breakpoint at q21 and q26, consistent with an aggressive type of AML. All treatment for NET was stopped and AML therapy with daunorubicin and Ara-C in a 3 and 7 combination was started. A year later, she passed away from immunosuppression complications.

Conclusions

Patients like this young woman have the potential to live longer and may live long enough to develop secondary malignancy. Leukemogenic potential of temozolomide should be further evaluated as its use increases. This is the first report showing possible development of AML in patients with metastatic NET treated with temozolomide.

It's Not Just Diarrhea, Cha Cha Cha!
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KU School of Medicine-Kansas City

Introduction

Gastrinomas are rare neuroendocrine tumors which cause gastric acid hypersecretion via high gastrin production, better known as Zollinger-Ellison syndrome. Clinically this presents as chronic diarrhea, abdominal pain, dyspepsia and ulcerations in the stomach and duodenum.¹ These tumors tend to occur in the duodenum but a small percentage are found in the pancreas (10 - 40%).^{2,3} Herein, we describe a patient who presented with chronic diarrhea and intractable nausea and vomiting with an extensive but unremarkable previous work-up.

Case Presentation

A 56-year-old white male presented to the ED with diarrhea of six months and acute intractable nausea, vomiting, and dyspepsia for four days. His past medical history was significant for multiple prior admissions with similar complaints for which no etiology could be found. His physical exam was remarkable for tachycardia, dry mucous membranes and mild abdominal tenderness. Laboratory work showed a hemoglobin of 17.5g/dL, platelets of 326K/uL, and white blood cells of 18.7K/uL. Potassium was 3.4mEq, chloride 96 mmol/L, BUN 44mg/dL and Cr 1.89mg/dL. Supportive therapy was initiated with fluid resuscitation, anti-emetics and antacids.

Initial work up for the chronic diarrhea included infectious, malabsorption and inflammatory causes but was negative. Further testing was pursued because of persistent symptoms and initial negative work up. Further work up included more rare causes of chronic diarrhea which revealed elevated serum gastrin (4195pg/mL), chromogranin A (138ng/ml) and urinary 5-HIAA levels (8.7mg/24 hr). An ultrasound of his abdomen showed a hypoechoic nodule in the right lobe of liver. CT of the abdomen and pelvis showed a low-density hepatic mass, a retroperitoneal mass and associated mesenteric adenopathy. EGD showed erosive esophagitis and diffuse inflammation, erythema and multiple non-bleeding ulcers in the entire duodenum. EUS showed a hypoechoic lesion in the pancreatic uncinata process. This was biopsied and showed neoplastic cells positive for Pancytokeratin, Synaptophysin, CD56, Chromogranin, and Gastrin consistent with diagnosis of a neuroendocrine tumor, gastrinoma subtype. Multiple Endocrine Neoplasm syndrome testing was negative. An octreotide scan showed somatostatin-receptor-positive tumor involving the left retroperitoneal mass with areas concerning for metastasis at the duodenum and in the liver.

Patient was started on octreotide and underwent Whipple procedure with symptom resolution and is currently on surveillance.

Conclusions

Work up of diarrhea is variable depending on resource availability (e.g. resource-rich versus resource-limited settings). A thorough history and physical exam may point towards a specific diagnosis. Nocturnal diarrhea, persistence despite fasting, large volume stools and non-responsiveness to empiric therapy are characteristics for secretory diarrhea. These characteristics are clues to broaden differential diagnoses to include secretory causes of diarrhea. While secretory causes of diarrhea are overall rare, they become more important once more common causes of diarrhea have been ruled out.

A Deadly Cause of Abdominal Pain
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Introduction

Infections with varicella, the herpes virus responsible for chicken pox and shingles, can occur in a primary or reactivated form and presents as cutaneous lesions in a vast majority of cases. Disseminated varicella is very rare but much more common in immunosuppressed and bone marrow transplants patients and is often fatal. Disseminated varicella is usually preceded by a rash, but in rare instances can present itself as abdominal pain, hepatitis or other clinical syndromes. This can make diagnosis very difficult, which is critical due to its high mortality and the importance of early antiviral treatment.

Case Presentation

A 51-year-old man with a past medical history of panhypopituitarism, adrenal insufficiency, diabetes insipidus and multiple myeloma post autologous stem cell transplant (ASCT) presented to ED with abdominal pain and fever. CT abdomen/pelvis showed a few bony lytic lesions but no acute process, lipase was within normal limits and basic labs were unremarkable. Shortly after admission his serum sodium declined from 138 to 130 and eventually to 123. Urine osmolality was high which was consistent with SIADH. AST and ALT climbed with a peak of 1562 and 1277 respectively. Creatinine elevated from 1 to 3 within 2 days. EGD showed gastric and duodenal ulcers but biopsies were non-diagnostic. While these symptoms seemed random and difficult to connect, on the second day a vesicular rash developed, starting on the scalp and spreading to the chest, abdomen and proximal lower extremities. Given his immunosuppression, empiric antiviral therapy was started and fluid was sent for varicella zoster virus (VZV), which was positive along with high serum levels of VZV found later. The patient was transferred for ICU for sodium correction. Shortly after transferring to the ICU, the patient became altered, hypotensive, and experienced respiratory failure. He was intubated and started on vasopressors and stress dose steroids. Patient required CRRT from renal failure and, despite supportive care, never recovered consciousness. Eventually his family transitioned to comfort measures and he passed away.

Conclusions

This case highlights the importance of looking for atypical presentations in immunocompromised patients. Disseminated varicella has been associated with a multitude of different clinical complications affecting the liver, kidneys, or even causing DIC. Our patient represents an exceedingly rare case in which both abdominal pain and SIADH presented before cutaneous lesions. This patient developed extensive organ damage including hepatitis, esophageal and duodenal ulceration, and severe thrombocytopenia, possibly due to DIC, all of which have been associated with varicella. Because of the havoc this process can wreak, it is important to keep a wider differential in our immunosuppressed patients and be aware of atypical presentations of infections in order for timely and life-saving treatments.

MSSA Bacteremia in an Immunocompromised Patient
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Introduction

Catheter-related bloodstream infections remain a significant cause of morbidity and mortality. Risk factors that contribute to the development of these bloodstream infections include chronic illness, immune deficiency, and malnutrition. Staphylococcus aureus accounts for a majority of these infections. S. aureus bacteremia often has a complicated clinical course and identification and treatment of complications is critical.

Case Presentation

A 37-year-old male with a medical history of End Stage Renal Disease on hemodialysis and a history of failed kidney transplant on chronic immunosuppression presents with complaints of chills, chest pain, and shortness of air. He had leukocytosis, fever, tachycardia, and lactic acidosis on admission. A pansystolic murmur was auscultated on exam. A dialysis catheter had recently been placed as an outpatient due to an inaccessible arteriovenous fistula. Cultures were obtained and he was started on broad spectrum IV antibiotics. Peripheral blood and dialysis line cultures grew Methicillin-Susceptible Staphylococcus aureus. His dialysis line was removed and he was transitioned to IV Cefazolin. His immunosuppression medications were held. Transesophageal echocardiogram showed vegetations in the right atrium and on the mitral valve with mild regurgitation of the mitral valve. CT angiography showed multiple cavitary lung lesions in the chest. Bronchoscopy was performed to evaluate the cavitary lung lesions and cultures from the abscesses grew MSSA. Acid fast bacilli cultures were negative and Histoplasmosis urine antigen was indeterminate. Blood cultures remained positive for seventy two hours. A new dialysis catheter was placed after negative blood cultures were obtained and dialysis was resumed. The patient was discharged to home to complete a six week course of IV antibiotics. A transesophageal echocardiogram will be repeated after completion of the antibiotic course followed by possible mitral valve replacement.

Conclusions

Patients with immunocompromise have a complicated course of Staphylococcus aureus bacteremia. Complications include endocarditis, persistently positive blood cultures, and septic emboli. All patients with S. aureus bacteremia should have an echocardiogram performed to evaluate for vegetations. A transesophageal echocardiography is more sensitive in identifying valvular vegetations and is more sensitive when performed five to seven days following the onset of bacteremia. While a transthoracic echocardiogram can be performed initially, in the setting of hemodialysis the transesophageal echocardiogram is necessary. Indications for replacement of an infected valve include heart failure, persistent infection, large vegetations, and peripheral embolism. Mitral regurgitation should also be a consideration for replacement.

A Case of Extrapulmonary Tuberculosis, Two-Ways
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Introduction

Tuberculosis (TB) is a widespread epidemic with an estimated 10.4 million new cases in 2015. The primary presentation of tuberculosis is pulmonary; however, in the United States in 2014, 21% of cases were extra-pulmonary. This case exhibited two different presentations of extra-pulmonary TB: abdominal/peritoneal TB and TB meningitis/encephalitis without concurrent pulmonary TB. The most common pathogenesis of extrapulmonary TB is from hematogenous spread from primary pulmonary TB, with only 15 - 25% of abdominal TB patients and 40% of TB meningitis patients having concomitant pulmonary TB.

Case Presentation

A 72-year-old Laotian male with known hypertension, gout, and stage 3 chronic kidney disease presented with acute encephalopathy and cyclic fever. He had abdominal ascites which resolved two days prior to admission. A previous workup showed omental caking and ascites, suggestive of peritoneal carcinomatosis, and multiple small intraparenchymal lesions with bilateral cerebral hemispheric involvement. The patient had a fever, visual hallucinations, a soft abdomen with no ascites, and was disoriented. He was started empirically on dexamethasone, vancomycin, and ceftriaxone for suspected central nervous system infection and encephalitis.

His workup included negative CEA, PSA, and CA19.9. IgM, histoplasma urine antigen, and HIV 1 and 2 antibodies were negative. Two blood cultures showed no growth. A tuberculosis Quantiferon test and toxoplasma IgG were positive. Chest x-ray showed no signs of cavitary lesions. A biopsy of brain lesions showed caseating granuloma, suspicious for TB, which was confirmed by mycobacterial cultures. The patient was started empirically on quadruple therapy for TB based on the pathology of the brain biopsy.

Conclusions

Peritoneal tuberculosis presents in three successive forms: wet type with ascites, fibrotic type with localized abdominal swelling, or dry-plastic type with the typical “doughy” abdomen and omental caking. TB meningitis is a more common presentation of extra-pulmonary tuberculosis with typical clinical presentation of stiff neck, headache, fever, vomiting, and altered consciousness. Risk factors for extra-pulmonary tuberculosis in the general population include HIV infection, young age, old age, especially if age greater than or equal to 65, Asian or African origin, and female sex.

A high level of clinical suspicion is needed with regards to diagnosing tuberculosis in a patient who is at high risk for developing extra-pulmonary tuberculosis. Abdominal ascites has a wide differential, as do intraparenchymal brain lesions; however, TB should be included in the differential when risk factors are present.

Sorting Out Genital Lesions
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Introduction

Sexually transmitted infections (STIs) have complex social and public health implications, in addition to their medical significance. Even with the introduction of effective treatments, such as penicillin for syphilis more than 60 years ago, syphilis remains an important disease. In fact, the rate of syphilis in men who have sex with men (MSM) is on the rise in some areas of the United States. STIs remain among the most common infectious diseases in developed and developing countries. The fact that diseases for which there are effective therapies, illustrates the complex nature of these diseases and the enormous challenges faced by communities. Here we describe a case of primary syphilis.

Case Presentation

A 36-year-old male was seen for pre-exposure prophylaxis for HIV. He had been on Truvada prior to his presentation, with no missed doses. His frequent STI screenings had been negative. He was on intermittent acyclovir treatment for genital herpes and denied any other STI history. Prior STI testing was negative. He is a MSM, and has sex with multiple partners with inconsistent condom use. Past medical history includes: genital herpes, right radical nephrectomy for renal mass, depression, TBI, headache, and PTSD. Medications include: Truvada, valacyclovir PRN, Strattera, Adderall, and Xanax. He is a tobacco user with rare EtOH use and no illicit drug use. He works in Information Technology. Review of systems was negative except for a genital penile lesion of 4 - 5 weeks, almost resolved with over-the-counter fungal cream. Objectively, Temp 98.5, HR 86, RR 14, BP 135/85. Physical exam was unremarkable except for two penile ulcers over the shaft of the penis with clean bases, painless, and no drainage or induration. Inguinal lymphadenopathy was present. Labs, including HIV ab, RPR, urine NAAT for gonorrhea and chlamydia, UA, CMP and CBC were all negative/unremarkable. Even though the patient's RPR was negative, given his genital ulcers and high suspicion for syphilis, he was treated with Benzathine PCN Gx1. He continued Truvada and repeat RPR Titer eventually came back reactive at 1:64. Repeat RPR was nonreactive at six months of treatment.

Conclusions

This case emphasizes the importance of thorough history and physical examination in a patient who seeks pre-exposure prophylaxis, even if they are HIV negative or on Truvada. Initial screening test for syphilis was negative. Patient had improved symptoms and attributed his genital lesion to herpes. If he had not undergone further evaluation, his diagnosis of primary syphilis would have been missed. In patients who present early in the course of the disease (ulcer), RPR may be negative as in our patient. Thus, when there is a high clinical suspicion for syphilis, repeat serology testing should be performed in 2 - 4 weeks and presumptive treatment should be administered.

Hyponatremia: Cardiac Salt Wasting Syndrome

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Introduction

The association of hyponatremia with pericardial effusion and its improvement following therapeutic pericardiocentesis has been described in some previous case reports. However, the severity of hyponatremia described is usually mild. This case highlights new onset of severe hyponatremia (<110) in patient with pericardial tamponade.

Case Presentation

A 70-year-old female with past medical history of dyslipidemia, hypertension, coronary artery disease, diabetes, schizoaffective disorder, and chronic kidney disease stage III presented to the emergency department (ED) with altered mental status. One day prior to presentation, she had several recurrent non-mechanical falls resulting in a head injury. She had a recent admission two weeks prior to the current presentation for atypical chest pain. At that time, a nuclear cardiac stress test as well as a computed tomogram of the chest were performed which were normal.

In the ED, she was hypotensive, tachycardic, and hypoxic. Additional pertinent examination findings included confusion, head contusion, and very distant and muffled heart tones. CT head was negative for acute intracranial process. Chest x-ray showed mild cardiomegaly. Bedside transthoracic echocardiogram demonstrated large pericardial effusion with tamponade physiology. Laboratory evaluation was significant for sodium concentration of 109 mmol/L (baseline 137), non-anion gap metabolic acidosis, serum creatinine of 4.09 mg/dl (baseline 1.6), leukocytosis of 15.6 k/ul, hemoglobin of 8 gm/dl (baseline 11). Other significant labs include normal TSH, serum osmolality: 257 mosm/kg, urine osmolality: 134 mosm/kg, urine sodium: 23 mmol/L, FeUrea: 10.6%. There was no recent change in medication to explain the hyponatremia. She was administered intravenous normal saline (total of 2 litres) and a repeat sodium level was 107 mmol/L. She underwent emergent pericardiocentesis with immediate improvement of hemodynamics. Serum sodium and creatinine returned to baseline within 72 hours of admission. Pericardial fluid analysis showed hemorrhagic fluid; infectious and autoimmune analysis of pericardial fluid was negative, thus reflecting likely traumatic pericardial effusion. Though the sodium correction was quite rapid, she did not suffer from any negative consequences.

Conclusions

In this patient the likely cause of her severe hyponatremia was cardiac tamponade and related acute CHF and acute renal failure. This case further consolidates the impressive reversibility of hyponatremia following pericardiocentesis. There have been few case reports suggesting the role of SIADH in hyponatremia due to pericardial effusion but definitive physiologic mechanism is still undetermined. We propose a cardiac salt-wasting syndrome, similar to SIADH that is due to pericardial effusion and subsequent high intracardiac pressure. Tamponade should be considered in the differential diagnosis of unexplained hyponatremia.

**Erythrocytosis and Clubbing in a Young Adult:
An Unusual Presentation of Adult Congenital Heart Disease
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Introduction

Congenital heart disease (CHD) affects nearly 1% of all live births annually in the US, and over 90% of children born with CHD survive into adulthood with appropriate intervention. Dual chamber right ventricle (DCRV) is a rare subtype of CHD, comprising 0.5 - 2.0% of all cases. Tetralogy of Fallot (ToF) is a severe CHD which portends a 50% mortality rate if unrepaired by age six. DCRV with features of ToF is incredibly rare and presents a unique set of diagnostic and operative challenges.

Case Presentation

A 25-year-old male from India with no past medical history presented to the ED with cough, dyspnea and fatigue for two weeks. On arrival, he was hypoxic on room air. Physical exam revealed a harsh holosystolic 3/6 murmur heard best at the left upper sternal border with a widely split S2, and bilateral clubbing of all digits. Carotid upstroke was diminished and no jugular venous distention was noted. Lung fields were clear bilaterally. Laboratory findings were notable for Hgb 22.1 and Hct > 60. Pro BNP, Troponin-I, ABG were unrevealing. Chest radiography was without cardiopulmonary abnormalities, and ECG showed an incomplete left bundle branch block. Echocardiography revealed perimembranous ventricular septal defect (VSD) with left-to-right shunting and transmembrane communication between left (LV) and right ventricles (RV). Moderate RV dilation, severe right-ventricular hypertrophy (RVH) and D-shaped septum were noted. Severe right ventricular outflow tract stenosis proximal to the pulmonic valve with peak velocity of 4.8m/s and peak gradient 94mmHg, mean gradient 50mmHg was seen. Cardiac MRI revealed findings consistent with ToF; overriding aorta, perimembranous VSD, and RVH with infundibular outflow tract stenosis. Patient underwent surgical correction approximately three months after initial presentation. Prior to repair, it was discovered that pt had DCRV. VSD was closed with a Hemashield patch and right ventricular outflow tract was repaired with RV infundibulotomy + patch augmentation using patient's own pericardium. Post-operative course was complicated by vasogenic shock and pulmonary edema, both of which resolved within six days of surgical correction.

Conclusions

This case of a young man with hypoxia and erythrocytosis highlights the importance of early access to medical care, as well as the unique morphologic abnormalities that result from improper embryogenesis. Specialized diagnostic tests such as echocardiography with color flow Doppler + bubble study and Cardiac MRI were utilized to diagnose this CHD variant. Prompt diagnosis and repair has provided the patient with an improved prognosis and quality of life.

Systemic Lupus Erythematosus Induced Pancreatitis
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Introduction

Systemic Lupus Erythematosus (SLE) is a chronic, multisystem, autoimmune disease. Acute pancreatitis (AP) is a rare but potentially fatal complication of SLE. The exact pathogenic mechanism of SLE-associated AP is unknown, possibly related to vasculitis of the pancreatic vessels, micro-thrombi of the pancreatic vasculature, vascular ischemia, viral infection, and drug-induced injury. Glucocorticoids and immunosuppressive medications have been used with reasonable success. Plasma exchange therapy as an adjunctive therapy also has been successful.

This case highlights our experience in the management of SLE associated with necrotizing pancreatitis and a pancreatic duct leak. Most literature focuses on endoscopic treatment of pancreatic duct leaks and pseudocysts. We present a young female with necrotizing pancreatitis and pancreatic duct leak secondary to the flare up of SLE, who responded significantly to plasma exchange and cyclophosphamide.

Case Presentation

A 28-year-old female was admitted with hemorrhagic necrotizing pancreatitis and pancreatic pseudocyst. She was diagnosed with SLE 12 years prior, but refused to any treatment. Upon admission, she was dyspneic, on two liters of oxygen, and was unable to walk a few steps without shortness of breath. Lab studies demonstrated hemoglobin of 7.7 mg/dL with schistocytes of 1.7 mg/dL, elevated lipase at approximately 800 units/L, elevated transaminases, and hypoalbuminemia (1.7 mg/dL). In addition, CT scans of her chest and abdomen showed pancreatic necrosis with hemorrhage and a pseudocyst, pleural effusion, and pericardial effusion. She started on IV steroids and plasmapheresis was initiated for six cycles followed by IV cyclophosphamide at 500 mg/m², with plans to continue the latter on a monthly basis. Follow-up imaging showed significant improvement in the pancreatic duct leak with medical management.

Conclusions

SLE-associated pancreatitis is an uncommon complication of SLE. The rate and the risk for increased mortality in pancreatitis is augmented by overall SLE activity. Treatment should begin as soon as the diagnosis is made. There are beneficial effects of corticosteroid therapy, with potential further benefits from cytotoxic therapy and/or plasmapheresis. Glucocorticoids should be started as soon as the other causes of pancreatitis are ruled out. The mortality rate among patients who received steroids immediately after the diagnosis was 20%, compared to 61% without steroid therapy. Plasma exchange may be a useful adjunct to steroids and cytotoxic drugs in the event of life threatening complications. Endoscopic treatment for pancreatic duct leak and pseudocyst formation also is recommended. Traditionally, endoscopic treatment has been the standard of care for severe complications such as pancreatic duct leak or pseudocyst formation. However, in our case, these manifestations were ameliorated or at least attenuated with medical therapy.

Bacterial Endocarditis Presenting as Leukocytoclastic Vasculitis
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Introduction

Subacute bacterial endocarditis is a potentially life-threatening condition that can, in rare instances, present as leukocytoclastic vasculitis owing to the effect of circulating immune complexes and micro-emboli on the vascular endothelium. A high index of suspicion needs to be maintained to differentiate between infectious versus noninfectious autoimmune vasculitides. In this case, a young female patient initially presented with a picture of idiopathic autoimmune cutaneous vasculitis delaying the diagnosis of an underlying infective endocarditis with aortic valve involvement.

Case Presentation

A 42-year-old female with medical history of hypothyroidism and recent cesarean section complicated by wound dehiscence and infection presented initially as an outpatient for worsening non-blanching purpuric rash involving the bilateral lower extremities.

Initial lab results revealed anemia with hemoglobin of 8.9 g/d. Her symptoms progressed over two weeks requiring hospital admission as she began to experience night sweats, fever and lethargy. Her hemoglobin dropped to 6.4 g/dl. Initial physical exam revealed the same rash, a temperature of 38.4° Celsius, pulse of 101 bpm, blood pressure of 90/40 mmHg, and O₂ saturation at 92%. No cardiac murmurs were noted on auscultation. She was worked up for possible hemolytic anemia and auto-immune vasculitis.

Laboratory evaluation revealed a white blood count of 7300 cells/mcL with 82% neutrophils and 8% bands and normal platelets, creatinine, lactic acid, coagulation studies, and creatine kinase. Hemolysis work-up was negative except for mild LDH elevation. ESR and CRP were elevated at 45mm/hr and 123 mg/L, respectively. Urinalysis showed trace protein, 0-3 RBCs, 4+ bacteruria, negative nitrite, and leukocyte esterase. HIV, hepatitis panel, ANA, Anti-phospholipids Abs, anti-GBM, c/p ANCA and anti-myeloperoxidase were negative. However, Cryoglobulins and anti-proteinase were positive and C3, C4 levels were low at 63 mg/dL and <2 mg/dL respectively. Skin biopsy showed leukocytoclastic vasculitis. The patient's condition rapidly deteriorated. She had hypotension, hypothermia, and respiratory failure requiring intubation and pressors. Antibiotics were initiated after blood cultures came back positive for *Enterococcus faecalis*. Transesophageal echocardiography revealed a 1.6 cm aortic valve vegetation with severe aortic insufficiency and aortic valve destruction with dissection into the left ventricle. She recovered well after antibiotics and valve replacement and was discharged home from the hospital.

Conclusions

Cutaneous vasculitis is a rare though potentially fatal presentation of bacterial endocarditis. The initial presentation can be misleading and a high index of suspicion needs to be maintained to avoid adverse and sometimes fatal outcomes derived from providing the wrong treatment. This can be problematic when directly competing etiologies are in the differential. Thus, we recommend paying close attention to features that would suggest an infectious versus an autoimmune etiology, such as complement levels, and obtaining timely blood cultures in suspected cases.

Tularemia after Squirrel Bite
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Introduction

Tularemia is a rare zoonotic illness caused by *Francisella Tularensis*. Humans may acquire the infection via several different routes. In North America, the most recognized mode of transmission is contact with ticks during the summer months. Other modes of transmission include contact with infected animal tissue, ingestion of contaminated food or water, and inhalation of contaminated aerosols, either through mowing over infected animal carcasses or improper handling of lab samples. We present a case of tularemia after squirrel bite to the fingers.

Case Presentation

A 21-year-old female presented to her primary care physician after being bitten by a squirrel on the right middle finger. At initial presentation, she complained of a red, erythematous lesion consisting of four puncture wounds where the teeth had penetrated the finger as well as a tender right epitrochlear, and axillary lymphadenopathy. Tetanus booster vaccine was administered and amoxicillin/clavulanic acid initiated. Rabies immunoglobulin series was started two days later. On the third day, she developed fever, nausea, chills, and diarrhea, though these could be attributed to the rabies immune globulin and amoxicillin/clavulanic acid, the latter of which the patient discontinued due to severe nausea. The patient presented to the emergency department after six days with worsening symptoms, including multiple pustules and ulcerations of her right middle finger and a visible epitrochlear bubo along with right axillary lymphadenopathy. She was given a single dose of piperacillin/tazobactam. The wound was lanced, drained, and dressed. Blood cultures were drawn and the patient was sent home on amoxicillin/clavulanic acid and oxycodone for pain. Four days later, two of two blood cultures were negative, however, tissue culture was positive for *Francisella tularensis*. The patient was hospitalized, started on daily intravenous gentamicin. After four days of gentamicin, she reported marked improvement in pain, fever had resolved, and the buboes were getting better. She was transitioned to oral doxycycline 100 mg BID for 15 days with a follow-up appointment in one week.

Conclusions

In North America, *Francisella Tularensis* is an infection acquired mainly from handling rabbits or through tick bites. We report the first known case that occurred after a squirrel bite. This case illustrated the importance of maintaining broad differentials that include uncommon etiologies when uncommon inciting factors occur.

Refractory Ascites and Pleural Effusions Post-Remission of Mantle Cell Lymphoma
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Introduction

Pleural effusions have been known to occur in about 20% of Non-Hodgkin's lymphomas.¹⁻³ Ascites is a common complication as well, occurring in as many as 10% of patients.⁴⁻⁶ While typically exudative in nature, in rarer cases they can be transudative for such reasons as venous compression, renal failure, CHF, or hypoalbuminemia.¹ Treating the primary disease usually treats the pleural effusions, but some cases require more aggressive techniques, as seen in the following case.

Case Presentation

A 71-year-old male presented to outpatient clinic with severe thrombocytopenia and a several month history of worsening abdominal distention, bilateral pleural effusions, and ascites. CT of the chest, abdomen, and pelvis, showed diffuse thoracic, supraclavicular, abdominal, and pelvic lymphadenopathy, as well as a right inguinal node, which was subsequently biopsied. This demonstrated mantle cell lymphoma. The patient was treated with rituximab 375 mg/m² weekly x four doses followed by rituximab/bendamustine for six cycles. During therapy, the patient developed refractory non-malignant pleural effusions requiring at least twice-weekly bilateral thoracentesis and paracentesis, totaling >50 thoracenteses in a four-month period. Pleur-X catheters were placed bilaterally for palliative drainage. The fluid from these pleural effusions was transudative and cytology was persistently negative. Diagnostic workup, including nuclear medicine lymph node exam, showed lymphatic obstruction along the channels in the abdomen and upper pelvis. Radiolabeled albumin showed no peritoneal-pleural fistula. A Denver shunt was then placed, which relieved abdominal ascites, but did not resolve the pleural effusions and was subsequently removed due to overlying infection. Left sided pleurodesis with intrapleural doxycycline was attempted without resolution of symptoms. The patient, as of this publication, was still suffering from refractory pleural effusions and declined consolidation therapy as a result. While continuing to have transudative effusions, conservative management was ultimately chosen for symptomatic treatment and has been well tolerated by the patient.

Conclusions

The literature has well described the association of lymphoma, particularly Non-Hodgkin's Lymphoma with pleural effusions,¹ but ascites is an uncommon presenting symptom of lymphoma.⁷ This also seemed to be a unique case due to the refractory nature of both the effusions and ascites to traditional therapies, as well as despite treatment of the malignancy. The likely explanation of this is lymphatic obstruction subsequently leading to ascites and pleural effusions. Given the patient went into remission but the effusions remained, we feel the etiology of impairment of lymphatic drainage may be therapy-related. This could represent a unique complication of standard mantle cell lymphoma therapy and thus presents an important diagnostic, treatment, and quality of life consideration for patients undergoing treatment for this disease. The major impact of this case is illustrating that clinicians must be cognizant of the potential for therapy-induced lymphatic obstruction as a potential complication of treating mantle cell lymphoma.

**Iatrogenic Cushing Syndrome Caused by an Interaction
Between Ritonavir and Inhaled Fluticasone**
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Introduction

Ritonavir is a protease inhibitor frequently prescribed as a part of highly active antiretroviral therapy (HAART). Ritonavir blocks the breakdown of other protease inhibitors by inhibiting the cytochrome P450-3A4 (CYP3A4) degradation pathway. By this same mechanism, Ritonavir can cause iatrogenic Cushing's syndrome (ICS) in patients using local corticosteroids by increasing corticosteroid bioavailability. We describe a patient with iatrogenic Cushing syndrome secondary to interaction between inhaled corticosteroids and Ritonavir that was initially misdiagnosed as adrenal insufficiency.

Case Presentation

A 48-year-old male patient with medical history of HIV, depression, and allergic rhinitis was brought to the Emergency Department with chest tightness, nausea, vomiting, intermittent diarrhea, and excessive fatigue. Physical exam revealed a pulse of 69 beats/min, blood pressure of 131/90 mmHg, temperature of 36.8 degrees Celsius, dry mucous membranes, truncal obesity, and mild facial puffiness. Laboratory work-up was remarkable for mild hyponatremia at 131 meq/L. Hospital stay was marked by frequent fluctuations in glycemic level and blood pressure. The patient was suspected to have adrenal insufficiency; his AM cortisol was consistently low and standard high-dose Cosyntropin stimulation test showed a stimulated cortisol to 10 mcg/dL at 30 minutes and 14 mcg/dL at 60 minutes. MRI of the brain was unremarkable for acute abnormalities. Hydrocortisone was initiated at a dose 20mg/day for the presumptive diagnosis of adrenal insufficiency.

The patient presented to the Emergency Department five months later with mouth ulcers and severe depression with suicidal ideation. Hydrocortisone was not well tolerated and he skipped multiple doses, although his dose was tapered to 5 mg BID prior to presentation. While on hydrocortisone, he gained 11 lbs. and had mouth ulcers, abdominal discomfort, nausea, skin thinning, and frequent bruising. Physical exam revealed Cushingoid features, including moon face, facial plethora, cutaneous atrophy, skin tenting, and multiple ecchymoses. Repeat Cortisol level was low, with subnormal response to Cosyntropin stimulation test. ACTH was low and 21-Hydroxylase Ab was negative.

After careful review of medications, an interaction was revealed between Fluticasone and Ritonavir, both of which patient used regularly. Additionally, his symptoms were aggravated by introducing Hydrocortisone.

Conclusions

Caution should be taken when caring for patients on Ritonavir-boosted HAART. While localized Fluticasone should not cause systemic toxicity, its long half-life increases its risk for adverse effects when used in conjunction with Ritonavir as described in our case. The resulting suppression of Cortisol level may misguide physicians into assuming a diagnosis of adrenal insufficiency owing to similarity in laboratory findings. We recommend a careful review of medications for patients on Ritonavir while avoiding any concomitant glucocorticoid use to establish a timely diagnosis of ICS and avoid its detrimental sequela.

Not So Fast...
Details to Consider Before a Community Acquired Pneumonia Diagnosis
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Introduction

We present a rare case of acute eosinophilic pneumonia (AEP). Diagnosis can be challenging because symptoms, including fever, dyspnea, non-productive cough, and inspiratory rales on lung exam, can be mistaken for severe community acquired pneumonia. Diagnosis includes arterial blood gas demonstrating hypoxemia and bronchoalveolar lavage (BAL) showing 25% eosinophils.

Case Presentation

A 47-year-old male with no significant past medical history presented with worsening cough and dyspnea of two days duration. Patient experienced malaise, fatigue and was having difficulty eating due to his symptoms. Review of systems was positive for fever, fatigue, and cough. The patient was a 15-pack year smoker, reported 12 cans of beer per week, recently released from prison, and had no drug allergies. The patient appeared to be in mild distress with a temperature of 100.9 F, pulse 125 beats per minute, blood pressure 138/72 mmHg, respiratory rate of 35, pulse oximetry of 95% on 5L of supplemental oxygen. The patient demonstrated tachycardia, injected conjunctivae, submandibular and tonsillar adenopathy. Inspiratory rales were auscultated on the left lung field. Arterial blood gas was 7.47/33/60/25. EKG showed sinus tachycardia with right axis deviation. CXR demonstrated interstitial pulmonary opacities within the mid and lower lungs, bilaterally. Further work up included a CT scan demonstrating diffuse interstitial prominence with small bilateral pleural effusions, interpreted as volume overload or CHF. Laboratory testing demonstrated WBC 16.5, neutrophils 59%, bands 28%, lymphocytes 2%, monocytes 8%, eosinophils 2%, negative troponin, and BNP of 51. Urinalysis demonstrated ketones and blood. Viral panel was positive for Rhinovirus. Ceftriaxone and azithromycin were started for community acquired pneumonia coverage. After 48 hours of care, patient had no symptom improvement and continued fevers. Antibiotics were expanded to vancomycin, piperacillin-tazobactam, and levaquin. A pulmonary consult was placed for bronchoalveolar lavage. BAL fluid demonstrated RBCs 2100, WBCs 530, and eosinophils 41, establishing the diagnosis of acute eosinophilic pneumonia. On the day of BAL, peripheral eosinophils increased to 13% and were 18% at discharge. Empiric antibiotics were continued and the patient was treated with 60 mg of prednisone.

Conclusions

AEP has been frequently mistaken with severe community acquired pneumonia. Diagnosis of AEP requires acute onset of febrile manifestations less than one-month duration, bilateral diffuse infiltrates on chest radiograph, hypoxemia, a BAL showing 25% eosinophils or eosinophilic pneumonia on lung biopsy, as well as absence of known causes of pulmonary eosinophilia. Treatment is mostly supportive, with little consensus on optimal dosage of glucocorticoids. Mortality is low. However, progressive respiratory failure may occur, requiring mechanical ventilation if not recognized in a timely manner. Approximately one hundred cases of AEP have been documented in the literature, demonstrating how rarely it is seen. It may often go undiagnosed, potentially making AEP a more common disease than is documented.

**Disseminated Histoplasmosis (DH) Presenting with Necrotic Finger Lesions
and Tenosynovitis in a Patient with Psoriatic Arthritis**
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Introduction

Disseminated histoplasmosis (DH) is a systemic fungal infection caused by *Histoplasma capsulatum*.¹ Patients receiving tumor necrosis factor (TNF) blockers are at increased risk of DH. Its cutaneous manifestations are rare, varied, and nonspecific. Immune Reconstitution Inflammatory Syndrome (IRIS) has been described in patients after TNF blockers discontinuation² and after initiation of HAART in patients with HIV.¹ We will discuss a patient with psoriatic arthritis who presented with finger ulcerations and necrosis and was treated with further immunosuppression for a suspected arthritis flare. He was initially misdiagnosed and as a result, he developed DH. This shows the importance of skin biopsy for accurate diagnosis before proceeding with further immunosuppression.

Case Presentation

A 61-year-old man with history of psoriatic arthritis maintained on methotrexate and prednisone presented with three months of bilateral hand and forearm swelling. He was treated for a flare with increase in methotrexate and prednisone with two doses of adalimumab. He developed tender, necrotic, ulcerative lesions on multiple fingertips that caused him to lose his job as a mechanic. He was admitted with syncope, fever and hypotension reaching systolic 80, so Methotrexate was held and prednisone was decreased for sepsis. MRI of hands showed tenosynovitis of the flexor tendons. The diagnosis of disseminated histoplasmosis was established on the basis of an elevated beta 1, 3 glucan test (424 pg/ml); the presence of histoplasma antigen in urine and serum; and growth of *H capsulatum* in blood and from a swab of one of the finger lesions. After 10 days of treatment with liposomal amphotericin B, oral itraconazole was begun. Over the course of two months there was improvement: resolution of fever, disappearance of antigen from blood and urine, and a marked decrease in the swelling of the fingers, hands and forearms and a decrease in beta 1,3 glucan to 74 (pg/ml). However, at that time the patient began to develop fluctuant eruptions on the dorsum of his hands and fingers. *H capsulatum* grew from a right ring finger biopsy. Therapy was changed back to liposomal amphotericin. The nodules and fluctuant lesions worsened. A component of IRIS was suspected and prednisone 40 mg per day was started. Swelling, pain and the nodules improved significantly but not completely.

Conclusions

Cutaneous presentation constitutes 6% of patients with disseminated histoplasmosis.¹ They pose a diagnostic challenge. This is the first case of disseminated histoplasmosis presenting as finger ulcerations and necrosis. Reducing immunosuppression resulted in IRIS. With an increased use in anti-TNF blockers for treatment of rheumatological disease, our case illustrates the importance of being aware of infections that can mimic rheumatological presentations and the importance of ruling them out before increasing immunosuppression.

**Purulent Pericarditis:
A Complication of Bacteremic *Enterococcus faecalis* Urinary Tract Infection
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Introduction

Purulent pericarditis is a rare clinical entity. The most common portal of entry is direct extension from a primary lung source and usually caused by *Staphylococcus aureus*, *Streptococcus pneumoniae*, or *Haemophilus influenzae*. We report the case of a man who presented with purulent pericarditis due to *Enterococcus faecalis* likely caused by haematogenous spread from a urinary tract source.

Case Presentation

A 69-year-old man with history of type 2 diabetes mellitus, coronary artery disease, and ischemic cardiomyopathy presented with fatigue, chills, and dysuria of several days duration. Medications included lisinopril, aspirin, metoprolol, atorvastatin and metformin. He was febrile and had tachycardia but stable blood pressure. Lungs were clear to auscultation, and there were no jugular venous distention or murmurs or rubs on cardiac auscultation. Laboratory evaluation revealed leukocytosis with a left shift and acute kidney injury. Urine and blood cultures grew *Enterococcus faecalis* susceptible to ampicillin. A transthoracic echocardiogram showed no evidence of endocarditis. Repeat blood cultures and urine cultures revealed no growth.

After two days, the patient became disoriented and hypotensive with increased leukocytosis and worsening kidney function necessitating hemodialysis and vasopressor support. The antibiotic coverage was broadened. Repeat blood cultures remained negative. CT scans were unremarkable. ECG showed atrial fibrillation with no signs of acute ischemia. Troponin I was elevated. A repeat transesophageal echocardiogram revealed a moderate pericardial effusion associated with significant mitral valve inflow variability with no signs of tamponade and no vegetation. CT scan of the chest confirmed a 1.8 cm pericardial effusion. Emergent pericardiotomy was performed and 500 mL of purulent material was drained. Tamponade was evident only on the third echocardiogram obtained during pericardiotomy. Gram stain revealed Gram-positive cocci and culture grew *E. faecalis* with the same sensitivities as the urinary organism. The patient experienced rapid clinical improvement and was discharged on parenteral antibiotics. Repeat transthoracic echocardiography showed persistent resolution of the pericardial fluid.

Conclusions

This case is interesting in terms of the microorganism involved and its unusual presentation. *Enterococcus faecalis* causing purulent pericarditis rarely has been reported. In our case, the most probable source of infection was urinary with haematogenous spread to the pericardial space. Haematogenous dissemination is rare. To the best of our knowledge, this is the first reported case of *E. faecalis* purulent pericarditis from a urinary source. Purulent pericarditis should be considered in patients who deteriorate, despite appropriate conventional management. Emergent pericardiotomy and drainage of the pericardial fluid is vital in purulent pericarditis with signs of tamponade. Despite appropriate therapy, mortality remains high partly due to a delay in diagnosis.

A Case of Polymyalgia Rheumatica Masquerading as Essential Thrombocytosis
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Introduction

Polymyalgia rheumatica (PMR) is one of the most common systemic inflammatory diseases of unknown cause affecting older adults, and interestingly, no universally accepted diagnostic criteria exists. There is a high degree of association with giant cell arteritis (GCA). About 20% of patients initially diagnosed with PMR will have subclinical GCA, and about half of GCA patients have symptoms of PMR. Clinical symptoms of PMR include bilateral morning stiffness lasting greater than 30 minutes with pain in the neck, shoulder, and hip girdle that may make it difficult to comb hair, rise from a chair, or even get out of bed. Insidious systemic symptoms such as low-grade fever, weight loss, fatigue, and malaise also may be present. There are no highly specific biomarkers for PMR. However, elevated markers of inflammation such as ESR and CRP support the diagnosis, level of disease activity, and relapse. Thrombocytosis can occur as a part of a general acute inflammatory response. We present a case of a patient with a spontaneous deep vein thrombosis (DVT) of the right lower extremity (RLE) and thrombocytosis.

Case Presentation

The patient was a 78-year-old male with a PMH of hypertension. He was traveling by car when he developed non-traumatic, acute swelling of his RLE. He went to a nearby emergency department where an ultrasound confirmed the presence of a right popliteal DVT. Routine blood work was normal except an elevated platelet count of $625 \times 10^3/\text{microliter}$, which was attributed to acute inflammation. He was discharged home on apixaban. At a two-week follow-up, his RLE edema had improved. However, his platelet count increased to $850 \times 10^3/\text{microliter}$. Upon presentation to a hematologist two weeks later, his platelet had count risen to $1,100 \times 10^3/\text{microliter}$. Workup was initiated: JAK2, calreticulin (CALR), and thrombopoietin (MPL) mutation were all negative, and bone marrow biopsy did not show evidence of a myeloproliferative disorder. Four weeks later, he presented with joint aches, pain, and difficulty getting out of bed. Labs showed a platelet count of $1,500 \times 10^3/\text{microliter}$ and ESR 90 mm/hr. A preliminary diagnosis of PMR was made. He was started on prednisone, and referred to a Rheumatologist where the diagnosis of PMR was confirmed. Recheck of his platelets three weeks after starting prednisone showed a normal count of $405 \times 10^3/\text{microliter}$. A temporal artery biopsy was not done, as the patient never exhibited symptoms of GCA.

Conclusions

The lack of universally accepted diagnostic criteria and the diverse presentation of PMR can make diagnosis difficult. Diagnosis is primarily clinical, supported by inflammatory markers, and response to treatment.

An Extremely Rare Case of Weakness
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Introduction

Liddle's Syndrome is a rare cause of early hypertension, hypokalemia and decreased plasma renin levels that can appear similar to a hyper-aldosterone state. Although the prevalence is unknown as of 2008, only 30 cases have been published in the literature. Its infrequent presentation makes it a challenging diagnosis.

Case Presentation

A 48-year-old man with a past medical history of hypertension, diagnosed in his 20's, presented to the emergency department with generalized weakness that started one week prior. On examination, the patient was only able to lift his extremities a few inches off the table. He was hypertensive with a blood pressure of 146/98. Laboratory evaluation showed potassium level of 1.4, a metabolic alkalosis and normal creatinine. The patient was admitted for the severe electrolyte abnormality and aggressive potassium replacement was started while the source of the hypokalemia was investigated. The patient received as much as 320mEq of potassium over 24 hours before plasma potassium levels returned to normal limits. A hyper-aldosterone state was suspected, however plasma renin and aldosterone levels were both low. This raised suspicion for Liddle's syndrome and the patient was started on Amiloride. Over the next few days, his potassium levels rose to within normal limits and his weakness resolved and he was discharged home. On follow up his potassium levels remained stable.

Conclusions

This case illustrated the importance of a complete history and maintaining a broad differential diagnosis. Hypokalemia has been found in over 20% of hospitalized patients and low levels of potassium can cause muscle necrosis, an ascending paralysis and/or cardiac conduction abnormalities. The causes of hypokalemia are diverse and a broad differential, that includes excessive mineralocorticoids, can help with the diagnosis and treatment of the disorder. Liddle's disease also provides a good review of renal physiology. This mutation causes an increase in ENaC channel activity in the distal convoluted tubule of the nephron. The activity leads to increased sodium and water reabsorption, which leads to hypertension. Consequently, there is increased potassium secretion into the lumen of the tubule, leading to hypokalemia. In summary, even though hypokalemia is common inpatient, always consider alternative causes.

Bi-Valvular Fungal Endocarditis: Challenging to Diagnose and Harder to Manage
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Introduction

Fungal Endocarditis (FE) is a rare, but serious disorder with mortality approaching 50%. Diagnosis can be difficult, is usually made post-mortem, and patients present resembling bacterial endocarditis.

Case Presentation

A 33-year-old man with a medical history of osteosarcoma, IV drug abuse, infected femur hardware, amputation and subsequent bacterial endocarditis requiring mitral valve replacement presented with complaint of arm weakness, intermittent fevers, and facial drooping two days prior to presentation. Physical exam revealed hypotension, tachycardia, and weakness in his left arm. He had an active precordium, loud mechanical mitral valve murmur, left above knee amputation, but otherwise normal exam. CT confirmed an MCA ischemic stroke, and echocardiogram showed aortic insufficiency, mitral stenosis from an obstructing vegetation, pulmonary hypertension, and an akinetic, dilated right ventricle. Cultures obtained on admission grew *Candida albicans*, confirming a diagnosis of FE.

Antifungal therapy was started, and surgery was consulted, as FE is an absolute indication for valve replacement. Informed of his high surgical risk, the patient chose medical treatment only. His cardiac function continued to decline. Repeat echocardiography showed worsening aortic insufficiency despite a decrease in size of the mitral vegetation. Shortly thereafter, he developed left arm paralysis, and MRI demonstrated new cerebral infarction. The patient then developed severe pain, loss of pulses, cool skin, and purple macules on his right leg. CTA showed complete occlusion of the popliteal artery, and a fungal embolus was removed. Despite these complications and poor prognostic events, the patient survived on a course of amphotericin B, flucytosine, and fluconazole. Even still, FE is a formidable diagnosis, and his long-term prognosis remains guarded.

Conclusions

FE is a serious infection, posing a >50% mortality to the patient, and prompt diagnosis can be a difficult challenge for the clinician. Here, diagnosis was made via culture; however, it is not always so straightforward. FE often presents identically to bacterial endocarditis or fever of unknown origin. To further complicate diagnosis, invasive infection can be present without fungemia, making blood cultures unreliable. It is important to maintain high suspicion for the disease in high-risk patient, particularly when no improvement is observed on antibacterial coverage.

This case also demonstrates the propensity for embolic phenomena in FE, which can further complicate management of the condition. It was clear in our patient that surgery was indicated due to his worsening cardiac function, but repeated embolic events, even with heparinization, complicated his treatment. Ultimately, though, the patient improved with medical treatment and lower limb embolectomy.

A Disease of Many Faces: HLH
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Introduction

Hemophagocytic lymphohistiocytosis (HLH) is a disorder of immune modulation resulting from impaired cytotoxic killing and unregulated inflammation. Reported incidence is 1.2 cases per one million individuals per year. Without treatment, reported mortality is up to 88%. Clinical presentation is often ambiguous, which poses a diagnostic challenge. We present an HIV-infected male with EBV viremia who developed secondary HLH.

Case Presentation

A 67-year-old male with recently diagnosed HIV, presented to the emergency department with confusion and fever. Infection and malignancy were main concerns, so imaging, lumbar puncture and work up for lymphoma were undertaken but were unrevealing. Eight days into his hospital stay, he was noted to have fever, splenomegaly, hypertriglyceridemia, pancytopenia, and high ferritin, raising concern for HLH. Bone marrow biopsy was done which showed possible phagocytosis but was not definitive. Blood work returned positive for EBV and CMV at which time he was started on antiretroviral therapy and valganciclovir to treat his viremia. However, the patient's clinical condition worsened and rituximab with dexamethasone were started due to concern for EBV-associated HLH. Reduced-dose rituximab was given to decrease prolonged B-cell impairment. Unfortunately, he developed an anaphylactic reaction to rituximab. He was then switched to the modified HLH94 protocol - etoposide, corticosteroids, cyclosporine A. He developed worsening renal and liver function, and cyclosporine and etoposide had to be stopped. He was then trialed on IVIG without improvement. His liver enzymes trended down, and he was changed back to low-dose etoposide with dexamethasone. Despite these efforts, he deteriorated quickly and was transitioned to comfort measures.

Conclusions

HIV in itself or in the setting of other infections or malignancies is associated with HLH. Other important causes of secondary HLH include viral infections (29%), other infections (20%), malignancies (27%), rheumatologic disorders (7%), and immune deficiency syndromes (6%). Reported low incidence is likely due to under-diagnosis. This is why a high index of suspicion for HLH should be kept in patients with clinical features who meet the diagnostic criteria (i.e. HLH-2004). To further complicate the diagnosis, bone marrow biopsies may be normal early on. Ferritin can be helpful when levels are >10,000 ug/L as this has been shown to be 90% sensitive and 96% specific for HLH. Treatment needs to be customized to each patient's underlying cause. Further studies are needed to see whether treatment with steroids or with an etoposide regimen is effective in cases that do not respond to antimicrobial therapy.

A Case of Lemierre Syndrome
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Introduction

Lemierre syndrome, also known as post-anginal septicemia or necrobacillosis, is usually caused by an acute oropharyngeal infection, resulting in thrombophlebitis of the internal jugular vein that can lead to metastatic septic embolization and bacteremia. The usual organism is *Fusobacterium Necrophorum*. Before the advent of antibiotics, this syndrome was often fatal. Due to the use of antibiotics, there has been a dramatic decrease in the incidence of Lemierre syndrome. The current mortality rate is estimated to be between 5% and 10%, with significant morbidity. We report a case of Lemierre syndrome in a young male who presented with complaints of neck pain and swelling.

Case Presentation

A 32-year-old male presented with complaints of worsening neck pain and swelling for seven days. He was seen at an outside hospital for these symptoms and was told that he had muscle strain and sent home. He reported symptoms of sore throat; however, he denied associated fever or chills. Baseline labs were unremarkable. On examination, the right side of neck was tender to palpation. A CT scan of the neck showed acute occlusive thrombosis of the right internal jugular vein extending into upper superior vena cava with reactive right axillary lymphadenopathy. Diagnosis of Lemierre syndrome was made and the patient was started on a course of Ampicillin/Sulbactam. Rivaroxaban was started for anticoagulation. Blood cultures were taken before the initiation of Ampicillin/Sulbactam and those came out negative. The patient was discharged and was scheduled for infectious disease follow-up.

Conclusions

Fusobacterium necrophorum is the most common pathogen associated with the syndrome. Other organisms isolated include *Bacteroides*, *Streptococcus*, *Peptostreptococcus*, and *Eikenella corrodens*. Tonsillitis is the most common primary infection, followed by mastoiditis and odontogenic infections. This is typically followed by invasion of the pharyngeal lateral wall and thrombophlebitis of the internal jugular vein that can result in neck pain and swelling. The most common site of metastatic infection is the lungs. Other site of metastatic infection can include septic arthritis, osteomyelitis, pericarditis, hepatic abscesses, and meningitis. There is no consensus on the antibiotic regimen. We used Ampicillin/Sulbactam in this case for four weeks. The patient was also anticoagulated with Rivaroxaban, the reported benefits of anticoagulation being increased resolution of the thrombus as well as penetration of antibiotics into the septic emboli.

In conclusion, Lemierre's syndrome should be suspected in a previously healthy young person who develops oropharyngeal infection and exhibits signs and symptoms of internal jugular vein thrombophlebitis with or without sepsis. Blood cultures should be obtained and CT imaging of the neck with IV contrast should be performed. This, in turn, will enable timely diagnosis and improved outcome.

Post Cardiac Catheterization Thyrotoxicosis
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Introduction

Over a million cardiac catheterizations are performed in the US annually. Coronary angiography requires the use of iodinated contrast media for opacification of the coronary arteries. Over 8 million liters of iodinated contrast are estimated to be used annually worldwide. Contrast media induced thyroid disorders are fortunately rare, but have been reported. This case highlights the increased risk of post-cardiac catheterization thyrotoxicosis in susceptible patients (especially those with underlying thyroid dysfunction).

Case Presentation

Our patient is a 67-year-old male with a history of Graves' disease (remote history of radioactive iodine ablation), hypertension, hyperlipidemia, diabetes and active smoking who presented with unstable angina. He had undergone cardiac catheterization at an outside hospital two days prior to this admission. Angiograms demonstrated severe multi-vessel disease including 90% stenosis of proximal left anterior descending (LAD), significant ulcerated right coronary artery (RCA) with preserved left ventricular (LV) function. Based on these findings it was recommended that he undergo coronary artery bypass grafting (CABG). On presentation to our institution, physical exam was notable for a tachycardia without a tremor. He denied any symptoms of palpitations, tremors, heat intolerance or increased bowel movements. Since he underwent RAI treatment 20 years prior for his Graves' disease, he had been asymptomatic and no active treatment. Pre-operative screening thyroid labs were drawn on admission and they demonstrated an elevated free T4 level of 3.2 ng/dL (0.6 - 1.6 ng/dL), elevated total T3 level of 226 ng/dL (87 - 180 ng/dL) and suppressed TSH level of 0.030 mcu/mL (0.35 - 5.00 mcu/mL).

Endocrinology was consulted and the patient was diagnosed with thyrotoxicosis secondary to recurrent Grave's disease, exacerbated by iodinated contrast received during cardiac catheterization. Metoprolol was initiated with a goal heart rate of less than 80 bpm. Thyroperoxidase inhibition was achieved with methimazole in order to decrease T3 and T4 levels. No uptake scan was possible in this case due to the large iodine dose with his previous contrast administration. Over the following three days, his T3 levels decreased from 226 down to 200 and his T4 levels decreased from 3.2 down to 2.7. This stabilization of his thyroid hormone levels allowed him to safely undergo his CABG.

Conclusions

This case highlights the importance of thorough risk assessment in patients undergoing cardiac catheterization and reminds us of the dynamic interplay between thyroid status and cardiac health. Thyrotoxicosis can be difficult to assess on exam in the elderly. A full endocrine history should be obtained from each patient prior to undergoing catheterization. While this case alone is not sufficient to recommend regular thyroid screening prior to catheterization, it demonstrates an opportunity for larger studies looking at this topic.

**Lupus Lymphadenitis as a Presenting Manifestation
of Systemic Lupus Erythematosus
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Introduction

Lymphadenitis is a known and relatively common feature of systemic lupus erythematosus (SLE). We report a case of a young female found to have diffuse lymphadenopathy as a presenting manifestation of SLE.

Case Presentation

Patient is a 24-year-old Asian female with no known past medical history who presented with diffuse, symmetric polyarthralgias, painful cervical and occipital lymphadenopathy, and constitutional symptoms of several weeks duration. The initial diagnostic considerations based on the patient's presenting complaints and exam included an autoimmune connective tissue disease, infection, or malignancy. Initial laboratory workup was significant for pancytopenia, elevated inflammatory markers, and an autoimmune panel that demonstrated an elevated ANA (1:1280), anti-dsDNA (1:1280), anti-Smith antibodies, and low complement levels. An extensive infectious workup evaluating for viral and bacterial etiologies was negative. A CTA chest was also obtained as a part of her initial evaluation that showed prominent cervical, axillary, and hilar lymphadenopathy. She was diagnosed with systemic lupus erythematosus (SLE) and started on Prednisone and Plaquenil, but there was also concern for coexisting malignancy given the presence of pancytopenia and lymphadenopathy. Further evaluation was obtained with the guidance of Rheumatology and Hematology and a PET scan revealed hypermetabolic adenopathy throughout the neck, chest, abdomen, and pelvis. Due to rising concern for potential lymphoma, a lymph node biopsy was performed and showed extensive coagulative necrosis and hematoxylin bodies consistent with lupus lymphadenitis.

Conclusions

Lymphadenopathy and lymphadenitis are established features of SLE with descriptions of the association dating back to the late 1800s. Estimations of frequency vary between sources, but many estimate that lymphadenopathy can be seen in 12 to 59% of patients with SLE. The most commonly affected sites are noted to be cervical, axillary, mesenteric, and inguinal lymph nodes. While the patient described above demonstrated clear diagnostic criteria for SLE, the differential diagnosis for lymphadenopathy is broad, including infection, malignancy, and other autoimmune/inflammatory disorders (sarcoidosis, Kikuchi-Fujimoto's disease) and it was important that other etiologies were entertained. Pathologic features of coagulative necrosis can be seen in a number of disease processes but the presence of hematoxylin bodies are pathognomonic for lupus lymphadenitis. Lastly, the presence of lymphadenitis in SLE seems to be a measure of disease activity and often times improves or resolves with treatment of the underlying disease, particularly with the use of corticosteroids.

Lemierre's Syndrome Following Extraction of Wisdom Teeth
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Introduction

Lemierre's syndrome is known as the forgotten disease as it is rare in the antibiotic era. It is potentially fatal most commonly due to oropharyngeal infections leading to thrombophlebitis of the veins, particularly the internal jugular vein. It is a form of septicemia commonly caused by *Fusobacterium Necrophorum* leading to metastatic infection. The infection often arises from tonsillitis and mastoiditis, but rarely as an odontogenic infection from tooth extraction.

Case Presentation

An 18-year-old male with no significant medical history presented with a three-day history of nausea and vomiting that began with left jaw pain and swelling. He had an uneventful extraction of four wisdom teeth 20 days prior. Physical examination revealed diffuse lower abdomen, left upper quadrant pain, and swollen left jaw. A maxillofacial CT scan showed abnormal soft tissue gas near the left mandibular socket, suspicious for underlying cellulitis with bilateral cervical lymphadenopathy. A CT of the abdomen revealed splenomegaly and was suspicious for mesenteric adenitis. Blood culture was positive for *Fusobacterium Necrophorum*. Broad antibiotic coverage was started.

Despite antibiotics, the patient deteriorated rapidly and developed septic shock with multi-organ failure. An ultrasound of the neck showed thrombosed superficial vein consistent with thrombophlebitis. CT angiography showed scattered nodular-appearing infiltrates throughout both lungs and moderate bilateral effusion. An echocardiogram showed an ejection fraction of 30%. No surgical intervention was recommended and medical management continued. A subsequent CT showed evolving cavitation of the numerous nodular densities consistent with septic pulmonary emboli.

The patient improved clinically and was discharged on day 12. Follow-up CT of the jaw showed a periosteal reaction, therefore, he was treated with six weeks of antibiotics. One month after discharge, the follow-up radiographic imaging demonstrated resolution of the pulmonary infiltration, superficial vein thrombosis, and improving of the left mandibular osteomyelitis.

Conclusions

There are few reports of Lemierre's syndrome following wisdom tooth extraction. Diagnosis is highly dependent on clinical presentation and supporting laboratory results including blood cultures and radiographic findings. CT scan with contrast confirms thrombosis vein, often the internal jugular vein. However, our case had thrombosis of the superficial vein. Since CT eventually revealed septic pulmonary emboli, the deep venous system likely was involved. Broad-spectrum antibiotics are key in the early treatment of sepsis and in Lemierre's disease it is important to cover anaerobic organisms such as *Fusobacterium*. There is no definite treatment for thrombophlebitis, therefore, the use of anticoagulation is controversial and varies by case. The presence of odontogenic infection with worsening pain and neck swelling should raise high suspicion to obtain necessary blood cultures and imaging for early diagnosis. Given the high mortality rate of approximately 17%, emergent imaging is crucial and there should be no delay in initiation of antibiotic treatment.

**Hypercalcemia as a Paraneoplastic Syndrome
of Squamous Cell Carcinoma of the Gallbladder**
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Introduction

Paraneoplastic Syndromes are symptoms caused by a chemical product released secondary to an underlying cancer. We report one of very few cases of ectopic PTHrP production in a patient with Squamous Cell Cancer of the gallbladder. Our patient had acquired hypercalcemia secondary to his gallbladder cancer.

Case Presentation

A 62-year-old patient with a recent diagnosis of Squamous Cell Carcinoma (SCC) of the gallbladder with involvements of the liver and the colon in November 2016 presented to the hospital with multiple complaints of confusion, lethargy, constipation, unintentional weight loss, and abdominal pain for several days. His admission labs showed profound hypercalcemia (corrected Ca²⁺ of 16.3), albumin of 3.0 and hypophosphatemia (PO₄³⁻ of 1.9). He was treated with intravenous fluids, calcitonin, Zometa (bisphosphonate), and electrolyte repletion. Intact Parathyroid Hormone (PTH) level was 6.2, which suggested a non-parathyroid mediated hypercalcemia. A Parathyroid Hormone Related Peptide (PTHrP) was ordered and returned significantly elevated with a value of 40. His bone scan and CT Head were negative for metastatic diseases. His hypercalcemia likely resulted from PTHrP secretions from his cancer. After the calcium was corrected, he was discharged. He subsequently followed up with his oncologist for initiation of chemotherapy.

Conclusions

SCC is a rare sub-type of gallbladder cancer and accounts for 2 - 10% of all gallbladder malignancies. PTHrP-secreting gallbladder cancers are even more uncommon. PTHrP essentially acts as an ectopic PTH hormone. This molecule stimulates bone resorption and calcium retention in the kidneys. Ultimately, the serum calcium rises. Management comprises of treating the underlying cancer and correcting the electrolyte abnormalities.

Most importantly, this case places a great emphasis on paraneoplastic syndrome as a differential in the management of cancer patients with metabolic abnormalities. These syndromes require a prompt diagnosis and management. Treatment improves patient's quality of life, creates efficient transition towards further cancer treatment, and increases survival rate.

Unexplained Anasarca: A Case of Supplement Induced Puffiness
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Introduction

Many herbal products have biologic activity that can lead to direct toxicity or interaction with other medications. Sometimes this toxicity presents in an organ-based, symptomatic fashion. However, in other instances, damage to multiple organ systems can create a murky clinical picture, such as the case described below. Knowledge of potential effect of supplements is particularly important due to their unregulated nature.

Case Presentation

A 41-year-old Hispanic woman with past surgical history of cholecystectomy and pancreatic cyst removal presented to the hospital with a two-week duration of abdominal distention associated with a 20-pound weight gain, diarrhea, and pedal edema. On physical exam, there was no abdominal tenderness. Initial labs found WBC, LFTs and INR to be unremarkable, however, albumin was undetectable. CT of the abdomen and pelvis showed cirrhosis, ascites and portal hypertension. IV albumin was initiated and Hepatology and GI were consulted. Viral hepatitis, HIV, paraprotein and autoimmune serologies including C3/C4, ANA, ANCA, as well as A1AT and ceruloplasmin were all within normal limits. Twenty-four hour urine protein collection showed 42g of protein loss. In order to improve patient comfort and aid in disease diagnosis, a paracentesis was performed. Although culture of the fluid ruled out spontaneous bacterial peritonitis, the patient's low serum albumin made it difficult to accurately interpret her SAAG.

Further patient questioning revealed that she consumed Herbalife energy supplements and phentermine for weight loss. Renal ultrasound to rule out nephrotic cause of ascites found structurally unremarkable kidneys, and renal biopsy was consistent with immune-mediated glomerular nephritis. Lisinopril and prednisone taper were initiated for nephrotic syndrome. The patient made several subsequent visits to the hospital in the weeks following her initial discharge for sequela of her toxin-induced nephrotic syndrome including an episode of angioedema from lisinopril, a lower GI bleed from anticoagulation and fluid overload requiring Bumex. Though symptoms are now stable, supplement adverse effects have drastically changed her life.

Conclusions

Although relatively rare in incidence, this case serves to remind of the potential serious side effects of non-FDA regulated dietary supplements, especially given that supplement use has been increasing in recent years. One questionnaire-based study of 989 patients found 39% of patients used some form of complementary or alternative medicine (CAM) at least once during the preceding month and 13% used herbs to treat their liver disease. Because patients may be reluctant to discuss CAM usage, it is more important than ever that physicians screen for the use of supplements.

**A Case of Successful Medical Management
of Bilateral Diffuse Xanthogranulomatous Pyelonephritis
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Introduction

Xanthogranulomatous pyelonephritis (XGP) is a rare disease process in which chronic infection leads to a suppurative, granulomatous response in the kidneys. It is most commonly diffuse, involving the entire kidney, but can be focal (10 - 17%). In extremely rare cases, both kidneys are involved. Risk factors include diabetes, immunocompromise, and urinary tract obstruction. Patients can present with fever, flank pain, lower urinary tract symptoms, and gross hematuria. Labs typically show leukocytosis, anemia, pyuria and hematuria. Proteus and E. coli are the most commonly isolated bacteria. Hallmark pathologic features are lipid-laden foamy macrophages (xanthoma cells) with plasma cell infiltration and histiocytes. Postulated mechanisms include chronic infection and obstruction, or an inadequate host inflammatory response. Treatment involves an extended course of antibiotics, directed by urine sensitivities. Surgical resection or nephrectomy is usually required as XGP often leads to nonfunctional renal parenchyma, and antibiotics rarely eradicate the disease. Bilateral XGP is commonly fatal without bilateral nephrectomy and subsequent dialysis or transplantation. Only one published case of successful management with antibiotics alone exists in the medical literature to date.

Case Presentation

A 42-year-old Hispanic woman with a history of diabetes and alcoholism presented with bilateral flank pain, malaise, fevers, and chills. She denied any history of dysuria, recurrent urinary tract infections, or nephrolithiasis. She was febrile and tachycardic, with bilateral CVA tenderness and 2+ bilateral lower extremity edema. Labs were notable for hemoglobin 8.4, MCV 92, WBC 16.1, BUN 34, Cr 2.21, GFR 24, sodium 130, electrolytes otherwise normal. Urinalysis showed pyuria, hematuria, leukocytes, and many bacteria. Blood and urine cultures grew Escherichia coli. CT showed bilaterally enlarged kidneys with hypoechoic renal cortices, without hydronephrosis or mass lesions. MRI did not show spread to adjacent organs. Renal biopsy was consistent with xanthogranulomatous pyelonephritis.

Consultations from urology, nephrology, and infectious disease were obtained, with the decision to treat medically with close monitoring of renal function. Ceftriaxone was started, which was ultimately de-escalated to cephalexin for a planned six to twelve month course. Creatinine peaked at 3 and improved to 1.8. She did not develop oliguria or anuria. At a follow-up appointment 2 months later, renal function remained stable.

Conclusions

Bilateral XGP is an extremely rare disease entity, which historically has been believed to be fatal without bilateral nephrectomy. This case demonstrates the second in the literature that has been treated medically with stable renal function. The authors in the first published case suggested that pyrexia may be an important indicator for nephrectomy in bilateral XGP. Our case is not consistent with this postulation, as the patient did have initial pyrexia. It is unclear if nephrectomy will ultimately be required. Despite this, the preservation of renal function with antibiotic suppression is in itself an achievement in care.

Acute Hepatitis C as a Cause of Acute Liver Injury
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Introduction

The CDC estimates that over 3.5 million people in the United States have chronic hepatitis C virus (HCV) infection with approximately 34,000 new infections contracted yearly. However, it's estimated as few as 7.1% of acute infections are diagnosed and reported to state health departments. This case report explores an instance of acute liver injury secondary to acute HCV infection and the patient's less-common risk factors for HCV infection.

Case Presentation

A 45-year-old male with past medical history of coronary artery disease, diabetes mellitus type II, hyperlipidemia, fibromyalgia, and polysubstance abuse (methamphetamine, cocaine, marijuana, and alcohol) presented to the emergency department with progressive dyspnea and fatigue with recent onset of nausea, vomiting, abdominal pain, and dark urine. He denied any alcohol or drug use for several years, but did endorse taking four to six tablets of oxycodone/acetaminophen 10/325mg per day and ten tablets of the herbal supplement fenugreek over a two-week period.

Physical examination on arrival was unremarkable; however, lab evaluation revealed AST 1041, ALT 815, alkaline phosphatase 315, and total bilirubin 4.0. Acetaminophen and alcohol levels were undetectable. Autoimmune and infectious hepatitis evaluations were performed and negative except for a positive HCV antibody with a viral load of 62 million IU/mL. Abdominal ultrasound revealed mild hepatomegaly with no significant masses or biliary ductal dilatation. Further history revealed the patient had tested negative for HCV one year prior, his girlfriend two months ago. He denied any IV drug use, recent tattoos, needle sticks, or exposure to another individual's blood. He was monogamous with his girlfriend, including practicing insertive anal sex, although she did have a recent extra-relational sexual encounter. After further questioning, he admitted to recent intranasal cocaine use. Hepatic enzymes continued to elevate during his hospitalization, with peaks of AST 1927, ALT 1168, alkaline phosphatase 315, and total bilirubin 10.2. He developed acute thrombocytopenia and jaundice, but his INR remained normal. Liver biopsy was performed which showed severe acute lobular and portal hepatitis, consistent with acute hepatitis C infection. He was managed conservatively, liver enzymes stabilized, and he was discharged home with plans to re-evaluate outpatient.

Conclusions

Acute HCV infection is rarely identified as only 17% of patients with new infections present with symptomatic acute hepatitis. The number of reported cases of acute HCV infection has been increasing since 2010, primarily due to increased injection drug use. While this patient denied IV drug use, his high-risk sexual behavior and intranasal cocaine use (an independent risk factor for HCV infection) increased his risk. This case is an important learning opportunity not only because HCV was identified as the cause of acute liver injury but also because it demonstrates the importance of non-injection drug use risk factors for new HCV infection.

**Melphalan Induced Nephrogenic Diabetes Insipidus:
A Rare Cause of Hypernatremia
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Introduction

Hypernatremia is a phenomenon less common than its often-encountered counterpart hyponatremia in the inpatient setting. This especially holds true in patients with access to free water and without mental impairment. Prompt recognition and reversal of hypernatremia is important in eliminating a condition associated with a high mortality rate in hospitalized patients. Nephrogenic diabetes insipidus occurs in response to kidney resistance to anti-diuretic hormone (ADH) action or aquaporin-2 abnormalities. There are a number of potential causes for nephrogenic diabetes insipidus (DI), but nephrogenic DI is not a known adverse effect of melphalan, a common chemotherapy agent.

Case Presentation

The patient was a sixty-two year old female who was admitted seven days post-stem cell transplantation for multiple myeloma. Prior to stem cell transplantation, the patient received ablative high dose melphalan. The patient reported increased thirst and urine output three days post transplantation. Outpatient labs noted an increase in serum sodium to 148 mEq/L on the day prior to admission. She was admitted with complaints of emesis and polyuria. The patient's vital signs were notable for a temperature 98.6, blood pressure 121/99 mm Hg, and pulse 130 beats per minute. The patient's physical examination was unremarkable except for poor skin turgor. Initial laboratory findings on admission included sodium 161 mmol/L, chloride 130 mmol/L, creatinine 0.94 mg/dL. Initial urine studies demonstrated random urine sodium 17 mmol/L, creatinine 42 mg/dL, osmolality 179 mOsm/Kg. The patient increased her fluid intake and intravenous dextrose infusion was initiated, which improved the serum sodium to 155 mmol/L. A 24-hour urine sample was collected and noted volume of 4.1 L, urine sodium 58 mmol/L, osmolality of 147 mOsm/Kg, creatinine 1442 mg/dL. There was concern for nephrogenic diabetes insipidus and intravenous desmopressin was administered to the patient. The urine osmolality was measured at 65 mOsm/Kg prior to desmopressin dosing, and 71 mOsm/Kg post desmopressin dosing. Repeat desmopressin dosing was completed with urine osmolality failing to increase above 100 mOsm/Kg. Complete nephrogenic diabetes insipidus was diagnosed and the patient was initiated on hydrochlorothiazide, amiloride, and indomethacin on subsequent hospital days. Her serum sodium improved to 137 mmol/L and urine volume decreased to 1.9 L prior to discharge.

Conclusions

This case demonstrated multiple unique features including temporality between medication administration and symptom onset with nephrogenic DI developing three days after melphalan administration. In addition, melphalan has not previously been reported to cause nephrogenic diabetes insipidus unlike other chemotherapy agents such as cisplatin. The diagnosis of nephrogenic DI can be made in a number of previously described manners including desmopressin challenge as in this case. Treatment options are supportive and seek to decrease urine volume and increase urine osmolality via thiazide, potassium sparing diuretics, and prostaglandin synthesis inhibitors.

**Spontaneous Coronary Artery Dissection due to Polyarteritis Nodosa
Presenting with Sudden Death
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Introduction

Spontaneous coronary artery dissection (SCAD) is a rare cause of acute coronary syndrome (ACS) and sudden cardiac death. It can be caused by vasculitis, such as polyarteritis nodosa (PAN). PAN is a systemic necrotizing vasculitis, which typically affects small to medium-sized muscular arteries. PAN-associated coronary artery disease rarely has caused ACS and sudden death has not been reported.

Case Presentation

A 62-year-old female presented with complaints of abdominal pain. The pain was constant, moving from periumbilical to epigastric, right upper quadrant, bilateral flanks, and back regions. Associated symptoms were nausea and vomiting. She had occasional night sweats and a 20-pound unintentional weight loss in the past six months. Past medical history was significant for hypertension and hyperlipidemia. She denied allergies, smoking, alcohol use, or illicit drug use. Her medications included Amlodipine, Atenolol, Quinapril, Furosemide, Clonidine, and Lovastatin.

On examination, she appeared alert, but in pain. Vital signs were normal. Abdomen was soft and non-distended, tender to palpation with no rebound tenderness or guarding. All other systems were normal. Her white blood count was elevated, but renal and liver function were normal. Urinalysis was unremarkable. Imaging revealed thickening of the gastric wall and mucosal enhancement and gall bladder wall thickening and suspicion of superior mesenteric artery dissection and complete occlusion.

The patient underwent laparoscopic cholecystectomy. On day four, she complained of headache but denied chest pain and was given pain medication. On day five, she was unresponsive and failed to respond after 30 minutes of resuscitation. On autopsy, she had marked narrowing of the posterior descending coronary artery. On microscopy, there was segmental fibroid necrosis with dissection, with blood within the media leading to narrowing or occlusion of the lumen. Cause of death was suspected to be due to arrhythmias from this occlusion since myocardial sections showed no evidence of ischemic injury. There was no significant atherosclerosis or thromboemboli in the proximal right, main, left anterior descending or circumflex arteries. She had vasculitis of the superior and inferior mesenteric arteries with dissection, celiac and pancreaticoduodenal arteries, hepatic arteries, and small artery branches in the kidneys.

Conclusions

SCAD is a rare cause of ACS and sudden cardiac death and typically affects young females with fibromuscular dysplasia. PAN-associated coronary vasculitis leads to intimal narrowing hence ischemic chest pain, occasionally ACS. This was an unusual presentation of PAN, though abdominal pain, weight loss, and CT scan findings were non-specific clues. Our patient denied chest pain possibly due to pain medications, but had no evidence of myocardial infarction on histology. While rare in this age group, SCAD should be considered in all patients with PAN, even in presence of traditional coronary disease risk factors.

Urethral Amyloidosis in a Patient with a History of IgA Nephropathy
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Introduction

Amyloidosis is an uncommon condition characterized by the deposition of abnormal β -sheet fibrillar proteins in various organs including the kidneys, spleen, and liver. It is found less commonly in the genitourinary tract, particularly in the urethra. Fewer than 60 cases of urethral amyloidosis have been described in the literature. Urethral amyloidosis can present at any age, but is found mostly in males with initial presenting symptoms including hematuria, dysuria, urethral discharge, penile induration or masses, and gross urethral bleeding. While urethral amyloidosis is a rare condition, there is no known association between IgA nephropathy and amyloidosis.

Case Presentation

A 35-year-old male with a history of IgA nephropathy presented to the urology clinic with complaints of intermittent gross hematuria for the previous two days associated with dysuria and perineal discomfort without penile discharge. His past medical history includes IgA nephropathy diagnosed nine years before this presentation. At the time of his IgA nephropathy diagnosis, he presented with gross hematuria and was in end-stage renal disease. He received a living, unrelated donor renal transplant the same year after several months of hemodialysis. His family history was significant for IgA nephropathy in his father. Physical examination was remarkable for a palpable, transplanted kidney in the left lower quadrant. Evaluation of the new hematuria included a urine analysis showing red blood cells too numerous to count; the specimen was grossly bloody and there was no proteinuria. Urine culture was negative for bacterial growth. A CT scan of the abdomen and pelvis showed atrophic native bilateral kidneys with minimal calcification. Flexible cystoscopy revealed an anterior, ulcerated mass three centimeters proximal to the urethral meatus. The mass was resected and measured 1.7 x 1.9 x.5 cm. The amorphous mass was positive for Amyloid P protein but negative for Amyloid A. Histopathology showed chronic inflammatory changes. Further hematologic workup ruled out primary amyloidosis and oncology recommended continued observation.

Conclusions

Urethral amyloidosis is a challenging diagnosis because it can mimic other conditions presenting with gross hematuria including urological malignancies and IgA nephropathy. The lesion can be found with cystoscopy. In most cases of localized urethral amyloid tissue, the only treatment required is local excision of the tissue. Recurrence of urethral amyloid tissue is uncommon. Once amyloid tissue has been identified on histopathology, further workup to distinguish primary versus secondary amyloidosis is recommended. There is no previously known association between IgA nephropathy and amyloid deposition, although IgA nephropathy may have a systemic inflammatory component that could contribute to the development of amyloidosis.

Invasive Disseminated CNS Aspergillosis
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Introduction

Aspergillus species is a significant cause of mortality in immunocompromised patients. Central nervous system (CNS) aspergillosis can occur through hematogenous or contiguous spread. Immunosuppressive therapy and neutropenia are the major predisposing factors leading to invasive aspergillosis. In CNS aspergillosis, mortality rates approach 100%. We present a patient with a recent diagnosis of granulomatosis with polyangiitis on systemic corticosteroids with development of invasive disseminated aspergillosis with hematogenous spread to the CNS confirmed postmortem.

Case Presentation

A 70-year-old female with a past medical history of granulomatosis with polyangiitis presented with acute encephalopathy and increased oxygen requirements. The patient was on a steroid taper after receiving rituximab and pulse dose solumedrol. A computed tomography (CT) of the head showed various hypodense lesions throughout both cerebral hemispheres. MRI of the brain showed innumerable ring enhancing lesions. CT of the chest showed innumerable pulmonary nodules and cardiomegaly with pericardial thickening and effusion. Patient further deteriorated, required intubation and had unsuccessful resuscitation. Autopsy showed invasive aspergillosis involving small arteries causing hemorrhagic infarcts and multiple fungal abscesses in the brain. Blood culture was positive for non-fumigatus species of Aspergillus.

Conclusions

The clinical presentation of CNS aspergillosis is nonspecific with fever, focal neurological deficits, changes in mental status, and seizures. These symptoms can be rapidly progressing with reports of a median time of seven days from the initial appearance of CNS symptoms to the diagnosis of CNS aspergillosis or death. Corticosteroids increase the risk of aspergillosis by their immunosuppressant effect on immune cells and the presumed direct trophic effect on Aspergillus.

Hypokalemia in Thyroid Dysfunction

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Introduction

Thyrotoxic periodic paralysis is an acquired sporadic form of hypokalemic periodic paralysis that can occur in association with hyperthyroidism. It is more common in males and Asian populations, but should be considered in the workup of a patient with significant weakness and profound hypokalemia.

Case Presentation

A 26-year-old Caucasian female patient with past medical history significant for recurrent nephrolithiasis, hyperthyroidism, and hypokalemia presented with acute onset of extremity weakness and numbness. Prior to this episode of weakness, the patient denied any signs or symptoms of illness. Her only medication was a potassium supplement. On presentation to the hospital, a physical exam showed sinus tachycardia, goiter, and decreased muscle strength in bilateral upper and lower extremities. Reflexes were intact as was sensation. On lab, the patient had non-anion gap metabolic acidosis with severe hypokalemia. She denied any family history of hypokalemia or hearing loss making autosomal dominant hypokalemia unlikely. She also denied any history of glue huffing or toluene ingestion. Urine pH was 7.0, and urine anion gap was calculated as 6, which was unlikely to be associated with distal renal tubular acidosis. Further history from the patient revealed that she had been diagnosed with hyperthyroidism in the past, but never followed up for further evaluation and treatment. The patient was started on IV electrolyte replacement with resolution of her weakness and IV fluids were administered with resolution of the non-anion gap metabolic acidosis. Workup was completed for cause, with notable values including a TSH of <0.01 uIU/mL, T4 >8.0 ng/dL, and free T3 28.6 pg/mL. Thyroid stimulating immunoglobulin was elevated at 542%. The patient was continued on potassium supplementation and started on methimazole for treatment of her severe hyperthyroidism. She had resolution of her generalized weakness and her potassium levels remained within normal limits on discharge.

Conclusions

The mechanism for hypokalemia in severe hyperthyroidism is not well understood. It is thought to occur due to elevated thyroid hormone causing increased responsiveness to sodium-potassium ATPase activity on skeletal muscle membranes. This leads to potassium being driven intracellularly. Patients should be treated with potassium supplementation as well as restoration of euthyroidism. It is important to consider reasons for hypokalemia and paralysis outside of typical renal, intoxication, neurologic, or familial causes, as this can lead to more appropriate long-term treatment and prevention.

Case of Pulmonary Tuberculosis within the State of Kansas
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Introduction

Tuberculosis (TB) is one of the world's deadliest diseases affecting one third of the world's population. In 2015, there were 10.4 million reported cases and 1.8 million TB-related deaths worldwide. TB is a leading cause of death in HIV infected patients. A total of 9,557 TB cases (a rate of 3.0 cases per 100,000 persons) were reported in the United States in 2015, which is an increase after having declined yearly during 1993–2014. Pulmonary TB should be suspected in patients with relevant clinical manifestations as well as epidemiologic factors including prior exposure, relevant risk factors, and travel to endemic areas where tuberculosis is ubiquitous.

Case Presentation

A 57-year-old Caucasian male with history of diabetes mellitus type 2, drug abuse, and history of incarceration presented to the emergency room with chief complaints of fever, chills, night sweats, hemoptysis, and productive cough for a month. On arrival, chest x-ray noted bilateral cavitory masses in the upper lung fields and labs noted white blood cell count of 18,000. A chest CT scan demonstrated large areas of cavitation and scattered nodular infiltrates throughout both lungs. Constellation of findings was consistent with tuberculosis. Sputum Acid-fast bacilli stain was positive and three weeks later cultures identified *Mycobacterium tuberculosis*. Further work up for infectious etiologies was negative – including negative HIV. The public health department was notified. Patient was started on rifampin, isoniazid, pyrazinamide, and ethambutol for two months. M TB strain was pan-susceptible. He then was transitioned to rifampin and isoniazid for additional four months. Follow-up identified negative sputum samples several weeks after initiation of antibiotic therapy.

Conclusions

Provisional data for 2016 demonstrate a slight decline in both TB case count and incidence in the United States compared with 2015. However, previously published epidemiologic modeling suggests that maintaining similar rates of decline in the future will not be sufficient to achieve TB elimination. Clinicians should implement systemic screening of high-risk populations. Epidemiologic models demonstrate that identifying and treating persons with Latent TB is critical to accomplishing the goal of TB elimination. The USPSTF characterizes populations at increased risk as those persons who were born in, or formerly resided in, countries with increased TB prevalence per World Health Organization; in the U.S., persons who currently live in, or have lived in high-risk congregate settings such as homeless shelters, correctional facilities, and long-term care facilities. Current TB control priorities, including early identification of TB, prompt initiation of treatment, and identification of exposed contacts remain critical to preventing a resurgence of TB and to achieve TB elimination.

Don't Underestimate the Power of Streptococcus!
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Introduction

Streptococcal toxic shock syndrome (TSS) is a clinical illness characterized by shock and multi-organ failure; it occurs because of capillary leak and tissue damage due to release of inflammatory cytokines induced by streptococcal toxins. Streptococcal TSS occurs most frequently in the setting of infection due to group A Streptococcus (GAS). There are an estimated 3.5 cases of streptococcal TSS per 100,000 persons, with a case-fatality rate of 30 to 60 percent. GAS TSS occurs among all age groups; Risk factors associated with development of severe GAS infection include minor trauma, NSAID use, recent surgery, viral infection, and postpartum state. GAS typically causes pharyngitis or skin and soft tissue infection; and can cause invasive disease in approximately one-third of cases. We describe a case of GAS pharyngitis with secondary pneumonia complicated by TSS.

Case Presentation

A 35-year-old female with PMH of Cohn's Disease and Asthma, admitted with complaints of fever, nasal congestion with drainage, sore throat, headache and weakness. SH married with two children, denies smoking, EtOH, illicit drug use. O: HR 160's, BP 80/60, T 104° F, RR 20. On examination, HEENT: oropharynx erythematous with significantly swollen uvula. No visible exudate. Neck: no tenderness to palpation, No JVD, No LAD. Lungs: CTAB, no wheezing, crackles or rhonchi, Heart: tachycardia without murmur, regular rhythm, Abdomen: BS +, soft, nontender, non-distended. Ext: No edema, cyanosis, clubbing Neuro; grossly intact. Admission labs: WBC 19.3, Hgb 13.8, PLT 196, HCO₃ 19, creat 0.85. Rapid Strep Throat swab was positive for Group A Streptococcus. Within 12 hours of admission, her condition suddenly deteriorated with hypotension, tachypnea, fevers, chills, diffuse erythematous skin rash, SOB, BP 80/50, prompting transfer to ICU, requiring Levophed, lactate of 3.5, HCO₃ 17. CT neck revealed fullness in the bilateral tonsillar pillars and prevertebral region without abscess formation. CT chest showing multi-lobar consolidation. BC negative. ECHO with normal EF; Her hemodynamics stabilized over the course of next days with fluid resuscitation, weaning of pressor support and de-escalation of antibiotics.

Conclusions

In TSS due to GAS, portal of entry of streptococci cannot be proven in at least half the cases and can only be presumed in many others. Treatment of streptococcal TSS consists of a beta-lactam agent in combination with clindamycin, which inhibits protein synthesis. Penicillin monotherapy is associated with high mortality and extensive morbidity in the setting of aggressive infections associated with TSS. Adjunctive therapies used for patients with streptococcal TSS include intravenous (IVIg), hyperbaric oxygen, and anti-tumor necrosis factor (TNF) antibody. Thus, Clinicians should consider Group A streptococcal (GAS) toxic shock syndrome (TSS) in any patient presenting from the community in shock in the absence of a clear etiology with multiorgan failure as timely antibiotic administration can be lifesaving.

Immunotherapy: Revolutionizing AML Treatment?
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Introduction

Molecular and cytogenetic profile plays a significant role in treating Acute Myeloid Leukemia (AML). The most common mutation in AML is FMS-like tyrosine kinase 3 (FLT3), which indicates a poor prognosis. Most therapeutic approaches are directed toward the cytogenetic and mutational profile of the disease. In this case, a relapsed case of AML managed with immunotherapy is presented which lead to complete remission of the disease.

Case Presentation

A 65-year-old female was diagnosed with AML with hyperleukocytosis in 2016. Bone marrow biopsy revealed acute myelomonocytic leukemia (AMML) with 80% hyper-cellularity and 30% blasts. Cytogenetic profile expressed isolated Trisomy 13. Myelodysplastic syndrome (MDS) panel was normal and FLT3-ITD mutation was positive. Induction chemotherapy with standard cytarabine/idarubicin (7+3) chemotherapy was initiated, but resulted in residual disease. The patient was given less intensity therapy with Azacitidine plus Sorafenib given her age and performance status. She attained complete remission after four cycles of therapy, but repeat bone marrow biopsy in early 2017 showed relapsed disease after developing leukopenia and thrombocytopenia after seven cycles. She was started on salvage therapy with FLAG (Fludarabine, Cytarabine and Granulocyte-colony stimulating factor) chemotherapy considering her cardiomyopathy with ejection fraction of 30%. She responded to therapy with complete remission and she was referred for allogeneic transplant. After considering the risks of an allogeneic stem cell transplant, the patient declined. Treatment with Decitabine with experimental Pembrolizumab was initiated. After two months of immunotherapy, bone marrow biopsy revealed complete remission of the disease and exhibited no side effects to therapy except for intermittent neutropenia and thrombocytopenia expected from decitabine.

Conclusions

Relapsed AML in general is known to pose a guarded outcome. FLT3 positive disease is the strongest indicator of short survival. Finding optimum AML therapies is challenging. Allogeneic transplant remains the mainstay therapy for AML for “fit” individuals. It is most effective in a younger population, but the age threshold continues to increase. Despite advances in therapy, treatment strategies for AML in the elderly are limited. In last 40 years, no treatment protocol has been generated for AML in the US that has proven more effective than the standard. Using the immune checkpoint inhibitor, pembrolizumab, complete remission was achieved in our patient. Studies show up-regulation of immune checkpoints in AML and its inhibition leads to maintenance and disease remission. However, larger clinical trials should be conducted to understand the disease process and treatment better.

**Seen on Closer Inspection:
A Rare Case of Pulmonary Intravascular Lymphoma Detected by 18FDG-PET/CT
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Introduction

Intravascular lymphoma (IVL) is a rare subtype of extranodal diffuse large B-cell lymphoma characterized by the selective growth of lymphoma cells exclusively within the lumina of small vessels. When IVL affects the lungs, parenchymal lesions on chest CT are almost always seen. We report an extremely rare case of pulmonary IVL detected by 18FDG-Positron Emission Tomography/Computed Tomography (PET/CT) without the patient having any corresponding chest CT abnormalities.

Case Presentation

A 60-year-old man with a nine-month history of 40-pound weight loss, fatigue, weakness, and night sweats presented with fever of unknown origin. Seven weeks prior, he received a splenectomy at another hospital for presumed splenic marginal zone lymphoma, detected solely by B-Cell gene rearrangement with minimal improvement. Physical examination demonstrated a fever of 38.7 C and new hypoxemia. Initial evaluation revealed a normal CTA chest. Further investigation for an autoimmune, neurologic, pulmonary, infectious, or malignant etiology showed no abnormalities except mild anemia, elevated LDH at 923 U/L, and elevated CRP at 10.57 mg/L. The negative workup and persistent pulmonary symptoms prompted further evaluation with 18FDG-PET/CT. Diffusely increased metabolic activity throughout both lungs without confluent parenchymal abnormality was seen suggesting intravascular lymphoma. A thoroscopic wedge resection of the right middle and upper lobes confirmed the diagnosis of pulmonary intravascular large B-cell lymphoma. Patient was initially treated with R-CHOP chemotherapy but was switched to methotrexate/cytarabine when further evaluation showed possible neurologic involvement. Patient was able to complete four cycles of treatment. However, his treatment course was complicated by recurrent infections. Patient ultimately died secondary to septic shock despite achieving complete remission from his disease.

Conclusions

Intravascular lymphoma (IVL) is a rare subtype of extranodal diffuse large B-cell lymphoma characterized by the disordered proliferation of neoplastic lymphoid cells exclusively within the lumina of small vessels. In Western countries, IVL predominantly affects the central nervous system and skin, but pulmonary involvement also has been described. Patients with pulmonary involvement face a difficult diagnosis as its presentation mimics various lung diseases including interstitial lung disease or a subacute or chronic infectious process. Thus, lung anomalies are usually detected at autopsy. Pulmonary IVL almost universally presents with some abnormality on CT imaging. The most common abnormalities are diffuse ground-glass opacities, but accompanying consolidations and nodules also can occur. However, in extremely rare cases, the diagnosis of pulmonary IVL is indicated by the diffuse uptake of FDG in the lungs on PET/CT without corresponding structural CT abnormality. This rare situation was seen in our case. Upon literature review, less than 10 other case reports with similar scenarios have been described. Thus, our case highlights the importance of utilizing 18FDG-PET/CT in the diagnosis of pulmonary IVL even if previous imaging show no anomalies.

Fatal Acute Liver Failure due to Primary Herpes Simplex Virus Infection
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Introduction

Herpes simplex virus is a well-documented, although very rare, cause of acute liver failure. It is more common in immunosuppressed and pregnant patients. Diagnosis is challenging as serologic, culture, and histologic techniques do not have rapid turn-around time and there may not be sufficient viremia to yield positive results with polymerase chain reaction. Clinical suspicion may be confounded by the lack of skin or genital findings, which may be negative in more than half of patients. This clinical suspicion is necessary to consider initiation of empiric acyclovir in the face of insufficient evidence. We present the case of an immunosuppressed man who presented with rapidly progressing acute liver failure due to primary HSV-1 infection, which was ultimately fatal.

Case Presentation

A 53-year-old white male was transferred from an outlying facility for jaundice of two days duration, with vague symptoms, including malaise and loss of appetite. Medical history was significant only for rheumatoid arthritis, which had been successfully controlled with infliximab and leflunomide. Routine laboratory monitoring two months prior showed normal liver function tests. The patient denied alcohol, illicit drug, and acetaminophen use which was corroborated on laboratory testing. On admission, his mental status was appropriate, without asterixis or signs of coagulopathy. There were no oral, rectal, or genital lesions evident. Serum AST and ALT were markedly elevated at 13,879 U/L and 3,023 U/L respectively. Bilirubin was mildly elevated at 3.1 mg/dL. INR was mildly elevated at 1.4. Serologic testing for hepatitis A, B, and C were negative. Markers of autoimmune hepatitis were ordered, as was serology and appropriate PCR for herpes simplex, varicella, Epstein Barr, and cytomegalovirus. Ultrasound of the liver showed echodense hepatomegaly suggestive of acute hepatitis, without splanchic thrombosis, or biliary dilatation. He was started on broad-spectrum antibiotics and N-acetylcysteine, though acyclovir was not considered at this time. His course rapidly deteriorated with rising INR, declining mental status, and anuric renal failure requiring emergent dialysis and transfer to liver transplant facility was sought. Transfer was complicated by seizure, arrhythmias, and shock; the patient passed away within 72 hours of presentation. Laboratory subsequently showed positive IgM Anti-HSV-1 antibody and positive HSV-1 DNA PCR consistent with acute primary infection. All other serologies were negative.

Conclusions

Prognosis for HSV induced acute liver failure is very poor, with an estimated 70 - 80% mortality. Due to rarity of disease, few data exist regarding mortality for those treated with empiric acyclovir. Despite this, empiric acyclovir should be considered in cases of acute liver failure without obvious cause, even with negative mucosal findings, and especially in immunosuppressed and pregnant patients.

Defying Expectations: A Unique Presentation of C. Difficile
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KU School of Medicine-Wichita

Introduction

Clostridium difficile is an anaerobic, gram-positive rod known for its propensity to cause colitis. Its pathogenicity is linked to antibiotic resistance, spore formation and toxin production, which allow it to invade mucosa and create pseudomembranes. Typical symptoms include watery diarrhea, abdominal pain, fever, and leukemoid reaction with recent antibiotic exposure. The organism is fastidious and difficult to isolate in culture. Contemporary diagnostic methods include enzyme immunoassays and PCR of toxin genes in stool. Management is guided by disease severity and is centered around Metronidazole or oral Vancomycin.

Case Presentation

A 54-year-old male with history of cirrhosis, chronic hepatitis B and C, and transjugular intrahepatic portosystemic shunt (TIPS) presented to his doctor with left upper quadrant pain, fever, and anorexia for one week. He denied associated vomiting, diarrhea, melena, or hematochezia. Five weeks prior to presentation, he had undergone a TIPS revision with splenic artery embolization. His temperature was 98.3° F, pulse 79 bpm, respiration rate 20 bpm, and blood pressure 125/63 mmHg. The physical exam was unremarkable except for left upper quadrant abdominal tenderness, shallow breathing and spider angiomas. He had no ascites. Comprehensive metabolic panel, lipase, and complete blood count were unremarkable. Ultrasound with Doppler of the liver revealed a patent TIPS. After symptoms continued for one month, a CT of the abdomen revealed a subcapsular, peri-splenic abscess, which measured 17.6 x 15.7 x 11 cm. Hospital admission to surgical team with medical consult, was arranged. Vancomycin and Piperacillin/Tazobactam were empirically administered. Laboratory studies were unremarkable. Blood cultures were negative. CT-guided aspiration with pigtail drain placement yielded 900cc of purulent drainage. Gram stain revealed white blood cells but no microorganisms. Six days later, *C. difficile* was isolated from culture. The patient was switched to oral Metronidazole and discharged with drain in place and plans for serial imaging, antibiotics, and infectious disease follow-up. Subsequent CT scans confirmed progressive decrease in abscess size.

Conclusions

C. difficile infections can present outside of the colon. Enteritis and reactive arthritis are most commonly cited. Other sites include abscesses and skin infections. These presentations are typically associated with bacteremia, prior colectomy, or immunodeficiency. This case of a monomicrobial *C. difficile* splenic abscess, in the absence of colonic disease or bacteremia, is exceedingly rare. The inciting event may have been the splenic artery embolization leading to transient bacteremia. As the incidence of *C. difficile* colitis and asymptomatic carriers rises, the rate of extracolonic disease is expected to rise. Mainstream EIA and PCR tests are limited to stool analysis. Considering this organism is difficult to isolate in culture, extracolonic *C. difficile* infections may often be misdiagnosed or entirely undetected.

Bing-Neel Syndrome: A Rare Presentation of Waldenstrom Macroglobulinemia
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Introduction

Waldenstrom Macroglobulinemia (WM) is an indolent B-cell lymphoma associated with overproduction of monoclonal immunoglobulin M (IgM) by a monoclonal plasma cell population. It presents with symptoms related to hyperviscosity, cryoglobulin precipitation, and bone marrow infiltration such as headaches, visual disturbances, mental status changes, hemolytic anemia, Raynaud phenomenon, renal insufficiency, peripheral neuropathy, cytopenias, and mucosal bleeding. While many disease manifestations of WM have been described, this case demonstrates a unique, yet important presentation.

Case Presentation

A 75-year-old male with a 12-year history of polyneuropathy presented to the hospital with three months of progressive lower extremity weakness, diplopia, left eye ptosis, weight loss, dyspnea, and inability to walk. He was started on prednisone 60mg for one month with slight improvement in symptoms, followed by an unsuccessful five day trial of pyridostigmine. Electromyography findings were consistent with axonal sensorimotor polyneuropathy without evidence of neuromuscular junction or motor neuron disease. A myasthenia gravis panel was negative. Additional laboratory studies were remarkable for elevated anti-nuclear antibody, and he was diagnosed with inflammatory polyneuropathy. This was treated with a five day infusion of intravenous immunoglobulin G, after which he had mild improvement in symptoms and was discharged.

Three weeks later, he was re-admitted with worsening mental status, dysarthria, dysphagia, and right facial droop. MRI brain revealed development of hazy fluid-attenuated inversion recovery (FLAIR) hyperintensities within the periventricular white matter, corpus callosum, and deep gray nuclei with concordant high diffusion signal. Lumbar puncture showed elevated protein and lymphocyte-predominant pleocytosis in the cerebrospinal fluid without evidence of malignant cells. Subsequent serum protein electrophoresis showed a probable monoclonal spike in the Beta/Gamma regions. Hematology was consulted for a bone marrow biopsy, which revealed findings consistent with low-grade B cell lymphoma involving 10% of the bone marrow, plasmacytoid differentiation, and hypercellularity. Genetic analysis revealed positivity for MYD88 gene mutation. He was diagnosed with WM and due to his clinical presentation was presumed to have Bing-Neel syndrome (BNS), despite the lack of pathological confirmation due to instability of patient's condition. In light of presumed BNS he met the criteria for treatment with Bendamustine and Rituximab, however declined despite treatment. Repeat MRI of the brain revealed progression of bilateral cerebral, callosal, basal nuclei, and brainstem signal abnormalities with superimposed areas of increased and decreased associated enhancement. These findings support our clinical diagnosis, although confirmatory autopsy results are pending.

Conclusions

WM is a rare disease with many different presentations and complications. Bing-Neel syndrome is a rare complication associated with central nervous system involvement by malignant cells and presentation can include headaches, cognitive deficits, paresis, cranial nerve involvement, gait disorders, and psychiatric symptoms. It is rarer still to see Bing-Neel as the initial presentation of WM, as seen in our patient.

Sickle Cell Trait as a Rare Case of Masked Polycythemia Vera
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Introduction

The combination of sickle cell hemoglobinopathy and polycythemia vera (PV) is an extremely rare finding. This unique combination has been infrequently described in previous case reports. We hereby report a patient with confirmed polycythemia vera and blunted erythropoiesis secondary to a coexisting sickle cell trait.

Case Presentation

A 66-year-old African American male with a history of hypertension, diabetes mellitus type 2, obstructive sleep apnea, hyperlipidemia, and Jak-2 positive PV (diagnosed 2012) presented to our clinic with complaints of mild fatigue and limited aquagenic pruritus. The patient had been treated with aspirin and infrequent therapeutic phlebotomies. Interestingly, he received several phlebotomies from 2012 to 2015, and once in January 2017, but has not required phlebotomies or cytoreductive therapy since that time. Family history was positive for a sister with sickle cell disease. On physical examination, the patient was found to have modest splenomegaly. Investigative studies at the time of diagnosis of PV in 2012 showed normal white blood cell and platelet counts. Hemoglobin was increased at 17.5 mg/dl and hematocrit was 53.8%. JAK-2 mutation testing was positive for JAK-2 V716F mutation and his serum erythropoietin level was decreased. There was no BCR-ABL rearrangement. Bone marrow biopsy was performed and was pertinent for absence of iron staining and hypercellular bone marrow with panmyelosis that is consistent with his previous diagnosis of polycythemia vera. The patient's hemoglobin electrophoresis was diagnostically consistent with sickle cell trait. Imaging confirmed splenomegaly on abdominal ultrasound. Current hemoglobin and hematocrit levels are 11.7 mg/dl and 39.3% respectively. The patient is currently undergoing active surveillance.

Conclusions

Polycythemia vera is a chronic myeloproliferative disorder characterized by a malignant clonal proliferation of erythrocytes and increased red blood cell mass. This increase in red blood cell mass is independent of erythropoietin. Hemoglobin and hematocrit are increased in polycythemia vera and treatment typically involves phlebotomy or other cytoreductive agents. Iron deficiency is commonly seen secondary to therapeutic phlebotomies and increased cell production demand. Sickle cell trait is an autosomal recessive inherited blood disorder in which patients carry one abnormal beta hemoglobin gene. The anemia commonly found in sickle cell patients is caused by the hemolysis of abnormally sickle-cell-shaped red blood cells. The diagnosis of PV in patients with concurrent sickle cell trait may be difficult to identify due to its masking effects in CBC studies, in which subclinical hemolysis may neutralize the malignant erythrocytosis. In our patient, his sickle cell trait has kept his extremely rare concomitant polycythemia vera in check without the need for regular management by essentially acting as an "auto-phlebotomy" treatment.

Azathioprine Hypersensitivity Mimicking Sepsis
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Introduction

Azathioprine hypersensitivity is less well known than toxicity, but has been documented to mimic certain life-threatening conditions including septic shock. In a case study of 900 adverse reactions to azathioprine, seven were determined to be hypersensitivity (0.8%). Timing of the reaction supports the possibility of a type III hypersensitivity reaction. The reaction can occur within one week, or up to 16 months after starting the medication.

Case Presentation

A 78-year-old female with history of scleroderma, heart failure, mitral valve stenosis, pulmonary hypertension, and CKD stage III. She was admitted from an outside hospital with diagnosis of sepsis. This was the third admission for septic shock requiring ICU care in three weeks. On the first visit, she was diagnosed with urinary tract infection treated empirically with cefepime and vancomycin, but no growth was found in cultures. She was sent home as she improved with oral antibiotics and instructed to remain off immunosuppression (azathioprine) until completing them. After completing antibiotic and resuming azathioprine, she again had fever and hypotension. She was started on vancomycin and cefepime again. All cultures were negative and she was discharged home to complete a 10-day course of cefepime and vancomycin. The day before the final admission, she completed antibiotics, again restarting azathioprine. She experienced vomiting, fever, and hypotension responsive to fluid and she was transferred to tertiary care facility. Blood and urine cultures again were negative. Any suspicion for aspiration was ruled out after a swallow study and esophagram showed no abnormalities. Initial procalcitonin was 0.6 ng/mL on arrival but spiked up to 34.8 ng/mL on day two. Chest x-ray was negative and the patient did not have any respiratory symptoms throughout hospitalization and remained afebrile. She was discharged to follow-up with rheumatology and developed a new medication regimen without azathioprine.

Conclusions

This case demonstrated a rare, potentially lethal adverse reaction to azathioprine. Elevated procalcitonin was a novel finding in this case. It is theorized that the immune response causing hypotension and fever was also responsible for procalcitonin release mimicking bacterial infection. Azathioprine hypersensitivity is a diagnosis of exclusion, but should be considered in patients taking the medication with symptoms of sepsis if exhaustive infectious work up is negative.

Focal Myopericarditis Presenting as Acute ST-Elevation Myocardial Infarction
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Introduction

The clinical presentation of myocarditis and pericarditis is variable and often mimic myocardial infarction. Distinguishing differential diagnoses for ST-Elevation myocardial infarction is challenging. The diagnosis of acute myopericarditis is frequently empiric, and is made based on the clinical presentation, electrocardiographic changes, elevated cardiac enzymes, and lack of epicardial coronary artery disease. Myopericarditis is one such diagnosis, if presented in focal form can mimic acute ST-Elevation myocardial infarction. We present the case of 18-year-old woman who presented with chest pain and inferior wall ST-elevation on electrocardiogram. The diagnosis of acute myopericarditis was eventually confirmed with cardiac magnetic resonance imaging (Cardiac-MRI).

Case Presentation

An 18-year-old female patient with no past medical history presented to the emergency room for crushing chest pain. Patient reported that she woke up with the mid-sternal chest pain radiating to the back and both arms with associated symptoms of shortness of air. Patient reported no change in quality of pain with position or on inspiration and also denied nausea, vomiting, drug abuse and diaphoresis. Focused physical examination in the emergency room was notable for regular heart rhythm with no audible murmur, gallop, or rub. Patient had clear lungs and no peripheral edema. Electrocardiogram done on presentation showed ST-Elevation in inferior wall region (Leads 2, 3, aVF) and non-specific P-R changes. Subsequently, patient underwent a CT-Angiography of the chest to rule out possible dissection and pulmonary embolism. Once the dissection and pulmonary embolism were ruled out, patient was taken for emergent cardiac catheterization.

Cardiac catheterization revealed normal coronary arteries with hypokinesis in the inferior wall left ventriculogram. Echocardiogram was subsequently performed, revealing left ventricular ejection fraction of 55-60% and showed mild degree of hypokinesis in the inferior wall. Troponin measured on arrival was 1.06 ng/mL (reference normal <0.07) and then peaked to 22.88 ng/mL. Erythrocyte sedimentation rates, C-reactive protein and, urine drug screen was normal which muddled the picture and the elevation in troponin and ST Elevation on an electrocardiogram was presumed to be due to coronary vasospasm. In order to confirm the diagnosis and look for other possible causes of non-ischemic cardiomyopathy, patient underwent a cardiac MRI which revealed hypokinesis in the lateral and inferior wall of the left ventricle apex with some epicardial and transmural delayed enhancement suggestive of focal myopericarditis.

Conclusions

ST-Elevation Myocardial Infarction is the most important differential diagnosis of focal myopericarditis. Myopericarditis shows a characteristic pattern of contrast enhancement on Cardiac MRI, which originates primarily from the epicardium, sparing the sub-endocardial layer. Myopericarditis need not always have elevated ESR and CRP as they are only positive in 60% of the cases.

**Challenges in Diagnosing Granulomatosis with Polyangitis
(GPA – Wegener’s Granulomatosis)
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Introduction

Granulomatosis with Polyangitis (GPA) and tuberculosis (TB) share similar clinical presentations. Patients may often present with nonspecific URI symptoms and hemoptysis. It is not widely known that anti-neutrophil cytoplasmic antibodies may be positive in GPA and TB about 40% of the time. Such a perplexing presentation warranted a biopsy for final confirmation.

Case Presentation

An 81-year-old Native Indian male with past medical history of metastatic prostate cancer, chronic kidney disease, diabetes, and hypertension presented to the hospital with productive cough associated with bilateral conjunctivitis, malaise, and hemoptysis. The patient was admitted for respiratory failure and suspected sepsis. His chest x-ray showed diffuse bilateral airspace opacities with a nodular pattern and heterogeneous appearance of the osseous marrow. He was started on broad spectrum antibiotics due to the chest x-ray findings in combination with leukocytosis and his past medical history.

As part of the work-up for developing renal failure, a urinalysis was obtained which revealed 3+ hematuria. In addition, a CT scan of the chest, abdomen, and pelvis was performed due to signs of multi-organ failure and concern of further prostate metastasis. The CT scan showed a 5cm x 4cm x 3cm right upper lobe (RUL) soft tissue mass with irregular lobulated margins and small central calcification. This cued us to broaden our differential diagnosis and lab tests such as PSA levels, ANCA Ab, GBM Ab, complement levels (C3, C4) levels, quantiferon testing, along with RUL lung and bone marrow biopsy were performed. The RUL lung biopsy showed granuloma with central caseating necrosis with no signs of vasculitis. The bone marrow revealed a normal marrow pattern.

The patient’s labs revealed abnormal results including C-ANCA positive (1:320 titer), Anti-proteinase-3 Ab positive (65 U/ml), and positive quantiferon. PSA levels were normal. Active TB treatment was started and three Acid-Fast Bacilli (AFB) smears with good sputum induction were performed. AFB smears, including the RUL lung biopsy specimen, were negative. Active TB treatment was stopped though the patient continued to experience worsening kidney function. Ultimately, a kidney biopsy was performed and was diagnostic of GPA revealing segmental necrotizing and crescentic glomerulonephritis consistent with pauci-immune type GPA. The patient was started on immunosuppressive therapy with Rituximab for GPA and isoniazid with rifampin for latent TB.

Conclusions

This case impacted the way we approached the differential diagnosis for GPA. Cross reactivity of ANCA, PR-3 and positive quantiferon labs muddled the picture between GPA and active TB infection. As non-invasive ANCA and PR-3 serology can be positive in 40% of TB patients, more invasive procedures, such as kidney biopsy, are necessary for confirmation of GPA in populations at risk for TB or a positive quantiferon test.

Differential Diagnoses and Lupus Lymphadenitis
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Introduction

Lymphadenopathy associated with systemic lupus erythematosus (SLE) is relatively common with an incidence of 26%, typically in cervical or axillary nodes. SLE patients with lymphadenopathy are more likely to demonstrate heightened disease activity. Differentiating clinical manifestations of SLE from concomitant diseases becomes complicated in cases of LAD. We present the case of a patient recently diagnosed with SLE who developed diffuse lymphadenopathy, and is eventually diagnosed with lupus lymphadenitis.

Case Presentation

A 24-year-old female, recently diagnosed with SLE, presented with diffuse musculoskeletal pain, sore throat, and fatigue for 36 hours. She denied any new medications, sick contacts, cough, neck stiffness, hematuria, urinary incontinence, diarrhea, or confusion. She discontinued azathioprine due to nausea. She is married, sexually active, denies tobacco or alcohol use, and has a family history of hypertension and heart disease. Review of systems was positive for chest pain, fatigue, decreased appetite, and 15lb unintentional weight loss in seven months. Physical exam findings notable for distress, tachycardia, normal breath sounds, suprapubic tenderness, generalized myalgias and arthralgias, non-tender bilateral cervical and axillary lymphadenopathy, AOx3, and diaphoresis. Laboratory studies revealed WBC 2.0, RBC 3.30, hemoglobin 9.2, HCT 27.1, MCV 82.1, MCH 27.8, RDW 15.1, platelet count 131, sodium 139, potassium 4.3, chloride 111, CO₂ 24, anion gap 4, BUN 13, creatinine 0.46, albumin 3.0, AST 76, and ALT 62. HIV, CMV, EBV, hepatitis panel, influenza, and mono screen returned negative. Serology reports were negative for mycoplasma pneumoniae. Urinalysis was positive for lactobacillus species and hematuria; she was started on empiric antibiotics for acute cystitis. Anti-dsDNA titer > 1280, ANA titer >1280, hypocomplementemia, positive anti-Smith Ab, positive anti-RNP Ab, positive anti-SSA/SSB, positive hexagonal lupus anti-coagulant, elevated cardiolipin IgM. CTA revealed bilateral cervical and axillary LAD with predominant bilateral hilar lymph nodes; mild nodular ground glass opacities were observed bilaterally. Peripheral smear demonstrated pancytopenia, normocytic anemia, anisocytosis, and granulocytopenia with normal neutrophil morphology. Serum leukemia/lymphoma flow cytometry panel indicated 34% lymphocytes with atypical B cells of unknown significance, and possible diagnosis of B cell lymphoma. Axillary lymph node biopsy revealed extensive coagulative necrosis with greater abundance of CD3 T cells than CD20 B cells. Presence of interstitial and vascular hematoxylin bodies pathognomonic for SLE, ruling out Kikuchi-Fujimoto Disease. Bone marrow biopsy did not reveal hemophagocytosis, ruling out hemophagocytic lymphohistiocytosis. A diagnosis of lupus lymphadenitis was made. Patient continued empiric treatment for acute cystitis and was discharged on hydroxychloroquine and prednisone.

Conclusions

Etiology of LAD in SLE patients has a broad differential. Hematoxylin bodies demonstrated on lymph node biopsy in the correct clinical context would be sufficient to diagnose lupus lymphadenitis. A systematic approach is essential for an internal medicine physician to rule out high-risk infections, drug interactions, secondary inflammatory processes, and malignancy, including non-Hodgkin lymphoma.

Ipilimumab-Induced Hypophysitis
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Introduction

Ipilimumab (Ipi)-induced hypophysitis is a well-known, though rare, complication of Ipi therapy, particularly in the treatment of melanoma. This condition subsequently leads to panhypopituitarism and is clinically difficult to diagnose due to the presence of only non-specific symptoms. The purpose of this case is to increase awareness of this condition, as the ability to diagnose quickly is closely tied to degree of clinical suspicion.

Case Presentation

A 62-year-old Caucasian male with a remote history of metastatic melanoma, diagnosed in 2008, presented to the hospital with a two-week history of fatigue, chills, abdominal pain, and nausea, accompanied by a 40 lb. weight loss over one month. His previous melanoma treatment included initial surgical resection and a short course of interferon therapy after which he was lost to follow up. Years later, he presented with an aggressive abdominal metastasis and underwent resection. Shortly after his last surgery, he started Ipilimumab and Nivolumab dual immunotherapy. He completed three cycles before the symptoms began. Of note, he had just been diagnosed with new-onset hyperthyroidism as an outpatient.

Initial admission physical and labs were notable only for a resolving autoimmune thyroiditis. A CT chest found consolidation consistent with pneumonia and he was treated with empiric healthcare-associated pneumonia coverage without improvement. A standard, high-dose cosyntropin stimulation test was performed and was consistent with secondary adrenal insufficiency; specifically, it revealed low ACTH of 7.5 pg/mL, low baseline cortisol of 3.3 ug/dL, and a low-normal cortisol peak of 21.2 ug/dL. An MRI was done to evaluate for potential brain metastases and discovered diffuse inflammation of the pituitary gland and stalk, indicative of hypophysitis. Antimicrobials were discontinued, and he was started on high-dose steroids with drastic improvement over 24 hours. He was discharged on physiologic replacement with good effect.

Conclusions

Ipi has been shown to increase life expectancy in patients with metastatic melanoma. Given the efficacy of this treatment, more patients will be seen by internists with autoimmune complications as the usage rate of Ipi rises. Though the exact target is unknown in Ipi-hypophysitis, Ipi leads to decreased T-cell self-tolerance, which promotes both anti-tumor effects and, unfortunately, increases the risk of autoimmune side effects. This complication can be seen in other immune checkpoint inhibitors such as Nivolumab and Pembrolizumab, both programmed cell-death protein 1 (PD1) inhibitors. This case is differentiated by the presence of hyperthyroidism at the time of diagnosis, obscuring the picture of panhypopituitarism. The timing of the case is typical, as studies have shown that average time to diagnosis is around eight to nine weeks. Recovery from hypophysitis has been poorly elucidated at this time, but one study suggests that panhypopituitarism will resolve with glucocorticoid treatment, and patients are likely to remain with corticotroph deficiency.