

**New York Chapter ACP
Annual Scientific Meeting**

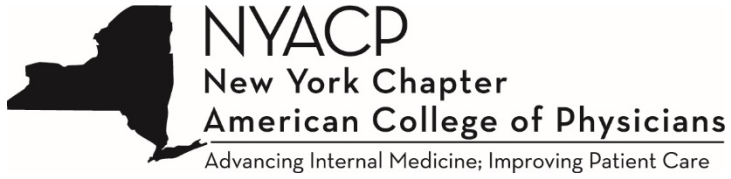
Poster Presentations

Saturday, June 3, 2017

Hyatt Regency Hotel

125 East Main Street

Rochester, NY 14604



**New York Chapter ACP
Annual Scientific Meeting**

Medical Student Clinical Vignette

Medical Student Clinical Vignette

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Title: PATIENT WITH CONGENITAL AGENESIS OF THE GALLBLADDER AND TETRALOGY OF FALLOT PRESENTING WITH TYPICAL BILIARY SYMPTOMS

A 34 year old female with PMH of Tetralogy of Fallot (TOF) presented with complaints of vague abdominal pain and vomiting exacerbated by eating fatty foods. She experienced this pain intermittently in the past with other exacerbating or alleviating factors. Her TOF was corrected surgically in childhood with pulmonic valve replacement and loop recorder for syncope. Initial work up revealed mildly elevated direct bilirubin, AST and ALTs with normal alkaline phosphatase, amylase, and lipase levels. As the patient had no history of prior surgeries, a biliary etiology of this pain was suspected.

Ultrasound of the right upper quadrant revealed no intra- or extra-hepatic biliary duct dilation, common bile duct measuring 6mm, and non-visualization of the gallbladder concerning for contraction of gallbladder or congenital absence of gallbladder. Subsequent computed tomography (CT) and magnetic resonance cholangiopancreatography (MRCP) imaging showed complete absence of the gallbladder, no intrahepatic or extrahepatic bile duct dilatation, and absence of any common ductal stones. A prominent 1 cm ampulla was visualized which did not appear to obstruct the distal common bile duct or pancreatic duct. The pancreas and its ducts were also unremarkable.

Appropriate diagnostic workup thereby saved the patient from an unnecessary operation. Subsequent upper endoscopy revealed evidence of mild distal esophagitis and two superficial ulcers in the antrum of the stomach in the background of moderate gastritis and mild duodenitis. Our patient was placed on a trial of Proton Pump Inhibitors and the patient was subsequently discharged with plans for follow up with Gastroenterology.

Agenesis of the gallbladder is an extremely rare congenital anomaly and its combined presentation with a PMH of TOF is an even rarer occurrence. Lack of gallbladder visualization in these patients following initial ultrasound imaging should raise the index of suspicion for gallbladder agenesis; diagnosis should be confirmed with additional imaging. MRCP was selected in order to noninvasively visualize variations in the biliary tract system and search for a possible ectopic gallbladder.

A lack of awareness of this condition among surgical, gastroenterologic, and radiologic staff can lead to unnecessary operative intervention in patients with symptoms consistent with presumed gallstones. This case highlights the need of a greater appreciation for agenesis of gallbladder as a cause of biliary symptoms.

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Title: Skin popping and chest pain: An unusual case of Klebsiella oxytoca tricuspid endocarditis

Case Presentation

A 36-year-old woman with ongoing heroin abuse presented to our hospital with one day of fever, chest discomfort, and dyspnea. Initial examination was notable for fever to 101.4F, a soft systolic murmur at the right upper sternal border, and large ulcerations with erythema and scarring over her forearms bilaterally suggestive of chronic cellulitis. She acknowledged intravenous and subcutaneous drug injection (skin popping) at these sites. Peripheral stigmata of endocarditis were not present.

Within 12 hours of collection, blood cultures grew gram-negative rods ultimately speciating as ampicillin-resistant *Klebsiella oxytoca*. Echocardiogram revealed a 1 x 0.4cm shaggy vegetation on a prolapsing tricuspid valve with suggestion of leaflet perforation. No other organism was isolated in eight sets of cultures collected during hospitalization.

Ceftriaxone and ciprofloxacin were begun, and her fevers subsided after ten days. Surveillance cultures were negative, and repeat echocardiogram at two weeks showed diminution of the vegetation. However, she left against medical advice after approximately three weeks of treatment.

Discussion

Klebsiella species are a rare cause of endocarditis despite being the second most common cause of gram-negative bacteremia, perhaps reflecting these organisms' limited ability to colonize valve tissue. To our knowledge, this is the first reported case of tricuspid valve endocarditis in an intravenous drug user caused by *K. oxytoca*, and the seventh case of *K. oxytoca* endocarditis overall.

As in this case, previous reports of community-acquired *K. oxytoca* endocarditis describe a relatively acute presentation of fever and valve dysfunction; immunologic and vascular phenomena are consequently absent. The biliary, urinary, and gastrointestinal tracts are the most commonly identified sources for *K. oxytoca* bacteremia; acquisition through skin infection is rarer, but has been described in cases of drug use with non-sterile injection technique.

Earlier cases of *K. oxytoca* endocarditis have generally been treated successfully using combination therapy with cephalosporins and aminoglycosides or fluoroquinolones, without early surgical intervention; this approach appeared effective in our patient as well. The natural history and optimal management of endocarditis caused by *K. oxytoca* may therefore be distinct from that caused by *K. pneumoniae*, which characteristically causes malignant illness with metastatic disease and high mortality. Additional reports are needed to confirm this impression.

Conclusion

Although most often associated with *S. aureus*, native valve endocarditis in intravenous drug users can also be caused by enteric gram-negative organisms such as *K. oxytoca*, with distinct implications for prognosis and management.

Medical Student Clinical Vignette

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Title: Severe Warm Autoimmune Hemolytic Anemia in a Young Female Refractory to Multiple Evidence-Based Therapies: A Case Report

Introduction: Warm autoimmune hemolytic anemia is a rare but serious condition which often presents with nonspecific symptoms and is most frequently idiopathic. We present a case of a patient with rapidly progressive warm autoimmune hemolytic anemia who responded poorly to numerous literature-based standards of care.

Case presentation: A 25 year old female with no past medical history presented with a headache, generalized weakness, and fatigue for one month. Vital signs were within normal limits and physical exam was remarkable for conjunctival pallor with no hepatosplenomegaly or scleral icterus. CBC was significant for a severe anemia, with a hemoglobin of 5.4g/dL and marked leukocytosis. Her lactate dehydrogenase and erythrocyte sedimentation rate were found to be elevated and she had a decreased haptoglobin level. The patient's Direct Antiglobulin Test was positive for IgG and C3, demonstrating warm autoimmune hemolytic anemia. Upon presentation and throughout her hospital course, the decision to transfuse the patient was made with consideration to the patient's symptomatology as well as the concern for alloimmunization to foreign blood.

On hospital day one, she was started on high-dose corticosteroids as accepted first-line therapy for her hemolytic anemia. She also received one unit of packed red blood cells due to her severely low hemoglobin level. The patient's symptoms did not resolve, and on hospital day 9 she received rituximab as second-line therapy. On hospital day 10, her hemolytic anemia worsened and her clinical picture further deteriorated with symptoms of diaphoresis and lightheadedness at rest. On hospital day 12, she was urgently transfused and underwent an emergent splenectomy, which temporarily improved and stabilized her anemia. By hospital day 24, she was started on a third-line therapy of cyclophosphamide due to worsening hemolysis.

On hospital day 32, her hemoglobin reached a nadir of 2.3g/dL. As a temporary stabilizing measure, she received IVIG and her hemoglobin ultimately improved to 5.6g/dL. Her lightheadedness improved and she was able to tolerate minimal exertion, and by hospital day 37 she was considered stable for transfer to higher-level care.

Discussion: Warm autoimmune hemolytic anemia can be challenging to manage in a patient with rapidly progressive disease. As this case illustrates, patients may quickly deteriorate despite treatment with traditional first-, second- and third-line therapies. Despite limited evidence for their use temporizing measures such as IVIG may be life-saving in a patient with severe hemolysis and may stabilize a patient until immunomodulatory therapies exhibit a clinical response. This case demonstrates the current need for guidance in the setting of severe cases of warm autoimmune hemolytic anemia refractory to traditional therapies.

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Title: C. difficile Colitis, When in Doubt Time for a Colonoscopy

Introduction: Pseudomembranous colitis is an inflammatory condition of the colon often characterized by elevated yellow-white plaques that coalesce to form pseudomembranes on the colonic mucosa. There is a strong association between pseudomembranous colitis and C. difficile infection (CDI) requiring the initiation of stool testing and empiric antibiotic treatment whenever suspected. If tests are negative and symptoms persist despite empiric treatment, early gastroenterology consultation and colonoscopy is considered the next step.

Case: An 85-year-old female with a history of diabetes mellitus, hypertension, hyperlipidemia, dementia, and hypothyroidism was admitted due to lethargy and dyspnea after vomiting several times. One month prior to admission the patient completed a course of Vancomycin for Methicillin-resistant-Staph aureus osteomyelitis. At initial presentation the patient had one large loose bowel movement. Physical exam showed a soft and non-distended abdomen which was tender to palpation with hypoactive bowel sounds. Stool guaiac was positive and laboratory evaluation was significant for leukocytosis. Computed tomography showed small fluid in the colon but no findings consistent with colitis. The patient was started on Metronidazole with oral Vancomycin for possible CDI. Stool samples were collected for testing. The patient continued to be febrile, with no improvement in leukocytosis and had numerous daily loose bowel movements despite treatment. Test results for C. difficile were negative. In view of the high clinical suspicion for CDI Metronidazole and oral Vancomycin were continued. C. difficile testing was repeated, which was negative. Colonoscopy was performed to determine the cause of diarrhea which showed severe ileocolitis with skip areas consistent with inflammatory bowel disease (IBD). Tissue samples were obtained for biopsy. Histopathologic features of the biopsy specimen were in favor of pseudomembranous colitis. After approximately 3 weeks of treatment for C. difficile with Vancomycin and Metronidazole, the patient began to show signs of improvement.

Conclusion: The development of CDI is almost always associated with prior antibiotic therapy. The diagnosis should be suspected in all patients who present with diarrhea who were recently treated with antibiotics and then confirmed with a stool test for toxigenic C. difficile or colonoscopic or histopathologic findings demonstrating pseudomembranous colitis. Typical colonoscopy findings in CDI include bowel wall edema, erythema, friability and inflammation. The absence of pseudomembranes does not exclude CDI, however when pseudomembranes are visible, these findings are highly suggestive of a CDI. This case demonstrates a patient who had a classic clinical presentation with contradictory laboratory results, suggesting that with the rise in prevalence of C. difficile infection not only should there be high clinical suspicion for CDI, the utilization of other modalities such as colonoscopy, should also be considered.

Medical Student Clinical Vignette

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Title: Pulmonary Sarcomatoid Giant cell carcinoma with Paraneoplastic hypertrophic osteoarthropathy in a HIV positive patient: A case report

Introduction

Sarcomatoid carcinoma (SC) is a rare primary malignant tumor. Its incidence is estimated to account for 0.3-1.3 % of all lung malignancies. Statistically, pulmonary sarcomatoid carcinoma commonly present in males with a heavy smoking history, and an average age at diagnosis of 60 years.

Case presentation:

A 57 year old female with a past medical history of HIV on HAART (last CD4 count was 621 cell/µl) presented with complains of a two months history of productive cough with yellowish sputum containing streaks of blood. The patient also complained of a twelve pound weight loss, bilateral hand swelling, and knee pain with noticeable finger clubbing on physical examination. Chest CT revealed prominent slightly rounded consolidation of the inferior right upper lobe with adjacent ground glass and interstitial opacities. Bronchoscopy was performed and revealed a protruding endobronchial lesion almost completely obstructing the right upper lobe bronchus limiting any further visualization of the RUL segments. Initial pathologic analysis of specimens biopsied during bronchoscopy revealed non-small cell lung cancer (NSCLC). A definitive diagnosis was established by way of a RUL lobectomy. Pathologic analysis of the resected specimen showed sarcomatoid giant cell carcinoma, tumor size 9.5 cm with invasion of the visceral pleura and 1/13 hilar lymph node involvement. The pathological stage was pT3N1Mx based on the tumor node metastasis (TNM) staging system.

Discussion

This case highlights a possible paraneoplastic syndrome associated with pulmonary SC, and the impact of HIV on carcinogenesis and prognosis. Hypertrophic osteoarthropathy is a paraneoplastic syndrome consisting of a triad of symptoms including clubbed fingers, symmetric polyarthritis, and periostitis of the long tubular bones. The above patient presented with signs and symptoms suggestive of this paraneoplastic syndrome which has not been reported to be associated with this cancer in the literature.

HIV/AIDS substantially increases the risk of certain malignancies including Kaposi sarcoma and non-Hodgkin's lymphoma classified as AIDS-defining cancers (ADC). However, in the post-HAART era the non-ADCs including anal cancer, lung cancer, liver cancer and head/neck cancer constitutes an increasing majority. Thus the role of HIV and HAART therapy in pulmonary carcinogenesis needs to be examined.

There are currently no standard effective chemotherapy but traditionally platinum based chemotherapy is used if the patient is not a surgical candidate or if adjuvant chemotherapy is indicated. An important factor in prognosis was found to be tumor size and presence or absence of metastases. The prognosis is poorer in HIV-infected patients than in the general lung cancer population as a substantive number of patients have died despite non-detectable HIV viral loads and well preserved immunity measured by CD4 cell counts.

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Title: RECURRENT OF PAPILLARY THYROID CARCINOMA AS ANAPLASTIC CARCINOMA: A RARE CASE

Introduction:

Papillary thyroid carcinoma (PTC) is the most common form of well-differentiated thyroid cancer and typically has a good prognosis. Anaplastic thyroid cancer (ATC) comprises only 2% of all thyroid cancers, yet is responsible for 40% of thyroid cancer related deaths. It has been reported that recurrent PTC presents more aggressively than its initial presentation. Furthermore, it is not well understood which well-differentiated thyroid cancers will transform to ATC. Effective treatment strategies can be difficult due to its aggressive nature, and propensity to present with advanced disease. In our report, we present a unique case of a patient who was diagnosed with stage IVA PTC, treated with total thyroidectomy and radioactive iodine ablation, with subsequent local recurrence and transformation to ATC.

Case:

A 65-year-old female with PMH of hypertension and anxiety initially presented in December 2014 with a neck mass and dysphagia. She underwent fine needle aspiration of the right thyroid, and subsequent total thyroidectomy. Pathology reports indicated the mass was papillary thyroid carcinoma (PTC), with local and central neck lymph nodes positive for a pT3(m)N1bM0, overall Stage IVA disease. She was subsequently treated with radioactive iodine ablation therapy. Approximately one year later, she presented with a right-sided neck mass and dysphagia. Whole body imaging with Iodine-131 and PET/CT demonstrated a 17x10 mm solitary metabolically active mass in the right thyroid fossa that was not radiiodine avid. Serum thyroglobulin was elevated (75.6 ng/mL). The patient underwent right neck dissection with 3 of 22 lymph nodes positive for metastatic PTC. MRI of the neck six months later revealed a complex 3 cm space-occupying lesion in the thyroid bed. Mass effect was present. A repeat PET/CT revealed a hypermetabolic right neck mass in the region of the previously resected mass, measuring 3.6x3.4 cm. Repeat biopsy of the new lesion was performed consistent with anaplastic/undifferentiated thyroid carcinoma, negative for TTF-1 and thyroglobulin. The patient was then started on a new chemo-radiation regimen, which has since been completed.

Discussion:

Anaplastic thyroid carcinoma (ATC) is an undifferentiated, aggressive tumor, with a median survival of only 6 months. Due to this aggressive nature, all ATC are considered Stage IV. We present a case of a patient who presented with PTC that was treated with surgery and radioactive iodine ablation, with transformation to a much worse prognosis with local recurrence of ATC one year later. It is not well understood which well-differentiated thyroid carcinomas will recur as more aggressive, poorly differentiated tumors. Approximately 20% of patients with ATC had a history of a well-differentiated thyroid carcinoma. Thus, physicians should be aware of the potential for PTC to transform to ATC, even after aggressive treatment measures have been taken.

Medical Student Clinical Vignette

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Title: REACTIVATION OF PANCREATIC TUBERCULOSIS IN A MAN UNDERGOING CHRONIC COLCHICINE THERAPY FOR ACUTE PERICARDITIS

A 69-year-old male with type 2 diabetes mellitus, prior smoking history, and a 3-month history of acute pericarditis undergoing treatment with colchicine presented to our hospital medicine service with 2 months progressive right-sided chest pain and dyspnea accompanied more recently by epigastric discomfort. He denied other constitutional symptoms, cough, nausea, vomiting, melena, or hematochezia. Workup revealed elevated inflammatory markers and a mixed anemia of chronic inflammation and bone marrow suppression. The patient's symptoms were attributed to his pericarditis, colchicine was discontinued, and a moderate dose prednisone taper was initiated. Despite improvement in his presenting chest pain and dyspnea, the patient continued to report poor feeding and abdominal discomfort. CT revealed a large, cystic mass in the pancreatic head. Endoscopic ultrasound was performed with biopsies obtained from the mass and an adjacent lymph node, and the patient was discharged with outpatient follow-up. Pathology of the mass revealed chronic inflammation, and the lymph node revealed necrotizing granulomas with negative acid-fast bacteria (AFB), fungal, and Gram staining.

The patient was re-admitted two weeks later with fever, rigors, tachycardia, hypertension, 12 pounds weight loss, and delirium. Repeat CT revealed rapid enlargement of the pancreatic mass. The patient initially defervesced on broad-spectrum antibiotics, but he experienced frequent breakthrough fevers with delirium. Further history revealed a brief jail term and military service during the Vietnam era. CT-guided biopsy of a second lymph node again revealed necrotizing granulomas with negative organism staining. Laboratory testing for sarcoidosis, autoimmune pancreatitis, and pancreatic cancer were negative. Ultimately, the mass was aspirated under CT guidance. Gram stain showed gram-positive cocci in pairs, and AFB stain was positive. AFB cultures subsequently grew *Mycobacterium tuberculosis*. HIV serology was nonreactive and chest X-ray revealed clear lung fields.

Isolated pancreatic tuberculosis is a rare presentation of tuberculosis almost exclusively reported in endemic countries. Our patient likely experienced primary infection during his jail term or military service in Vietnam. While anti-tumor necrosis factor agents are associated with tuberculosis reactivation, to our knowledge, this case represents the first report of tuberculosis reactivation on colchicine. Interestingly, this patient's disease acutely worsened when prednisone therapy was initiated. Fortunately, pancreatic tuberculosis responds well to standard anti-tuberculosis regimens. This patient responded to anti-tuberculosis therapy, and he continues to do well with follow-up by the county public health department.

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Title: A Case of Evans Syndrome: Determining Primary vs. Secondary Disease

Introduction:

Evans syndrome is a rare autoimmune disease involving simultaneous or sequential development of autoimmune hemolytic anemia (AIHA) and immune thrombocytopenia (ITP), characterized by frequent exacerbations and remissions. It was first described in 1951 and has a reported prevalence of 0.8%-3.7% in patients with either AIHA or ITP at onset. We report a case of Evans syndrome associated with lymphocytosis of CD8+ T-cells.

Case Presentation:

A 71 year-old male with immune thrombocytopenia of ten years was initially treated with corticosteroids, IVIG and ultimately went into remission after treatment with rituximab. Three years ago he developed severe autoimmune hemolytic anemia, resistant to corticosteroids. He underwent a splenectomy and again received rituximab, achieving a second remission. Last year, he relapsed with autoimmune hemolytic anemia and was treated again with rituximab resulting in remission. Currently he presents as an outpatient with no complaints. Physical exam showed no cyanosis or lymphadenopathy. Laboratory results revealed hemoglobin 12.5g/dL, platelets 355,109/L, total WBC 17x10⁹/L with a differential of 28% granulocytes and 66.3% lymphocytes. He has had persistent lymphocytosis and flow cytometry showed decreased CD4/CD8 ratio (0.2) due to increased CD8+ T-cells which were not immunophenotypically aberrant. T-cell gene rearrangement was negative. Bone marrow biopsy revealed an increase in cytotoxic T-cells and a T-cell gene rearrangement assay with an oligoclonal pattern. Serologies and CT scans failed to reveal an autoimmune disorder or lymphoproliferative disorder.

Discussion:

Evans syndrome was initially thought to be an incidental finding. There is increasing evidence suggesting that Evans syndrome reflects a state of profound immune dysregulation as opposed to a coincidental combination of immune cytopenias. Retrospective review has determined that up to 50% of cases are associated with other disorders. Therefore, Evans syndrome should be classified either as primary (idiopathic) or secondary (associated with an underlying disease). In a recent analysis of sixty-eight cases of Evans syndrome, thirty-four were determined to be secondary, with lymphoproliferative disorders represented by ten. Systemic lupus is most commonly associated with the syndrome in younger patients, while Non-Hodgkin's lymphoma (NHL) is most commonly associated in patients over age fifty. Despite an exhaustive evaluation, our patient is best classified as idiopathic Evans syndrome. However, the presence of an oligoclonal T-cell gene rearrangement pattern demands continued close follow up to diagnose an evolving lymphoproliferative disorder.

Conclusion:

Evans syndrome is a rare autoimmune disorder and an important consideration for physicians when AIHA or ITP is present. Furthermore, there is a high degree of association with other autoimmune diseases as well as lymphoproliferative disorders. Given the low incidence of Evans syndrome, our understanding of the disorder remains observation-based. This case demonstrates that Evans syndrome warrants lifelong follow-up to monitor for development of lymphoproliferative disease, especially in the elderly.

Medical Student Clinical Vignette

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Title: AN UNCOMMON ETIOLOGY OF ASCITES

Introduction:

Pancreatic ascites is seen in 1% of all cases of ascites.

Etiologies include pancreatic duct disruption and leaking pseudocyst, where patients may present with positive fluid wave and shifting dullness. Pancreatic ascites is often confused with spontaneous bacterial peritonitis (SBP) or portal hypertension. In our report, we present a patient with history of chronic pancreatitis in the setting of pancreatic ascites.

Case:

A 64-year-old female, with PMH of pancreatitis, COPD, large ascites s/p paracentesis, chronic alcohol and tobacco abuse, presented for abdominal pain, increasing abdominal girth, and non-bilious vomiting over the past 3-5 days. Patient described the pain as dull and worsening to 7/10 upon movement. Patient denied fever, chills, shortness of breath, and chest pain. Patient's temperature was 98.6F, pulse 88 beats/min, BP 96/56, and 18 breath/min. Upon physical exam, patient appeared cachectic with distended abdomen, shifting dullness, and diffuse abdominal tenderness. Ascitic fluid revealed absolute PMN of 238 cells/mm³, amylase 2755 IU/L, total protein 3.2, and negative culture results. Liver panel, PT/INR, and platelets were normal. Abdominal CT was consistent with multiple small pancreatic pseudocyst, cirrhotic liver and peripancreatic stranding. Patient was kept NPO and treated with IV fluids, while being referred to tertiary care center for endoscopic retrograde cholangiopancreatography (ERCP). ERCP demonstrated fresh blood flowing from the ampulla and contrast was noted to extravasate at the proximal body of the pancreas. Patient was diagnosed with acute hemorrhagic pancreatitis with ductal disruption, and was treated with pancreatic sphincterotomy and stent placement.

Discussion: Pancreatic ascites comprises 1% of all ascites cases. Etiologies include: disruption of main pancreatic duct, rupture of a pseudocyst, pancreatic trauma, or after an acute pancreatitis episode. In patients with history of alcohol abuse, chronic pancreatitis is often the cause, although symptoms of pancreatitis may not be present. Thus, these patients are likely to be misdiagnosed with portal hypertension and liver cirrhosis. Diagnostic criteria for pancreatic ascites includes: ascitic fluid amylase >1000 IU/L, total protein >3, and SAAG <1.1 g/dL. Paracentesis can exclude SBP by absolute PMN count and culture. CT of the abdomen is important to evaluate the presence of pancreatitis or a pseudocyst, while ERCP is useful to visualize ductal disruption and placing a stent to prevent leakages. Although diffuse abdominal tenderness and history of chronic alcohol abuse is typically seen in SBP secondary to liver cirrhosis, we illustrate an uncommon presentation of pancreatic ascites. In addition to its rarity, the symptoms of pancreatic disease were absent, making ascites due to SBP or portal hypertension appear to be more likely. Thus, clinicians should include pancreatic ascites as a differential when GI symptoms are otherwise unexplainable.

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Title: DIAGNOSTIC DILEMMA: ATYPICAL VASCULITIS-LIKE PRESENTATION OF PULMONARY ADENOCARCINOMA

INTRODUCTION: Adenocarcinoma is the most common type of non-small cell lung cancer with histopathological variants of presentation. Biopsy determines cell type and variant of adenocarcinoma. Common paraneoplastic syndromes of adenocarcinoma include hypercoagulability, thrombophlebitis, and Libman-Sacks endocarditis. Our report reveals an unusual association of adenocarcinoma with positive anti-neutrophil cytoplasmic antibody (ANCA) titers and focal necrosis.

CASE: A 69 year old male presented with non-productive cough for two weeks. He denied chest pain or shortness of breath. Past medical history included hypertension, prostate cancer and pacemaker. Patient reported a 30 pack year smoking history, but quit 15 years ago. Physical examination revealed equal airway entry into lungs bilaterally, no crackles or wheezing, and no palpable cervical or supraclavicular lymphadenopathy. A chest x-ray revealed a 6 cm mass in the left upper lobe of the lung periphery. Positron emission tomography (PET) scan showed hypermetabolic opacity in posterior segment of upper lobe and in left hilar lymph node, highly suggestive of primary pulmonary malignancy. Mediastinoscopy revealed large pre-tracheal nodes, negative for malignancy. Bronchoalveolar lavage was significant for nuclear atypia and multinucleated giant cells. Subsequently, a core needle biopsy reported dense fibrosis and focal necrosis with no definitive malignancy. Repeat lavage and biopsy 2 months later found no malignancy without change in clinical picture. An ANCA test was positive. Patient was sent for rheumatology workup due to positive ANCA and giant cells. Several months later, follow-up non-contrast computed tomography (CT) revealed features of bronchogenic carcinoma. Lobectomy and lymphotomy showed poorly differentiated adenocarcinoma of the lung, Stage Ib (T2b N0 M0) and all 3 lymph nodes without malignancy. Patient recovered well from surgery and management with chemotherapy was started with oncologist.

DISCUSSION: Clinical suspicion leaning towards primary lung malignancy with laboratory results referring to a secondary cause leads to delayed diagnosis and treatment. In our diagnostic dilemma where lab values contradicted the clinical picture, the more common diagnosis proved to be correct. Researchers have identified the presence of vasculitis markers in patients with solid tumor malignancy and theorize the tumor produces antigens resulting in antibodies which can cross react with ANCA testing. Accurate guidance and depth of biopsy must also be considered, as going too deep past the mass margin into the center increases false negative results. Our case report highlights an atypical presentation of adenocarcinoma with focal necrosis and elevated ANCA levels as both are uncharacteristic findings. Adenocarcinoma should not be excluded despite positive ANCA results when clinical suspicion for primary pulmonary mass is present.

Medical Student Clinical Vignette

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Title: Skull Base Hyperdense Lesion- A Diagnostic Challenge

Introduction: Neurenteric cysts are congenital cysts, that arise due to errors in notochord development. These cysts can present with focal or non-specific signs of neurological pathology. Neurenteric cysts can present variably on noncontrast CT depending on the intrinsic intracystic mucin content, and may be mistaken for intracranial hemorrhage in the emergency setting, thereby affecting patient management. Report: A young 36-year-old Hispanic female G9 P9 L9 presented to the emergency department with dizziness, left sided weakness and blurry vision. She progressed to near syncope and mild left sided ataxia. Physical Examination revealed mild left sided motor weakness (muscle power grade 3/5). Initial non-contrast head CT demonstrated an infratentorial, hyperdense, oblong extra-axial lesion ventral to the brainstem, causing mild mass effect. There was no hydrocephalus, midline shift or evidence of suspicious osseous abnormality. MRI of the brain was performed and demonstrated this lesion to be hyperintense on T1 and hypointense on T2 and FLAIR series, without abnormal diffusion restriction. There was no demonstrable enhancement on postcontrast T1 series. Susceptibility weighted series failed to demonstrate blooming or evidence of acute or chronic blood breakdown products. Subsequently, intracranial hemorrhage and epidermoid cysts were excluded as differentials. In the absence of abnormal enhancement or abnormal calvarial marrow signal, alternative differentials including cystic or metastatic neoplastic processes were also excluded. The patient was managed conservatively and showed resolution of mild left sided weakness on two subsequent six monthly examinations. Conclusion: We present a teaching case of a skull base atypical neurenteric cyst for emergency physicians, residents and medical students, to emphasize the importance of location based differential diagnosis, when encountering multitude of presentations in the busy emergency setting. Although only 10% of the neurenteric cysts demonstrate low T2/FLAIR imaging signal and most of them occur in the spine rather than in the brain (3:1), given the morphology and location at imaging, the primary differential diagnosis entertained was neurenteric cyst. However, alternative differential diagnoses included hemorrhage given the acute clinical presentation of left-sided weakness, as well as other pathologies such as endodermal cysts like Rathke's cleft cyst/colloid cysts, ecchordosis physaliphora, epidermoid cyst, melanotic metastatic disease is, infectious neurocysticercosis, amongst others.

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Title: Cardiac Myxedema: A Case Report

Myxedema effusion resulting in cardiac tamponade is an uncommon, but serious, complication in patients with severe hypothyroidism. We present a case of severe hypothyroidism presenting with tamponade physiology. A 68 year-old female with hypothyroidism was brought in for evaluation of altered mentation and dyspnea. She reportedly had hallucinations and subtle changes in mentation for almost 12 months prior. She also endorsed fatigue, cold intolerance, weight gain, and constipation. Her only medication was Synthroid 88 mcg daily, which she did not adhere to. She previously smoked, denied alcohol use, was unemployed, resided with family, and performed daily activities with minimal assistance. In the emergency department, she was in mild respiratory distress. Her SO₂ was 95% on 2 L O₂ via nasal cannula, temperature was 98.8°F, respiratory rate was 24 breaths/minute, heart rate fluctuated between 60-80 bpm, and pressure was 141/67 mm Hg. Examination revealed an elderly female appearing older than stated age with generalized facial swelling, thin eyebrows, and macroglossia. She was lethargic, but able to answer simple questions. She had decreased intensity of S1 and S2 and no appreciable murmurs. Her skin was dry and reflexes were low in amplitude. CBC and BMP were normal. Troponin was 0.085 ng/mL, CKMB was 11.1 ng/mL, and pro-BNP was 564 pg/mL. TSH was 58.5 micro IU/mL and free T4 was 0.19 ng/dL. Thyroglobulin was > 1000 Intl Units/mL and TPO was 444 Intl Units/mL. ECG showed accelerated junctional rhythm, fusion complexes, electrical alternans and low voltage precordial and limb leads. Chest x-ray revealed left mild atelectasis and a small effusion. CT of the head was unremarkable. A chest CT showed small bilateral pleural effusions and a moderate pericardial effusion. Echocardiogram revealed LVEF of 70%, a large pericardial effusion, and collapse of both atria and right ventricle. Hypothyroidism complicated by cardiac myxedema and metabolic encephalopathy was diagnosed and she was restarted on Synthroid. Pericardiocentesis was performed and 1.3 liters of serous fluid were removed. Atypical mesothelial cells and lymphocytes were isolated, but bacterial cultures and gram stains were negative. She had dramatic clinical improvement and was discharged. During her six week follow up, TSH was 4.170 micro IU/mL, free T4 was 1.51 ng/dL, her mental status had returned to baseline, and a repeat echocardiogram showed resolved effusion. Thyroid hormone has a significant effect on cardiac myocytes and pericardial effusion has been associated with hypothyroidism. Although rarely documented in literature, cardiac tamponade secondary to myxedema should be considered when patients present with clinical, laboratory, and imaging features of tamponade in the setting of severe hypothyroidism.

Medical Student Clinical Vignette

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Title: The Case of the Seizure-less Cysticercosis

An organism that lives within another organism and benefits at the host's expense is defined as a parasite.

Neurocysticercosis refers to infection of the central nervous system with a specific parasite, *Taenia Solium*, a tapeworm that is found in pigs. The most common presentation of this disease is epilepsy (70% of cases). It is currently endemic in Central and South America, Asia, and sub-saharan Africa, and estimated to affect 50 million people worldwide. In the United States, a higher prevalence of infection is seen in the Hispanic immigrant population. We present a case of neurocysticercosis with a complete absence of epilepsy or any seizure activity.

A 39-year-old Indian female, with a past medical history of vitamin B12 deficiency, presented to her primary medical doctor with complaint of severe headache. 2 days prior to this visit, the patient had visited the emergency department with the same complaint and was discharged home with Tylenol. At the visit, the patient also endorsed a sudden inability to use the cash register at her workplace, where she works as a cashier. Her husband informed the doctor that his wife was acting confused and not speaking much, although her basic speech was intact. She denied any fever, seizures, visual disturbances, paresthesias, and dysarthria. Blood work was negative. It is notable that 6 months prior to her initial visit, she had spent 2 months in India, giving suspicion to a possibility of infection due to poor sanitation and consumption of meat products that are not adequately prepared.

The patient was sent to get a computed tomography (CT) scan of the brain, which showed a lesion. Subsequently, a magnetic resonance imaging (MRI) was done, showing that the patient had a lesion indicative of parasitic infection in the form of cysticercosis in the left temporal occipital region along with significant surrounding edema. EEG (electroencephalogram) showed no findings. IgG Serology to *T. Solium* was found to be positive. Due to these findings, the patient was started on treatment with 8 days of albendazole and 10 days of dexamethasone. A MRI 2 weeks later showed significant reduction in swelling and the lesion became smaller and less enhancing. A one-year follow up showed no lesions and resolved edema.

Neurocysticercosis is becoming increasingly prevalent in North America, and the most common avertable cause of epilepsy in the developing world, and thus an important diagnosis to keep in mind to avoid the abysmal outcomes of this disease. This case highlights the importance of having a thorough differential diagnosis, which considers the possibility of something beyond what we see clinically day to day.

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Institution: South Nassau Community Hospital

Title: A RARE CASE OF PAPILLARY THYROID CARCINOMA WITHIN A BRANCHIAL CYST: A CASE REPORT

A 31-year-old male with past medical history of medication-controlled hypothyroidism, autism, and seizure disorder presented with a complaint of recurrent left-sided cervical swelling, which he started noticing one year ago. Nine months prior to admission, CAT scan of the neck showed a cystic lesion measuring 6.9 x 6.7 x 4.9 cm on the left cervical region. This cystic lesion was drained twice, but still recurred. He had no pertinent surgical history. He denied both a family history of thyroid cancer and prior exposure to head and neck radiation. Clinical evaluation demonstrated a large cystic mass in the left lateral neck with no lymphadenopathy. Office laryngoscopy showed bilateral mobile cords.

Fine needle aspiration (FNA) of the lesion was consistent with cystic contents and showed no evidence of malignancy. Based on clinical and radiological findings and reports from FNA, we believed that this patient had a left branchial cleft cyst. Patient underwent a branchial cystectomy; the excised mass grossly measured 7.0 x 5.2 x 1.5 cm, contained 60 mL of dark brown fluid, and occupied levels 2, 3, 4, and 5 of the neck. Pathology revealed a 1 cm tumor positive for malignant cells consistent with papillary thyroid carcinoma that did not extend beyond the cyst wall. Two adjacent lymph nodes were negative for malignancy. Follow up care will entail physical exams and the use of serial ultrasounds every three to six months for the first two years to monitor the status of the thyroid gland. If new or recurrent cancer is suspected within the gland, the patient will be advised to undergo thyroidectomy.

This case illustrates the unusual and extremely rare occurrence of papillary thyroid cancer (PTC) in ectopic thyroid tissue within a branchial cleft cyst (BCC). There have been only five prior documented cases worldwide. This stems from the fetal development of the associated structures. The branchial arches and pouches overlap with the descent of the thyroid gland, and during this developmental stage, ectopic thyroid tissue can be abnormally taken up by branchial arches or pouches. When a branchial arch fails to obliterate, it commonly forms a BCC. In rare cases, ectopic thyroid tissue can be incorporated into the branchial cleft cyst wall while retaining its malignancy potential. PTC accounts for 80% of all thyroid malignancies and although well-differentiated, it has the potential to invade lymphatics and metastasize, making its detection and monitoring essential. Traditionally, PTC is diagnosed with FNAB; however with this patient, FNAB was negative for malignancy because the PTC was small and adherent to the cystic wall. In this case, the PTC within the BCC was discovered only after a complete excision and analysis of the cyst.

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Title: A NOVEL CAUSE OF SPONTANEOUS SEPTIC ABORTION IN AN IVF PREGNANCY

Introduction:

Septic abortion is defined as an infection of the placenta, with the possibility of spreading to the uterus where bacteria can gain access into the maternal intervillous space or become systemic, causing septicemia. It usually occurs in the setting of predisposing risk factors like intrauterine tissue damage from retained products of conception after spontaneous or induced abortion, an unsafe abortion involving instruments or chemicals, or a pregnancy with an intrauterine device in place. The usual bacteria causing intrauterine fetal death are *Peptostreptococcus*, and toxin-producing strains of *Clostridium perfringens*, *Staphylococcus aureus*, and *Escherichia coli*. We present the first case of septic abortion at 16 weeks in an IVF pregnancy due to *Streptococcus bovis* and *Acinetobacter baumannii*.

Case:

A 25 year-old G2P0010 presented at 15 weeks 6 days gestation referred by Maternal Fetal Medicine for absent fetal heart tones on ultrasound. Pregnancy was through In Vitro fertilization. Her previous pregnancy resulted in a spontaneous abortion early in the first trimester. Patient complained of abdominal cramps, dizziness, and vaginal bleeding. Throughout her current pregnancy she took oral micronized progesterone.

Admission vital signs revealed a temperature of 39.2°C; C, pulse of 150 bpm, blood pressure 79/49 mm Hg, respiration rate 22 bpm, and oxygen saturation 99%. Ultrasound exhibited a singleton fetus in the vertex position, but an absence of fetal cardiac activity, or somatic movements. The remainder of the physical examination was benign. Intravenous fluids, ampicillin, and gentamicin were started.

Approximately 3 hours after admission, following induction with misoprostol and oxytocin, the patient delivered a 100g male stillborn fetus vaginally. The placenta was intact and delivered spontaneously. Placental culture was taken. Both fetus and placenta were sent to pathology.

Initial maternal blood culture results revealed *Streptococcus bovis*. The urine culture was contaminated. Placental cultures from both maternal and fetal sides grew *S. bovis* and multi-drug resistant *Acinetobacter baumannii*. Pathology diagnosis was second trimester placenta with extensive acute villitis, chorioamnionitis, and focal funisitis (i.e., inflammation of Wharton's jelly). Based on sensitivities of *A. baumannii*, meropenem was started, ampicillin was discontinued, and gentamicin continued. On hospital day 11, the patient was discharged with a peripherally inserted central catheter to continue 6 weeks of meropenem.

Discussion: This pregnant patient presented with sepsis secondary to endometritis due to multi-drug resistant *A. baumannii* and *S. bovis* along with bacteremia secondary to *S. bovis*. This case highlights two bacteria that very rarely cause septic abortion (zero previous cases of co-infection), especially with a lack of identifiable risk factors. Such cases require immediate initiation of broad-spectrum antibiotics, fluid resuscitation, and prompt removal of retained products of conception as early treatment. In order to properly identify and manage sepsis in pregnancy, it is critical to consider these two pathogens as possible causative agents.

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Title: First Case of Brucella ovis in Human

INTRODUCTION: Brucellosis is a zoonotic infection that was first identified in 1887. Several species have been identified. *Brucella ovis* (*B. ovis*) is known to infect sheep, with prevalence in endemic areas estimated to be as high as 43.7% [1]. *B. ovis* has been associated with sheep epididymitis and orchitis [2]. Brucellosis may be identified in humans through direct culture of blood, cerebral spinal fluid (CSF), bone marrow and wounds [3], or through serologic assays. To date, no human infection with *B. ovis* has been identified [3, 4]. Here, we present the first case of *B. ovis* infection documented in a human.

CASE REPORT: Our patient is a 42 year old male with past medical history of recurrent sinus infections, who presented to our hospital with 1 day of dizziness with palpitations. He was found to have second-degree heart block, and was presumptively diagnosed with Lyme carditis based on ELISA and Western blot showing several reactive bands. He completed 21 day treatment with ceftriaxone followed by doxycycline. 2 months later, the patient developed worsening fatigue, malaise and diplopia. He underwent a lumbar puncture with CSF analysis demonstrating 56 mg/dL glucose, 24 mg/dL protein 5 red blood cells/µL, and 4 white blood cells/µL. Culture grew *Brucella* species, and was sent to the NYS lab for confirmation, where real-time PCR assays suggested *B. ovis*. The isolate was sent to the Centers for Disease Control (CDC), where conventional PCR was performed, and *B. ovis* was detected. He was subsequently started on combination antimicrobial therapy with ceftriaxone, doxycycline, and rifampin.

DISCUSSION: The reason that *B. ovis* has not previously been associated with disease in humans remains unclear. Genomic analysis has identified an increase in unstable elements including pseudogenes and transposable elements when compared to the known zoonotic *Brucella* species. In addition, several important segments required for essential functions are absent, including lipopolysaccharide biosynthesis, urease function and uptake of nutrients [5]. *Brucella* may be transmitted through environmental, occupational and foodborne exposure, and human-to-human transmission is rare [3]. The role of blood-sucking insects as vectors has been hypothesized [6]. It remains unknown how the patient we present was exposed to *B. ovis*. In addition, as genomic sequencing is a relatively new technique, it may also be possible that *B. ovis* has previously infected humans but was mistaken for another *Brucella* species. Additional study using genomic sequencing techniques may be needed to identify more human cases, characterize their features, and evaluate the response of *B. ovis* to antibiotic treatment.

**New York Chapter ACP
Annual Scientific Meeting**

Medical Student Research

<p>Author: Raynata Ramkhelawan, MS3 Additional Authors: Salam Hawa, Ravneet Randhawa, Niket Sonpal MD Institution: Kingsbrook Jewish Medical Center</p> <p>Title: An Expensive White Coat</p> <p>An Expensive White Coat Introduction: A dream is a mere goal sometimes cut short by reality; this is one realization made by many medical students striving towards their career. As the years pass and the financial burden grows, the reason of dreaming of a medical degree, whether an innate passion, parental demand, or simply a change in career path, is no longer the only significant factor in making career decisions. The purpose of this survey is to collect data to investigate the level of impact of how accumulated debt imposes on the choice of specialty by medical students and residents. Studies have shown a trending decline in primary care with a simultaneous inclination towards specialty medicine.</p> <p>Methods: The survey consisted of 26 questions administered via Google forms and made accessible for 20 days. The questions were designed to acquire the demographics of each student pertaining to the accumulated loans, level of financial understanding, and furthermore how accumulated debt affects their medical specialty choice.</p> <p>Results: There were 118 responses collected; of those some responses were excluded due to unspecified answers, thus percentages given account for such exclusions. Only 117 responded regarding total accumulated debt with 30.09% having an accumulated debt of less than \$60,000, 34.95% have between \$60,001 to \$140,000, 22.33% have between \$140,001 to \$220,000, and 12.6% have above \$220,001. When asked if loan amounts were sufficient to cover their obligations, there were 116 responses. Of these responses, 60.22% stated their loans were sufficient and 39.77% stated they were not able to meet their financial obligations with their loans. On questioning, if accumulated debt provoked a change in the choice of specialty, 117 responses were collected with 52% stating their choice did not change and 48% stating it did.</p> <p>Conclusion: Upon review, most students fall within the debt range of \$60,001 to \$140,000 and the majority of students stated loans sufficiently covered their needs and furthermore indicated debt was not a factor in choice of field. However, there is a substantial 39.77% of students who counteract this notion and a greater 48% who indicated their interest in pursuing a high paying specialty serves as a means to pay their accumulated debt. Further comparisons in the changing rates of medical school costs versus compensation rates for physicians will be essential in defining the root of the problem.</p>	
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**New York Chapter ACP
Annual Scientific Meeting**

**Resident/Fellow
Clinical Vignette**

Resident / Fellow Clinical Vignette

Author: Obed Adarkwah, M.D.

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Institution: Wyckoff Heights Medical Center

Title: An uncommon presentation of supraventricular tachycardia in severe hypothyroidism

Introduction

Hypothyroidism typically presents with fatigue, cold intolerance, constipation, weight gain, and a slow heart rate. An increased heart rate as seen with supraventricular tachycardia is typically found in patients with hyperthyroidism. Here, we present an unusual case of severe hypothyroidism presenting with supraventricular tachycardia.

Case presentation

A 65 year old male presented to the emergency department complaining of cough, dyspnea, and palpitations. These symptoms persisted intermittently for years; however, he noticed the symptoms worsening over the past few weeks. He had a past medical history of hypothyroidism and was noncompliant with levothyroxine. On physical examination, his vitals were stable. He showed 2+ right pedal pitting edema, with the rest of physical examination unremarkable. The patient exhibited runs of supraventricular tachycardia (SVT) when speaking that disappeared at rest. His EKG showed left bundle branch block. Laboratory values showed a normal WBC count, TSH of 113.139 UIU/mL, free T3 of 1.29 ng/dL and free T4 of 0.5 ng/dL, D-dimer of 2293.97 ng/mL, and BNP of 1594.79 pg/mL. A CT angiogram of chest was negative for pulmonary embolism, but an infectious process with bilateral atelectasis noted. The patient received ceftriaxone for community-acquired pneumonia.

Endocrinology was consulted, and they recommended IV steroids and titrated doses of levothyroxine by mouth due to increased heart rate. Over the course of treatment, the patient had intermittent episodes of tachycardia. An echocardiogram demonstrated an ejection fraction (EF) <20% with marked changes in the left ventricle with severe mitral regurgitation. Cardiology was consulted, and the patient was also started on furosemide, metoprolol and aspirin. Cardiac catheterization showed nonobstructive CAD with severe cardiomyopathy. Medical therapy was continued with optimization of heart failure therapy. The patient followed up with resolution of hypothyroidism and was placed on Bi-ventricular ICD with current follow up in the clinic.

Discussion Hypothyroidism typically presents with slowed heart rate. Instead of presenting with the typical symptoms of hypothyroidism, the patient presented with symptoms of hyperthyroidism. Secondary causes of tachycardia should be ruled out in patients with severe hypothyroidism that present with tachycardia. This patient had a very low EF, which predisposes the patient to arrhythmias. In addition, aggressive treatment with levothyroxine can precipitate tachycardia and arrhythmias, which presents obstacles in treating hypothyroidism. Our case emphasizes the importance of treating hypothyroidism cautiously in scenarios where patients are predisposed to arrhythmias. With significant elevations of TSH, it is recommended to start intravenous steroids with oral levothyroxine while monitoring T4 very closely.

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Title: A rare presentation of bilateral anterior cerebral artery infarction in a healthy young male

Introduction

Concurrent bilateral anterior cerebral artery infarction is a rare presentation in terms of neurological diseases. It is usually a result of ruptured anterior communicating artery aneurysm. Here we present a case of bilateral anterior cerebellar artery infarction caused by anterior communicating artery aneurysm.

Case Presentation

A 35 year-old male presented to the emergency department with altered mental status and fever. A week prior to hospitalization, he had been feeling fatigued with no appetite. He had no significant past medical history except for chronic alcohol abuse which resulted in multiple prior visits to the ED for alcohol intoxication or fights. However, all CT head findings were unremarkable on those visits. On physical examination, he was conscious, febrile (102.3°F), with no pallor. He was unresponsive to verbal commands, however, made minimal reactions when his name was called and responded to tactile and noxious stimuli. He had normal tone and +2 reflexes in upper and lower limbs, with Babinski positive. Pupils were equal and reactive to light. Given the patient's altered mental status and fever, a lumbar puncture was performed to rule out possible meningitis. The results of the lumbar puncture showed 5700/mm³ RBCs and positive for xanthochromia. Initial head CT was unremarkable. Urine toxicology was unremarkable and alcohol level <10mg/dL. All other laboratory workup was unremarkable except an increased trend of CPK. He was admitted to the medical ICU initially with meningitis and rhabdomyolysis. Neurology was consulted and initial differential diagnoses were status epilepticus vs meningitis. A bedside EEG was performed and status epilepticus was ruled out. He made eye contact but was non-verbal and psychogenic disorder such as elective mutism was considered. Since patient initially had xanthochromia on lumbar puncture a CTA of brain was performed which showed bilateral anterior cerebral occlusive stroke with extravasations. Neurology reevaluation suggested that there was a high likelihood of anterior communicating aneurysm present, which explains the ACA infarcts as well as fever due to subarachnoid hemorrhage secondary to aneurysm. Patient was transferred to tertiary care hospital where he received coiling of anterior communicating artery.

Discussion

A closer reevaluation of the previous head CT scans by a neuroradiologist does reveal a subtle change that might represent an anterior communicating artery aneurysm. However, this was very difficult to diagnose and could be a hindsight bias. The initial clinical features of fever and altered mental status were suggestive of meningitis, encephalitis, or status epilepticus. Clinical symptoms of quadriparesis, a complete loss of communicative ability with xanthochromia should trigger a further evaluation such as rupture of an anterior communicating artery aneurysm.

Resident / Fellow Clinical Vignette

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Title: Epistane: not all it is bulked up to be

Introduction: Drug induced liver injury (DILI) can develop from the use of prescription or over the counter medications and is the most frequently cited reason for withdrawal of medications from the marketplace. Anabolic androgenic steroid use is associated with variable clinical liver toxicity. However, there has been no case reported thus far with the administration of Epistane, a prohormone and derivative of dihydrotestosterone. Below is the first case reported of DILI with the use of this medication.

Case: A 34-year-old male presented with symptoms of yellowing skin, nausea, fatigue and right upper quadrant fullness. He admitted to using six cycles of a pro-hormone called Epistane over the past year, with the last dose being the day prior to hospitalization. He denied use of other medications, travel, sexual activity or intravenous drugs. On admission, vital signs were stable and physical exam was notable for jaundice and scleral icterus. Laboratory studies were significant for total bilirubin of 9.5 mg/dl, direct bilirubin of 7.5 mg/dl, gamma-glutamyl transferase of 131 U/L, a mild transaminitis with aspartate transaminase of 73 U/L and alanine aminotransferase of 130 U/L. Serum studies for acute hepatitis and pancreatitis were negative. Ultrasound of the abdomen showed probable cholelithiasis, mild gallbladder wall thickening with 0.3cm polyp at the anterior wall of the gallbladder. Computerized tomography abdomen and pelvis with contrast showed cholelithiasis with a bulky pancreatic head. Subsequent magnetic resonance cholangiopancreatography was negative for obstructive pathology, ductal dilatation, or pancreatic mass. Liver biopsy was performed, revealing mild acute and chronic hepatitis without steatosis, cholestasis, iron or fibrosis, consistent with drug reaction. He was seen by gastroenterology, bilirubin levels stabilized and was discharged with outpatient follow up. Prior to normalizing as an outpatient, his peak total bilirubin level reached 36 mg/dl without worsening symptoms and despite discontinuation of Epistane.

Discussion: DILI is the most common cause of acute liver failure in the United States, accounting for approximately 10 percent of all cases of acute hepatitis. It is classified into hepatocellular injury, cholestatic injury or a mixed injury. The above case depicts the first report of cholestatic injury caused by Epistane or 2a,3a-epithio-17a-methyl-5a-androstan-17b-ol which is an oral prohormone derivative of the anabolic steroid dihydrotestosterone used for bulking and as a cutting cycle agent by binding to androgenic receptors. Epistane and other prohormones have been banned from the United States market; however, are still available through the internet and overseas. This makes it easily accessible for our patients and therefore requires increased physician awareness and diligent patient education of the potential for life threatening hepatic toxicity.

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Title: Spontaneous Coronary Artery Dissection in a Post Partum Breast Feeding Mother

Introduction: Spontaneous coronary artery dissection (SCAD) is a rare but lethal cause of acute coronary syndrome that occurs most commonly in young women during the peri/post-partum periods.

Case: A 41-year-old female with history of e-cigarette smoking and uncomplicated pregnancy with C-section two weeks prior presented with sudden onset stabbing chest pain, dyspnea, and diaphoresis which began while breastfeeding. On presentation, her blood pressure was 148/105 with a heart rate of 88 and unremarkable physical exam. Initial electrocardiogram showed ST elevations in V2-V4 with ST depressions in the inferior leads. Troponin T and CPK levels peaked at 15.1 and 5330 respectively.

An emergent cardiac catheterization revealed dissection of the left anterior descending artery (LAD) with compromise of the first and second diagonal arteries. A drug eluting stent was placed in the mid LAD while the diagonals received balloon angioplasty. The patient had resolution of chest pain and ST segment elevations. A post-catheterization transthoracic echocardiogram revealed an ejection fraction of 50% with akinesis of the antero-septal, apical and distal anterior walls. A rheumatologic workup was sent as there is a link with SCAD in literature, and was notable for positive Beta-2 Glycoprotein I IgA Antibody and Cardiolipin IgA Antibody.

The patient was discharged on aspirin, clopidogrel, atorvastatin, metoprolol and lisinopril with cardiology follow up.

Discussion: The incidence of SCAD is underreported as many cases are diagnosed at autopsy. The post-partum state is associated with an elevation of progesterone leading to weakening of the tunica media increasing the risk of dissection. Whether breast-feeding is an independent risk factor for SCAD is currently unknown. Estrogen, which has a protective effect against atherosclerosis and ischemic injury of the myocardium, falls after parturition while the act of suckling stimulates the secretion of both oxytocin and prolactin. Thus, it can be hypothesized that as progesterone levels rise and estrogen levels fall there is an increased risk of SCAD. Prolactin and oxytocin themselves have not been associated with an increased risk for cardiovascular disease.

There have been four case reports of positive anti-phospholipid antibodies and SCAD. In this case, our patient had elevated titers of anti-cardiolipin IgA and Betaglycoprotein IgA. Anti-phospholipid syndrome is associated with hypercoagulable states that increase risk for coronary events. The significance of isolated elevations in antiphospholipid antibodies without disease in post-partum women with SCAD is unclear and requires further research.

Conclusion: Clinicians should consider SCAD as an etiology for chest pain in peri/post-partum women without known cardiac history. Physiologic changes during the peri/post-partum period may contribute to an increased risk for SCAD and links with antiphospholipid antibodies should be further explored.

Resident / Fellow Clinical Vignette

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Title: ENCEPHALITIS OF UNKNOWN CAUSE? THINK SARCOIDOSIS

Introduction:

Neurosarcoidosis occurs in 5% of patients with sarcoidosis, and in half of these it is present at the time of diagnosis. We present a case of neuro-sarcoidosis presenting with encephalitis and extensive brain lesions.

Case report: A 42-year-old woman presented to the emergency department (ED) with severe headache and dizziness. Four months prior, she was admitted to the hospital after an MRI obtained for persistent headaches showed an elevated FLAIR signal suggestive of cerebellitis with encephalitis, involving hippocampi, frontal lobes, insular cortices, middle cerebellar peduncles and bilateral cerebellar hemispheres. CSF encephalitis panel and evaluation for autoimmune and paraneoplastic syndromes were negative. Acyclovir and antibiotics were stopped and her clinical condition improved. A follow-up MRI after two months was unchanged.

In the ED, physical examination revealed a new wide based gait, hyperreflexia, crossed abductors, bilateral Hoffmann signs, prominent pectoral reflexes and jaw jerk. Laboratory studies revealed a sodium of 153 mEq/L, serum osmolality of 310 mOsm/kg H₂O and urine osmolality of 124 mOsm/kg H₂O, consistent with diabetes insipidus. Repeat brain MRI showed new involvement of the hypothalamus, brainstem and temporal lobes in addition to previous changes. Chest, abdomen and pelvis CT were unremarkable. CSF analysis showed elevated protein in the CSF with no pleocytosis. Temporal lobe biopsy showed non-specific inflammation, and this was followed by a pituitary stalk biopsy that revealed non-caseating granuloma with lymphocytic infiltration suggestive of sarcoidosis. She was started on plasmapheresis and pulse steroids with initial improvement, followed by a relapse, prompting treatment with infliximab and prednisone.

Discussion: Encephalitis with extensive brain parenchyma, brainstem, cerebellum and hypothalamic involvement usually occurs with infectious, autoimmune, neoplastic or paraneoplastic etiologies and is uncommon with neuro-sarcoidosis. In our patient, the discrepancy between the radiological and clinical findings is very remarkable. Despite this extensive brain involvement, it took four months to be evident clinically. This may be specific for neurosarcoidosis. There are few cases in the literature with such extensive and isolated brain sarcoidosis to compare with. Neurosarcoidosis most commonly presents with mono-neuropathy but can present with focal neurologic deficits, psychiatric symptoms, aseptic meningitis and hydrocephalus. It also can involve the hypothalamus and/or pituitary glands which may manifest with diabetes insipidus and multiple endocrine abnormalities. There are no specific radiological or CSF findings in encephalitis due to sarcoid. Brain biopsy remains the gold standard diagnostic modality especially in cases with atypical presentations. Hence, neuro sarcoidosis should be considered as a potential etiology in patients presenting with neurological symptoms not explained by routine evaluation even when evidence of systemic sarcoidosis is absent. Early diagnosis with prompt treatment could potentially improve the morbidity and mortality associated with this devastating condition.

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Title: Alpha Gal Induced Anaphylaxis to Herpes Zoster Vaccination

Introduction: Reported incidence of tick borne illness has progressively risen over the last decade. Galactose-alpha-1,3-galactose (alpha gal) allergy is a novel presentation of a tick borne illness induced by the Lone Star Tick, which is a well known vector for ehrlichiosis, tularemia, and Southern Tick-associated Rash Illness (STARI). Individuals with tick borne illnesses in the Lone Star Tick distribution of southern, midwestern and northeastern states are at greater risk for developing this condition. Three major hypotheses exist to describe how ticks might induce an IgE response through transmission of alpha gal: 1) alpha gal may be a component of tick saliva, 2) alpha gal may be residual from prior blood meal, and 3) alpha gal may be transmitted through commensal organism transmission. Clinical presentation of alpha gal allergy includes gastrointestinal symptoms, itching and urticaria, and delayed anaphylaxis. There has been one prior report of alpha gal induced anaphylaxis after administration of herpes zoster vaccination in the literature. We describe a second, unique presentation of this allergic condition observed at our institution.

Case Description: A 73 year old female with a past medical history of hypertension, chronic hepatitis C, and alpha gal allergy presented to our Emergency Department for near syncope after epinephrine administration for upper lip, mouth and tongue swelling experienced 45 minutes after obtaining her herpes zoster vaccination. Review of the vaccination contents revealed use of porcine gelatin - a meat product felt to have induced an IgE response in the setting of alpha gal allergy. The patient was admitted, managed with intravenous fluids, and discharged the following morning.

Discussion: To increase physician awareness of alpha gal allergy and increased incidence in populations exposed to tick-borne diseases. We report a case of alpha gal anaphylaxis by herpes zoster vaccine due to use of porcine gelatin. We suggest avoiding administering the herpes zoster vaccine in patients with a history of a tick borne illness.

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Title: Giant Cell Tumor of Bone with co-existing aneurysmal bone cyst in a non-Asian patient

Giant cell tumor of bone (GCTB) is a rare and benign tumor that accounts for three to five percent of all bone tumors in the US. Comparatively, Asian populations have a high percentage of GCTB, where it represents twenty percent of all primary bone tumors. The tumor itself can be locally aggressive, often recurring after curettage alone, and is often difficult to differentiate from other bone tumors such as aneurysmal bone cyst, chondroblastoma and non-ossifying fibroma. We present a case of an African American male with no past medical history who presented with a left pathological lateral condylar fracture, biopsy proven to be GCTB and with a concomitant aneurysmal bone cyst.

A 24 year old male with no significant past medical history presented with complains of left knee pain, 8/10, non-radiating, not associated with tingling and numbness, which occurred while he leapt for a shot while playing basketball. He denied constitutional symptoms including weight loss, night sweats, fevers, chills, nausea, vomiting, diarrhea, sick contacts or trauma to the knee. He had no family history of cancer, was not on medications, denied any previous fractures, smoking or illicit drug use. Since the patient was immediately non-weightbearing on that leg, he was brought to our hospital where an XRAY confirmed a pathological fracture of left lateral femoral condyle and an MRI showed a destructive 7.1 cm lesion related to lateral femoral condyle with transverse dimensions 5.2 x 5.3cm with cortical disruption and aggressive features. His blood work including a complete blood count, chemistry, erythrocyte sedimentation rate, C-reactive protein, CT chest, abdomen and pelvis were all normal. A bone scan confirmed a pathological fracture and he underwent an open biopsy of the lesion, followed by extended curettage and cementation with open reduction and internal fixation of the fracture by Orthopedics. Surgical pathology report was positive for GCTB in addition to aneurysmal bone cyst, thus not necessitating medical oncological treatment. The patient did well post-operatively.

GCTB is a benign tumor, which comprises about three to five percent of bone tumors in the US. It normally occurs after skeletal maturity has been achieved and is associated with a female predominance. In Asian countries however, the incidence of GCTB is as high as twenty percent, for reasons that are unknown.

When GCTB are associated with cystic components, it is termed an aneurysmal bone cyst, with a fourteen percent incidence rate. On a positron emission tomography/computed tomography (PET/CT) these lesions can appear to be heterogeneous and hypermetabolic. In our case, we have an African American male, with no prior medical history, who was diagnosed to have GCTB and an aneurysmal bone cyst whose PET CT was homogeneous.

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Title: RECURRENT FRACTURES IN A YOUNG WOMAN ASSOCIATED WITH HYPOGONADOTROPIC HYPOGONADISM

INTRODUCTION:

Hypogonadotropic hypogonadism (HH) is defined as a deficiency of the pituitary secretion of gonadotrophins including follicle-stimulating hormone and luteinizing hormone which results in the impairment of pubertal maturation, reproductive function and decreased bone mineral density. We describe a case of a young woman who presented with left periprosthetic femur fracture due to fall and was found to have panhypopituitarism.

CASE

A 25 year old woman from Dominican Republic (DR) with medical history of left femoral neck fracture presented with left thigh pain after she sustained a fall. The patient was diagnosed with left peri-prosthetic femur fracture and had surgical repair on admission. She mentioned history of left femur fracture ten years prior to presentation after a bike accident which was treated conservatively in the DR and she underwent left proximal femur repair five years ago after migrating to the US. Upon further questioning she reported chronic weakness, inability to participate in sports and recurrent falls. She had primary amenorrhea which was never evaluated. Review of system was negative except for fatigue. On examination, the patient appeared younger than her reported age and had underdeveloped breast, Tanner stage- 2 with complete absence of axillary hair and pubic hair but normal appearing external genitalia. There was bitemporal hemianopia and signs of visual compromise. Labs revealed panhypopituitarism: low FSH (1.2 mIU/ml), low LH (0.5 mIU/ml), low cortisol (2.6 ug/dL) and low Free T4 (0.7 ng/dL). Growth hormone, estradiol and prolactin were undetectable. Pelvic USG showed vaginal cuff but no ovaries or uterus. Brain MRI revealed 3 cm mass within the sella turcica extending to the suprasellar cistern and interpeduncular cistern and compressing the optic chiasm. Patient was treated with hydrocortisone and levothyroxine initially then referred to outpatient endocrinology and neurosurgery for further management.

DISCUSSION:

Acquired hypo gonadotropic hypogonadism (HH) can be due to several causes including mass effect from a pituitary tumor, infiltrative or infectious pituitary lesions and hyperprolactinemia. The clinical characteristics of HH is usually hallmarked by a deficiency in reproductive hormones and delay in pubertal sexual maturation. Long-term estrogen deficiency may manifest as early onset osteopenia and osteoporosis leading to increased risk of fracture as was observed in this patient. Fracture in a young woman, especially when associated with other manifestations such as primary amenorrhea should be evaluated with further work up.

LEARNING POINTS:

- Fractures associated with primary amenorrhea, hypogonadism and delayed puberty in a woman warrant complete work up to find the underlying etiology.
- HH may manifest as recurrent fractures in young woman.
- Early diagnosis and treatment can prevent long term functional impairment.

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**Title: ARRHYTHMOGENIC RIGHT VENTRICULAR
CARDIOMYOPATHY**

ARRHYTHMOGENIC RIGHT VENTRICULAR CARDIOMYOPATHY

A Rare Cause Of Life Threatening Cardiomyopathy
Abstract

Zaid B. Al Jebaje , Osman Saleem and Robbie D. Wall
Arrhythmogenic right ventricular cardiomyopathy (ARVC) is a clinical entity characterized by ventricular arrhythmias and a specific ventricular pathology. Over 30 genes have been linked to its pathogenesis usually code for proteins involved in desmosomal structures between cardiac cells. The incidence of ARVC is unknown, but the prevalence in the general adult population is estimated to be approximately 1 in 2000 to 1 in 5000.

Our case is about a 27-year-old athletic female with no significant past medical history who collapsed while playing Frisbee. EMS assessment revealed a pulseless patient, in ventricular fibrillation, was successfully resuscitated after an extended-30 minutes CPR. Received 8 DC shocks, on-scene intubation and was transferred to ED. Anterior T wave inversions in leads V1-V3 during sinus rhythm, QRS duration in lead I =120 milliseconds during ventricular arrhythmia, QRS notching during ventricular arrhythmia and precordial transition in lead V5 or later during ventricular arrhythmia were seen on EKG findings. Initial TTE showed LVEF 30%, moderate enlargement and reduced RV function. Follow-up TTE revealed a preserved LVEF 50-55%. Cardiac catheterization was negative for coronary artery disease. Cardiac MRI revealed mild dilatation of the right ventricle with no regional wall defect. Genetic testing showed heterozygosity for a novel variant of uncertain significance in the DSC2 gene that codes for Desmocollin-2 (a desmosomal protein), the pathogenic variants of which are found in autosomal dominant forms of ARVC. The patient wore a wearable defibrillator for 30 days, was treated with beta-blockers and received a single chamber subcutaneous ICD, she continues to follow our clinic regularly.

Palpitations “ 67 %, Syncope “ 32 %, Atypical chest pain “ 27 %, RV failure “ 6 % are the common presenting symptoms of ARVC. Life style modifications including refraining from high intensity physical activities, beta blockers, and ICD implantation for primary or secondary prevention. Genetic test can be helpful in diagnosis and as screening tool for family members in patient with confirmed diagnosis. Although many patients including ours do not show evidence of right ventricular damage eventually nearly all will, which requires thorough follow up by physician and timely intervention.

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**Title: LARGE BRAIN MASS PRESENTING AS SUBTLE
BEHAVIORAL CHANGES**

A 61-year-old male patient with a medical history of diabetes and stage III rectal cancer treated with surgery and chemotherapy in 2014. The patient presented to ED at midnight via EMS after his wife reported unusual behavior. She was concerned about a progressive alteration in his behavior. That included urinating in cups and buckets next to him as well as emptying his colostomy bag in the sink. The patient denied any abnormal behavior and attributed those bizarre actions to his urinary urgency and generalized fatigue that prevented him from getting to the bathroom in time. Physical examination was generally unremarkable, and so was his neurological exam. He scored 29/30 on mini-mental status examination. Overall, he seemed clear and sharp in terms of mental function and orientation. Laboratory investigations only revealed mild hyponatremia but were otherwise unremarkable. His wife was called at home so we could clarify her concerns. She described progressive behavioral changes for the past 7-8 months. Those could be easily linked to a possible depression that developed after losing his job 8 months prior following a car accident. However, that could not explain few other odd behaviors, which raised concerns for Executive Dysfunction Syndrome. A brain CT was done and showed a 5x3 cm left temporal lobe enhancing mass with compression of the temporal lobe and lateral ventricle, and a large amount of surrounding hydrostatic edema. He was later found to have stage IV diffuse large B-cell lymphoma. He refused surgery and opted for medical management instead. His capacity to make decisions was deemed to be intact at that point.

Discussion:

Behavioral changes and executive functions are important components of history taking as they can signal a more sinister intracranial process that might otherwise go undetected for a long time. Direct questioning and careful observation of responses during history-taking and physical examination are key. However, as seen with our patient, family inputs are crucial for this matter. Additionally, several other brief tests have been developed to help physicians assessing the cognitive and executive functions of their patients. Examples include the Wisconsin card sorting test, Montreal Cognitive Assessment, Behavior Rating Inventory of Executive Function and others. These tests have a pooled sensitivity of 75-92% and a specificity of 81-91%. Due to time constraints, emergency physicians are inclined to use quicker tools such as the Mini-Mental Status Examination. However, subtle behavioral changes might not get picked up by this test, as seen in our patient. Thus, further studies are needed to develop more sensitive and reliable quick tests to assess executive functions at emergency rooms, especially for patients with little or no neurological findings and for those who lost follow up with their primary doctors.

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Title: RARE PRESENTATIONS OF PARAGANGLIOMAS

BACKGROUND:

Paragangliomas are benign, hypervascular neoplasms accounting for 0.012% of all tumors and 0.6% of all neoplasms in the head and neck region. These neoplasms are insidious in onset, making the diagnosis more challenging. In this report, we have presented three unique cases of paragangliomas.

CASE REPORT:

Case 1: A 65 year-old woman presented with hoarseness and hearing loss of two months duration. On physical examination, she was found to have left cranial nerve VIII, IX and X paralysis. Laryngoscopy also revealed left vocal cord paralysis. MRI of the brain was consistent with Glomus Vagale Tumor. Pheochromocytoma work up was negative. She was successfully treated with IR guided embolization and surgical debulking of the tumor.

Case 2: A 31 year-old woman with a history of recurrent left ear canal polyps of four year duration, presented with bloody otorrhea and pulsatile tinnitus. MRI showed a left middle ear mass extending through the jugular foramen consistent with Glomus Jugulotympanicum Tumor. She underwent IR guided embolization followed by surgical debulking of the tumor with complete resolution of the symptoms.

Case 3: A 50 year-old woman with a history of pulsatile tinnitus in the left ear, presented with left sided facial numbness. MRI revealed a mass which on biopsy showed Glomus Jugulare Tumor. Her symptoms resolved after embolization and surgical debulking. However, tumor relapsed requiring radiotherapy.

DISCUSSION:

Paragangliomas are rare, slow growing tumors, arising from paraganglionic tissue of neural crest origin. They usually presents between the 5th and 6th decade of life with a female predominance. Common presentations include a mass in the middle ear (Glomus Tympanicum) causing pulsatile tinnitus and conductive hearing loss, or as a pulsatile painless mass in the neck (Carotid Body Paraganglioma). Our second case was diagnosed with Paraganglioma in the second decade of life, which is very unusual. She presented with recurrent external ear polyps which is a unique finding. In 2.5% of cases, paragangliomas present with hoarseness and vocal cord paralysis (Glomus Vagale) as mentioned in our first case. The unique feature of the third patient, unlike the first two, was the recurrence of the tumor after resection reaching the same initial size in 7 months. This suggests the possibility of a malignant Paraganglioma, which occurs only in 1-5% of Paragangliomas.

CONCLUSION:

We present a broad spectrum of clinical presentations of this extremely rare tumor. These presentations include dysphagia, hearing loss, pulsatile tinnitus, hoarseness, pain and cranial nerve palsies. A high index of suspicion and clinical awareness could help in early diagnosis and with improved outcomes in the treatment of paragangliomas.

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Title: DO QUESTION FROM MEDICAL LICENSING AND BOARD EXAMINATIONS PREPARE PHYSICIANS FOR SHARED DECISION MAKING?

Background

Many decisions in medicine involve a comparable trade off between harms and benefits such that patient preference should be considered to determine the correct option. Shared decision making is a method considered by most professional societies as the optimal approach to handling preference sensitive decisions (PSDs), yet clinical implementation is impeded by the challenge of recognizing such scenarios in practice. Standardized examination practice questions teach how to assess and/or manage clinical situations, yet the extent to which they mitigate or increase the challenge of recognizing PSDs is unknown. Our aim was to assess the extent to which management-oriented practice questions included patient preference as a necessary component in arriving at the predefined correct answer.

Methods

Practice questions were purchased from National Board of Medical Examiners (NBME) as well as three popular online question banks (USMLEWorld, USMLE-Rx, and MKSAP), for Medical Licensing Examination (USMLE) I, II, and III as well as American Board of Internal Medicine (ABIM). These were then reviewed by trained assessors. Questions were excluded that did not involve clinical management (e.g. diagnosis questions). Preference-sensitive question-answer pairs were defined by whether they presented more than one correct way to handle a situation and the patient's preferences were used to arrive at the final answer.

Results

A total of 937 questions were reviewed: 6% from NBME, 17% from USMLEWorld, 41% from USMLE-Rx, and 35% from MKSAP. Of these, 444 (47.4%) were management-related practice questions, comprised of 68 (16%) for USMLE I, 123 (27%) for USMLE II, 23 (5%) for USMLE III, and 230 (52%) for ABIM. No questions for USMLE I, II or III, and only four questions (1.7%) for ABIM met the definition of preference-sensitive.

Conclusion

Practice questions have a role in teaching novice physicians how to approach clinical situations. From this broad sample of questions we conclude that students and trainees are almost exclusively exposed to scenarios suggesting that the correct medical choice can be arrived at independent of the patient's preferences. This may contribute to the difficulty physicians have in recognizing preference sensitive decisions in which they should involve their patients.

<p>Author: Tanureet Arora Additional Authors: Dr.Karthik Vadamalai; Dr.Stephen Silver Institution: Rochester General hospital</p> <p>Title: A Dilemma: Pseudohyperkalemia or Pseudohypokalemia?</p> <p>Background: An array of metabolic derangements is seen in patients with acute myeloid leukemia (AML) having hyperleukocytosis and tumor lysis syndrome (TLS). Here we present a patient with hyperleukocytosis who had low and high potassium levels reported from two different samples drawn at the same time. Directly analyzed blood sample or sample transported on ice can help prevent misdiagnosis and inappropriate management of these patients.</p> <p>Case: A 56 year old woman presented to our hospital with complaints of nausea, vomiting and altered mental status for one week duration. Laboratory data on admission, sent to the lab in the usual manner via the pneumatic tube system, showed a leukocyte count of 513,000/L with 95 % myeloid blasts on peripheral smear. Uric acid 24.7mg/dL; lactate dehydrogenase 4309 U/L and creatinine 3.2mg/l; serum potassium was 3.3mEq/L and whole blood potassium was 8.1mEq/L. A repeat analysis of the blood specimen placed on ice and walked by hand to the lab revealed serum potassium 3.6 mEq/L and whole blood potassium 3.7 mg/dL. Based on these findings, the decision was made to defer emergent dialysis. Computed tomography of head was negative for intracranial hemorrhage. Her course was complicated by persistent blast crises and spontaneous tumor lysis syndrome. Patient was treated with plasmapheresis for hyperleukocytosis and started on hydroxyurea to decrease the cell turnover rate. Even with aggressive measures, patient developed disseminated intravascular coagulopathy and multi-organ failure leading to death.</p> <p>Discussion: Hyperleukocytosis occurs in 10 to 20 percent of patients with newly diagnosed AML. In these patients ongoing tumor lysis causes multiple metabolic abnormalities including hyperkalemia, hyperphostemia, hyperuricemia and hypocalcemia. During blast crisis the massive tumor genesis can reutilize the released potassium from the ongoing cell lysis leading to hypokalemia. However, there can be spurious electrolyte abnormalities during handling or processing of the blood samples. Patient can have pseudohypokalemia due to intracellular shift of potassium by sodium-potassium pump in the tumor cells if sample is left in room temperature for significant amount of time. Interestingly the phenomenon of pseudohyperkalemia has also been described in patients with hyperleukocytosis. This is due to the lysis of tumor cells after coming in to contact with heparin in the blood sample tubes or even with mechanical disruption of the tumor cells in the tube. It appears in our patient that the most important of these factors was the mechanical disruption that occurs with usual delivery of the lab sample. The normal serum potassium on the repeat sample helped us to safely exclude the need for emergency dialysis. The recognition of these factitious electrolyte abnormalities in patients with hyperleukocytosis is important to aid with appropriate treatment decisions.</p>	<p>Author: Fadi Asfahan Additional Authors: Asfahan F, Abdullah A, Hoefen R, Patel T, Institution: Unity hospital</p> <p>Title: Cardiac arrest and the ghost in the room</p> <p>A 36 years old female with a history of missed abortion at 17 weeks gestation and Deep Venous Thrombosis (DVT) on enoxaparin was admitted for elective dilatation and evacuation (D & E). DVT was diagnosed 3 months ago and involved the femoral and popliteal veins. A repeat Doppler US showed resolution of the clot burden after one month of enoxaparin therapy; the last dose was administered 24 hours before the procedure. Previous thrombophilia screen revealed heterozygosity for prothrombin gene mutation. Shortly into the procedure, the patient developed desaturation followed by circulatory collapse with asystole and cardiac arrest. Cardiopulmonary resuscitation was commenced immediately for 5 minutes with return of spontaneous circulation.</p> <p>An urgent limited bedside transthoracic echocardiogram {Fig. 1} showed dilated right atrium (RA) with evidence of floating structure most likely a thrombus. An urgent trans-esophageal echocardiogram was performed which showed severely dilated (RA) and moderately dilated right ventricle (RV), but no evidence of intra-cardiac thrombus {Fig 2}. The patient was intubated and transferred to the intervention radiology suite for possible thrombolysis/clot extraction. Surprisingly, an invasive pulmonary angiogram showed no evidence of any obstructive lesion along the pulmonary arteries {Fig.3}. Repeat transthoracic echocardiogram on the same day showed resolution of the RA and RV dilatation. The patient regained full cognitive function and was extubated after 24 hours. On day 4 she was discharged on full anticoagulation with enoxaparin and later on warfarin.</p> <p>Discussion In this case report we describe the striking events occurring during an elective (D &E) procedure in a patient with history of DVT. An urgent limited bedside echocardiogram was able to demonstrate dilatation of the RV and RA which reflects acute pulmonary hypertension consistent with embolism. The demonstration of the mobile thrombus in the right atrium is very rare, and the rapid resolution of the thrombus in subsequent angiography and echocardiogram is even more striking.</p> <p>The case highlights the increased risks associated with venous thromboembolism during pregnancy, pregnancy loss, and related procedures.</p> <p>Conclusion -Limited echocardiogram is a helpful modality in the emergent assessment of acutely unwell patients. - Venous thromboembolism may be associated with high surgical risk despite adequate anticoagulation</p>
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<p>Author: William Carroll, MD Additional Authors: Astha Muttreja, Stony Brook University Hospital, Astha.Muttreja@Stonybrookmedicine.edu Alice Fernan, Stony Brook University Hospital, Alice.Fernan@Stonybrookmedicine.edu Ivan Crnosija, Stony Brook University Hospital, Ivan.Crnosija@Stonybrookmedicine.edu Institution: Stony Brook University Hospital</p> <p>Title: Making it count twice: QI to improve chronic pain management and efficiency in a resident continuity clinic Background: Chronic non-cancer pain management with opioids challenges training physicians. Chronic pain not caused by cancer is a highly prevalent, debilitating condition and it is challenging for physicians to know when, and how to prescribe opioids for chronic pain without increasing public health risks. We aimed to assess our adherence to the 2009 American Pain Society/American Academy of Pain Medicine opioid guidelines and the effectiveness of a quality improvement intervention to improve adherence and office visit utilization in our resident clinic. Objective: To improve delivery of care for chronic pain and increase clinician adherence to guidelines for safe opioid prescribing. Methods: We implemented a multifaceted quality improvement intervention, including an electronic template to standardize documentation of chronic pain management and workflow redesign for electronic opioid renewal to decompress office visit utilization. We conducted a retrospective study pre- (7/2014-6/2015) and post-intervention (7/2015-6/2016). Process outcomes measures included documentation of annual toxicology and narcotic agreement. Clinical and utilization measures included morphine milligram equivalents (MME) and number of annual office visits. Bivariate analysis was conducted using SAS software for continuous variables using the independent samples t-test, paired samples t-test, and Wilcoxon Signed-Rank test where appropriate, and McNemar's test and Fisher's exact test for categorical variables. Results: 60 charts of patients on opioid therapy for >3 months were reviewed. 10 were excluded due to treatment for cancer pain, inadequate follow up, or opioid prescription from pain management, and 50 were used for analysis. Annual toxicology increased from 52% to 82% (p < 0.01) and annual narcotic agreement increased from 12% to 56% (p < 0.0001). Average morphine equivalent dose decreased significantly from 100.6MME to 75.2MME (p = 0.0049) and 7 patients were tapered off opioids. The number of annual office visits also decreased from 11.1 to 8.9 (p < .001). Women were more likely to have narcotic agreements than men (76% vs. 36%, p<0.01) and younger patients <65 were more likely to have annual toxicology (90% vs. 38%, p<0.01) and risk stratification documented (86% vs. 50%, p = 0.04) than those with age =65. Outcomes did not otherwise vary by age, gender or race. Conclusions: Chronic pain and opioid management is a significant public health issue, with increasing overdose-related deaths and the need for safer systems for opioid prescribing. QI interventions that standardize documentation can be effective in increasing adherence to annual toxicology and narcotic agreements. Our post-intervention decrease in MME and discontinuation of opioids in some patients suggests we are moving towards effective and safer care. Our re-designed workflow, with visit frequency based on risk stratification, allowed for fewer office visits and increased clinic efficiency. QI is a useful strategy for enhancing the quality, safety and efficiency of care for patients with chronic pain.</p>	<p>Author: Roberto Cerrud-Rodriguez, MD Additional Authors: Quinteros, Maria; Azam, Mohammed Institution: SBH Health System</p> <p>Title: INTERNAL CAROTID ARTERY OCCLUSION WITH SUBSEQUENT ISCHEMIC STROKE AS A COMPLICATION OF CISPLATIN-BASED CHEMOTHERAPY FOR METASTATIC TESTICULAR GERM-CELL TUMOR: A CASE REPORT</p> <p>Introduction Testicular tumors are the most common solid tumors in males between ages 15-35. Germ-cell tumors (GCT) account for 95% of all testicular cancers and the non-seminomatous (NSGCT) type accounts for half of all GCTs. Cisplatin-based chemotherapy is curative in up to 90% of NSGCT, but its use has been associated, in rare cases, to ischemic cerebrovascular events. We describe a rare case of an internal carotid artery occlusion with a subsequent ischemic stroke associated to cisplatin chemotherapy.</p> <p>Case Report 36-year-old male on chemotherapy presents with a 30-minute history of the sudden onset of left hemiparesis, left facial droop, dysarthria. Physical exam was notable for dysarthria, left hemiplegia, left hemi-hypoesthesia, anosognosia without asomatognosia. Blood pressure was normal. NIHSS score was 11. Former smoker (20 pack-year). Had a known history of left testicular NSGCT diagnosed 2 years before, initially treated with a left orchiectomy. Follow-up with serial CTs showed new retroperitoneal lymphadenopathy, 4 months before the onset of the stroke. Cisplatin and etoposide were started at that time. 3 cycles of chemotherapy were given, the last one 3 days before the stroke. Family history was unremarkable. CT brain in the ED was normal. Thrombolysis was given, without improvement. Labs showed hypomagnesemia, normal coagulation and lipids. MRI brain revealed acute infarct in the right MCA distribution. USG Duplex of the carotids suggested a right internal carotid occlusion. CT angiogram confirmed a complete occlusion of the right internal carotid artery. Hypercoagulable workup was obtained, being unremarkable. Transesophageal echo and Holter monitoring were unremarkable. Patient was managed with aspirin. The patient was discharged to short-term rehab on day 10 with moderate improvement of his neurological deficit. He was reevaluated 3 months after discharge and had recovered motor strength in his left lower extremity, regaining the ability to ambulate, with severe residual weakness in his left upper limb. NIHSS 5.</p> <p>Discussion A few cases of CVAs in young male patients affected by NSGCT after cisplatin-based chemotherapy have been reported. These events most commonly involve the internal carotids or the cerebral arteries. This was the case with our patient, who developed a right internal carotid occlusion with subsequent right MCA stroke 3 days after receiving cisplatin-based chemotherapy. Chemotherapy against NSGCTs is associated to increased platelet aggregation, increased levels of vWF, and direct endothelial lesion. Cisplatin, in particular, has been associated to an increased alpha-adrenergic tone, as well as hypomagnesemia, which in conjunction could trigger an arterial spasm. Hypomagnesemia and vasospasm could have played a role in our patient's presentation. There is still no consensus on what the preventive strategy for this population should be - but some advocate prophylactic heparin use. The prognosis in this patient population is usually good, with most regaining their neurologic function.</p>
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<p>Author: Yanming Chen, MD Additional Authors: Farhana Polara MD Anjana Pillai MD Andrew Burger MD Institution: Lincoln Medical and Mental Health Center</p> <p>Title: Rare variability of adrenoleukodystrophy confounded with alcohol abuse</p> <p>Introduction X-linked adrenoleukodystrophy (ALD) is peroxisomal disorder caused by ABCD1 gene mutation, which results in accumulation of very long chain fatty acids mostly in myelin and adrenal cortex. Based on brain MRI features, age of onset and progression of symptoms, ALD is classified into different phenotypes including cerebral ALD and adrenomyeloneuropathy (AMN) with no phenotype-genotype correlation. We present a case of adult-onset cerebral ALD with initial clinical features of AMN which is exceedingly rare and confounded with alcohol abuse history.</p> <p>Case report A 35-year-old male with alcohol abuse was admitted with new onset seizures and subacute lower extremity weakness, urinary incontinence. Deterioration of patient was started with personality changes consisted of apathy, pailialia and declining daily functions for one year. Detailed family history revealed mild cognitive dysfunction of his nephew. Neurological exam revealed gait ataxia, increased muscle tone of lower extremities with diminished deep tendon reflexes. Initial brain computed tomography (CT) demonstrated a mass lesion occupying left caudate extending into frontal lobe. Brain Magnetic Resonance Imaging (MRI) revealed extensive, bilateral and symmetric signals on axial fluid-attenuated inversion recovery (FLAIR) sequence involving corpus callosum, periventricular white matter and internal capsule with enhancement. Blood and cerebral spinal fluid test ruled out common infectious, demyelinating or malignant etiologies. Empirical nutritional supplement for toxic encephalopathy based on his alcohol abuse history was non-responsive. An inherited condition was suspected and ALD was diagnosed by elevated long chain fatty acid and confirmed by ABCD gene testing.</p> <p>Discussion AMN usually manifests in male patients between 20 to 30 years. Weakness and spasticity of lower extremities, sphincter dysfunction and impotence are common symptoms. The neurologic dysfunction usually developed slowly within years, with only moderate increase of brain MRI signal of the white matter without enhancement. Cerebral ALD usually has an age of onset between age 5 to 12. The cognitive function will deteriorate rapidly after evidence of demyelination on MRI, accompanied by motor deficits, ataxia, cortical blindness or seizure. Vegetative state will ensue within 2 to 5 years. While 20% of patient with AMN develops cerebral ALD later, cerebral ALD with presentation of typical symptoms of AMN as in our case is very rare, and initial impression of acute to subacute clinical picture may mimic acquired demyelinating diseases. In such cases. Detailed family history, personal history and radiographic images with high index of suspicion are crucial for diagnosis.</p> <p>Conclusion We described a young adult patient with alcohol abuse history who had cerebral ALD with initial clinical feature of AMN. The clinical phenotypes of ALD can be diverse and overlapping with each other confounded with other etiologies, and this rare diagnosis should be entertained early to provide genetic counselling in a timely fashion.</p>	<p>Author: Saurabh Chitnis, MD MS Additional Authors: Andrea Popescu-Martinez MD & Shobhana Chaudhari MD, FACP Institution: NYMC- Metropolitan Hospital Center</p> <p>Title: Correlation between Vitamin D deficiency and Metastatic Breast carcinoma in a predominantly Hispanic population - A retrospective study</p> <p>Background: There have been numerous studies conducted linking Vitamin D deficiency to various cancers, including cancers of the breast. Studies associating Vitamin D deficiency and breast cancer have shown mixed results in patients with no clear consensus. In-vitro studies have shown that the active form of Vitamin D can inhibit the metastatic capability of breast cancer cell lines to bone. Ablation of Vitamin D receptor on breast cancer cells has also shown to accelerate tumor growth and enhance the development of tumor metastases. Based on this we aimed to elucidate whether there exists any correlation between Vitamin D deficiency at diagnosis and metastatic breast cancer.</p> <p>Materials & Methods: Retrospective analysis of the electronic medical records for women diagnosed with breast cancer and enrolled in Oncology database at our hospital from 2010-2016 was done. Patients were grouped into either breast cancer with metastases or without metastases and their Vitamin D levels at diagnosis were reviewed. Patients with 25- Hydroxy Vitamin D levels measured within one year of diagnosis of breast cancer were included for the study. Patients with 25-Hydroxy Vitamin D levels <20ng/ml were considered as Vitamin D deficient. Study was planned from 2010-2016 specifically as Vitamin D levels for patients were not monitored as frequently prior to 2010.</p> <p>Results: From total of 102 patients who started follow up with Oncology clinic during the study time period, 2 were referred to our center for further management with diagnosis made prior to 2010 and were not included in the study. Out of the 100 patients considered, 58 patients had Vitamin D levels measured within a year of diagnosis of breast cancer and were included in the study. 70% of the population was Latino/Hispanic. Total patients with Vitamin D deficiency were 30; out of which 5 were with metastatic disease and 25 with non-metastatic breast cancer. Out of 28 patients with normal Vitamin D levels, 5 had metastatic disease and 23 had non-metastatic breast cancer. Odds ratio for a patient with Vitamin D deficiency to develop metastatic breast cancer was 0.92 and relative risk was calculated to be 0.93.</p> <p>Discussion: Based on above results, the outcome was similar in both groups. The odd's ratio and the relative risk imply that there was no difference evidenced between both groups. These findings signify that there was no direct correlation between metastatic breast cancer and Vitamin D deficiency at the time of diagnosis in the population seen at our center. Though findings with in-vitro studies for Vitamin D have been promising, it is unclear how screening for Vitamin D deficiency in the general population would benefit primary healthcare.</p>
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Title: AN UNUSUAL CASE OF STAGE IV ADENOCARCINOMA OF THE LUNG

Introduction

Adenocarcinoma is the most common subtype of non-small cell lung cancer (NSCLC) and represents the majority of all lung cancers. NSCLC staging is based on size, location, and metastasis with stage I and II describing smaller tumors without metastasis and stage III and IV describing larger tumors with metastasis to local and distant sites, respectively. We are presenting an unusual case of a metastatic adenocarcinoma of the lung.

Case Description

A 46-year-old female with a history of obesity and anemia was referred by her hematologist/oncologist for further evaluation of outpatient ultrasound findings of liver lesions. She reported that she had been experiencing shortness of breath for six weeks which worsened to the point where she was barely able to ambulate from one room to another. In addition, she endorsed an unintentional 60-pound weight loss during this time. Pertinent physical examination findings included: tachycardia, clear lungs on auscultation, mild epigastric tenderness, and right calf tenderness. Initial laboratory tests revealed microcytic anemia and leukocytosis. Computed tomography (CT) of the chest, abdomen and pelvis revealed a 1.5 cm noncalcified nodule in the left lower lung, upper abdominal/retroperitoneal adenopathy, and multiple hepatic nodules with the largest nodule in the right hepatic lobe measuring 7.4 cm. Additional findings were bilateral iliac vein deep vein thrombosis (DVT) and bilateral pulmonary emboli. She was treated for pulmonary emboli and DVT with enoxaparin. A CT-guided core biopsy of the right hepatic lobe revealed poorly differentiated adenocarcinoma suggestive of lung primary. Immunostaining of tumor cells was positive for cytokeratin 7 and thyroid transcription factor-1. A diagnosis of stage IV adenocarcinoma of lung was made. She was discharged home on treatment with enoxaparin and was scheduled for outpatient palliative chemotherapy.

Discussion

Both NSCLC and small cell lung cancer present late in the disease course with similar symptoms including cough, hemoptysis, dyspnea, and chest pain. Treatment depends on staging and patients with stage I, II, or III are treated with surgical resection with curative intent, chemotherapy, and/or radiation therapy while palliative care is recommended for stage IV. Tumor size is an important prognostic factor for staging as several studies have shown a correlation between tumor size and 5-year survival rate. Tumors <3 cm have a significantly higher 5-year survival rate than those >3 cm. In this case, a lung nodule as small as 1.5 cm was shown to have metastasized. Tumors of such size are normally stage 1 and rarely have this potential to metastasize. Our case signifies the need for more studies on other aspects for assessing the metastatic potential of a lung nodule.

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Title: ACUTE ISOLATED HYPERBILIRUBINEMIA AS A PRESENTATION OF ALCOHOLIC LIVER DISEASE – A CASE REPORT

Isolated hyperbilirubinemia as a manifestation of alcoholic liver disease without significant liver abnormalities is seen very rarely. We report such a case where patient presented to ER with acute jaundice with bilirubin 24.8 mg/dl, mostly conjugated without significant abnormalities of liver function or extrahepatic cholestasis.

A 63 year old male with past medical history of chronic alcoholism presented to ER after noticing yellowish discoloration of his skin, dark urine and worsening nausea and anorexia for last 7 days. He denied associated abdominal pain or abdominal distention. There was no pertinent family history of jaundice or related liver disease. Physical exam was notable for deep icteric sclera and abdominal exam was unremarkable and there were no peripheral stigmata of liver disease. Laboratory findings were significant for total Bilirubin 24.8 mg/dl with direct bilirubin 18.8 mg/dl, AST- 76 IU/L and Platelet count of 28 K, albumin 2.7 gm/dl, other labs were within normal limit including ALT, ALP, GGT, MCV, INR and PT. Abdominal sonogram showed small right hepatic cyst (1.2 cm) without evidence of cirrhosis or extra hepatic duct obstruction. CT abdomen without IV contrast and MRI with MRCP were unremarkable. Hepatitis Panel initially was negative for Hepatitis A,B and C virus. Repeat panel showed HBV core IgM antibody positive but negative HBD DNA, HBsAg, HBeAg, HBe Ab. Other viral panel – CMV, EBV, parvovirus were negative. Anti-mitochondrial antibody, anti-smooth muscle antibody, sickle cell screen were negative as well. Liver biopsy showed focal steatosis, focal nonspecific chronic inflammation and portal fibrosis and non-specific increase in lipochrome pigment in hepatocyte. It excluded hemochromatosis, dysplasia or malignancy. Bilirubin gradually trended down to 7.3 mg/dl and patient was discharged with instructions and recommendations to follow up in GI clinic.

Patients with alcoholic liver disease are typically asymptomatic initially and as the disease advances, they can present with symptoms including jaundice and other signs of peripheral stigmata of liver disease or signs of hepatic decompensation but rarely present with isolated intrahepatic cholestasis. The exact pathology behind alcohol induced intrahepatic cholestasis is not well established but experts identified the possible causes as interference with basolateral uptake and intracellular transport of bile acids or compression of intrahepatic biliary tree. In most cases, alcohol induced liver disease can be diagnosed easily with reliable history, simple laboratory and imaging tests. However, some cases like ours, diagnosis can be challenging due to very atypical presentation and may need more invasive studies such as liver biopsy or ERCP to diagnose.

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Title: It's All In The Head- SLE Flare Presenting with Neuropsychiatric Symptoms

Introduction

Neuropsychiatric systemic lupus erythematosus (NPSLE) includes heterogeneous and rare neuropsychiatric (NP) manifestations involving both the central and peripheral nervous system. It can be primary from pure auto immune mediated vasculitis or secondary from disease related infections, drug or metabolic effects. Due to lack of a gold standard, diagnosis of primary NPSLE represents a clinical challenge in spite of the widespread prevalence of NP symptoms. It is a severe complication and early diagnosis has prognostic, socio-psychological and better quality of life implications. We present a case of NPSLE who presented with acute confusion, psychosis and cognitive impairments and developed fulminant SLE flare leading to a fatal outcome.

Case: Patient was a 29 year old female with long standing history of SLE, APLA (anti phospholipid antibody syndrome) and depression who was admitted with mild acute pericardial effusion, polyarthritis and was non-compliant on medications. It was noted that besides her depression and refusal of therapy, she manifested progressively worsening cognitive decline and psychotic behavior. Antipsychotic therapy was initiated and she underwent neuroimaging for work up of acute encephalopathy. MRI brain revealed innumerable strokes highlighting concern for CNS vasculitis. CSF analysis revealed elevated CSF protein $\hat{=}$ 294; CSF cell count $\hat{=}$ 1; CSF glucose $\hat{=}$ 50. Extensive rheumatological workup was suggestive of acute SLE flare namely, elevated speckled pattern ANA >6250 and severe hypocomplementemia. The patient was treated aggressively with IV steroids and IV cyclophosphamide but she continued to worsen and developed cardiorespiratory insufficiency, worsening mental status and multiple hematological abnormalities. In spite of aggressive multi systemic symptom management, she developed multi organ failure and complications from ICU stay and eventually was transitioned to comfort care and passed away.

Discussion: According to a set of definitions of 19 NPSLE syndromes and their diagnostic criteria by the American College of Rheumatology (ACR), approximately 40-50% of events are due to primary NPSLE. The long term management of SLE is often complicated by secondary depression and anxiety common to chronic diseases. In this setting, mood and cognitive changes in patients can often be misleading, especially when multiple specialties are involved, creating a diagnostic dilemma. Our patient presented with neurological and psychiatric symptoms secondary to immune mediated vasculitis and went on to develop a fulminant course with multi organ involvement. Onset of NPSLE has a grim prognosis if it presents in the setting of an acute flare up as highlighted here. A higher threshold for this diagnosis can lead to initiation of timely aggressive treatment and may be lifesaving. On the other hand, any new psychiatric changes with a subtle presentation should also be evaluated with a high suspicion since NPSLE is associated with higher morbidity and has implications on quality of life.

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Title: MULTIPLE SCLEROSIS MASKED BY A STROKE

Multiple sclerosis (MS) is a demyelinating disease of the central nervous system, which clinically presents with focal neurological symptoms and may be challenging to differentiate from or may coexist with acute cerebrovascular accident (CVA). Multiple sclerosis carries increased risk of CVA especially in those under 40 years old. Here we will discuss a case of a young woman, who was diagnosed with multiple sclerosis, had a CVA in the past which was likely masking the initial MS presentation.

Case:

A 40-year-old African American, pre-menopausal woman presented with chief complaint of hand clumsiness and unilateral left facial numbness of 3 days duration. Her past medical history was significant for hypertension and CVA 5 years ago. She didn't report interval symptoms after the CVA, and her right sided weakness had completely resolved. However on further questioning she endorsed some loss of color vision and intermittent numbness in her left hand. Her initial CT of the head didn't reveal signs of hemorrhage or acute ischemic changes, but was notable for left parietal lobe white matter disease. On further review of imaging patient was noted to have right frontal lobe white matter disease in addition to lacunar stroke on MRI 5 years ago. Repeat MRI was significant for interval development of greater than 9 periventricular and subcortical white matter lesions, in addition to the previously seen left posterior capsular lesion. Her cerebrospinal fluid analysis revealed presence of oligoclonal bands.

Discussion:

Transient focal neurological deficit is a frequently encountered clinical scenario by the general internist. In this case the patient initially presented with symptoms and MRI findings compatible with a stroke; however, she also had signs of T2 flair white matter hyperintensity. The latter may be attributed to gliosis, demyelination or microvascular ischemic changes. As the patient had a history of hypertension, these changes were attributed to uncontrolled blood pressure and further work up of demyelinating disease was not pursued. This likely led to delay in diagnosis by more than 5 years.

Pathophysiology of lacunar stroke remains incompletely understood but is thought to be related to atherosclerotic disease of penetrating arteries. In the case presented the only risk factor for atherosclerotic disease was hypertension. But hypertension appeared to be mild and well controlled and therefore atherosclerosis as a cause of T2 flair hyperintensity might be put lower on the differential. Moreover, MRI 5 years later revealed demyelination focus in the same area where patient had the lacunar stroke. Thus it remains unclear the focal neurologic symptoms during initial presentation were due to lacunar stroke or underlying demyelination.

Learning objective:

Demyelinating disease should be one of the top differentials in young person presenting with focal neurologic symptoms. CVA may complicate or coexist with multiple sclerosis.

Resident / Fellow Clinical Vignette

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Title: The Dangers of Herbal Supplements: A Case of Acute Liver Injury From Fenugreek

Introduction: Fenugreek (*Trigonella foenum graecum*) is a long established medicine plant that was traditionally recommended for increasing milk production in lactating women. However, research has shown numerous other health benefiting properties such as antioxidant, anti-inflammatory, antidiabetic, hypocholesterolemic, among many others. Although numerous benefits have been suggested with its use, the toxic profile of Fenugreek continues to increase as more research uncovers unintended toxicological effects. Hepatotoxicity has been noted, including necrosis of liver hepatocytes, acute hepatitis and early liver degeneration, especially noted at higher doses.

Case Description: A 34 yo G1P1 female three months s/p uncomplicated vaginal delivery, with no significant medical history, presented with acute onset right upper quadrant abdominal pain that started several hours prior to her arrival in the emergency department, accompanied by several episodes of emesis. Patient was in her usual state of health prior to presentation, only reporting mild difficulty with lactation, for which she had been taking Fenugreek to enhance her milk production for the past 6-8 weeks; she denied alcohol use in setting of breast feeding and was not taking any other over-the-counter medications. During hospital course, patient was noted to have severe transaminitis, with AST/ALT levels reaching 5720/2164. Her physical exam revealed tenderness to palpation of right upper quadrant with no organomegaly and without signs of peritonitis; no jaundice or scleral icterus were noted. A CT abdomen and pelvis with contrast revealed subhepatic fluid consistent with acute liver injury without significant dilatation of common bile duct or stones visualized in a post-cholecystectomy patient; findings from right-upper quadrant ultrasound were similar. All viral causes of hepatitis were excluded, including HBV, HCV, HDV, HEV, EBV IgM (IgG positive in this patient), CMV, HSV, VZV; iron studies were normal, as were Ceruloplasmin levels, quantitative immunoglobulin levels, kappa/lambda ratios, microsomal antibody assays, mitochondrial antibody, smooth muscle antibody, and ANA.

Patient was monitored over several days, with conservative therapy for symptomatic pain control; Fenugreek was discontinued on presentation to emergency room. Liver enzymes continued to downtrend without further intervention. Patient was discharged home and told to avoid any herbal supplements indefinitely.

Discussion: This case illustrates the potential hepatotoxic effects seen with use of Fenugreek, as well as the necessity to consider Fenugreek as a possible etiology of liver injury. When a patient presents with acute liver injury in the absence of common etiologies of acute hepatitis, it behooves clinicians to take a thorough history including over-the-counter remedies, especially non-FDA regulated herbal supplements. As no OTC supplements are currently federally regulated, there is no monitoring system in play to track unwanted side effects, warranting clinicians to broaden their understanding of commonly used supplements.

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Title: Case Report: Streptococcus pneumoniae-related Hemophagocytic Lymphohistiocytosis Treated with IVIG and Steroid therapy

Background

Hemophagocytic Lymphohistiocytosis (HLH) is a rare life-threatening condition that can be a primary or acquired disorder of uncontrolled immune activation of normal T-cells, NK-cells and macrophages. It is characterized by clinical and laboratory evidence of severe inflammation. Here, we report a case of an adult with acquired HLH secondary to *Streptococcus pneumoniae* that responded well to IVIG and steroid therapy.

Case summary

A 41 year-old African-American female with a history of diabetes mellitus type 2 presented with nausea, vomiting, diarrhea and fever, 2 hours after eating Chinese food. She also had a mild upper respiratory tract infection one week prior. Initial physical examination was remarkable for tachycardia of 127 beats per/min, tachypnea of 28 respirations per/min, and fever of 101F. She was admitted to the intensive care unit with a diagnosis of sepsis complicated by disseminated intravascular coagulopathy, respiratory, liver, and renal failure.

Initially, she was treated with broad-spectrum antibiotics with limited clinical improvement. The sepsis workup came back with two blood cultures positive for pan-sensitive *Streptococcus pneumoniae*. Extensive work-up for autoimmune, viral, or other bacterial etiology were unremarkable. Clinical course was complicated by acute pancreatitis and dry foot gangrene despite intact distal pulses. Lab findings were remarkable for: leukocytosis 48,700cell/mm³, hemoglobin 6.8g/dl, platelets 29,000cell/mm³, ferritin 11,000ng/ml, AST 7,913 mg/dl, ALT 2,247mg/dl, fibrinogen 51mg/dl, BUN/Cr 137/13 mg/dL, and triglycerides 1050mg/dl. Clinical suspicion for acquired HLH was high due to her cytopenias, high lipids, low fibrinogen and elevated ferritin level. Modified clinical criteria for HLH was met and then further supported by a 99% probability for HLH (H-score of 261). Based on these findings, IVIG and high dose dexamethasone therapy was initiated and the patient responded to treatment. The final diagnosis of HLH was confirmed with an elevated CD25 soluble receptor assay. Chemotherapy was initially planned but the patient continued to improve clinically and chemotherapy was deferred. She was eventually transferred from the intensive care unit to a subacute rehab facility to continue therapy for critical illness induced myopathy.

Conclusion

From this case, we conclude that prompt recognition and high index of suspicion for HLH is essential for any patient who is acutely ill presenting with unexplained fever, cytopenia, and hyperferritinemia but does not respond to stand of care. Once HLH is suspected, cytogenetic studies and/or bone marrow biopsy is helpful in the diagnosis. However, early treatment with immunosuppressive agents is crucial in improving survival even with an unconfirmed diagnosis as in this case.

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Title: History repeating itself: A case of recurrent stroke from a massive aortic thrombus

Introduction

Atherosclerotic aortic plaques (AAP) are known to increase the risk of stroke.

Current evidence postulates that plaques with certain characteristics are more prone to thrombo-embolic events. In this report we describe the case of recurrent strokes associated with AAP and premature termination of therapy.

Case description

Four years ago, a 77 year old, right handed male, with a history of CAD, HLD, HTN and former tobacco abuse, presented to the emergency department with headache and aphasia. Clinical examination lacked any further neurological deficits. At that time, the CT scan showed a temperoparietal stroke consistent with middle cerebral artery infarct. Subsequent CT angiogram of the head and neck was absent for aneurysm, dissection or carotid stenosis. Neither 24-hour Holter monitoring nor cardiac telemetry during his admission revealed atrial fibrillation. A trans-esophageal echocardiogram showed normal left ventricular function; no intra-cardiac thrombus, but showed multiple mobile plaques, with ulcerated surfaces along the ascending, descending, and origin of the left subclavian arteries. The largest plaque was a 10mm intramural thrombus with adjacent mobile calcified plaque. He was discharged on Warfarin.

A follow up TEE two months later demonstrated improvement with only one mobile plaque but persistent intramural thrombus. Warfarin was to be continued indefinitely.

More recently, he presented with left facial droop, dysarthria and left upper limb weakness. This happened four weeks after he stopped his warfarin secondary to nausea. Initial CT showed no new event, but evidence of an old stroke.

We proceeded with intravenous thrombolysis with alteplase (tPA) with some improvement of his weakness, facial droop and dysarthria. His warfarin was restarted again after two weeks of aspirin therapy.

Discussion

In our case, recurrent ischemic stroke occurred within four weeks of stopping anticoagulation therapy. This highlights the high-risk profile of our patient.

The striking size of the aortic plaque is very unusual, as most of TEE procedures yield no clear cause for "cryptogenic stroke". Although there was no clear benefit over dual antiplatelet therapy, we opted to start warfarin therapy. There are few reports that suggested warfarin therapy improve the plaque burden in these patients and this was demonstrated in our case, but further prospective studies are needed.

Other issues this case brings to the forefront; the indication for TEE in the workup of cryptogenic stroke and follow up of aortic plaques.

Conclusion:

Large mobile ulcerated aortic plaques reflect an extremely high-risk profile for stroke. In patients that have negative cardiogenic and carotid causes of their stroke, a TEE or advanced imaging of the aorta, may be helpful.

More studies are needed to clarify the optimal antithrombotic approach and follow up imaging strategies.

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Title: A Case of Complicated Native Valve Endocarditis due to Corynebacterium Striatum

INTRODUCTION: Corynebacterium striatum is an aerobic gram-positive bacillus that is part of the normal flora of skin and mucosal membranes. In a hospital setting it is usually considered non-pathogenic contaminant. However, it still can cause serious infections such as endocarditis affecting native or prosthetic valve.

CASE PRESENTATION: A 69-year-old female with a previous history of hypertension, Chronic Kidney Disease on hemodialysis and metabolic syndrome presented with worsening renal function and right lower extremity cellulitis. Patient denied any fever, chills, chest pain, shortness of breath, abdominal pain, headache or focal deficits. Vital signs on presentation included blood pressure 106/64, pulse 81, oral temperature 36.8 °C (98.3 °F), respiratory rate 22 breath/minute and Oxygen saturation 94 % on room air. Physical exam revealed a morbidly obese lady, no acute distress, normal cardiopulmonary and abdominal exam. There was right thigh eschar and surrounding erythema. Laboratory workup revealed WBCs 11.1, Hemoglobin 8.2, Creatinine 3.2 (baseline creatinine was 1.5), urinalysis was unremarkable. X-ray tibia didn't show any osteomyelitis. She was treated empirically with Clindamycin. Blood culture grew Corynebacterium Striatum sensitive to Vancomycin (MIC <=1 4 mg/L) but resistant to Penicillin (MIC = 4 mg/L), Therefore antibiotic was switched to Vancomycin. Initial transthoracic echocardiogram (TTE) and transesophageal echocardiogram (TEE) showed mild aortic regurgitation and no evidence of endocarditis. Hospitalization was complicated with encephalopathy for which MRI brain performed and revealed bilateral frontal punctate infarct. Also patient was found to have free air in the abdomen confirmed by CT abdomen, underwent exploratory laparotomy which revealed perforated sigmoid that was resected and she was transferred to the ICU for mechanical ventilation. She remained encephalopathic, repeated blood cultures again grew Corynebacterium Striatum. Repeated TTE showed severe aortic valve regurgitation and raised a suspicion for vegetation, so TEE was done and confirmed aortic valve vegetation that correlated with the brain emboli and patient's mental status. Patient didn't show any significant improvement despite aggressive antibiotics treatment. Family chose comfort care for her and patient passed away.

DISCUSSION: Corynebacterium species are usually considered contaminants when isolated from blood culture, and, hence ignored. Our case is instructive because it demonstrates that Corynebacterium can cause significant disease, and when isolated from multiple blood cultures should be considered a pathogen and treated.

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Title: ANEMIA AND HYPOTHYROIDISM ; STOP THE THYROID INSANITY .**Introduction:**

Hypothyroidism can cause various types of anemia and comprehensive workup is essential to identify the underlying etiology and direct management. We report a case of severe macrocytic anemia secondary to hypothyroidism per se in the absence of vitamin B12 and folic acid deficiencies which is an association that has been rarely reported in the literature.

Case:

A 22-year-old lady with history of primary hypothyroidism presented to the emergency with worsening fatigue and dizziness over one week, associated with exertional dyspnea. She denied any blood loss, constipation or cold intolerance. She was off levothyroxine for over seven years due to non compliance. On physical exam: BP 85/50 mmHg, HR 111 bpm, BMI 24.1, she had bilateral conjunctival pallor, ejection systolic murmur, delayed relaxation phase of tendon reflexes and neck exam showed no palpable thyroid. Her Labs showed HB 3.6 gm/dl, HCT 11.8 %, MCV 105.4, TSH 524.38 uIU/ml, FT4 0.2 (0.9-1.8 ng/dl) and T3 42.8 (60-180 ng/dl). She was admitted as a case of severe anemia and hypothyroidism. Further work up for the anemia showed normal iron studies, normal B12 and methyl malonic acid, normal folic acid and her hemolytic work up (T. bilirubin, Haptoglobin, corrected reticulocyte count, LDH) was negative. She received total of four units of PRBCs during her hospitalization. She did not meet the criteria for myxedema coma diagnosis and was started on oral levothyroxine replacement. Her anemia was attributed to severe chronic hypothyroidism by exclusion. Her symptoms improved over the next two days, her hemoglobin was 8.1 g/dl, she was discharged to follow up with endocrinology.

Discussion:

Anemia is reported in 20-60% of the patients with hypothyroidism (overt and subclinical). Anemia in hypothyroidism is characteristically normochromic normocytic related to inhibition of erythroid colony development, reduction in oxygen distribution to tissues and low erythropoietin level in the absence of thyroid hormones. Normalization of hemoglobin level occur with replacement of thyroid hormone. Iron deficiency resulting from iron malabsorption and menorrhagia is the second common cause. Macrocytic anemia can occur secondary to vitamin B12 deficiency (pernicious anemia) or rarely to hypothyroidism per se. Association between the degree of severity of the anemia and the severity of hypothyroidism has been a controversial issue and in our patient the severity of anemia and the macrocytosis can be explained by the long standing untreated hypothyroidism.

This case highlight the importance of recognizing hypothyroidism as a cause of anemia and the assessment of thyroid function as a part of workup of anemia of unknown etiology.

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Title: Reversible Findings on Early CT Head Imaging Leading to Delay in Diagnosis of Cerebral Air Embolism

Introduction: Paradoxical cerebral air embolism (CAE) is a rare, underdiagnosed cause of stroke that can be associated with manipulation of central venous catheters. Initiation of hyperbaric oxygen treatment in less than eight hours has been associated with improved neurological outcomes (1). Therefore, a high clinical suspicion and early diagnosis of CAE are of paramount importance.

Case Description:

A 37-year-old woman with history of quiescent systemic lupus erythematosus, seizure disorder, and end-stage renal disease came to the ED after having witnessed, generalized tonic-clonic seizure. The patient had just finished routine hemodialysis via established central venous catheter. Following the seizure, she awakened with left hemiplegia, left homonymous hemianopsia, right gaze preference, and right leg weakness. Her National Institutes of Health Stroke Scale (NIHSS) score was 14, consistent with moderate-to-severe stroke.

Non-contrast CT head 90 minutes after seizure was negative except for diffuse hypodensities along sulci of right frontal, parietal, and occipital lobes, suggestive of fat or air (versus artifact). CT angiography was negative and there were no other obvious mechanism(s) for stroke. Repeat CT Head performed 150 minutes post-seizure no longer demonstrated abnormalities seen on initial CT scan. Brain MRI on day of admission showed no acute findings. Given her seizure presentation, presumptive diagnosis of postictal Toddâ€™s paralysis was entertained. Patient was admitted to the general medical floor and treated with supplemental oxygen and anticonvulsants.

Her mental status further deteriorated requiring transfer to the ICU. Her hemiplegia and hemianopsia persisted.

Subsequent MRI Brain done two days after admission now showed unusual right hemispheric gyral hyperintensities, bright on diffusion-weighted imaging (DWI) but dark on apparent-diffusion coefficient, consistent with ischemic stroke. Given lack of clear mechanism and source, with negative CTA, further review of initial head CT suggested cause was air embolism, likely introduced through her dialysis catheter. Given suspicion for presence of likely shunt [patent foramen ovale (PFO)], transthoracic echocardiogram with bubble was performed; it was strongly positive.

Discussion:

Diagnosis of paradoxical CAE is a difficult diagnosis to make. In our case, there was delay in diagnosis due to reversible findings on early imaging. Repeat head CT done 60 min after the first CT scan no longer showed diffuse hypodensities suggestive of fat or air embolus. It is important to recognize that follow-up imaging may be negative as soon as 2.5 hours after CAE(1). A PFO was found in our patient. This suggested venous air introduced through dialysis catheter, shunted to arterial side via PFO, went to brain vessels causing stroke. A high index of suspicion for paradoxical CAE should be entertained in the setting of recent central venous catheter access in patients with no other obvious sources of stroke.

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Title: A SUDDEN CLOTTING DILEMMA – A CASE REPORT OF ACQUIRED FACTOR VIII DEFICIENCY

Introduction

Acquired Factor VIII deficiency is rare with estimated incidence 1.3 to 1.5 cases per million populations per year. Case

78 year old female with history of hypertension and hyperlipidemia presented with intermittent hematuria following a mechanical fall at home 2 weeks prior. Patient complained of weakness, fatigue, light-headedness and loss of appetite. She presented to her physician who referred her to hospital for low Hemoglobin/Hematocrit (Hb g/dL/Hct %). Patient presented with Hb/Hct of 6.4/22.1 and elevated creatinine of 3.26 mg/dL. She received 1 unit of packed red blood cells (PRBC) but Hb continued to drop to 5.5. She was noted to have partial thromboplastin time (PTT) of >200sec with no history of anticoagulation or clotting deficiency. CT scan of abdomen without contrast showed peritoneal and retroperitoneal hemorrhages bilaterally and suspected right renal hemorrhagic products in collecting system with moderate hydronephrosis. She was transferred to intensive care unit for monitoring. Mixing studies were done which showed non correction with pooled normal plasma and increasing with incubation. Rheumatological work up was unremarkable. Patient received multiple units of PRBC, fresh frozen plasma, platelets and 3 units of Recombinant Factor VII with eventual stabilization of Hb/Hct. Bethesda assay was sent and Factor 8 activity was <1%. Patient started on rituximab 600mg x 3 doses with 4th dose as outpatient and prednisone 60mg daily. Repeat CT abdomen showing stable retroperitoneal bleed. Patient was discharged. At 3 month follow up, she remained in remission after completion of prednisone/rituximab therapy.

Discussion

The approach to treatment for acquired factor VIII deficiency is two-fold. Initially the aim is to stop the potentially life threatening bleeding event and secondarily to reduce the antibody titers to factor VIII. Spontaneous resolution of Factor VIII inhibitors occurs in up to 30% of patients however because average time to spontaneous resolution is 21 months physicians usually recommend immunosuppressive treatment. The use of Rituximab for the treatment of this condition was first reported in 2000 with a single case report. It has been proposed in combination with steroids as a less toxic regimen for persons not able to tolerate other immunosuppressive regimens like cyclophosphamide. When steroids are used alone, patients usually relapse once tapered completely off while retrospective analysis show persons who use combination rituximab and steroids are able to maintain remission when steroids stop. The relapse rate of regimens after a first complete remission has been estimated at about 20 percent however 70 percent of such relapsing patients achieve a second complete remission and is not affected by choice of 1st regimen. For this reason combination Rituximab with steroids appears to be a safe first line option especially in older patients who cannot tolerate other regimens.

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Title: INVOKANA: A RARE CAUSE OF PERSISTENT ANION GAP IN DIABETIC KETOACIDOSIS

Introduction

Sodium-glucose co-transporter 2(SGLT2) inhibitors are FDA approved in patients with type 2 diabetes mellitus(DM). They block glucose reabsorption in the proximal convoluted tubules and increase glucosuria. Very rarely, their use leads to Diabetic Ketoacidosis(DKA) in type 2 DM, at glucose levels of <250 mg/dl with a distinctly difficult to close persistent anion gap.

Case: 64-year-old male with type 2 DM presented with progressive fatigue, myalgias and worsening appetite of three-week duration. He denied any fever, chills, cough, nausea, vomiting, diarrhea or recent surgery. His medications included Metformin (1000mg BID), Invokana (300mg daily), Victoza (1.8mg daily). Physical examination was unremarkable except for dry mucous membranes. On initial assessment, he had glucose level of 213 mg/dl, HCO₃ of 16 mmol/L, anion gap of 24 mmol/L, beta hydroxybutyrate of 3.7 mmol/L, WBC count of 18x10³/L. Urine examination showed ketonuria and glucosuria. He was managed with insulin drip, IV fluids and other supportive care in medical ICU. This was the first documented episode of DKA in this patient. All the body fluid cultures eventually turned out to be negative. Despite aggressive medical therapy and comprehensive workup of common precipitating factors, the anion gap could not be closed for over a week. Reviewing his medications revealed Invokana as the only new introduction in the last few months. Thus, a final diagnosis of Invokana induced DKA was made.

Discussion: Ketones like acetoacetate and betahydroxybutyrate are acidic fuel molecules produced through fatty acid oxidation when dietary carbohydrates are limited. DKA results from a combination of glucagon elevations, which promote a shift to fat metabolism, and insulin deficiency which may manifest as either absolute or relative, coupled with severe insulin resistance. The glucose lowering property of SGLT2 inhibitors leads to a decrease in circulating insulin levels, which increases the rates of lipolysis in adipose tissue and ketogenesis in the liver, ultimately resulting in elevated circulating ketone body levels. A subsequent compensatory increase in gluconeogenesis in the liver accounts for the increased urinary excretion of glucose. The resultant glucagon elevation drives the production of ketone bodies. According to recent reports, SGLT2 inhibitors also act by increasing preproglucagon gene expression by acting directly upon pancreatic α -cells. Furthermore, phlorizin (a nonselective inhibitor of SGLT1 and SGLT2) has been demonstrated to increase renal tubular reabsorption of acetoacetate. If selective SGLT2 inhibitors mimic this action of phlorizin, it is possible that they could also decrease renal clearance of ketone bodies thus promoting ketoacidosis.

Conclusion: Clinicians need to be aware of this rare but life-threatening adverse effect of Invokana in patients with type 2 DM. The unique feature of this entity is that the glucose levels are generally within 250 mg/dl and thus are likely to be overlooked.

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Title: A Rare Case of Tuberculous Ileitis in An AIDS Patient in the Absence of Pulmonary Involvement

Introduction: Tuberculous ileitis (TI) is a rare entity even in an immunocompromised patient. Its diagnosis is often challenging and difficult especially without pulmonary symptoms. We present a case of tuberculous ileitis in an AIDS patient without any pulmonary syndrome.

Case Description: A 32-year old Gambian woman, who immigrated 11 years ago with newly diagnosed AIDS, CD4 T cell count=18/cumm and viral load=77,500 copies/ml on antiretroviral therapy for a month presented with generalized intermittent cramping abdominal pain, fever, unspecified weight loss, intermittent vomiting as well as constipation for three weeks. This was patient's third visit for the same complaint. Her laboratory results were remarkable for white blood cells of 7.3/nL with a left shift, no eosinophilia, Hemoglobin of 8g/dL, positive Quantiferon test from an outside hospital.

The rest of laboratory tests and chest radiograph were normal. Fungal cultures and blood Acid fast bacilli (AFB) were negative. She otherwise denies constitutional symptoms. Physical examination was unremarkable except for abdominal distention and tenderness in right lower quadrant. Computer tomography showed transmural wall thickening in terminal ileum. She showed no improvement on antibacterial treatment. As part of a work up for ileitis in an immunocompromised patient she had colonoscopy with biopsy showing erythematous mucosa in the terminal ileum, ulceration and friability and possibly fistulous lesion with poorly formed non-necrotizing granulomata, but AFB and fungal stains were negative. However given the high suspicion for TI a regimen of isoniazid, rifabutin, pyrazinamide and ethambutol were started. Eventually her biopsy cultures and AFB stool culture became positive for Mycobacterium Tuberculosis Complex. Patient eventually showed significant clinical improvement, and was discharged with tuberculosis regiment.

Discussion: Extra pulmonary tuberculous (EPTB) accounts for about 50% of cases in HIV positive patients, and intestinal TB is the sixth most prevalent form of EPTB. The ileocecal and jejunoileum are the most common sites involved, accounting for about 90% of ITB cases. The diagnosis is often difficult and challenging to establish without high clinical suspicion, moreover AFB stains are positive on tissue samples in less than 30% of cases, and TB culture remains the gold standard for diagnosis which may take up to eight weeks to grow. Therefore, clinicians should maintain a high suspicion and consider the possibility of TI in all patients with abdominal complaints, particularly in immunocompromised patients as misdiagnosis or delay in diagnosis can have consequences.

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Title: Evolution from Mixed Connective Tissue Disease to Systemic Lupus Erythematosus: A Case Report with Eight-Year Follow Up

Introduction: Mixed connective tissue disease (MCTD) is an uncommon disease with overlapped clinical characteristics of certain connective tissue diseases in presence of high titer anti-ribonucleoprotein antibodies (anti-U1RNP). The evolution of MCTD to systemic lupus erythematosus (SLE) is rare and very few cases have been reported. Here we present a case of MCTD that later on developed into SLE during a period of eight years.

Case Description: A 44 years old Hispanic woman presented with six months' Reynaud's phenomenon, generalized myalgia and arthralgia. Physical examination showed multiple joints tenderness. Serology evaluations revealed high titer of anti-U1RNP, antinuclear antibodies, complement levels were normal, and rest rheumatologic test including anti-smith, anti-double strand DNA (anti-dsDNA), anti-cyclic citrullinated peptide antibodies and rheumatic factor were all negative. Patient was diagnosed with MCTD. The case was also complicated with thrombotic thrombocytopenic purpura. Patient had a prompt response to glucocorticoids therapy together with plasmapheresis with rapid normalization of hematological test results. The serologic markers remained stable; however the arthralgia persisted despite different regimens of immunosuppressants. Eight years later patient presented again with complicated syndromes, including renal dysfunction, perimyocarditis and pulmonary hypertension. The anti-U1RNP titer had decreased; meanwhile both anti-SM antibodies and anti-dsDNA antibodies became positive and complement level decreased significantly. Renal biopsy also showed features of lupus nephritis. The diagnosis of SLE was made by systemic lupus international collaborating clinics classification criteria.

Discussion: The progression of MCTD to SLE is usually rare. The patient in our report demonstrated classical clinical and serologic features of the two diseases that occurred sequentially during its prolonged course. Along with it there were also multiple rare complications, including TTP, pulmonary hypertension, and perimyocarditis. Physicians should be aware of this rare transformation during long term follow ups as different organ systems may be involved and different strategy of management should be considered accordingly.

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Title: LIFE THREATENING THROMBOCYTOPENIA SECONDARY TO TRIMETHOPRIM/SULFAMETHOXAZOLE

Thrombocytopenia is a common side effect of Trimethoprim/Sulfamethoxazole (TMP/SMX) but it is usually mild-to-moderate in severity. We report a case of severe and possibly life threatening thrombocytopenia associated with TMP/SMX therapy.

A 92-year-old female presented to the emergency department with intractable bleeding from a laceration on her left leg, following a mechanical fall. She denied any rash, bruising or bleeding from the gums or other orifices. She had a history of hypertension, hyperlipidemia and chronic leg edema with recurrent cellulitis. Her home medications included nebivolol, torsemide, losartan, atorvastatin and oxybutynin. On examination, vital signs were stable. Physical examination was unremarkable except for a small laceration on her left shin, with constant oozing of blood. No purpura or mucosal hemorrhage was noted. Stool guaiac test was negative. Complete blood count revealed WBC $9.4 \times 10^9/L$ ($9.4 \times 10^3/mm^3$), hemoglobin 12.5g/L (12.5 g/dL) and platelets $5 \times 10^9/L$ ($5 \times 10^3/mm^3$). A repeat platelet count was $4 \times 10^9/L$. Prothrombin time was 11 seconds and INR 1. BUN and creatinine were 26.07 mmol/L (73mg/dL) and 435.91 mol/L (4.93mg/dL) respectively. Peripheral blood smear showed severe thrombocytopenia with no platelet clumping and no evidence of fragmented cells or schistocytes to suggest thrombotic thrombocytopenic purpura. Serum LDH, thyroid functions and vitamin B12 levels were normal. One month ago, platelets count was $165 \times 10^9/L$ and creatinine 79.58 mol/L. It was later determined that 2 days prior to admission, the patient had completed a 10-day course of TMP/SMX prescribed by her primary care physician for cellulitis of the lower extremities. Naranjo algorithm score was 7, indicating a probable adverse drug reaction to TMP/SMX resulting in thrombocytopenia. TMP/SMX was discontinued and 1 unit of platelets was transfused, with increase in platelet count to $108 \times 10^9/L$. The patient's renal function also improved with IV fluids and discontinuation of TMP/SMX. She was discharged on day 3 of admission.

Thrombocytopenia associated with TMP/SMX is an immune-mediated process resulting in platelet destruction by drug-dependent platelet antibodies. Treatment of thrombocytopenia associated with TMP/SMX therapy includes discontinuation of the offending drug and the use of corticosteroids. Platelet transfusion and intravenous immunoglobulin may be required in some patients. Severely low platelet counts of $\approx 10 \times 10^9/L$ have been implicated in severe spontaneous bleeding on rare occasions. Thus, thrombocytopenia associated with TMP/SMX carries potential life threatening complications. This hematologic adverse effect of TMP/SMX, which appears to be dose/duration independent, should warrant careful monitoring of complete blood cell count, especially platelet count, before and during TMP/SMX therapy. In an era where TMP/SMX may be increasingly prescribed for possible community acquired methicillin-resistant Staphylococcus aureus (MRSA) infections, physicians should be aware of this potential life threatening toxicity.

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Title: Look in the abdomen for Eggerthella bacteremia

Learning objective

To evaluate for gastrointestinal (GI) pathology in a patient with Eggerthella bacteremia

Introduction

Eggerthella lenta is an anaerobic, gram positive bacillus commonly associated with GI illness. With improved diagnostic facilities, more E. lenta isolates are currently being recognized. We report a case of E. lenta bacteremia in a patient with gastroenteritis.

Case

A 70 year-old woman presented with respiratory distress in the setting of multiple episodes of vomiting and diarrhea for one day. Her past medical history included asthma, diabetes, hypertension, and anxiety disorder. She was alert and had bilateral rhonchi with coarse breath sounds. Arterial blood gas revealed metabolic acidosis with lactate of 3.8 mg/dl. Chest X ray revealed pulmonary vascular congestion and right infrahilar opacity. She was intubated for increased work of breathing after she failed noninvasive ventilation. She was treated with antibiotics for possible aspiration pneumonia, while awaiting the cultures. She was successfully extubated after 2 days. Meanwhile, her blood cultures grew gram positive cocci in pairs and chains along with gram positive bacillus. The organisms were identified to be Streptococcus mutans and Eggerthella lenta. Metronidazole was added for E. lenta coverage. CT abdomen was performed to identify any intra-abdominal source of infection, but the patient could not tolerate the test. She was discharged on oral Augmentin to cover both the organisms.

Discussion

Eggerthella lenta is an anaerobic, gram positive bacillus commonly associated with gastrointestinal tract pathology. It was first described in 1935 by Arnold Eggerth. E. lenta is a normal intestinal microbiome that is being increasingly recognized in the modern clinical microbiology laboratory including matrix-associated laser desorption ionization-time of flight mass spectrometry (MALDI-TOF). E. lenta causes a wide spectrum of disease ranging from asymptomatic bacteremia (seen in transient GI illness), to polymicrobial bacteremia from intra-abdominal source (perforated viscus), to severe monomicrobial disseminated disease. It has been associated with ulcerative colitis, crohn's disease, hepatobiliary diseases, abscesses, diverticular disease, appendicitis, malignancies, decubitus ulcers, and pelvic inflammatory disease. Abdominal imaging should always be performed for E. lenta bacteremia even though the diagnosis is not apparent at the time of isolation. E. lenta bacteremia has a high mortality rate ranging from 36% to 43% and often warrants ICU level of care. E. lenta is susceptible to ampicillin-sulbactam, metronidazole, and carbapenems. In our patient, E. lenta bacteremia was polymicrobial and was likely due to aspiration of the gastric contents during the GI illness, which responded well to beta-lactam and metronidazole. Thus, E. lenta should not be considered as a contaminant when identified in blood cultures and a detailed evaluation with appropriate abdominal imaging should be done.

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Title: Localized mesenteric vasculitis with response to adalimumab while tapering prednisone

Mesenteric vasculitis is rare in the absence of Takayasu or giant cell arteritis. Localized mesenteric vasculitis (LMV) is a syndrome of abdominal pain associated with computerized tomography (CT) angiographic findings of segmented narrowing, dilation, occlusion or aneurysm of gastroenterological vessels [1].

A 45-year-old Caucasian male with no past medical history presented to our Emergency Department with 10 days of progressively worsening abdominal pain. The pain was diffuse and burning in nature, and exacerbated by food. He denied any nausea, vomiting or change in bowel habits. He was afebrile, with blood pressure of 126/81, and normal sinus heart rate of 75. Abdominal exam was notable for normal bowel sounds, tenderness to palpation in all four quadrants; no rebound or guarding; stool was negative for occult blood. There were no vascular bruits and pulses were normal. Erythrocyte sedimentation rate (ESR) was 49mm/h, and CRP 3.9mg/dL, WBC and lactic acid were normal. Anti-nuclear antibody, rheumatoid factor, anti-neutrophil cytoplasmic antibody, and hepatitis panel were negative. Contrast enhanced abdominal CT showed a moderate length segment of superior mesenteric artery ectasia with subtotal occlusion and surrounding infiltration, which is consistent with LMV.

The patient was treated with prednisone 80 mg /day and symptoms improved by day 2, and resolved by day 3. While prednisone was tapered, 10 mg of weekly methotrexate (MTX) was added at 4th week. At week 20, when prednisone was tapered to 40 mg daily, he developed recurrent abdominal pain. A trial of leflunomide failed. At week 21-23, patient received two dose of adalimumab, with complete relief of abdominal symptoms. From month 5-10, prednisone was tapered to 10mg daily. Repeat CT at 5 months and 8 months after discharge showed resolution of the vascular abnormalities. At one year after discharge, his treatment includes prednisone 10mg/day, MTX 10mg weekly, and Adalimumab twice monthly. The patient has not had abdominal complains since 1 year discharge.

Vasculitis of the mesenteric artery is rarely diagnosed as an isolated disease. Prednisone and methotrexate have been used to treat this condition. To our knowledge, this is the first report of successful treatment of LMV with a combination of prednisone, MTX and adalimumab. The addition of Adalimumab should be considered while treating LMV, especially while tapering prednisone.

[1]Salvarani C, Calamia KT, Crowson CS et al, Localized vasculitis of the gastrointestinal tract: a case series. *Rheumatology* 2010;49:1326-1335

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Title: AN INTERESTING CASE OF SYSTEMIC MUCORMYCOSIS IN A 57 YEAR-OLD-MALE PRESENTING WITH UPPER GASTROINTESTINAL BLEEDING AND BILATERAL HYDRONEPHROSIS**Introduction:**

Mucormycosis is a rare fungal disease caused by members of the Mucorales with a high mortality. Mucorales species are vasotrophic organisms that may cause angioinvasive disease in immunosuppressed hosts. Risk factors include diabetic ketoacidosis, chronic kidney disease, organ or bone marrow transplantation, burns, malignancies and steroid therapy. Here we describe a case of Systemic Mucormycosis presenting with upper gastrointestinal bleeding and obstructive uropathy.

Case presentation:

A 57 year-old-male with past medical history of coronary artery disease, systolic heart failure, alcohol abuse and stage IV sarcoidosis on long-term prednisone presented with hematemesis and melena that started the night before his arrival to the Emergency Department. Patient was noted to have tachycardia, hypotension, and paleness. He was admitted to the medical ICU for hypovolemic shock secondary to massive bleeding that required immediate endotracheal intubation for airway protection. Fluid resuscitation, blood products replacement, therapy with vasopressors and pantoprazole drip was provided. An emergent endoscopy revealed a bleeding Dieulafoy lesion that was promptly clipped. CT of abdomen requested to rule out ischemic colitis in the setting of progressive abdominal distention showed a diffuse pericolic inflammation most pronounced at the rectosigmoid colon. Repeat endoscopy with biopsy revealed necrotic debris with acute leucocytic exudate and numerous variably sized, 90-degree angulated fungal hyphae favoring Phycomycosis species. Patient was started liposomal B-amphotericin however his renal function deteriorated and treatment was switched to isavuconazonium sulfate. Repeat CT of abdomen showed mild to moderate hydronephrosis with hydronephrosis and bladder wall thickening. Cystoscopy showed distorted anatomy of bladder lumen with multiple mounds of tissues all throughout the bladder most notable in the trigone with failure to locate ureteral orifices. Random biopsies showed necrotic debris with an active inflammation and numerous variably sized angulated fungal hyphae consistent with phycomycosis species. Hydronephrosis significantly improved after placement of bilateral nephrostomy tubes. Subsequently, continuous bladder irrigation with Amphotericin B for management of bladder mucormycosis was offered. After three weeks of systemic treatment with isavuconazole, a third endoscopy showed inflammatory changes with a pathologic report in which phycomycosis was no longer appreciated. His kidney function remained stable even after clamping and removal of his nephrostomy tubes. Patient remained asymptomatic after completion of his mucormycosis therapy for six months.

Conclusion:

Early diagnosis of mucormycosis is challenging given the diverse clinical manifestations. It is classified as rhino-orbital-cerebral, pulmonary, cutaneous, gastrointestinal, disseminated and miscellaneous types. The correction of the underlying conditions and interventions such as removal of infected tissue with appropriate antifungal therapy has shown to improve the survival outcomes on affected individuals. Current available therapies include Amphotericin B, posaconazole and recently approved isavuconazonium. High clinical suspicion and multidisciplinary team approaches are often required.

<p>Author: Rami Jabbour, MD Additional Authors: Shaheen Alvi, MD, Charbel Ishak MD, Sridhar Chilimuri MD Institution: Bronx Lebanon Hospital Center, Bronx, NY</p> <p>Title: CEREBROSPINAL FLUID LEAK: A Diagnostic algorithm for a rare illness</p> <p>BACKGROUND: Cerebrospinal fluid leak (CSF leak), although rare, is a well-recognized cause of headache. The annual incidence of spontaneous CSF leak was estimated to be 5/100,000. Since headache is a common condition it is unclear when to suspect CSF leak requiring additional workup. We propose a diagnostic algorithm based on our experience. We present four illustrative clinical cases to explain this algorithm.</p> <p>CASE REPORTS: Case 1: A 49 year-old woman presented with postural headache. She had recurrent spontaneous right ear fluid leak and right nostril rhinorrhea of 6 month duration. There was no history of trauma. Computed tomography (CT) cisternography revealed CSF leak. Case 2: A 57 year-old man presented with postural headache associated with neck, shoulder and back pain. Six months prior to presentation, he had history of a closed head trauma. CT cisternography revealed CSF leak in left maxillary and frontal sinuses. Case 3: A 55 year-old man presented with postural headache and photophobia one-month after cervical laminectomy. A diagnostic spinal tap revealed meningitis. CT cisternography revealed a 7x6x15 cm fluid collection overlying the cervical spine compatible with CSF leak. Case 4: A 50 year-old immunocompetent woman presented with headache, nausea and vomiting. She was diagnosed to have cryptococcal meningitis. She reported a remote history of closed head trauma and on further questioning reported intermittent rhinorrhea. CT cisternography revealed right nostril CSF leak.</p> <p>DISCUSSION: Postural headache is an important feature of CSF leak. However, by itself, lacks the sensitivity or specificity to suspect a CSF leak. Postural headache with a history of head trauma/surgery appears to increase the possibility of a CSF leak. The presence of postural headache, with history of head trauma and the presence of unusual meningitis should prompt a search for CSF leak. It is well recognized that postural headache with rhinorrhea should prompt search for a CSF leak. CT cisternography remains the gold standard for diagnosing CSF leak. CSF leak can lead to life threatening complications including meningitis in 25-50% of cases.</p> <p>CONCLUSION: Both spontaneous and head trauma related CSF leaks are rare conditions. However, when unrecognized could lead to life threatening complications. Early recognition could lead to interventions with excellent outcomes. Postural headache, history of trauma/surgery (recent and remote) and the presence of unusual causes of meningitis should prompt work up for CSF leak. CT cisternography remains the gold standard for diagnosing CSF leak.</p>	<p>Author: Tracian James-Goulbourne, MBBS Additional Authors: O Barrett-Campbell, C Goulbourne, R Hardie, G Briefel Institution: SUNY Health Science Center at Brooklyn (Downstate)</p> <p>Title: A RARE PRESENTATION OF LACTIC ACIDOSIS</p> <p>Introduction: Type B lactic acidosis (Type B LA) secondary to underlying malignancy is a rare paraneoplastic phenomenon and a life-threatening oncologic emergency. It is most commonly associated with hematological malignancies, but may be seen in solid tumors. The pathogenesis, which remains poorly understood, is likely multifactorial and possibly explained by enhanced aerobic glycolytic activity in malignant cells.</p> <p>Case: A 59-year-old man with a history of Helicobacter pylori gastritis presented to hospital with several weeks of epigastric pain associated with non-bloody emesis and a 35-pound unintentional weight loss. Physical examination was significant for a cachectic middle-aged man with epigastric tenderness and hepatomegaly. Initial labs were significant for anemia of chronic disease, transaminitis and a normal lactate. Abdominal computed tomography (CT) scan showed multiple liver masses with retroperitoneal lymphadenopathy. Ultrasound guided liver biopsy showed poorly differentiated adenocarcinoma. Tumor markers: alpha-fetoprotein, cancer antigen 19-9 and carcinoembryonic antigen were significantly elevated. He improved after conservative management and was discharged with outpatient oncology follow-up. He presented eight days later with dyspnea and right upper quadrant abdominal pain. On examination he was tachypneic and tachycardic with a blood pressure of 124/82 mmHg and oxygen saturation 99% on room air. He was icteric with right upper quadrant tenderness. Labs showed worsening anemia and transaminitis with a high anion gap metabolic acidosis (lactate - 11.1 millimoles/liter), for which bicarbonate infusion was commenced. CT pulmonary angiogram ruled out pulmonary embolism but showed right sided pleural effusion. Thoracentesis revealed atypical cells suspicious for malignancy. The patient deteriorated with multi-organ failure; lactic acid level trended up to 23 millimoles/liter. Nephrology was consulted for refractory metabolic acidosis and the patient was determined to have Type B LA. Renal replacement therapy (RRT) was deferred due to significant coagulopathy. He died in intensive care unit on the sixth day of admission.</p> <p>Discussion: This case highlights several important points. Type B LA secondary to underlying malignancy is a rare but important entity and should be considered early in patients who present without evidence of tissue hypoperfusion. The liver is an important site of lactate clearance and liver metastases with impairment has been reported as an attributable factor in decreased utilization of lactate. However, there have been several reported cases of Type B LA with no evidence of liver involvement. This signifies that lactic acidosis can occur without hepatic impairment and decreased hepatic clearance of lactate is not essential in the pathogenesis of Type B LA. Finally, bicarbonate infusions and RRT are important supportive measures for managing metabolic derangements. It is important to emphasize that mortality benefit and improved outcomes are less likely to be seen without successful treatment of the underlying malignancy.</p>
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**Title: STEROID RESISTANT NON-GRANULOMATOUS
ULCERATIVE JEJUNITIS: A RARE CASE OF PROTEIN LOSING
ENTEROPATHY**

Introduction

Non-granulomatous ulcerative jejunitis is one of the rare causes of protein-losing enteropathy. Steroid is the primary modality of treatment. There is very limited case reports on treatment options for patients with refractory symptoms after steroid therapy. We are presenting a case of non granulomatous ulcerative jejunitis resistant to steroid therapy, but responsive to azathioprine treatment.

Case description

A 62 year old female was transferred for symptoms of diarrhea, nausea, vomiting, diffuse abdominal discomfort and generalized anasarca (causing 30lb weight gain) for 3.5 months. She was found to have hypoalbuminemia (1.4gm/dl; baseline 4.0gm /dl) and was started on IV albumin infusions and furosemide for symptomatic treatment. Albumin levels improved initially to 3.4gm/dl on IV albumin supplementation. She had no proteinuria and no signs of liver disease to explain hypoalbuminemia. Other pertinent laboratory data included normal CRP, ESR, SPEP, UPEP, as well as negative tissue transglutaminase IgA and IgG, negative HIV serology and negative CMV and HSV serologies. Stool studies were negative for bacteria, ova and parasites, giardia. She underwent two esophagogastroduodenoscopies (EGDs) which revealed normal gastric folds and were overall endoscopically normal. Colonoscopy showed diverticulosis of descending colon but was otherwise normal. Biopsies of stomach, duodenum and colon were all normal. MR enterography revealed normal small bowel anatomy. Small bowel video capsule endoscopy demonstrated denuded, erythematous mucosa with superficial ulcerations and erosions. A push enteroscopy then revealed jejunal mucosa with erosions and erythema. Jejunal biopsies showed focal partial villous blunting associated with loss of brush border along with marked reactive/reparative epithelial changes. No granuloma or dysplasia was seen. Based on the symptoms, laboratory data, imaging and endoscopic studies, and biopsy results, patient was diagnosed with non-granulomatous ulcerative jejunitis and was started on 14 day steroid therapy (60mg prednisone daily) and taper over 6-8 weeks. Patient improved symptomatically and albumin levels improved and IV supplementation was stopped. After the steroid taper was initiated, albumin levels trended down and she was supplemented intermittently with IV albumin. TNF alpha inhibitors and alternative therapeutic agents were considered. Infliximab was not started due to patient's history of shingles and her unwillingness to take antiviral prophylaxis. Patient was started on Azathioprine, after which her symptoms resolved, including her anasarca, and her albumin levels normalized. She is now coming off of her albumin infusion while steroids are nearly fully tapered off of her regimen.

Conclusion

Non granulomatous ulcerative jejunitis is a rare disease, with poor prognosis. Azathioprine can be considered a viable option for treatment among steroid resistant population.

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Institution: Westchester Medical Center**Title: The Role of Complement Inhibition in Catastrophic
Anti-Phospholipid Syndrome (CAPS)**

Introduction: CAPS is a severe form of anti-phospholipid syndrome (APS), characterized by diffuse vascular thrombosis leading to multi-organ failure, usually occurring over a short time. Steroids and rituximab, along with plasmapheresis, have long been the standard of care. Recently, the role of complement inhibition in CAPS has been explored. We present a case of refractory CAPS successfully treated with Eculizumab, a terminal complement inhibitor. Case: A 65 year old male presented with intermittent abdominal pain for 5 months and altered mentation. He had a history of positive anti-phospholipid antibodies. On presentation, his vital signs were within normal limits. Physical examination was positive for malar rash and bluish discoloration of the left 5th toe. Laboratory examination showed a leucocyte count of 19,700/mm³, hemoglobin of 6.6 g/dl, platelet count of 108,000/mm³ and acute kidney injury (AKI) with creatinine rising from 1.98 to 6.98 mg/dl over a period of one week. Further work-up revealed positive anti-neutrophilic antibody (1:38), anti-double-stranded DNA antibody (1:66), lupus anticoagulant (LA) (1:1.69), anti-cardiolipin IgM (1:23), anti- β -glycoprotein (β -GP) (1:20) and low C3 (52 g/dl) and C4 (5 g/dl). Peripheral blood smear demonstrated schistocytes, which, along with thrombocytopenia, suggested thrombotic microangiopathy (TMA). ADAMTS13 activity was 18%, with no inhibitor, ruling out thrombotic thrombocytopenic purpura. Renal biopsy demonstrated multiple arterial microthrombi, confirming TMA in the kidney, which, along with positive antibodies suggested CAPS. Patient was started on pulse dose steroids, plasmapheresis and rituximab, and achieved adequate immunosuppression as evidenced by leucopenia. Subsequent clinical course was complicated by acute systolic heart failure requiring inotropic support, hypoxic respiratory failure requiring ventilatory support, bilateral adrenal hemorrhages, and worsening AKI requiring hemodialysis (HD). LA titers remained elevated with worsening organ failure and hence the decision was made to start patient on Eculizumab, a monoclonal antibody against complement 5 (C5). The patient received 4 doses of 900 mg weekly, followed by 1200 mg weekly. In addition, he also received Azathioprine 100 mg daily and weekly plasmapheresis. Following the initiation of this regimen, LA titer dropped to 1.2 and β -GP dropped to 9.4. Patient's renal function started recovering with improving urine output. He continues to remain on HD with close monitoring for renal recovery. Discussion: Our patient met the criteria for CAPS (more than 3 organs involved in less than one week with pathognomic laboratory and histopathologic features). Conventionally, steroids, plasmapheresis and rituximab have been the first line of therapy, therapeutic options for refractory cases are limited. Eculizumab, a C5 inhibitor, has been approved for use in paroxysmal nocturnal hemoglobinuria and atypical hemolytic uremic syndrome. Studies indicate a role of complement activation in CAPS. We present a very rare case of CAPS successfully treated with Eculizumab, with resolution of TMA and ongoing clinical recovery.

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Title: Right Atrial Primary Angiosarcoma Presenting as Cardiac Tamponade

Introduction: Primary cardiac tumors are extremely rare. The most common tumor arising in the right atrium is myxoma. However, primary angiosarcoma has been rarely reported. Right atrial tumors can lead to obstruction of blood flow mimicking tricuspid stenosis. Tumor fragments may also embolize to the pulmonary vasculature, causing pulmonary embolism. Cardiac tamponade is a relatively uncommon presentation of right atrial tumor. We present a case of right atrial primary angiosarcoma presenting as cardiac tamponade. Case: A 60 year old female presented with progressively worsening dyspnea, orthopnea and substernal chest pain. On admission, her vital signs were normal. Physical examination revealed pulsus paradoxus and jugular venous distension. She had clear lungs, normal heart sounds and no murmur rubs or gallops. Neurological examination revealed no focal deficits. Electrocardiogram demonstrated normal sinus rhythm without any ST-T wave abnormalities. Laboratory examination was unremarkable. A chest radiograph revealed enlarged cardiac silhouette, but no focal consolidation or pleural effusions. An echocardiogram revealed a large immobile mass, measuring 45.5 x 30.8 mm, in the right atrium. There was a moderate to large circumferential pericardial effusion with right ventricular collapse, suggestive of tamponade physiology. The inferior vena cava was dilated and retrophasic changes were blunted. The patient underwent diagnostic and therapeutic pericardiocentesis; 450 cc of hemorrhagic pericardial fluid was drained, cytology was negative for malignant cells. Computed Tomography scan of the chest and abdomen revealed bilateral multiple pulmonary nodules and a non-enhancing lesion at the dome of the liver. The differential diagnosis at this point included primary cardiac tumor (benign or malignant) versus metastatic disease. Cardiac magnetic resonance imaging (MRI) revealed a 57 x 51 x 72 mm mass adjacent to the right atrial free wall, partially obstructing the superior vena cava and extending up along the proximal ascending aorta. Image characteristics favored sarcoma as the most likely diagnosis. The patient underwent surgical resection of the mass but had positive margins. Her symptoms improved after the surgery. Biopsy revealed primary cardiac angiosarcoma. She has been scheduled to receive radiation to the heart due to positive margins as well as systemic chemoimmunotherapy with liposomal doxorubicin and olaratumab, a platelet derived growth factor receptor (PDGFR) alpha antibody. Response to treatment will be assessed as outpatient. Discussion: Primary cardiac angiosarcoma is extremely rare with limited treatment options. Surgical resection is the mainstay of treatment, but may not be an option in patients with advanced disease. Nevertheless, surgical resection can provide symptomatic relief. Response to chemotherapy has been traditionally poor. However, systemic chemoimmunotherapy with anthracyclines and olaratumab (a PDGFR alpha antibody) has shown to improve survival compared to anthracyclines alone. Potential for subsequent cardiomyopathy needs to be considered. Targeted therapy with tyrosine kinase inhibitors such as pazopanib may be better tolerated.

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Title: Idiopathic Granulomatous Mastitis: Case Series

Idiopathic granulomatous mastitis (IGM) is a rare benign chronic inflammatory condition of the breast of unknown etiology. It mimics breast cancer or abscess, causing diagnostic dilemma and potential treatment delay. Diagnosis is established after ruling out other causes of granulomatous inflammation and with histological finding of lobulo-centric noncaseating granulomas. We present three patients with IGM seen in our clinic.

Case 1:

31 y/o Hispanic nulliparous female presented with painful left breast for a year and palpable left breast mass for 3 months. Examination showed two, soft, mobile masses in the upper medial portion of left breast. Ultrasound (US) revealed a well-circumscribed hypoechoic mass of 2.8 x 1.1 x 2 cm extending medially from the nipple. Core needle biopsy revealed complex fluid collection which was evacuated. Histopathology showed granulomatous mastitis. Her symptoms improved with NSAIDs and no relapses were noted on follow up.

Case 2

26 y/o nulliparous Hispanic female presented with a progressive painful mass in the left breast for six months. Left breast was denser than right breast over the retroareolar region. US showed an ill-defined hypoechoic nodule measuring 2.8x3x1.8 cm. Histopathology revealed necrotizing granulomatous inflammation. ANA was 1:80. There was improvement with a tapering dose of prednisone initially, but an abscess formed later requiring antibiotics and drainage.

Case 3

30 y/o Hispanic female, gravida 2 para 1, presented in her second trimester with 3 months of left breast pain. Examination revealed an indurated erythematous lesion. US showed a well-circumscribed mass on the medial left breast measuring 6.1 x 5.0 x 6.2 mm. Granulomatous tissue was noted on core biopsy. Postpartum, prednisone 40 mg daily was started and her symptoms improved. She had multiple recurrences and is being considered for Methotrexate therapy.

Discussion:

The true prevalence of IGM is unknown. Recent studies have been published with increasing number of cases, possibly due to increased awareness of the diagnosis and the use of biopsies. Although pregnancy and breastfeeding have been linked to the disease, two of our patients were nulliparous. All of our patients were Hispanic; studies have demonstrated increased incidence in this ethnicity among US population.

One of the hypotheses about the cause of IGM is autoimmunity, however serological tests like ANA and anti-dsDNA have been positive only in few patients. Cultures, stains for mycobacteria and fungi were negative in all three cases. The treatment of this disease remains empiric due to lack of large studies. Management involves observation, antibiotics, corticosteroid therapy, immunosuppressive drugs and surgical intervention depending on the extent of lesions and severity of symptoms.

Clinicians need to have a high clinical suspicion of IGM in a young childbearing Hispanic and Asian women presenting with unilateral breast pain, mass or recurrent sterile breast abscesses.

Resident / Fellow Clinical Vignette

<p>Author: Kavneet Kaur Additional Authors: Lavneet Chawla Shikha Broker Savneek Chugh Brij Ahluwalia Institution: Westchester Medical Center</p> <p>Title: A very rare case of Pontine ischemic stroke involving complex gustatory and neural pathway</p> <p>CASE: A 51 year old diabetic male admitted from neurology clinic for sudden loss of hearing in right ear for the past 2 days, which later resolved spontaneously. He also has been complaining of numbness and tingling of right tongue for the last 2 weeks with loss of taste sensation especially to salty food for which he was referred to neurology clinic. On examination, he had decreased sensation on right face and tongue with taste impairment in right anterior 2/3rd of the tongue. MRI brain revealed a right dorsolateral pontine late sub-acute infarct and right pontine ischemic stroke in the distribution of long circumferential branch of basilar artery. CSF studies revealed normal biochemistry and negative for WBC or RBC. Further hypercoaguable workup showed an elevated factor VIII level and patient was placed on dual anti-platelet therapy with slow improvement of his neurological deficit over 2 months.</p> <p>BACKGROUND: Gustatory Pathway originates from the nucleus of solitary tract (NST) at the level of the pyramidal decussation near the cervical spinal cord and extends rostral to the caudal part of the dorsal cochlear nucleus. The rostral extreme of NST is medial to the spinal trigeminal nuclei and receives gustatory stimuli from anterior 2/3rd of the tongue. The chorda tympani innervate the anterior 2/3rd of the tongue and palate, respectively and project to the most rostral portions of the nucleus. Neurons within the rostral NST also receive input from medial parabrachial nucleus. The projection from the primary pontine taste-responsive area is parabrachial nucleus which terminates primarily within rostral central and ventral division. Oro-sensory input enters the rostral NST in a topographic fashion from the trigeminal, facial, glossopharyngeal and vagus nerve. The blood supply of dorsolateral pons is mainly from the long circumferential branch of basilar artery.</p> <p>Conclusion: A single small dorsolateral pontine infarct involving rostral NST, trigeminal nucleus and ascending pathways to parabrachial nucleus caused ipsilateral hemiaglesia (Loss of taste) and hemisensory impairment of face in this patient. Minimal sensory dysfunction of face may be associated with taste sensation loss due to the close proximity of taste fibers to trigeminal nuclei. Pontine infarct lesions should be considered and be thoroughly evaluated in these cases. Patients with such rare lesions can eventually progress towards malnutrition due to loss of taste and sensation together or can develop hypertension due to excess intake of salt in food.</p>	<p>Author: Rana Raheel Khan, M.D. Additional Authors: Chime, Chukwunonso M.D. 1, Raiyani, Henish, M.D. 1, Chilimuri, Sridhar M.D. 1, Cindrich, Richard M.D. 1 1. Bronx Lebanon Hospital Center, Bronx, New York. Institution: Bronx Lebanon Hospital Center</p> <p>Title: A DISCONCERTING RISE OF CARBAPENEMASE PRODUCING ORGANISMS IN HOSPITALS</p> <p>Background: Carbapenemase producing organisms pose a serious therapeutic and infection control challenge. We describe the epidemiology of carbapenem resistant organisms, including Acinetobacter, Pseudomonas, Klebsiella, and Morganella in our safety net hospital.</p> <p>Methods : We reviewed 597 cultures and subcultures of organisms with resistance to either or both imipenem and meropenem from the year 2016. It is important to note that isolates routinely tested for carbapenem sensitivity do not include those with inherent resistance to carbapenem e.g Burkholderia, S. maltophilia etc. Duplicate isolates were eliminated by comparison of antibiograms with reference to species and patients.</p> <p>Results : There were 290 unique isolates demonstrating some degree of resistance to carbapenems identified. Acinetobacter (37%), pseudomonas (32%), Klebsiella (8%) and Morganella (7%) constituted approximately 85% of the isolates. The remaining 15% of isolates in order of decreasing frequency include Enterobacter (4%), Providencia (2%), Serratia (2%), Proteus (1.4%), Streptococcus (1%), Citrobacter (0.3%), Empedobacter (0.3%), and Kluyvera (0.3%). Acinetobacter was mostly isolated from respiratory (59%) and urine (14%) cultures and predominantly located on step down ventilator floors (50%) and ICU (39%). Pseudomonas was most commonly isolated in respiratory (73%), wound (14%) and urine (11%) cultures. Among carbapenem resistant Enterobacteriaceae isolates, Klebsiella (33%), Morganella (26%) and Enterobacter (13%) were predominant and mostly isolated in urine (33%), respiratory (28%), diabetic foot ulcer (20%) and blood (11%) cultures.</p> <p>Conclusion : We observed extreme resistance isolates of Acinetobacter and Pseudomonas in chronic respiratory infections. Although Acinetobacter and Pseudomonas are the predominant isolates, there is a disconcerting increase of carbapenem resistant Enterobacteriaceae constituting nearly a quarter of the isolates. Though the most common location for all carbapenem resistant isolates were in step down ventilator care floors and ICU, we cultured isolates from all inpatient floors. Routine infection control protocols may be inadequate in controlling the spread of carbapenemase producing organisms. New innovative approaches in infection control practice and antibiotics stewardship programs are warranted.</p>
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Institution: Winthrop-University Hospital

Title: Ovarian Steroid Cell Tumor, a Rare Cause of Hirsutism in a Post-menopausal Female

Introduction: Ovarian steroid cell tumors are rare functioning sex cord-stromal tumors, comprising <0.1% of all ovarian tumors. Not-otherwise-specific (NOS) accounts for 60% of tumors and are associated with androgenic changes. Most common presentation is amenorrhea and virilization in a young female, with mean age of 43. We present a unique case of a post-menopausal female with slowly progressive hirsutism and virilization.

Clinical case: A 65 year old post-menopausal obese female presented with 1 year history of coarse dark hair, male pattern baldness and deepening of voice. She denied muscle weakness, galactorrhea, weight changes or symptoms of flushing. She had regular menstrual cycles until menopause at age 55 and denied having fertility issues or history of diabetes.

On presentation, she was normotensive with a BMI of 43.9. Hirsutism was present on chest, back, abdomen and forearms with a calculated Ferriman-Galway score of 12 (normal <8). Skin examination was notable for acne on face and back and absence of signs of cortisol excess.

Frontotemporal alopecia was noted, without clitoromegaly. Initial total testosterone level was 371 ng/dl (<45 ng/dL) which increased to 502 ng/dL in a subsequent visit. Free AM cortisol level found to be normal at 11.5 ug/dL, with normal suppression with 1 mg of dexamethasone.

Dehydroepiandrosterone sulfate (DHEAS) found mildly elevated at 221 mcg/dL (<133 mcg/dL). Estradiol level was 34 pg/mL (<31 pg/mL menopausal), with FSH of 39.6 mIU/mL (post menopausal 116.2 mIU/mL) and LH of 12.8 mIU/mL (post menopausal 54.7 mIU/mL). Plasma metanephrine levels and renin to aldosterone ratio levels were normal.

Transvaginal ultrasound noted an abnormal appearance and increased vascularity of left ovary. MRI abdomen-pelvis showed a 2 cm left ovarian mass and a 1 cm left lipid-laden adenoma. Bilateral adrenal sampling was not pursued, given the small size of the adrenal lesion and only mildly elevated levels of DHEAS, which favored an ovarian source. She underwent a total abdominal hysterectomy with bilateral salpingo-oophorectomy. Histopathology revealed a left ovarian steroid cell tumor. One week post-operatively total testosterone levels were 16 ng/dL and four weeks later she noted improvement in hirsutism and temporal baldness with consistent resolution of testosterone level.

Conclusion: Although rare in nature and mostly found in pre-menopausal females, steroid cell tumors are commonly benign ovarian tumors and should be investigated as a potential cause for virilization in post-menopausal females as seen in our case. Timely diagnosis and surgical treatment, specifically bilateral surgery in a post-menopausal patient, will allow for effective normalization in symptoms and testosterone levels.

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Title: Does genetic testing say it all for familial hypocalciuric hypercalcemia?

Familial hypocalciuric hypercalcemia (FHH) is a diagnosis that can be missed. It is an autosomal dominant trait with high penetrance. We report an interesting case with all clinical features of FHH with negative genetic testing.

A 37 year old fireman on no medications was sent for long standing hypercalcemia.

His physical examination was unremarkable and his neurologic examination was also normal.

On review of the medical records, sestamibi scan was not able to localize a lesion. Contrast enhanced CT scan of the neck (done in view of the negative sestamibi) was suggestive of possible 4th gland hyperplasia. Eventually the patient underwent parathyroid exploration.

Frozen section showed hypercellular parathyroid (left and right). Intraoperative PTH measurements done 0, 5 and 10 mins after each parathyroid excision) ranged from 38 to 78 pg/ml. We noted that the higher levels of intact PTH were even after excision.

Before Surgery

Calcium-11.4 mg/dl

Ionized calcium-1.49 mmol/l

Intact PTH-78 pg/ml

24 hour calcium/creatinine ratio-0.005

After Surgery

Calcium-11.1 mg/dl

Ionized Calcium-1.30 mmol/l

intact PTH-72 pg/ml

24 hour calcium/creatinine ratio-0.00315

His deceased mother had hypercalcemia and underwent two surgeries for her parathyroid glands and was never surgically cured. Furthermore, his 4 years old daughter also has unclear etiology of hypercalcemia. He is currently being managed non-surgically with a diagnosis of FHH.

Albumin-4

Intact PTH-7

PTHrP-18 pg/ml

25 Vitamin D -18

1,25 vitamin D-<8

SPEP-Normal pattern

Consistent with autosomal dominant features of FHH, our patient has family tree consistent with FHH in all generations. In contrast to primary hyperparathyroidism, our index patient did not have symptoms of hypercalcemia.

Representation of different genes in three different types of FHH can explain why the genetic testing in our case (Calcium sensing receptor gene (CaSR))was negative.

About 65% of FHH cases are caused by inactivating mutations of (CaSR) gene. FHH3 was recently found to be caused by AP2S1 gene mutation. A significant number of patients suspected of having FHH but proven negative for CASR mutation have AP2S1 p.R15 mutations.

Another possible reason that CaSR gene can be negative is that linkage analysis showed that the predominant locus of the FHH disease gene (e.g. the CaSR gene) resided on the long arm of chromosome 3 (band q21-q24). However, two families with clinical features similar to FHH showed linkage to the short and long arms of chromosome 19, respectively; one of these was called the "Oklahoma variant" that may be present in a minority of the ~30% of FHH cases.

It is important to diagnose FHH, both in the index case and in family members because these patients need to be advised and prevented from unnecessary surgical intervention considering at least 9% of the patients referred after unsuccessful parathyroidectomy had FHH.

Resident / Fellow Clinical Vignette

<p>Author: Venkata Sandeep Koripalli, MD Additional Authors: Libardo Rueda Prada MD, Oscar Carazas MD, Ana Bechara MD, Kaniz N Banu MD Institution: St Barnabas Hospital</p> <p>Title: A RARE CASE OF NEUROSYPHILIS PRESENTING WITH MENINGITIS AND OCULAR INVOLVEMENT IN AN HIV PATIENT WITH NEW ONSET SYPHILIS</p> <p>Neurosyphilis (NS) is the involvement of the central nervous system by the spirochete <i>Treponema pallidum</i>. It has been classified into early (involvement of cerebrospinal fluid, meninges, and vasculature) and late forms (involvement of brain and spinal cord parenchyma). During the pre-antibiotic era, NS was most commonly seen, and it used to present as general paresis or tabes dorsalis. Nowadays, NS is most frequently seen in patients with HIV infection. HIV patients at higher risk for developing NS are the ones who are not on antiretroviral therapy or have low CD4 cell count. Ocular syphilis, an early form of NS, can involve any eye structure. Posterior uveitis and panuveitis are the most commonly involved structures, which manifest as diminished visual acuity. Lumbar puncture should be considered in patients who present with neurologic or ocular disease that could be caused by syphilis. A reactive CSF-VDRL establishes the diagnosis of NS but a nonreactive test does not exclude the diagnosis, as it may be falsely negative in a high percentage of patients with NS.</p> <p>We present a 47-year-old male with five-day history of intermittent frontal and retro-orbital headache, blurry vision, photophobia associated with redness, tearing and pain of the left eye. His medical history was remarkable for HIV on HAART (last CD4 cell count 1022 cells/&#181;L) and chronic kidney disease. He had a history of unprotected anal sex with a new sexual partner four months before presentation. On clinical examination, ophthalmic exam revealed a bilateral decreased visual acuity, bilateral papilledema, and uveitis in the left eye. Initial workup revealed positive rapid plasma reagin (RPR) test with a titer of 1:128 and positive microhemagglutination assay for <i>Treponema pallidum</i> (MHA-TP). RPR was negative 5 months prior to this visit. Cerebrospinal fluid analysis revealed 45 white blood cells/mm³ with 42 lymphocytes, glucose of 49 mg/dL and protein of 126 mg/dL. CSF-VDRL was negative. A diagnosis of NS was made. Patient was started on intravenous Penicillin G for 12 days along with cyclopentolate and prednisone drops for the affected eye. Patient's ocular symptoms gradually improved. Patient was discharged after completion of antibiotic therapy. Outpatient follow up showed decreasing RPR titers from 1:128 at the time of presentation to 1:16 at 3 months and 1:8 at 6 months from discharge. Uveitis and papilledema completely resolved with back to baseline visual acuity. The diagnosis of NS in HIV patients remains a challenge. A high index of suspicion for NS should be maintained in HIV-infected patients with new onset syphilis, especially if presenting with ocular symptoms. Physicians must consider a lumbar puncture on such patients and start intravenous penicillin therapy as soon as possible, if findings of CSF are suggestive of NS, which can prevent further neurovascular complications.</p>	<p>Author: Afua Kunadu, MD Additional Authors: Leonel Carrasco, MD; Milton Ray, MD; Pranit Shrestha, MD; Harseerat Rataul, MD; Camille Edwards, MD; Gene Pesola, MD; Raji Ayinla Institution: HARLEM HOSPITAL CENTER</p> <p>Title: HEPARIN-INDUCED THROMBOCYTOPENIA AND THROMBOSIS (HITT) PRESENTING AS MULTIPLE ARTERIAL THROMBOSIS IN A SEPTIC PATIENT</p> <p>Introduction: HITT is a rare but life-threatening complication, which can result in about 5% of patients exposed to heparin. Many are asymptomatic, only 25% of patients present with thrombosis. Half of those patients have venous thrombosis while arterial thrombosis occurs in a mere 3% of patients presenting with thrombosis.</p> <p>We present the case of a patient managed for septic shock from legionella pneumonia who was anticoagulated with unfractionated heparin (UFH) due to a high clinical suspicion for pulmonary embolism (PE) and subsequently developed multiple arterial thromboses due to HITT.</p> <p>CASE DESCRIPTION: 83-year-old woman, former smoker, with a past medical history of Hypertension, Ischemic Stroke, Peripheral Vascular Disease (PVD) and Dementia brought to the ER due to cough, shortness of breath and confusion. On presentation, she was febrile, tachypneic, tachycardic and hypotensive. She had left sided inspiratory crackles on lung exam. Labs were significant for elevated BUN, creatinine and high WBC with left shift. Urine legionella antigen was positive. Chest DX showed left pleural effusion with retro cardiac consolidation, echocardiogram had signs of right heart strain suggestive of a PE for which intravenous UFH was started. She was managed for multi-organ failure from septic shock and received aggressive fluid resuscitation, broad-spectrum antibiotics, vasopressors and dialysis. 48 hours into admission, her right hand, which had previously had an arterial line, became cyanotic and cold with absent radial and ulnar pulses even on Doppler. Platelet count dropped by more than 50% and Platelet-factor (PF4) antibody returned positive. UFH drip was switched to argatroban for the management of HITT. Nonetheless, she developed bluish discoloration of toes bilaterally on day 5 of admission which progressed to dry gangrene. Argatroban drip was eventually bridged with warfarin. Patient had right hand and foot amputated after the gangrenous areas got clearly delineated.</p> <p>Discussion: HITT results from autoantibody directed against endogenous PF4 in complex with heparin. The risk of HITT depends on the type of heparin and the patient population. The incidence is up to 10 times as high in patients receiving UFH compared to those receiving low-molecular-weight heparin. Risk factors for development of thrombosis (several of which our patient had) include female sex, antecedent trauma, surgery, prior thrombosis and PVD. The risk of thrombosis persists until an alternate non-heparin anticoagulant is substituted. Our patient developed multiple arterial thrombi to the feet even while therapeutic on argatroban drip. The 4 T's score is used for estimating the likelihood of HITT and diagnosis is confirmed by HITT antibody testing.</p> <p>Conclusion: Key interventions in patients with highly suspected or confirmed acute case of HITT are the prompt cessation of heparin and the initiation of an alternate anticoagulant at a therapeutic dose and patient should avoid heparin for life.</p>
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Resident / Fellow Clinical Vignette

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Institution: Northwell Health

Title: RANDOM ACCESS MEMORIES: A RARE CASE OF AUTOIMMUNE LIMBIC ENCEPHALITIS

Case Presentation: A 35 year-old female with a past medical history significant for type 2 diabetes mellitus, polycystic ovarian syndrome and migraine headache presented with alteration of mental status for 2 weeks. Patient endorsed progressive, generalized fatigue with intermittent episodes of confusion, behavioral disturbance, hypersomnia and short-term memory loss. Vital signs on admission were significant for tachycardia to 111. Physical examination was significant for confusion and short-term memory loss. Neurologic examination was otherwise unremarkable. Laboratory studies revealed leukocytosis (19.5) and thrombocytosis (506). Basic metabolic panel was initially within normal limits though patient developed hyponatremia throughout the course of hospitalization. Urine and blood cultures were negative. Toxicology screen was negative. Acyclovir was initiated given concern for herpes encephalitis. Lumbar puncture was performed and studies were significant for mild glucose elevation. An extensive infectious evaluation of cerebrospinal fluid (CSF) was unremarkable. Acyclovir was discontinued. MRI brain revealed abnormal signal of the bilateral medial temporal lobes. Findings were suggestive of limbic encephalitis. Patient was found to have voltage-gated potassium channel (VGKC) antibody (Ab), leucine-rich glioma inactivated 1 (LG1) Ab and glutamic acid decarboxylase (GAD) Ab positivity. Remaining autoimmune and paraneoplastic markers were negative. CT chest, abdomen and pelvis was negative for primary malignancy. EEG revealed rhythmic delta activity concerning for electrographic seizure. Patient was without clinical seizure activity. Levetiracetam and IVIG therapy was initiated. Patient completed 5 days of IVIG followed by 5 days of solumedrol. Repeat MRI revealed mild improvement in temporal lobe enhancement. Gradual improvement in short-term memory was noted. Patient was discharged to home with plan for outpatient neurology follow up and continued IVIG infusions.

Discussion: Limbic encephalitis is broad diagnosis that comprises infectious, autoimmune and malignant pathologies. VGKC is a rare subset of non-paraneoplastic limbic encephalitis characterized by cognitive decline and seizure. Patients may additionally develop hyponatremia secondary to SIADH. Treatment involves immunotherapeutic modalities including steroids, plasma exchange and IVIG. GAD positivity may be associated with treatment-resistant epilepsy.

Conclusions: Here we report a case of VGKC complex antibody-associated limbic encephalitis in a patient presenting with memory loss with subsequent development of hyponatremia. VGKC encephalitis is often overlooked due to the rarity of this condition and its clinical similarity to other infectious, autoimmune and malignant encephalitides. SIADH is associated with VGKC encephalitis and hyponatremia may be an important diagnostic element. A high index of suspicion is required in the diagnosis of VGKC encephalitis; prompt diagnosis is required in order to prevent seizure and amnesia.

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Title: A RARE CASE OF VASCULITIS-ASSOCIATED CVA IN AN ADULT PATIENT WITH HENOCH-SCHONLEIN PURPURA

Case Presentation: A 64 year-old male with a past medical history significant for COPD presented with fever, abdominal pain and rash. Patient endorsed fever and chills for 2 weeks prior to inpatient hospitalization. Patient subsequently noted intermittent generalized abdominal pain associated with hematochezia. Patient noted onset of bilateral lower extremity rash the day of hospital admission. Vital signs on admission were significant for tachycardia and hypotension responsive to intravenous fluids. Physical examination revealed mild, diffuse abdominal tenderness, palpable purpura of the lower extremities and fine petechiae of the lower extremities and abdomen. Laboratory studies revealed leukocytosis, hematuria and proteinuria. Skin biopsy was performed given concern for Henoch Schonlein Purpura (HSP). Biopsy revealed granular IgA deposits within dermal vessel walls. Prednisone therapy was initiated. Hospital course was complicated by arthralgias, progressive vasculitic rash and AKI. Twenty-four hour urine protein was elevated to 1358 mg. Patient was placed on IV steroids and underwent renal biopsy. Biopsy revealed mesangial and focal proliferative glomerulonephritis with scattered mesangial deposits. Findings were consistent with mildly active HSP. Renal function improved on IV steroids. Course further complicated by blurred vision and diplopia. MRI revealed punctate acute infarcts in the right midbrain and left occipital lobe. CTA was significant for high-grade stenosis of the intracranial left vertebral artery. Pulse steroid therapy was administered given concern for a vasculitic etiology of cerebrovascular accident (CVA). Patient improved on high dose steroid therapy and was transitioned to oral prednisone. Patient was discharged to home with plan for multidisciplinary outpatient follow up.

Discussion: HSP is a small vessel vasculitis that typically presents with purpura, arthralgias, abdominal pain and nephritis. HSP is primarily a disease of childhood and rarely presents in adults. CVA in the setting of HSP vasculitis is exceedingly rare. Four similar reports are described in the current medical literature with only one case report noted in an adult patient. Given the paucity of available literature, treatment options for HSP-associated CVA are ambiguous. Pulse dose steroids and cyclophosphamide have been reported as proposed therapeutic modalities.

Conclusions: Here we report a rare case of HSP and vasculitis-associated CVA in an adult patient presenting with fever, abdominal pain, rash, nephritis and arthralgias. This is the second report of its kind and the first to be reported in the United States. Therapeutic guidelines for the management of HSP-associated CVA are nonexistent given the rarity of this condition. Pulse steroids, however, are often used and were effective in the treatment of this patient.

Resident / Fellow Clinical Vignette

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Institution: Wyckoff Heights Medical Center

Title: Hemophagocytic lymphohistiocytosis: a rare and fatal condition as a cause of pancytopenia

Introduction

Pancytopenia is a common laboratory findings seen with acutely ill patients, often secondary to malignancies, infections, drugs and autoimmune disorders. Here, we describe a rare and fatal condition of adult hemophagocytic lymphohistiocytosis (HLH) in the context of sepsis as a cause of pancytopenia.

Case presentation

A 44-year-old female with no significant past medical history presented to the emergency department complaining of body aches, abdominal pain, and subjective fever. She was recently treated with levofloxacin for an uncomplicated urinary tract infection. Upon examination, she was oriented to person only with a fluctuating mental status and white plaque lesions on her tongue; the rest of her physical examination was unremarkable. Head computed tomography showed hydrocephalus with suspicion of obstruction at the third ventricle, possibly secondary to an intracranial mass. Laboratory workup revealed a platelet count of 128,000/ $\times 10^9$ /L and sodium level of 126 mEq/L. She was admitted to the medical ICU with a differential diagnosis of SIADH, cerebral salt wasting syndrome or adrenal insufficiency. Despite an appropriate correction of her hyponatremia, no improvements were seen in her mental status. Also notable was a cortisol level of 10.4 μ g/dL with a non-HIV related, low CD4 count of 134 cells/ $\times 10^6$ /L. She was started on treatment for suspected panhypopituitarism with hydrocortisone. Her mental status improved slightly, and a ventriculostomy with biopsy was scheduled. A platelet transfusion given prior to surgery further decreased her platelet count to 59,000/ μ L without platelet clumping. Further tests also illustrated ferritin levels of 2548 ng/mL. Antiplatelet factor 4 antibody and serotonin release assays were negative. Subsequently, the patient developed disseminated intravascular coagulation (DIC) and acute liver failure, followed by severe acute respiratory distress syndrome, septic shock, and cardio-respiratory arrest leading to her death. The bone marrow biopsy confirmed adult HLH secondary to sepsis, with possible alteration of immune system homeostasis leading to immunodeficiency, hemolytic anemia and DIC.

Discussion

HLH is a challenging diagnosis with a very high mortality if left without treatment. This condition can be triggered by infection and has variable clinical presentations. The patient met 5 out of 8 HLH-2004 trial criteria, which confirms the diagnosis of HLH. However, nonspecific clinical and laboratory findings may delay an earlier diagnosis of the disease, resulting in poor outcomes in affected patients. Our case aims to emphasize the importance of considering this condition as a cause of pancytopenia. A bone marrow biopsy should be performed early if a patient is showing no signs of improvement with medical treatments. Early diagnosis and treatment may improve chances of survival.

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Title: Severe anion gap metabolic acidosis masquerading as septic shock

Introduction:

Anion gap metabolic acidosis can be seen in numerous cases with variable presentations such as lactic acidosis. Lactic acidosis can be secondary to sepsis, resulting in higher chances of mortality if not treated early. It has been known that the higher the lactate level, the higher the mortality. Here we describe a case of severe anion gap metabolic acidosis masquerading as septic shock.

Case presentation:

A 52 year old male presented to the emergency room because of altered mental status. Further history revealed that the patient had been binge-drinking alcohol prior to admission. He had a past medical history of HIV, hypertension, and type 2 diabetes mellitus in which he had been taking metformin and glipizide. On physical examination, his heart rate 105/min, blood pressure 87/70 mm Hg, respiratory rate 20/min, and temperature 33.3 $^{\circ}$ C. He appeared severely dehydrated, with the rest of his physical examination being unremarkable. The patient had an episode of seizure and was intubated for airway protection. Laboratory values revealed a WBC of 19.5 $\times 10^9$ /L, lactic acid of 30.0 mg/dL, creatinine of 13.1 mg/dL, BUN of 61 mg/dL, serum osmolality of 343 mosm/kg, and an anion gap of 46. Arterial blood gas showed a pH of 6.83 with pCO₂ of 10 mmHg and HCO₃ $<$ 3 mmol/L, indicating metabolic acidosis with compensatory respiratory alkalosis. The patient was admitted to the medical ICU for septic shock, severe metabolic acidosis, severe lactic acidosis, acute respiratory failure and acute renal failure. The patient was treated with empiric antibiotic therapy, aggressive IV fluid resuscitation and bicarbonate for his acidosis. A temporary femoral dialysis catheter was placed; subsequently, emergent hemodialysis was initiated. Further workup did not reveal any signs of an infectious process; focal lesions were ruled out with imaging, and blood cultures and urine cultures were negative. The antibiotics were discontinued. With the hemodialysis treatment and the fluid resuscitation, his creatinine had improved to his baseline level. The patient was hemodynamically stabilized and was discharged home with normalization of symptoms and laboratory values.

Discussion:

Lactate levels have been a predictor for disease severity in critically ill patients. Depending on the cause, severe lactic acidosis can have mortality up to 56.8% in metformin-associated lactic acidosis (MALA) population vs 88.1% in non-metformin related lactic acidosis. The patient's severe metabolic acidosis was precipitated by binge-drinking, causing dehydration with progression to acute renal failure and metformin toxicity. Although septic shock can also present in a similar manner, the mortality rate is much higher as compared to MALA. Our case aims to emphasize the importance of early aggressive treatment with hemodialysis for those patients with MALA. Even though high lactate levels correlate with high mortality, MALA patient may have a better overall prognosis.

Resident / Fellow Clinical Vignette

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Title: Fabry Disease: An Uncommon Cause of Renal Failure

INTRODUCTION

Fabry's Disease (FD) is a X-linked disorder caused by glycosphingolipid accumulation due to α -galactosidase (GLA) deficiency. Data is limited in patients with Latin American ancestry.

CASE PRESENTATION

A 30-years-old Mexican man without known medical conditions was referred to our center due to abnormal renal function tests. He had intermittent mild lower extremity edema for 10 years, which progressed significantly over a 2-weeks period. Review of systems revealed arthralgias, acroparesthesias and hypohidrosis since early childhood. He denied any family history, however his mother and maternal grandfather had similar, milder acroparesthesias.

Physical examination revealed marked teeth discoloration, mild neck vein distention and regular tachycardia. A papular rash was observed on the dorsolateral aspect of his hands and forearms. He had 3+ lower extremity edema up to the knees, with chronic skin changes. The rest of the physical exam was unremarkable. The patient's laboratories revealed renal failure, and were suggestive of chronicity. His electrocardiogram was consistent with significant left ventricular hypertrophy. The patient was started on fluid restriction and hemodialysis, which led to clinical and serological improvement.

The presentation of end-stage renal disease in a young adult, in association with acroparesthesias, hypohidrosis, isosthenuria, and left ventricular hypertrophy suggested the diagnosis of FD. It was established by a serum leukocyte α galactosidase level of 0.001 (reference range 0.074-0.457). Genetic analysis confirmed the patient was hemizygous for a nonsense c.748C>T mutation in the GLA gene. Enzyme replacement therapy was successfully started.

DISCUSSION

FD leads to premature death, most significantly due to renal, cardiac and cerebrovascular involvement. During childhood and teenage years, acroparesthesias, hypohidrosis, nausea, abdominal pain and postprandial diarrhea predominate. After age 20, these symptoms worsen and proteinuria often develops in men. Lastly, renal failure and life-threatening cardiac and cerebrovascular manifestations arise, with an approximate 20-year reduction in life expectancy.

FD data in Latin America is limited to Brazil, Mexico and Colombia. In Brazil, 38 alterations of the GLA gene have been described; most patients present with acroparesthesias and the disease appears to be linked to European ancestry, as it is less prevalent in the Afro-Brazilian ethnicity. In Mexico, the most frequent and consistent findings are acroparesthesias, heat/cold intolerance, and hypohidrosis/anhidrosis; both men and women develop proteinuria and can progress to end-stage renal disease. Genetic analysis in the Mexican population is limited to a four-patient series, and suggests genetic heterogeneity.

We present a case of FD type 1 with a novel nonsense mutation, with possible familial involvement. It illustrates the importance of seeking etiologic workup in young patients with renal failure, and highlights the role of routine primary care.

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Title: Neuroendocrine carcinoma of the breast with endobronchial metastases and syndrome of inappropriate anti-diuretic hormone secretion.

Introduction

We present the case of a woman with neuroendocrine carcinoma of the breast (NECB) with syndrome of inappropriate antidiuretic hormone secretion (SIADH) and endobronchial metastases. In our knowledge, no cases of NECB with SIADH and endobronchial metastases have been reported.

Case Report

A 67-year-old woman presented with back pain. Further evaluation with bone scan showed abnormal tracer concentration throughout the axial and appendicular skeleton. Computerized tomography (CT) of the chest showed left neck adenopathy. A right apical lung mass measuring 1.7 x 1 cm was seen. Extensive osteoblastic and osteolytic metastases were noted. She developed lethargy, confusion, dyspnea and cough and was seen in emergency department. Laboratory work revealed sodium of 118mmol/L and a urine osmolality of 505 mOsm/kg. SIADH was diagnosed. Bronchoscopy showed numerous tumor deposits in the proximal right bronchus. Bronchial biopsy showed intermediate-grade neuroendocrine carcinoma. PET scan revealed hypermetabolic malignancy. She was treated with 4 cycles of cisplatin and etoposide. Hyponatremia resolved. Improvement in skeletal metastases with interval development of sclerotic bone changes was seen.

She was first seen in our clinic one year after diagnosis. Physical examination was notable for a 1 cm hard mass in the right breast. Diagnostic mammograms and right breast ultrasound showed a heterogeneously hyperechoic mass in the right breast measuring 1.1 x 0.9 x 1.2 cm. PET/CT showed the primary mass of the right breast to be hypermetabolic. Right breast mass biopsy showed epithelial neoplasm arranged in nests and trabecular patterns with intervening slender, elongated collagenous stroma resembling neuroendocrine pattern. Neuroendocrine markers showed focal positive staining for CD56, and rare cells positive for synaptophysin. Immunohistochemistry panel showed diffuse staining of estrogen receptor, progesterone receptor, cytokeratin 7 and GATA3 binding protein. Neuroendocrine markers showed focal positive staining for CD56. Next-generation sequencing of tumors was done using the OmniSeq Comprehensive 2 platform. Both breast and lung specimens, showed mutations of ISH1 c.548A>G (Y183C) and NOTCH c.6170A>G (Q2057R). No translocations or activating mutations involving EGFR, KRAS, BRAF, ALK, RET or ROS1 were found.

We initiated treatment with letrozole, palbociclib and zoledronic acid. Treatment was well tolerated. Skeletal pain resolved and serum sodium remained normal. PET/CT after 3 months of treatment showed decrease in mass size and abnormal FDG uptake in the right breast and mixed lytic/sclerotic skeletal metastases. CA27.29 tumor marker fell from 115.9 U/ml to 60.1 U/ml.

Conclusion:

This case report highlights the clinical presentation, diagnostic challenges and treatment of NECB. Our patient presented with skeletal, endobronchial, lung and nodal metastases. She had SIADH. She was diagnosed at outset with atypical carcinoid lung tumor and responded to treatment with carboplatin and etoposide. Upon progression of her malignancy, we revised her diagnosis to hormone receptor-positive, NECB. She responded to treatment with letrozole and palbociclib.

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Title: Pericardioesophageal Fistula: A Rare Complication of Radiofrequency Catheter Ablation

Introduction:

Atrial fibrillation is one of the most common cardiac arrhythmias with an incidence of more than 2.7 million individuals in the United States. Radiofrequency ablation is reserved for patients who fail pharmacological management. Pericardioesophageal fistula is one of the rare complications related to the procedure. Here we present a case of pericardioesophageal fistula following radio frequency ablation.

Case:

We present a 72-year-old female with a past medical history of chronic paroxysmal atrial fibrillation who failed to respond to pharmacological treatment. She underwent an elective catheter ablation procedure in January 2017. Ten days later, she presented with sudden onset of sharp substernal chest pain associated with shortness of breath. Physical exam revealed an alert female with tachycardia along with irregularly irregular rhythm and tachypnea. EKG revealed atrial fibrillation with a rapid ventricular rate. Laboratory findings revealed leukocytosis ($20.7 \times 10^3/\text{micro liter}$) with normal cardiac markers. She was started on broad-spectrum antibiotics for possible sepsis due to an unknown source. Given her uncontrolled heart rate, she was started on intravenous amiodarone. Due to the nature of her severe chest pain, aortic dissection was strongly suspected. Emergent CT angiogram of chest did not reveal any evidence of aortic dissection; however it revealed pneumopericardium with a small pericardial effusion along with subcarinal air suggestive of pneumomediastinum. Given the recent radiofrequency ablation, pericardioesophageal fistula was suspected. Further evaluation with esophagogram revealed a fistulous connection between the middle-third of the esophagus and pericardium. She subsequently underwent pericardial window followed by esophageal stenting. She was discharged home after a 2 week of hospital stay with complete recovery.

Conclusion:

We present a patient with pericardioesophageal fistula as a consequence of a recent radiofrequency ablation for the management of atrial fibrillation. This is one of the mainstay approaches in the management of atrial fibrillation as a second line therapy. Although overall it is considered effective and safe, it does carry potentially serious adverse effects. A nationwide survey reports the prevalence of an atrial-esophageal fistula formation to be 0.03%, although reports on pericardioesophageal fistula as a complication are rare.

Since it is a rare complication, clinicians should have a high index of clinical suspicion for the development of a fistulous connection between pericardium/atria in patients who underwent ablation presenting with chest symptoms. This makes early recognition and prompt treatment crucial in preventing life threatening complications such as mediastinitis.

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Title: Glycogenic hepatopathy presenting predominantly as a cholestatic liver abnormality: A rare presentation

Introduction

Glycogenic hepatopathy (GH) is a rare and under-recognized complication of uncontrolled diabetes mellitus leading to accumulation of glycogen in the hepatocytes. GH predominantly occurs in patients with type I diabetes mellitus. GH usually presents with a hepatocellular pattern of liver injury however we report a case that presented with the cholestatic pattern.

Case Presentation

A 44-year-old male with a longstanding history of uncontrolled type I diabetes mellitus was admitted with hyperglycemia and forearm abscess. Physical examination revealed hepatomegaly and confirmed by abdominal sonography which showed hepatomegaly without altered liver echogenicity and no evidence of hepatobiliary obstruction. Laboratory analysis was compatible with cholestasis with predominantly increased alkaline phosphatase of 1100u/l, AST 80u/l, and ALT of 70u/l. Total bilirubin and coagulation tests remained normal. Workup including Viral hepatitis panel, ANA, ASMA, AMA was negative. The patient underwent liver biopsy which showed hepatocellular glycogenosis and periportal hepatocellular glycogen nuclei which was consistent with Glycogenic hepatopathy. The liver function tests returned to normal after appropriately controlling the blood sugars in six months.

Discussion: Glycogen hepatopathy (GH) is a rare cause of elevated serum transaminase, mostly seen with type 1 diabetics. Incidence and prevalence of GH are not well established. GH presents with abdominal pain, tender hepatomegaly, and mostly hepatocellular pattern of liver injury. Significant elevation of ALP compared to transaminases has been reported only in few case reports. The main pathology in GH is the entrapment of glycogen in the liver leading to liver injury. There is no liver cell death or necrosis involved. GH usually occurs in people with type 1 diabetes whereas NAFLD/NASH predominantly seen in people with type II diabetes. In NASH/NAFLD liver function tests show mild elevation in aminotransferases. However, extreme range and wide fluctuations in liver function tests are more common in GH. NASH and GH can reliably be distinguished only by liver biopsy. Histopathology in GH shows large glycogen filled hepatocytes which can appear pale with glycogen acted nuclei, sinusoidal compression. PAS-Diastase staining can demonstrate glycogen accumulation. Steatosis is usually minimal to absent. GH has not known to cause cirrhosis of the liver. Achieving glycemic control is the best way of treating GH which can normalize LFTs and hepatomegaly. Clinicians' awareness of GH should prevent diagnostic delay and disease progression.

Conclusion

It is important to recognize GH as one of the differentials in patients with type I DM presenting with a cholestatic pattern of liver injury. Adequate management of glycemic control can result in complete remission of clinical, laboratory and histological abnormalities.

Resident / Fellow Clinical Vignette

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Title: Look Again! T cell Lymphoma presenting as a testicular mass. A rare case.

Introduction

T cell lymphoma of the testis is very rare and aggressive. Very few cases are reported in literature. Testicular lymphoma comprises 1% of all lymphomas and 5% of testicular malignancies. Testicular lymphomas are usually B cell lineage (70 to 90% of reported cases). We report a case of T cell lymphoma presenting as a testicular mass with metastasis.

Case Report

A 51 year old man presented to the Hospital with 5 days of vomiting and decreased oral intake and a history of scrotal swelling. No family history of cancers and patient denied tobacco or recreational drug use. He had presented with the same symptoms and underwent right total orchiectomy in 11/2015 in his country. He developed complications and decided to seek better care in the United States. On physical examination, he had right nystagmus, no hepatosplenomegaly, no lymph nodes palpable. Right testicle was absent. Labs revealed a low sodium level (117). Wbc count of 3.9, Hemoglobin of 11. Platelet count of 126. HIV negative, HTLV 1 and 2 antibody negative, EBV IgM negative. LDH- elevated at 1349. ferritin- 27,210. AST- 361, ALT- 365. Alkphos- 347. Bilirubin -2. Ultrasound of the abdomen showed a hypo echoic mass of the right supra renal fossa, likely of adrenal origin and a hypo echoic mass of the right lobe of the liver. CT Chest- scattered ground glass opacities and mild interstitial thickening. MRI could not be performed due to body shrapnel from prior gunshot wound. CT abdomen showed bilateral suprarenal lesions, matted peri aortic and retroperitoneal lymphadenopathy with encasement of the IVC and bilateral renal veins, 2.3cm hypoattenuating lesion in the liver with hepatic venous flow, soft tissue density obscuring the bilateral adrenal glands. Lymph node Biopsy repeated was consistent with peripheral T cell lymphoma positive for CD3. He was started on Ifosphamide, Carboplatin and Etoposide and intrathecal methotrexate.

Conclusion- Testicular lymphomas are a rare and aggressive form of NHL with high incidence of extra nodal occurrence. They comprise about 1-2% of tumors in men with a mean age > 60 years. Only a few cases of primary T cell lymphoma presenting in the testis have been reported in the US. Our case was present in a male with typical elevated LDH and biopsy results, abdominal and leptomeningeal metastasis. His International Prognostic Index score was intermediate-high. Education is needed for diagnosis and better treatment because of poor prognosis for patients.

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Title: Pulmonary Extramedullary Hematopoiesis Masquerading as Idiopathic Pulmonary Hypertension

Myelofibrosis (MF) is a chronic myeloproliferative disorder caused by clonal proliferation of abnormal myeloid cells and is associated with bone marrow fibrosis, severe cytopenias and marked splenomegaly. In MF, the hematopoietic niche in the marrow becomes uninhabitable to the hematopoietic progenitor cells. As such, these progenitor cells are forced to occupy unconventional territory, most commonly the spleen, liver and lymph nodes. In rare cases, patients can develop extramedullary pulmonary hematopoiesis. These pulmonary manifestations are associated with a poor prognosis and there are limited treatment options available. An 84 year old female with past medical history significant for atrial fibrillation not on anticoagulation, HTN, CHF, myelofibrosis, pulmonary hypertension and breast cancer s/p mastectomy was sent to the hospital from her cardiologist's office for worsening shortness of breath. She was initially seen in 2013 for worsening anemia with a peripheral smear showing teardrop cells and nucleated red blood cells. Bone marrow biopsy was done and was consistent with myelofibrosis. She was treated with erythropoietin stimulating agents, however no improvement was noted so patient was subsequently switched to Ruxolitinib, with marked improvement. She continued on Ruxolitinib for approximately 18 months at which point she experienced progressively worsening dyspnea on exertion; labs revealed worsening anemia and thrombocytopenia. Ruxolitinib was tapered off but patient remained anemic and thrombocytopenic.

On admission, physical examination was notable for respiratory distress (requiring supplemental oxygen), splenomegaly, JVD, and diminished breath sounds bilaterally. Further workup revealed severe pulmonary hypertension with a PA pressure of 105 mmHg as seen on echocardiography. CT imaging revealed alveolar septal thickening and ground glass opacities, prompting a concern for extramedullary hematopoiesis, specifically in the lungs given progressively worsening pulmonary hypertension. She underwent bone marrow scan which revealed uptake in both lungs confirming the suspicion of extramedullary hematopoiesis. Patient's pulmonary hypertension was initially medically managed however minimal response was seen. Radiation oncology was then consulted for evaluation and the patient received whole lung radiotherapy, with marked improvement. After two weeks, the patient was able to ambulate at her baseline without supplemental oxygen. The case highlights the necessity for considering extramedullary hematopoiesis in the lungs when pulmonary hypertension is present in the setting of primary myelofibrosis. Additionally, it reinforces the value of a complete history including past therapies, especially in patients with atypical (or puzzling) presentation. Prompt recognition of this disease can lead to appropriate treatment, such as in our case where the patient benefited from radiation therapy. Our patient's improvement further demonstrates the value of radiation therapy in patients with EH for palliation of symptoms.

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**Title: A RARE CASE OF CARDIAC SARCOID SHOWING
COMPLETE RESOLUTION WITH STEROID TREATMENT**

Background:

Worldwide prevalence rates of sarcoidosis are 4.7-64 for every 100,000 persons. It is a granulomatous disease most commonly involving the lungs but also causes cardiac, hepatic, renal, neurological and skin involvement. We present a unique case of cardiac sarcoid (CS), manifesting as isolated right bundle branch block (RBBB) that reversed with steroid treatment.

Case presentation:

A 42-year-old African-American man with history of pulmonary sarcoidosis was referred to cardiology clinic for new RBBB on his electrocardiogram (EKG). Sarcoidosis was diagnosed three-years earlier on trans-bronchial biopsy. EKG was done as a part of routine pre-employment exam and patient was completely asymptomatic. His physical exam and laboratory tests were unremarkable. EKG was unremarkable except for complete RBBB, with normal sinus-rhythm and normal PR interval. Transthoracic echocardiography and 24-hour holter monitoring were unremarkable. CT chest revealed extensive mediastinal and peri-lymphatic nodules with calcification. Cardiac MRI (CMR) showed a myocardial nodule with a dark center in the mid-anterior wall of the left ventricle at the hinge point, which is consistent with myocardial sarcoidosis. Patient was started on prednisone, which was tapered off after 6 months. Repeat CMR noted complete resolution of cardiac sarcoidosis. A repeat EKG revealed narrowing of the QRS complex with only an incomplete RBBB.

Discussion:

In the United States, annual incidence of sarcoidosis is 35.5 per 100,000 African-Americans. Only 5% of patients with pulmonary or systemic sarcoidosis exhibit symptomatic cardiac involvement. But the actual prevalence of CS is much higher, 25% on autopsy studies and up to 54.9% on imaging studies like CMR. Therefore, it is important to have a high index of suspicion for cardiac involvement in sarcoid patients, especially with new EKG manifestations, even if they are asymptomatic.

CS manifestations include conduction abnormalities, ventricular arrhythmias and heart failure. The most commonly reported conduction abnormalities are Mobitz Type-II or third-degree AV blocks. Our case presented with RBBB, which is an uncommon presentation; prevalence rate being 3.2-8.6% in CS. CMR should be pursued in suspected CS cases, as echocardiography is not sensitive. CS tends to have a predilection for interventricular septum and sub-epicardium. It is particularly important to recognize this entity as patients with CS have a poorer prognosis as compared to patients without cardiac involvement. Further, this patient's RBBB and CMR findings reversed with prednisone, which highlights the importance of early initiation of steroids in patients with CS.

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**Title: HIGH CAN COME WITH LOW!!! LEVAMISOLE
INDUCED CYTOPENIA**

Introduction

Levamisole, an immunomodulator mainly used as a veterinary anti helminthic, is a common adulterant of cocaine that can cause agranulocytosis and cutaneous vasculitis. Almost 70% of cocaine is now adulterated with levamisole to increase the euphoric effect. Here we present a case of levamisole induced ANCA associated cytopenia.

Case

64-year-old female cocaine user presented with a month-long history of low grade fever, loss of appetite and dry cough. Past medical history was significant for a chronic leg ulcer associated with elevated ANCA and biopsy evidence of vasculitis, and prior skin lesions with biopsies suggestive of pyoderma gangrenosum, which had been treated with intermittent steroids. She also had multiple emergency department visits for ear lobe pain.

Physical examination showed tachycardia, tachypnea, hypotension and left lower extremity chronic ulcer.

Laboratory data revealed new onset normocytic anemia (Hgb- 6.3 g/dl) and leukopenia (2100/micro liter) with absolute neutrophil count (ANC) of zero. Chest X-ray showed right lower lobe infiltrate; broad spectrum antibiotics were started for possible septic shock pneumonia.

Further evaluation included normal Vitamin B12, folic acid, haptoglobin and reticulocyte count. Peripheral blood smear showed agranulocytosis without blasts. Infectious workup was negative for HIV, HCV, CMV, B19 Parvovirus, EBV and HBV. Rheumatologic workup demonstrated negative ANA and lupus anticoagulant, while c-ANCA, p-ANCA and anti MPO ab were positive.

Given the history of cocaine use, skin manifestations and positive ANCA, levamisole-induced agranulocytosis and anemia were strongly suspected. She was transfused 2 units packed red blood cell (PRBC) and was treated with 4 doses of granulocyte colony stimulating factor (G-CSF). She was discharged after 2 weeks with complete recovery of cytopenia.

Discussion

Levamisole can lead to a constellation of clinical features including agranulocytosis and vasculitis-like purpuric skin eruption. Multiple case series have shown that levamisole-induced vasculitis usually associated with p-ANCA. The exact mechanisms responsible for the formation of these autoantibodies remain elusive. Multiple studies have suggested that levamisole acts as a hapten and may cause auto-antibodies. However, the specific antigens responsible for positive ANCA patterns are not yet clearly identified. Also, ANCA antibodies were directed against antigens borne on the surface of normal donor neutrophils is probably the underlying mechanism for agranulocytosis.

Thorough social history is important when physicians encounter patients with unexplained cytopenia; levamisole-induced cytopenia should be considered in patients with cocaine use. However, it remains a diagnosis of exclusion. First line treatment includes abstinence from cocaine. Granulocyte stimulating factor (G-CSF) can shorten the duration of levamisole-induced neutropenia but its mortality benefit has not been firmly established.

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Institution: SUNY Upstate Medical University**Title: LAUNDRY BLUES: A RARE CASE OF METHEMOGLOBINEMIA WITH LAUNDRY DETERGENT AND ACETAMINOPHEN INGESTION**

21y/o female with PMH of bipolar disorder, self-mutilation was transferred to our hospital for higher level of care after she ingested laundry detergent At the outside facility the patient was hypotensive, tachycardic and developed acute hypoxic respiratory failure with encephalopathy, needing intubation. A chest x-ray was unremarkable. She was put on airway pressure release mode of ventilation, continued to have oxygen saturation in the range of 70-75%. Initial workup at the outside facility showed hemoglobin of 14.2, WBC count 16.5, and an unremarkable BMP. INR was 1.4, AST/ALT was 68/40. A serum acetaminophen level was 109 mcg/ml 5 hours after ingestion (normal < 20 mcg/ml), and salicylate level was 26 (normal < 30). Given persistently low oxygen saturation levels she was transferred to higher level of care

At the time of presentation examination showed marked bluish discoloration of the perioral area, tongue, ear lobules, and extremities. ABG showed pH of 7.61, PaO₂ of 418, pCO₂ of 29. Given the mismatch, methemoglobin level was obtained and was 40.1%. She was then given methylene blue, 20 mins after the administration the patient's oxygen saturation readings increased to > 90%, and hemodynamics improved. A repeat level 8 hours later was 8%. She developed liver dysfunction 14-16 hours after ingestion; which improved with conservative management.

Discussion:

Household cleaning products are amongst the top 5 most common accidental exposures reported by the National Poison data system in 2011. Laundry detergents are cleaning products that may contain strong acids, alkalis, or phosphates. Though there have been cases of pediatric laundry detergent overdose, on our limited review of literature we could not find any cases reported in adults associated with methemoglobinemia. Very limited data is available over the mechanism of toxicity induced by these laundry detergents and the severity is believed to vary by the brand as well. They are implicated in causing eye irritation, nausea, vomiting, choking and CNS depression from unknown mechanism.

Acetaminophen in therapeutic doses, 90% of the acetaminophen is metabolized by glucuronidation or sulfation to nontoxic metabolites which are then excreted in the urine. Approximately 2% is excreted in the urine unchanged and 5% is metabolized by cytochrome P450 (mainly 2E1 and other sub-families) to electrophile N-acetyl-p-benzoquinone imine (NAPQI) which is toxic. The most commonly known manifestation of tylenol toxicity recognized is liver dysfunction. However methemoglobinemia is another rare manifestations believed to be secondary to oxidative stress. In our case we believe that the change in pH induced by the oxidant compounds lead to this presentation.

Conclusions:

Given the staggering number of acetaminophen overdose cases, it is important that clinicians are aware of methemoglobinemia as a potential complication, especially in cases of mixed ingestion, for timely management

Author: Leslie Seijo, MDAdditional Authors: Margaret Baran
Institution: Mount Sinai Beth Israel**Title: Do You Have A Stabbing Headache? Melatonin can help!****Introduction**

Primary stabbing headache (PSH) is an uncommon headache also known as ice-pick headache or jab and jolts syndrome. PSH is characterized by transient short stabs of pain localized in various areas of the scalp. We present a case of this uncommon type of headache and its treatment.

Case report

A 59 year old woman with history of breast cancer presents for evaluation of headache. The patient reports of suffering from headaches for over a year. The headache is described as sharp and stabbing, severe, that last for seconds. The headache occurs several times a day almost daily. This headache starts at different parts of her head, but primarily in the parietal region. She denies any nausea, vomiting or changes in vision. No neurologic deficit when headache starts. Due to the short-lasting headache, she has not taken anything to alleviate them. However, because of the frequency of the headache, she was starting to be fearful of having future episodes. Lastly, she also complained of chronic insomnia that had not improved on trazodone. On physical exam, the patient did not have any neurologic deficits. She was started on melatonin 3 mg at bedtime. On a month follow up, patient reports of having resolution of symptoms after 3 days of taking melatonin. She also reported improvement in her sleep.

Discussion

PSH is characterized by stabs of sudden, severe pain typically lasting for less than 3 seconds. The pain usually involves the orbit, temple or parietal region. Attacks moves from one are of the head to another. Spikes of pain can range from rare episodes to up to 50 times per day at regular or irregular intervals. The individual stabbing pain is so severe and sudden in onset that the patient will immediately stop any activity. Patients who have chronic attacks, may live with fear waiting for the next stabbing headache. The etiology of PSH is not well understood. Structural abnormalities must be excluded if the pain is invariably localized at one particular area. Diagnosis is based on clinical features and distinction from other types of headaches. Treatment modalities include observation or pharmacotherapy. In patient with rare episodes of PSH, treatment is not necessary. However, for those patients with chronic PSH indomethacin 75-150 mg can be use. For patients that can not tolerate indomethacin due to side effects of gastrointestinal and renal, melatonin is an alternative. A recommended dosing strategy is starting melatonin 3 mg at bedtime and increasing by 3 mg every 3 to 4 nights until response is achieved with maximum of 24mg per night. In our patient, who not only was suffering from PSH, but also had insomnia, melatonin was the best treatment modality.

Resident / Fellow Clinical Vignette

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Title: A case of cocaine induced ischemic hepatitis.

Introduction:

Cocaine is the most common cause of drug-related emergency department visits in the United States and its deleterious effects have been reported to affect almost every organ system. Recent data provide evidence of cocaine consumption being associated with hepatotoxicity, ranging from transient transaminase elevation to rare cases of acute liver failure. However, the latter seems to be associated with other culprits, such as alcoholic liver disease, concurrent rhabdomyolysis or viral hepatitis.

Case presentation:

A 50 year-old male with a past medical history of coronary artery disease, congestive heart failure, paroxysmal atrial fibrillation, chronic kidney disease stage 3, type 2 diabetes mellitus, alcohol and cocaine abuse presents to the emergency room with altered mental status secondary to hypoglycemia. Patient partially regained consciousness after intravenous push of dextrose and admitted cocaine and alcohol use prior to this episode. Initial lab work revealed hyperlactatemia, metabolic acidosis, acute on chronic renal failure, elevated troponins, and abnormal liver profile. He was then admitted and given supportive treatment and antibiotics for suspected sepsis for which work up was eventually unrevealing.

Patient parameters continued to improve dramatically while in the hospital. Cardiac work up failed to prove heart failure exacerbation while hepatitis work up was negative as well. Imaging studies showed enlarged liver on sonogram with minimal ascites. Liver enzymes returned to baseline after 8 days of hospitalization. He was discharged with a diagnosis of ischemic hepatitis likely secondary to a brief hypotensive episode induced by cocaine use, in addition to direct organ toxicity by cocaine augmented by alcohol consumption.

Discussion: Cocaine can cause direct tissue damage, which appears to be dependent mainly on N-oxidative metabolites of cocaine and can also produce other metabolites which deplete the antioxidant defenses, contributing to further liver damage. In addition to this, cocaine has a negative inotropic effect resulting in acute left ventricular failure which can ultimately lead to multiorgan failure due to hypo perfusion, evidenced in our case by high lactic acid, mild acute on chronic kidney failure and troponins release. This effect can be exacerbated by ethanol consumption. This patient was managed as a case of shock liver, which is usually a self-limiting condition and its treatment depends on the severity of the underlying disorder. Conservative management may include N-acetylcysteine, lactulose, vitamin K, fresh frozen plasma for reversal of coagulopathy, but liver transplant may be indicated in some cases when the appropriate criteria are met.

Conclusion: Cocaine can rarely lead to a picture of shock liver due to vasoconstriction and direct tissue damage. Our case underscores the importance of obtaining a complete work-up for all the possible factors that could trigger acute liver disease, including a complete history and a low threshold for ordering toxicology tests.

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Title: A Christmas miracle indeed

Introduction:

Immunotherapy in cancer has seen resurgence with development of anti-Programmed Death-1 antibodies and their approval for use in various solid tumors like melanoma, lung, kidney and head/neck cancers. FDA recently approved the use of nivolumab, (anti-PD-1 antibody) for treatment of advanced Hodgkin's lymphoma. Their use for non-Hodgkin's lymphoma is being studied under various clinical trials.

Case Description:

An 89-year-old male presented with atraumatic right arm swelling for 2 weeks associated with severe pain and weakness. An ultrasound was negative for DVT but CT scan revealed an axillary mass that on biopsy revealed a high grade large cell lymphoma. PET scan revealed disease in the right neck with extension into right arm, chest. FISH analysis showed MYC, Bcl-2 and Bcl-6 gene rearrangement in tumor cells consistent with "triple hit lymphoma".

He was started on dose modified rituximab, cyclophosphamide, mitoxantrone, vincristine and Prednisone. A PET scan after completing 4 cycles of treatment revealed a near complete response but a PET scan after 6 cycles of treatment, revealed disease progression with new soft tissue masses and cutaneous involvement of shoulder girdle confirming refractory disease.

Given his age and refractory disease, salvage chemotherapy was not considered to be the best option. Based on the encouraging data on nivolumab in Lymphoma, a decision was made to treat his disease with Nivolumab which usually is well tolerated. PET scan following 6 cycles of nivolumab therapy revealed a striking response with near resolution of disease except for a single 1.5 cm focus in the right chest wall which resolved with ongoing treatment after 8 cycles. Patient however developed Nivolumab related colitis and hence he was started on Prednisone and further therapy was interrupted temporarily.

After witnessing the striking response, we ordered PD-L1 expression testing on his tumor tissue which was shockingly negative.

Discussion:

Triple hit lymphomas are rare and carry prognosis of about four months as they are associated with an aggressive clinical course. Due to the complicated clinical course and gene rearrangements, standard chemotherapy used for Diffuse Large B-cell lymphoma is often ineffective (as in our patient) and can have many side effects. Hence, by achieving a remission with nivolumab, our patient had both an improved quantity (over one year thus far) and quality of life.

The role of anti PD-1 antibodies in NHL is evolving and ongoing clinical trials will shed more light on its benefit. However, all the studies are being conducted in patients with tumours positive for PD- L1 expression which was not the case in our patient making the response even more curious.

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Title: A Rare Case of Intramural Esophageal Hematoma

Introduction:

Intramural esophageal hematoma (IEH) is a rare cause of submucosal esophageal bleeding and it is on the spectrum of esophageal wall injury along with Mallory-Weiss tear and Boerhaave syndrome. Its risk factors include coagulopathy, trauma (foreign body ingestion or food impaction), iatrogenic usually secondary to esophageal instrumentation or it can happen spontaneously. It presents with a triad of retrosternal chest pain, dysphagia and hematemesis, however the triad is only present in 35% of patients with 50% having only 2 symptoms. Diagnosis can be missed as the presentation can be confused with cardiac/pulmonary diseases that's way high index of suspicion is crucial. Here we are presenting a case of IEH secondary to food impaction and subsequent retching.

Case Report:

A 75-yr-old man with a history of intermittent dysphagia to solids and liquids over the last 3 years, presented with sudden onset retrosternal chest pain and hematemesis associated with acute onset dysphagia after he ate a fish sandwich 1 day prior to admission. He denied having any abdominal pain, odynophagia, nausea or fever. On admission, he was stable with BP 130/82 mmHg and pulse rate 90 bpm. Laboratory data showed hemoglobin concentration of 12.5 g/dl, platelets were normal 308,000/uL with normal coagulation profile. CT Thorax showed esophageal luminal narrowing at distal third of esophagus with mural thickening and a soft tissue density extending to the gastroesophageal junction concerning for esophageal hematoma. He was managed conservatively with NPO, maintenance IV fluids and pantoprazole drip. Subsequently he improved with conservative management and diet was advanced to clears. Few days after admission, he underwent EGD which showed esophageal stenosis, a non-bleeding esophageal ulcer, resolving hematoma and a large hiatus hernia with normal stomach and duodenal mucosa. No biopsies were taken given recent bleeding. He was started on a pureed diet with oral pantoprazole twice daily which he tolerated well without any recurrence of his chest pain or hematemesis and he was discharged home.

Discussion:

IEH is usually diagnosed with a Barium swallow or CT which shows an intraluminal filling defect or a 'double barrel' sign. CT can also show intramural soft tissue density as in our case. Early upper endoscopy can be considered especially if hematemesis is the primary concern. Typical findings include obliteration of the esophageal lumen and visualization of a bluish longitudinal mass with a friable mucosa with or without a visible tear. It is important to differentiate this from an aorto-esophageal fistula as that will require urgent surgical management. Most cases resolve spontaneously in 1-3 weeks with conservative measures including NPO, acid suppression and correction of coagulopathy if present. A soft diet may be started in stable patients. Surgical intervention is rarely needed and associated with poor outcomes.

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Title: Acquired thrombotic thrombocytopenic purpura in a patient with pernicious anemia

Introduction

Acquired thrombotic thrombocytopenic purpura (TTP) characterized by the presence of anti-ADAMS13 antibody, has been associated with different autoimmune disorders. However, its association with pernicious anemia is rarely reported.

Case Report

A 46-year-old male with past medical history significant for bronchial asthma presented with blood-smear sputum on clearing his throat and blood in urine for one day. Rest of the review of symptoms was negative. There was no similar episode in the past, and no family history of any bleeding disorder or malignancy. Vitals were stable with a temperature of 98.2 F, pulse rate of 72, respiratory rate of 18 and blood pressure of 140/94 mm Hg. Physical examination was significant for icterus and absence of any petechial rash, lymphadenopathy or hepatosplenomegaly. Labs were significant for WBC of 15,000/ µL, hemoglobin of 10.7 g/dL, hematocrit of 32%, mean corpuscular volume of 102 fL, mean corpuscular hemoglobin of 34 pg, platelet of 13,000/ µL, blood urea nitrogen of 41 mg/dL, serum creatinine of 1.9 mg/dL, total bilirubin of 3.1 mg/dL with unconjugated bilirubin of 2.6 mg/dL. Subsequent tests showed an elevated LDH (1499 IU/L), low haptoglobins (<10 mg/dL) with many schistocytes, nucleated RBCs and reticulocytes (2.3%) on peripheral smear. ADAMTS13 activity of less than 10% with an elevated ADAMTS13 antibody (>140 u/mL) clinched the diagnosis of severe acquired TTP, and the patient was started on plasmapheresis. Furthermore, the initial serum Vitamin B12 was low (202 pg/mL) in the presence of anti-intrinsic factor (IF) antibody. So with a concomitant diagnosis of pernicious anemia, the patient was supplemented with 7 days of parenteral Vitamin B12. Rest of the immunological workups and thyroid function tests were negative.

Over the subsequent days, his symptoms resolved and there was a significant improvement in his hematological parameters, LDH, serum creatinine and bilirubin. The improvement, however, was not sustained after discontinuation of plasmapheresis, with the platelet count dropping from 171,000/ µL to 25,000/ µL. So a diagnosis of refractory TTP was made, and he was resumed on daily plasmapheresis with the initiation of weekly Rituximab for four doses. The frequency of plasmapheresis was then slowly tapered off, and he continued to remain asymptomatic while his hematological parameters stabilized with a platelet count of 257,000/ µL at discharge.

Discussion

Acquired TTP is immunologically mediated, and has been associated with several autoimmune disorders like systemic lupus erythematosus (SLE) and Hashimoto's thyroiditis. While several cases of TTP-like features in patients with pernicious anemia have been reported, actual TTP in such patients is rarely reported. This association between TTP and pernicious anemia needs further evaluation given the reported overlap in features of the two entities.

Resident / Fellow Clinical Vignette

<p>Author: Hong Shi, MD Additional Authors: Hong Shi, MD; Chris Adubor, MD; Daniela, Levi, MD; Dariush Alaie, MD; Richard Petrillo, MD, Yun Wang, MD Institution: Montefiore Mount Vernon Hospital</p> <p>Title: Severe Lactic Acidosis Induced by Septic Shock and Metformin Treated with Early Intermittent Hemodialysis: a Case Presentation</p> <p>Introduction: Lactic acidosis is generally defined as a serum lactate concentration above 4 mmol/L, is common among emergency room and ICU. Metformin-associated lactic acidosis (MALA) is an important cause of type B lactic acidosis. Although incidence of MALA to be fewer than 5.1 cases per 100,000 patient years, when it occurs, it is fatal in approximately 50% of cases. We present a case of severe lactic acidosis induced by septic shock and metformin toxicity.</p> <p>A 59-year-old male with a history of hypertension, diabetes mellitus cerebrovascular accident and peripheral vascular disease s/p right lower extremity angioplasty, stage 3 chronic kidney disease on metformin admitted with confusion. Upon arrival, patient was found to have hypothermia 88.6 F, bradycardia, dyspnea, hypotension BP 84 /53 mmHg. Arterial blood gas revealed a lactate of 14.5 mmol/L, with pH 6.76. WBC count 20,000/microL, BUN 71 mg/dL, creatinine 3.97 mg/dL, anion gap 31 mmol/L, glucose 173 mg/dL. Chest xray showed mild perihilar and interstitial opacities, and patchy and streaky opacities in the right lung base. Septic shock protocol was started, however remained refractory to treatment. The patient was subsequently intubated and placed on mechanical ventilation. The lactic acidosis worsened 21 mmol/L, pH 6.98. Emergent hemodialysis was started and resulted in rapid correction of the lactic acidosis and pH. On 3rd day of ICU the patient was extubated and transferred from ICU. Subsequently the urine Streptococcus pneumoniae antigen and urine Legionella antigen were positive. Blood culture grew Staphylococcus simulans and Aerococcus viridans, sputum culture showed gram (+) cocci in chains. Metformin concentration was 8.5 mcg/mL (normal range 1-2 mcg/mL) before hemodialysis.</p> <p>Discussion: Unexplained severe lactic acidosis is not rare in ICU. As some of the laboratory tests are either time consuming, or special equipment (eg LC-MS/MS method for serum metformin level) is needed, results are rarely available when the patient is critically ill. Therefore, a thorough consideration of the possible etiologies and prompt empiric treatment are important. Septic shock is associated with type A lactic acidosis. In patients with kidney injury metformin may further induce lactic acidosis type B. Early renal replacement therapy might be life-saving because it can eliminate both lactate and metformin. Timely consideration of hemodialysis in patients with severe lactic acidosis will help rapid recovery.</p>	<p>Author: Tannaz Shoja, MD Additional Authors: Samira Molai MD, Samir Sarkar MD Institution: Jamaica Hospital Medical Center</p> <p>Title: DIAGNOSIS AND MANAGEMENT OF NEUROLEPTIC MALIGNANT SYNDROME BROUGHT ON BY HEAT EXHAUSTION IN A PATIENT ON LONG TERM ANTIPSYCHOTICS</p> <p>DIAGNOSIS AND MANAGEMENT OF NEUROLEPTIC MALIGNANT SYNDROME BROUGHT ON BY HEAT EXHAUSTION IN A PATIENT ON LONG TERM ANTIPSYCHOTICS</p> <p>Tannaz Shoja MD, Samira Molai MD, Samir Sarkar MD Neuroleptic malignant syndrome (NMS) is a fatal idiosyncratic reaction to any medication with dopamine receptor-antagonist properties. Typical symptoms include fever, altered mental status, muscle rigidity and autonomic dysfunction. The onset of symptoms varies from hours to days after drug initiation, however nearly all cases develop within 30 days of starting medication. NMS is unpredictable and its diagnosis should be considered in patients with suggestive symptoms who are taking dopaminergic medications, even if the symptoms presents outside the usual diagnostic window. We report a case of a woman who developed signs and symptoms of NMS who was on the same neuroleptic regimen for more than a year. Her symptoms seemed to be precipitated by an episode of heat exhaustion.</p> <p>A 69-year-old female with bipolar disorder type I, PTSD, and schizophrenia, presented to the ER with altered mental status and temperature of 104.8&deg;F. Her son reported that she went for a walk the day before and was exhausted by the heat. As the day advanced, he noticed she was progressively talking less and was found unresponsive by the next day. Her psychiatric medications included quetiapine 400 mg, fluphenazine 10 mg, and mirtazapine 30 mg, which the patient had been taking for more than a year. At the ER, she had abnormal involuntary movements including perioral movements, was sweating and shivering, was tachypneic and tachycardic, and had muscle rigidity. The patient was intubated and was started on a benzodiazepine drip for agitation. The lumbar puncture was negative for meningitis and CPK was found to be 3954 (30-135 U/L) at its highest. All antipsychotic drugs were put on hold. She was managed in the intensive critical care unit and her mental status did not improve. A full dose of Dantrolene along with hydration and cooling blanket was given to reduce the core body temperature. The temperature came down to 100&deg;F in the first 24 hour. On day 6 of admission, the patient’s presenting symptoms had subsided and she was extubated. After stabilization she was transferred to her primary hospital for continuity of care and mental health management.</p> <p>Heat stroke can occur during treatment for psychiatric conditions and can precipitate NMS. Clinicians should be proactive to reduce the risk of heat stroke in such patients.</p>
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Title: A Rare Case of Mediastinal Hematoma Leading to Right Ventricular Outflow Tract Obstruction**Introduction:**

Patients with blunt chest trauma are subject to myriad cardiac complications including tamponade, myocardial contusion and cardiac rupture which could be life threatening (1). We report an unusual case of blunt chest trauma with subacute course and development of severe right ventricular outflow tract (RVOT) obstruction resulting from a large retrosternal hematoma.

Case Description:

A 55-year-old male with no comorbidities presented to the emergency room with mid sternal chest pain after sustaining a "Jack Hammer" injury. He denied any dizziness, palpitations, syncope or shortness of breath. On presentation, he was hemodynamically stable. Physical examination was non-revealing. Bedside focused assessment with sonography for trauma (FAST) was non-significant. CT scan showed well-defined 6.8 x 4.4 cm substernal hematoma. Surgical evacuation of hematoma was recommended but patient refused to undergo surgery and was discharged. On outpatient follow up, physical examination revealed new onset holosystolic murmur in left second intercostal space. Lab investigation revealed stable Hgb/Hct. Kidney, liver and cardiac enzymes panels were normal. EKG showed new onset right bundle branch block. Repeat chest X-ray revealed cardiomegaly with left sided pleural effusion. Repeat CT scan revealed enlarging substernal hematoma (8.5 x 4.5 cm) with severe extrinsic compression of right ventricular outflow tract with signs of active bleeding. 2-D ECHO was consistent with severe obstruction with gradient of 36 mmHg across RVOT. Patient agreed for surgery and underwent video assisted thoracoscopic surgery with evacuation of hematoma with thoracotomy and mediastinotomy. Post-operative course was unremarkable and he was discharged home in a stable condition with uneventful recovery.

Discussion:

Mediastinal hematoma after blunt trauma are most often seen after motor vehicle accidents and result from aortic or other major vessels injuries. Most patients die before reaching the hospital due to hypovolemic shock or extra cardiac tamponade (1). This case was unusual because of absence of any external or bony injury and the insidious course of mediastinal hematoma which led to right ventricular outflow tract obstruction. The diagnosis was missed initially on FAST screening. The low threshold for chest CT due to persistent pain (2), aided in diagnosis and timely management of a potentially life threatening complication.

References:

1. Coleman GM, et al. Blunt chest trauma. Extrapericardial cardiac tamponade by a mediastinal hematoma. *Chest*. 1989Apr;95(4):922-4
2. Hsu LW, et al. Traumatic mediastinal hematoma: a potentially fatal condition that may be overlooked by traditional Focused Assessment with Sonography for Trauma. *Am J Emerg Med*. 2013Jan;31(1):262.e1-3

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Title: Topiramate-induced secondary angle closure with myopic shift**Introduction:**

Topiramate is an anticonvulsant that is used commonly to treat patients with epilepsy, for migraine prophylaxis, and off label for cluster headaches. Although the side effects are more from its effects on the central nervous system like drowsiness and paresthesias. We present a very uncommon presentation of Topiramate-induced secondary angle closure with myopic shift

Report

54-year-old female with a history of anxiety, chronic headaches, depression, CKD and chronic low back pain presented with complaints of bilateral blurry vision and eye pressure. She was started on Topamax (Topiramate) a couple weeks ago for her "low back pain". She stated that she felt like she had grit in her eyes. She reported no other complaints.

The bedside ocular ultrasound showed that left optic nerve sheath was widened at 6 mm and the right optic nerve sheath was at the cutoff of normal at 5 mm. Intraocular pressures readings showed a pressure of 55 in the right eye and 53 in the left eye. Repeat showed 48 in the right eye and 46 in the left eye.

Ophthalmology frequently evaluated the patient did several rounds of multiple eye drops - 4 rounds of timolol, brimonidine, and latanoprost and 2 rounds of cyclopentolate OU and 3 rounds atropine OU with the final IOP before discharge being 33/35 in an attempt to lower the patient's intra-ocular pressures from 55/53 at presentation to the ED.

She was seen in the clinic the following day and she was found to have an IOP 29/26 without gtt since seen in ED. A diagnosis of Topiramate induced secondary angle closure with myopic shift was made and the patient was made and the patient was told to not take Topiramate anymore.

Discussion:

Topiramate is a Sulfamate-substituted monosaccharide. Since it is a sulfonamide derivative it has been, in rare occasions, implicated in the development of acute angle closure glaucoma. The mechanism of action seems to be due to induction of uveal effusion and increase in intraocular pressure. These sulfonamide medications lead to increased extracellular fluid around the ciliary body and choroid and this is believed to be an idiosyncratic reaction. The first presenting symptom is always blurring of vision. There are many case reports of AAC glaucoma and very rare uveitis as well. The treatment involves discontinuation of the offending sulfonamide drug, in this particular case Topiramate along with management of the intraocular pressure. The review of 115 case reports showed that the cases can vary from blurring of vision to acute angle closure with myopic shift all of which can be resolved acutely and are reversible with drug discontinuation. The review also found that peripheral iridectomy is ineffective in these cases for management.

Resident / Fellow Clinical Vignette

<p>Author: Spencer Tavares, DO Additional Authors: Lara Kollab, OMS III Institution: Orange Regional Medical Center</p> <p>Title: Intractable Abdominal Pain in a Neurofibromatosis Type 1 patient secondary to a GIST</p> <p>Gastrointestinal Stromal Tumors (GIST) are sub-epithelial tumors most commonly arising in the gastrointestinal tract. The majority of GISTs are sporadic in origin and are associated with activating mutations in PDGFRA or KIT proto-oncogenes. However, a small subset of GISTs are seen in the context of autosomal dominant familial disorders such as Neurofibromatosis Type 1 (NF1), and do not typically display these particular mutations. This report presents a case of a NF1 patient with intractable abdominal pain caused by multiple GISTs in the small bowel.</p> <p>A 45 year-old-female with a past medical history of NF1 was hospitalized with a two day history of progressively worsening right sided abdominal pain, bloating, nausea, vomiting, diarrhea, and anorexia. Patient was noted to have a leukocytosis, non-anion gap metabolic acidosis, but normal LFTs and lipase. An abdominal CT scan with IV contrast was significant for bowel wall thickening of the proximal jejunum. A CT angiogram was negative for ischemia. Her bowel pain persisted despite bowel rest and treatment with IVF, abx, and analgesics. On the tenth hospital day, patient underwent a single balloon enteroscopy with biopsy and found to have focal edema with erythema in the jejunum along with a linear clean based ulcer in the jejunum. Biopsy of the ulcer showed acute enteritis. Patient’s diarrhea and vomiting subsided but she continued to experience worsening right sided abdominal pain, early satiety, bloating, and distention. Patient underwent a rheumatologic workup for vasculitis as cause of the pain, which was only positive for elevated ESR & CRP, but a normal C1, INH, C2, C4. No TTG IgA, TTG IgG, or cryoglobulins were detected. On the 22nd hospital day, patient underwent an exploratory laparoscopy, converted to open exploration after inability to dissect significant adhesions of omentum to mid anterior abdominal wall, with a small bowel appearing to fistulize into the omentum. A one foot long section of small bowel with an emanating large tumor was resected and sent for pathology. The open exploration was also notable for smaller non-obstructive tumors. Patient improved very quickly after surgery and was discharged from the hospital in good condition. Tumor pathology was consistent with a GIST.</p> <p>This case demonstrates the benefit of early suspicion of a stromal tumor in NF1 patients, and outlines which tests did and did not aid in identification. The identification of a stomach ulcer in this case may have delayed the identification of the GIST. Other studies that were discussed during hospitalization such as CT enterography, MRI with oral contrast, PET scan, and capsule endoscopy, could hypothetically have yielded diagnostic results earlier in the course of the hospitalization, but ultimately, surgery was required for repair.</p>	<p>Author: Julia Tian, MD Additional Authors: Nabil Zeineddine Institution: Staten Island University Hospital</p> <p>Title: Gastrointestinal bleeding as initial presentation in organ specific amyloidosis</p> <p>Introduction: Non-systemic, biopsy proven amyloidosis isolated to the gastrointestinal tract is particularly rare. Bleeding is the presenting symptom in less than half of all patients with gastrointestinal tract amyloidosis. This case describes a 76-year-old male with AL amyloidosis who manifested with a lower GI bleed secondary to amyloid induced colonic ulcers. Case presentation: A 76-year-old Caucasian male presented with an acute onset of hematochezia with symptomatic anemia related to blood loss. His vital signs on presentation were normal except for sinus tachycardia. After receiving initial volume resuscitation, the patient was taken for a colonoscopy, which revealed multiple colonic ulcers. Histopathological examination of biopsies taken during the procedure revealed thick walled blood vessels with acellular eosinophilic material, consistent with amyloid on congo red staining.</p> <p>Workup for ruling out other organ involvements including electrocardiogram, transthoracic echocardiography, liver function tests and urinalysis for proteinuria failed to reveal any systemic involvement. Multiple myeloma was also ruled out after normal serum and urine protein electrophoresis, serum immunofixation and absence of urine free light chains.</p> <p>The patient was diagnosed with primary AL amyloidosis localized to the gastrointestinal tract, and was treated conservatively without any need for chemotherapy or immunomodulators.</p> <p>His case represents a rare manifestation of amyloidosis that is unrelated to plasma cell dyscrasias and localized only to one organ system.</p> <p>Conclusion: Symptomatic amyloidosis of the gastrointestinal tract is unusual. Including amyloidosis within the differential diagnoses of gastrointestinal ulcers may prove befitting, particularly in the elderly population. Once diagnosed using histopathological tools, follow up management should include organ specific workup to rule out liver, heart, and kidney involvements. Our patient represented an isolated case of amyloidosis localized to the gastrointestinal tract, manifesting as col ulcers and non-life threatening bleed.</p>
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Resident / Fellow Clinical Vignette

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Title: SEVERE HYPERKALEMIA WITHOUT EKG CHANGES!

Introduction:

Potassium plays a key role in maintaining electrical stability across cellular membrane and processes of depolarization and repolarization. Elevation in potassium level correlates with electrocardiographic changes and severe hyperkalemia causes fatal dysrhythmias.

Our case is of severe hyperkalemia (potassium level >7 mEq/L) with no associated electrocardiographic changes in a patient with chronic renal failure.

Case Description:

A 69 years old male with chronic renal failure due to diabetic nephropathy presented to our emergency department for evaluation of a pruritic rash. Vital signs were normal. Emergency department work up revealed hemoglobin level 10.2mg/dl, white cell count 8.7, potassium level of 9.1mmol/L with BUN (blood urea nitrogen) of 68mg/dl, creatinine of 5.52 mg/dl, sodium 153mmol/L, chloride 131mmol/L, carbon dioxide 13mmol/L. EKG was without any T wave changes. P waves were present, PR interval was 160ms and the QRS duration was 112ms. The patient was treated aggressively with intravenous (IV) insulin, 50% dextrose, albuterol inhalation, kayexalate and IV calcium gluconate. The potassium level came down to 8.4mmol/L. Plasma potassium was reported as 8.1mmol/L. Multiple electrocardiograms recorded no changes. He was admitted to the Intensive Care Unit and started on dialysis via quinton catheter. He had long standing chronic kidney disease and had not followed up with a nephrologist and so had not been getting any dialysis. With dialysis, his potassium corrected to 3.8mmol/L and then 4.1mmol/L; EKG continued to show no dysrhythmias.

His hospital stay was complicated by bilateral lower extremity deep venous thrombosis, anticoagulation with heparin was initiated which he later refused. Patient also refused AV fistula creation. He was discharged to follow up with a nephrologist closely. He has presented to the emergency room since then with hyperkalemia because of refusal for long term dialysis.

Conclusion: According to literature, the most prominent effect of hyperkalemia is on the myocardium and even mild hyperkalemia is associated with T wave changes, known as tenting. More severe hyperkalemia results in a decrease in membrane potential, leading to a decrease in myocardial cell velocity which results in increased repolarization and increased PR interval, increased QRS duration and flattening of the p wave according to literature, AS Narula et al (Severe hyperkalemia with normal electrocardiogram; Indian J Nephrol 2005;15: Supplement: S34-S36).

Hyperkalemia can lead to fatal cardiac arrhythmias and few cases of severe hyperkalemia without EKG changes have been reported. The absence of EKG changes may possibly be secondary to chronicity of hyperkalemia in those instances. Our patient also had a normal EKG with no T wave, p wave, PR interval or QRS complex changes and his hyperkalemia was successfully treated with dialysis and medications.

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Title: Postoperative hypotension: Were you using a nasal spray?

Introduction:

Postoperative hypotension can be present in one of every six patients who undergo bilateral knee replacement. It is usually secondary to effect of anesthesia and pain medications. We present a case of persistent post operative hypotension in a patient who had relative adrenal insufficiency secondary to preoperative fluticasone nasal spray use.

Case Presentation

A 57 year-old female with past medical history of hypothyroidism status post partial thyroidectomy and osteoarthritis was admitted after bilateral knee replacement surgery. Medications at home included Levothyroxine. Preoperative TSH level was normal. Intraoperative, patient had stable vitals with minimal blood loss. After surgery, her vitals were stable with blood pressure (BP): 108/64 mmHg, heart rate: 70/min and respiratory rate: 12/min. Overnight, her BP dropped to 69/37 mmHg. She did not have dizziness or palpitation; and the physical examination was normal. Epidural anesthesia was held. She was bolused with 500ml of normal saline (NS) and was placed on maintenance fluid at 100ml/hr of fluid. BP repeated after 2 hours was 72/45 mmHg. She was re-bolused with a liter of NS. On further questioning, patient reported daily use of fluticasone nasal spray for rhinosinusitis for the last 8 weeks. Morning cortisol level was sent but presumptive glucocorticoid replacement was avoided to prevent complications including impaired wound healing. Patient's BP remained low for the next 48 hours and required IV fluids. Her serum cortisol was reported to be 0.7 mcg/dl (normal range: 4-22), suggesting suppressed hypothalamic-pituitary-adrenal (HPA) axis from her nasal corticosteroid use. Patient's blood pressure gradually stabilized and was subsequently discharged to rehabilitation facility.

Discussion:

Fluticasone is highly lipophilic and has high glucocorticoid receptor affinity. This causes increased tissue distribution, prolonged pulmonary receptor occupancy and longer plasma half-life. After repeated doses, it demonstrates a moderate degree of accumulation in the adipose tissues. It has been postulated that such accumulation, rather than peak plasma concentration, suppresses the adrenocorticotropin hormone via negative feedback control. This leads to the adrenal atrophy and loss of cortisol secretory capability. During a time of stress, like surgery, the adrenal gland is unable to respond maximally. This causes relative adrenal insufficiency and it commonly manifests as hypotension. Hence, it is imperative for medical professionals to be aware that prolonged use of corticosteroids as nasal spray can cause HPA suppression.

Resident / Fellow Clinical Vignette

<p>Author: Wen Zhang, DO Additional Authors: Franco Vallejo, MD Institution: Mount Sinai St. Luke's and Mount Sinai West</p> <p>Title: Eszopiclone overdose leading to hypothermia and shock</p> <p>Eszopiclone (brand name Lunesta) is a non-benzodiazepine hypnotic for use in insomnia. It has a rapid in onset and has less daytime sedation, withdrawal and dependence effects in comparison to benzodiazepines, which makes it an attractive drug for use. There are few published cases of significant eszopiclone overdose in literature to date, and there is only one case report found of eszopiclone overdose with associated hypothermia requiring pressor support, which was presented in an elderly female who had co-ingested other substances including benzodiazepines and had been found down outside after an unspecified period of time.</p> <p>We present a case of a 35 years-old female with history of bipolar disorder and alcohol abuse. The patient and her husband had an altercation at their hotel room, whereupon the patient took around 15-20 pills of lunesta (home dose 3 mg/day). The patient then became very drowsy and eventually unresponsive after 10 minutes, whereupon the husband called 911. Patient noted to also have had at least 4 martinis at dinnertime that day. In the emergency department she was noted to be hypothermic to 93.2F, respiratory rate of 6 ppm and heart rate of 125 ppm. The patient required intubation for airway protection. Laboratory findings were remarkable for alcohol level of 154. Other labs, including hepatic function panel were normal. Urine toxicology panel (opiates, methadone, benzodiazepines, PCP, cocaine, amphetamines) was negative. EKG showed sinus tachycardia. She was admitted to the ICU and initially was hypotensive requiring norepinephrine. Blood pressure and temperature normalized within 1 day permitting stop vasopressors. Upon extubation, the patient was initially disoriented and confused, but eventually returned to her baseline mental status within a couple of days.</p> <p>Chronic insomnia is noted to occur in as many as one quarter of the population and can be present in as many as 9 in 10 patients with depression. In a multicenter study, eszopiclone was noted to have improvement in insomnia in a diverse population of patients, although had smaller effects in patients with Major Depressive Disorder in comparison to patients with primary insomnia only. In the US, there is no clear toxic concentration levels as yet established for eszopiclone. Overdoses however have been known to lead to hypoxia, pulmonary edema, respiratory failure, methemoglobinemia, renal insufficiency and coronary vasospasm. This case report highlights the importance of considering lunesta as a potentially easily available drug of abuse that is often overlooked and is also the only case study where lunesta overdose was thought to be the main culprit of hypothermia and distributive shock.</p>	<p>Author: Gurjwan Virk, MD Additional Authors: Mikram Jafri, MD; Sajid Hussain, MD; Christopher Ashley, MD Institution: ALBANY MEDICAL CENTER</p> <p>Title: Screening for Colorectal cancer (CRC) in Octogenarians and Nonagenarians: Nationwide Study of US Veterans</p> <p>Background: USPSTF recommends against continuing screening for colorectal cancer past 75 years in adequately screened individuals [1]. Research has shown that one time screening for elderly who have never been screened appears to be cost effective till 86 years of age [2]. Age should not be the sole criteria for screening recommendations and stratification should be made based on life expectancy and risk benefit ratio [3]. According to SEER registry data from 2012, incidence of CRC is 48/100,000 for males under 65, 246/100,000 for males aged 65 and older, 319/100,000 for males 80-84 and 353/100,000 for males aged above 85 [4]. Objective: To determine prevalence of colon cancer screening and incidence of colon cancer in octogenarians and nonagenarians. The primary end point is determining the incidence of CRC in these groups. Early detection may benefit patients who have life expectancy greater than 10 years.</p> <p>Design: Retrospective study Setting: Male veterans greater than 80 years with or without prior screening who underwent screening colonoscopy at a Veterans affairs hospital between 2000 and 2015. Data was extracted using VINCI* National database using ICD 9/Codes. Inclusion criteria includes male age >80 years with or without prior screening. Exclusion criteria includes age<80, history of CRC, previous colorectal surgery, known genetic predisposition to CRC.</p> <p>Methods and Results: 458,224 patients with Age>= 80 were obtained from the database between years 2000 to 2015. This was divided into age 80-84 years (89,621 patients), 85-90 years (248,155 patients), >90 years (120,448 patients). A total of 81,946 (17.88%) underwent screening colonoscopies of which 9365 (11%) were diagnosed with colon cancer. Reported Colon cancer among these patients receiving screening colonoscopies were as follows;1889 in group 80-84, 4463 in group 85-90 and 3013 in group >90 years old. Colon cancer prevalence among these different age groups was calculated based on total population at risk within these age groups.</p> <p>Conclusions and discussion: There is an increase in colonoscopies and prevalence of CRC cancer for people of age > 90 compared to other age groups. Our results showed a significant drop in prevalence of CRC in age group 80-85 years with subsequent increase after 90 years of age suggesting screening may be beneficial in 80-85 age group to avoid future development of CRC.</p> <p>REFERENCES and TABLES : To be available on poster.</p>
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Resident / Fellow Clinical Vignette

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Title: A Rare Case of Weil's Syndrome in a Slaughterhouse Worker

Introduction:

Leptospirosis is generally a mild and self-limiting zoonotic disease. Rarely, it may progress to a potentially fatal form known as Weil's syndrome which is characterized by the classic triad of jaundice, acute renal failure, and bleeding complications. In these cases, speedy identification and treatment as well as prompt notification of the CDC are crucial.

Case:

A 46 year-old male slaughterhouse worker presented to our institution with six days of subjective fevers, chills, shortness of breath, fatigue, and lower extremity myalgias. He developed multiple episodes of watery diarrhea for three days and had one episode of epistaxis on the day of presentation. He reported that two weeks prior to this presentation, he had a coworker who suddenly became ill with similar symptoms and expired. On exam, the patient was noted to have conjunctival suffusion and worsening respiratory distress. He was subsequently admitted to the medical intensive care unit for a rapidly progressive systemic illness with new renal failure, hepatic dysfunction, rhabdomyolysis, marked thrombocytopenia and mild coagulopathy. He progressed to septic shock with profound multisystem organ failure, briefly requiring intubation for acute respiratory distress syndrome and pressor support. He was started on empiric antibiotic coverage and weaned off of pressors. Hemodialysis was initiated for acute renal failure. On hospital day five his urine leptospira PCR returned positive and his antibiotics were narrowed to ceftriaxone and doxycycline. The patient made a remarkable recovery of pulmonary, cardiovascular, and gastrointestinal organ systems and was discharged home on hospital day 17 in good condition though still requiring hemodialysis.

Discussion:

First characterized by Adolf Weil in 1886, leptospirosis is a zoonotic infection transmitted from the urine of an infected animal (usually livestock or rodents) to humans via abraded skin and rarely by ingestion. Infection in the United States is rare with most occurring in the southern and pacific coastal states. Symptoms typically start after a 10 day incubation period. The mortality of Weil's syndrome despite treatment is estimated to be 10%, with 52% for those admitted to the intensive care unit. After the initial tubulointerstitial nephritis causing acute renal failure, complete renal recovery typically takes three to six months. As our patient contracted a rare zoonosis, the Center for Disease Control (CDC) was alerted. Through the CDC, we learned that our patient's colleague expired from leptospirosis. Given the obvious occupational concerns, the slaughterhouse was closed by the NY Health Department. Our patient is one of three cases in the first cluster of leptospirosis in New York City, with only 26 cases reported from 2006 to 2016.

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Title: A Curious Case of Acute Liver Injury

INTRODUCTION:

Drug-induced hepatotoxicity is a frequent cause of liver injury and accounts for approximately one-half of the cases of acute liver failure. It predominantly presents as acute hepatitis and/or cholestasis but it can essentially mimic all forms of acute and chronic liver disease.

CASE PRESENTATION

A 48 year old incarcerated man with a history of Hepatitis C and polysubstance abuse was admitted for painless jaundice, chronic diarrhea and unintentional weight loss of 15 pounds in the past one year. Denied recent substance abuse since past 6 months. Vitals were normal and physical exam was unremarkable except for presence of icterus. Labs revealed transaminitis with cholestasis (ALT- 133, AST- 176, ALP-143, total bilirubin-16.4, direct bilirubin 9, INR- 1.1). Tests for acute hepatitis A, B, CMV, HSV, and EBV were negative. Autoimmune markers for anti-nuclear, anti-smooth muscle and antimitochondrial antibodies were negative. Serum Ceruloplasmin, alpha-1 antitrypsin and TSH were normal. USG abdomen, CT abdomen/pelvis, MRCP and subsequent ERCP were unremarkable. Liver biopsy revealed toxic injury (predominantly Zone 3) in addition to changes associated with chronic Hepatitis C (Grade 2, Stage 2-3). On further questioning patient denied any medication use but did reveal recent cocaine use while in custody. During the hospital course the bilirubin peaked to 26 and then trended down with the other liver enzymes. Patient experienced symptomatic relief with conservative management and was followed up as outpatient.

DISCUSSION

Although investigative research of animal models in cocaine metabolism and associated liver cell injury has been fairly extensive during the past 10 years, little evidence of hepatotoxicity has been documented in man. The mechanism of liver injury is related to the production of reactive metabolites, with peroxidation, free radical formation, and covalent binding to hepatic proteins. Hepatotoxicity usually arises hours to a few days after an acute overdose. Initially, serum aminotransferase and LDH levels are markedly elevated with minimal increase in alkaline phosphatase. The serum bilirubin begins to rise after 2 to 3 days. Liver histology usually shows centrilobular (zone 3) necrosis and fatty change, features that resemble liver injury due to drugs. The liver injury due to cocaine is usually self-limiting and resolves rapidly on discontinuation of the drug. There is no specific antidote for acute cocaine toxicity but infusions of N-acetyl cysteine are often given because of the similarity of the injury to acetaminophen hepatotoxicity.

CONCLUSION

Drug induced liver injury has a wide histological and clinical spectrum. Outcomes of cocaine intoxication may vary from acute fulminant hepatic failure to significant transaminitis and hyperbilirubinemia which may resolve on its own in 1-2 weeks without any treatment. It is important for clinicians to consider substance abuse as one of the differentials in cases of unexplained transaminitis and hyperbilirubinemia.

Resident / Fellow Clinical Vignette

<p>Author: Matthew Wieder, MD Additional Authors: Margarita Chernovolenko, MD; Veevek Anand Agrawal, DO Institution: Mount Sinai Beth Israel</p> <p>Title: A CURIOUS CASE OF SCLERITIS: AN AGGRESSIVE MALIGNANCY MASQUERADES AS SCLERITIS</p> <p>Case Presentation: A 56 year old West African man with HIV on HAART (CD4 694, undetectable viral load) presents for evaluation of a lung mass found during a workup for vision loss of the left eye (OS). He was recently diagnosed with anterior and posterior scleritis, exudative retinal detachment, and a sub-retinal mass OS by his ophthalmologist and underwent a chest X-ray revealing a right upper lobe mass, for which he was referred to the ER. The patient describes a one month history of progressive visual loss OS associated with tenderness to palpation and lacrimation. He denies headaches, trauma, or a history of similar symptoms in either eye. He endorses a 20 pound weight loss, but denies fevers, chills, night sweats, cough, hemoptysis, epistaxis, change in bowel habits or skin lesions. PPD five years prior was negative. On exam, he is afebrile with mild tachycardia, normal blood pressure and respiratory rate with an oxygen saturation of 100% on room air. Eye exam OS is notable for injection and visual acuity limited to hand motion. He has clear lungs to auscultation, no rashes and no palpable cervical, axillary or inguinal lymphadenopathy. Workup is notable for elevated ESR, but normal ANA, RF, RPR, ACE, and ANCA. CT chest shows a large central right upper lobe mass, a right lower lobe nodule, and multiple mediastinal lymph nodes. CT abdomen reveals multiple masses in the adrenals, a solid 1.5 cm left kidney mass, a mass indenting the liver, as well as a nodular lesion in the superficial abdominal fat pad. MRI brain shows multiple metastases throughout the brain. Findings are consistent with metastatic disease of pulmonary or renal origin. Biopsy of the mass abutting the liver confirms adenocarcinoma.</p> <p>Discussion: Scleritis is often the initial presentation of an underlying systemic illness. Although the eye is generally a rare site of malignancy, when cancer is present, it is most often due to metastatic disease. Our case demonstrates two potential vision-compromising complications of choroidal metastasis, specifically exudative retinal detachment as well as anterior and posterior scleritis. While exudative retinal detachments due to choroidal metastasis are well documented, malignancy associated scleritis is rare and has been documented in only a few case reports.</p> <p>Conclusion: While scleritis as an initial presentation of metastatic cancer may be rare, it has been described in the literature. There may be times when scleritis is the first and perhaps only sign of deeper underlying pathology. It is thus prudent for an astute clinician to investigate each case of scleritis in depth. If routine treatments do not promptly lead to resolution of symptoms, then the differential should expand to include the possibility of systemic diseases such as metastatic malignancy.</p>	<p>Author: Christopher Wilcox, DO Additional Authors: Robbie Wall, DO Institution: CHS Internal Medicine Training Program University at Buffalo</p> <p>Title: Possible Mahaim fiber in Wolff-Parkinson-White Syndrome</p> <p>Mahaim fibers are accessory cardiac conduction pathways that possess unique properties in regard to signal transmission between the atria and ventricles. They are often associated with second accessory pathways and complicate the success of cardiac ablation procedures. The patient is a 47 year old Caucasian male that experienced episodes of palpitations with associated dizziness and lightheadedness without chest pain, shortness of breath, or syncope. He has a medical history of hyperlipidemia and hypertension. He works as a machinist and reports no illicit drug, alcohol, or tobacco use. He reported no arrhythmias or sudden cardiac death in his family. The Physical exam and laboratory values were within normal limits. His EKG demonstrated Wolff-Parkinson-White pattern. The EKG as well as intra operative electrophysiology mapping system predicted the patient to have left atrial accessory, or pre-excitation, pathway. Extensive left atrial ablation based upon intraoperative pre-excitation signal detection did not eliminate pre-excitation but did change EKG pattern. A subsequent ablation of a single spot in the right atrium completely eliminated pre-excitation. The possibility of Mahaim fiber connecting to left posterior fascicle was considered due to unusual behavior during electrophysiology study. After a review it seems there were either two accessory pathways or less likely an extensive bilateral oblique pathway. The right atrial pathway did not demonstrate decremental conduction, a defining property of Mahaim fibers, however the left atrial pathway was not able to be isolated and therefore Mahaim fiber cannot be ruled out. Forty percent of Mahaim fibers are associated with second pathways and in this case a Mahaim fiber in the left atrium may have been the reason ablation and isolation of the pathway was difficult.</p> <p>The case highlights the need for retrospective analysis of unusual cases in the field of electrophysiology to hopefully increase accuracy for targeted ablation in the future. In this case, the patient's right atrial pathway was not detected by either electrophysiology mapping system or pre-op EKG analysis causing an extended but successful ablation procedure.</p>
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Title: Emotional Malady

Bahishta Yaqubi, PGY-2, Department of Medicine, New York Presbyterian - Brooklyn Methodist Hospital, Brooklyn, NY.

A 69 year old woman with past medical history of hypertension, hyperlipidemia and nephrolithiasis presented to the Emergency Department for acute onset chest pain and dyspnea after attending her granddaughter's wedding. Pain was described as substernal, continuous, pressure-like, 5/10 in severity, radiating towards her right shoulder, worse with exertion and improved with rest. Patient reported she was emotionally distressed with her husband's recent diagnosis of malignancy and the wedding preparation. Patient's ECG showed ST elevations in I, II, aVF, V5, V6, and Q waves in II, III, and V6. Troponin I and CKMB was elevated. Patient underwent cardiac catheterization which was consistent with right dominant circulation and patent coronaries. Patient's echocardiogram was obtained which showed an EF of 60% and moderate hypokinesis of the apical septal and apical lateral walls. Subsequent ECGs were consistent with T wave inversion in lead I, II, V4, V5, V6 and QT prolongation and resolution of prior Q waves in II, III and V6. Patient was diagnosed with Takotsubo Cardiomyopathy. Our patient's left ventricular function returned to normal 2 months after discharge with resolution of the ECG findings.

Takotsubo Cardiomyopathy which is also known as "broken heart syndrome" and "transient left ventricular apical ballooning syndrome". This condition is characterized by severe but transient left ventricular dysfunction caused by a wide variety of emotional or physical stressors. It is hypothesized to be caused by diffuse catecholamine-induced microvascular spasm/inflammation causing myocardial stunning or direct catecholamine-associated toxicity. This condition has 90% female dominance and most commonly seen in postmenopausal women with suspected acute coronary syndrome (ACS). Mean age is about 62-76 years. Clinically, differentiating between Takotsubo cardiomyopathy and ACS is always a challenging task. However, ECG findings, Echocardiography and negative cardiac catheterization can help. Most common ECG findings are ST segment elevation in the precordial leads and T wave inversion in most leads. With time, T waves evolve and deepen further within 3 days and transiently turn shallow and then become significantly deeper in 2-3 weeks. T wave changes may revert to normal in 2-4 months. Prolongation of QTc may occur within first 24-48hrs. Q waves are seen in 56% of the cases. The prognosis of takotsubo cardiomyopathy is generally considered favorable. Our patient demonstrated many of the features of this syndrome and has had an excellent outcome.

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Title: THYROID STORM AND AFIB WITH RVR IN THE SETTING OF RECENT MI, SEVERELY REDUCED EF AND LV THROMBUS: A MANAGEMENT DILEMMA

Introduction

Thyroid storm is an endocrine emergency which requires prompt diagnosis and early aggressive treatment. However alternative, more invasive treatment options may be necessary to achieve clinical stabilization. Here we present a case of thyroid storm presenting with life-threatening cardiovascular manifestations.

Case

This is a 68-year-old male with a PMHx of afib treated with amiodarone, and recent NSTEMI s/p PCI with drug-eluting stents to mid and distal LAD who presents with dyspnea and admitted for symptomatic heart failure and afib with RVR. TSH was 0.004, free T4 was 6 and free T3 was 445 likely related to amiodarone use and iodinated contrast from recent cardiac catheterization. Methimazole, prednisone, and metoprolol were started. TTE revealed an EF of 15% with a LV thrombus. Rate control was not achieved with low dose metoprolol and higher doses were not tolerated. Concurrent use of digoxin was also unsuccessful in controlling the heart rate. Of note, potassium iodine was not given to try and further suppress thyroid hormone release as there was a concern for exacerbating already present iodine-induced thyroid disease. Electrical cardioversion was to be done however the patient later spontaneously converted back to normal sinus rhythm on metoprolol and digoxin. Vital signs improved subsequently and soon after the patient was discharged with close follow up with outpatient Cardiology and Endocrinology.

Discussion

Patients diagnosed with thyroid storm should urgently be placed on a beta-blocker, thionamide, and steroids. Particularly in afib, with concern for tachycardia-induced decline in myocardial function, beta-blocker's anti-adrenergic and anti-thyroid effects are paramount. However beta-blockers should be used with caution in patients with decompensated HF, especially HFrEF. In our case, digoxin had proven very useful. If afib is still refractory to medical management, cardioversion should be done. With regards to cardioversion in patients with LV thrombus, there is a general hesitancy to do so given fear of thromboembolic phenomena. However data is limited with regards to the rate of thromboembolic events in these patients. In fact there is evidence to suggest that cardioversion in these patients is safe. Lastly, if life-threatening symptoms still persist despite all the above measures, radioactive iodine therapy and/or thyroidectomy should be considered which may be life-saving in patients with persistent thyroid storm.

Conclusion

Thyroid storm has a relatively low incidence, however it has a mortality of 20-50%. Most cases of thyroid storm can be managed with standard first line treatments; however our case required consideration of other more invasive approaches. Given this, an increased knowledge and awareness of the disease manifestations and of the treatment options are of extreme importance.

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Title: Use of Mifepristone in ACTH-dependent Cushing's Syndrome due to Small Cell Carcinoma of Prostate Origin

Purpose: Small cell carcinoma of the prostate is a rare, high-grade malignant neoplasm with neuroendocrine differentiation. Prostate adenocarcinomas can undergo neuroendocrine differentiation from genetic alterations after androgen deprivation therapy, and they can cause Cushing's syndrome. In most cases of Cushing's syndrome caused by ectopic ACTH-secretion, surgical excision of the tumor is first considered; if nonresectable, then ketoconazole is often used. Mifepristone is an alternative drug that can be used to block the effects of hypercortisolism.

Case Presentation: Patient is a 74 year-old male with metastatic prostate cancer, diagnosed in 2008, treated with radiation and chemotherapy. Biopsy results in 2008 and 2012 showed prostate adenocarcinoma. He was recently found to have metastasis to the liver, and underwent a biopsy showing high grade neuroendocrine carcinoma of small cell type. 3 weeks prior to admission, the patient was started on denosumab and leuprolide. He presented to Winthrop University Hospital due to weakness and unsteady gait accompanied by numbness of his fingers and face, bilateral lower extremity edema, and hyperpigmentation of face and right hand. His physical examination was notable for palpable, enlarged liver, moderate to severe edema of lower extremities bilaterally, and hyperpigmented plaques on right hand and face. Otherwise, there was no clear evidence of Cushingoid appearance such as muscle weakness, moon facies, dorsal fat pads or striae. His laboratory results showed hypokalemia (potassium=2.8mEq/L), hypocalcemia (calcium=6.1mEq/L), metabolic alkalosis (pH=7.56), and transaminitis (ALT=219IU/L, AST=178IU/L, ALP=431IU/L). His baseline AM cortisol level was 64.7mcg/dL, with an increase in the level to 92mcg/dL both post low-dose (1mg) and high-dose (8mg) overnight dexamethasone suppression test. His 24-hour urinary free cortisol was 1502ug, and his ACTH level was 167.2ng/dL. His chromogranin A level was 465nmol/L. Small cell carcinoma of prostate origin causing ACTH-dependent Cushing's syndrome was considered. Due to the patient's transaminitis, mifepristone was initiated as a treatment of choice.

Conclusion: Mifepristone blocks the glucocorticoid receptor at high dose, leading to significant clinical improvement in patients with Cushing's syndrome who are poor candidates for surgery and other adrenal enzyme inhibitors. Patients taking mifepristone may experience adrenal insufficiency and should be monitored for clinical manifestations of adrenal insufficiency since biomarkers are not available to monitor its effect. Hence, clinical guidelines to monitor the response to mifepristone should be investigated to better care for the patients.

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Title: Serum Potassium Level and Length of Hospital Stay in Acute Decompensated Heart Failure

Purpose: An abnormal potassium level is common in patients with heart failure (HF) and associated with worse outcomes. We assessed the association between admission level of potassium and length of stay (LOS). Method: Electronic medical record of a cohort of 127 consecutive patients was retrieved with the diagnosis of acute decompensated heart failure (ADHF) from 2015 to 2016. Chart review was performed to determine demographics, lab parameters on admission, and LOS. The cohort was divided into two groups based on potassium level (K =5.0 mEq/L or >5.0 mEq/L). Statistical analysis includes descriptive statistics and multivariable regression analyses. Results: The cohort includes 127 patients (54% males) with the mean age of 69 ±17; 13 years. Hyperkalemia (K >5.0 mEq/L) was present in 24 patients (18%). After stratification, no significant difference was found in demographic and clinical characteristics among the two groups. Serum creatinine(Scr), blood urea nitrogen (BUN), and BUN/Scr ratio were higher in hyperkalemia patients [median (IQR): 1.6 (1.3-2.0) vs. 1.2 (1-1.6) mg/dL, p=0.01; 38 (29-50) vs. 23 (16-36) mg/dL, p<0.01; 23 (21.1-30.4) vs. 20.3 (16.7-24.5), p=0.04], and hemoglobin (Hb) was lower in hyperkalemia patients (11.2±1.7; 2.5 vs. 12.1±1.7; 2.1 g/dL, p=0.03). A larger proportion of patients with K =5.0 mEq/L were on an angiotensin-converting enzyme inhibitors(ACEI)/angiotensin- renin blockers(ARB) (68% vs. 25%, p<0.01). Patients with hyperkalemia had longer LOS [median (IQR): 10.5 (6-14) vs. 7 (4-10) days, p=0.01], and the higher fraction of hyperkalemia patients had LOS >5 days (83% vs. 63%, p=0.04). Linear regression analysis demonstrated an association between potassium level and LOS after multivariate adjustment with age, sex, race, chronic kidney disease (CKD), use of mineralocorticoid antagonist, use of ACEI/ARB, BUN/Cr ratio, baseline GFR, and baseline creatinine (ß=0.23, p=0.04). Compared with K =5.0 mEq/L, patients with hyperkalemia had significantly higher odds of prolonged LOS (>5 days) [OR (95% CI): 3.76 (1.03-14.93), p=0.035] after adjustment. Conclusion: Admission potassium level is associated with LOS in ADHF patients after multivariate adjustment. Surprisingly, a lower proportion of hyperkalemia patients were on an ACEI/ARB, suggesting another mechanism could contribute to the potassium abnormalities. Aggressive measures to control potassium such as dietary modification and newer medications to control potassium may aid in the optimal management of ADHF. More studies are needed to confirm our findings.

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Title: Recurrent radial artery thrombosis and polydigital ischemia due to ESBL E-Coli septicemia

BACKGROUND:

Sepsis-induced purpura fulminans is a rare but life-threatening disorder, characterized by hemorrhagic infarction of the skin caused by hypotension and complement activation leading to dermal vascular thrombosis which is usually seen in children <2 years, commonly in the setting of meningococcal or pneumococcal sepsis. Purpura fulminans associated with E-coli septicemia is rare in adults, and has only been reported twice in literature to the best of our knowledge. We present a case of purpura fulminans associated with Extended-Spectrum-Beta-Lactamase (ESBL) producing E-Coli septicemia complicated with development of recurrent radial artery thrombosis.

CASE:

A 64-year-old male with past medical history of bladder cancer status post resection, urostomy, and atrial fibrillation, on Warfarin, presented with fever and chills. He was diagnosed with septic shock and multiorgan dysfunction syndrome. Renal ultrasound, as part of renal failure work up, revealed severe hydronephrosis prompting urgent insertion of bilateral nephrostomies. He was transferred to the Intensive Care Unit where he required Norepinephrine and Vasopressin for 72 hours during which his blood pressure was monitored via a right radial arterial line. Urine and blood cultures grew ESBL E-coli which were treated with imipenem. Two days after the removal of arterial line, he developed mottling and ischemia of right hand, and was found to have radial artery thrombosis diagnosed by Doppler ultrasound. He underwent thrombectomy and was started on therapeutic heparin infusion. Twenty four hours later, he developed re-occlusion of radial artery despite heparin. He required repeat thrombectomy, and tissue plasminogen activator infusion. Fifteen hours later, his radial artery occluded for a third time and he developed cyanosis and mottling of his left hand and bilateral feet with hemorrhagic bullae. Concomitantly, his platelet count dropped from 111 to 65 which prompted work-up for Heparin induced thrombocytopenia, however, PF4 assay was negative. Other labs revealed elevated Fibrin-split products, elevated Fibrinogen, normal LDH and haptoglobin and negative anti-cardiolipin antibodies and lupus anticoagulant. Polydigital ischemia of the left hand and feet improved with anti-coagulation but patient suffered from gangrene of right hand necessitating future amputation.

CONCLUSION:

Cardinal manifestations of sepsis induced purpura fulminans include presence of skin ecchymosis followed by development of gangrene and formation of vesicles and bullae, which marks the development of hemorrhagic necrosis all of which were present in our patient. While radial artery thrombosis may have been secondary to radial artery cannulation, it is worth mentioning that the patient developed recurrent thrombosis after thrombectomy with absent radial and palmar arch signals on sonography despite being on therapeutic anticoagulation. Simultaneous involvement of all other extremities is further supportive of the diagnosis. Our case demonstrates the importance of recognizing early signs of skin necrosis in patients with septic shock, and considering the diagnosis of purpura fulminans with uncommon organisms

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Title: Plugging the Filter: Thrombosed Inferior Vena Cava Filter Causing Acute Renal Failure

Current guidelines recommend retrievable inferior vena cava (IVC) filters when both the risk of recurrent pulmonary embolism (PE) and the risk of bleeding with anticoagulation are high. The substantial burden of filter thrombosis warrants their removal once it is safe to resume anticoagulation therapy. We present a unique case of extensive filter thrombosis resulting in acute renal failure. An 86 year-old obese African American female presented with acute lower back pain, bilateral lower extremity swelling, and decreased urine output over two days. Her medical history included hypertension, hyperlipidemia, diastolic congestive heart failure, untreated deep vein thrombosis one year prior and remote bilateral PE complicated by diverticular lower gastrointestinal bleeding, requiring placement of an infrarenal IVC filter. Physical examination was notable for severe bilateral asymmetric pitting lower extremity edema up to the thighs. Abnormal laboratory results included: Sodium 133 mmol/L, BUN 33 mg/dL, and Creatinine 1.84 mg/dL. Urinalysis showed hematuria without red cell casts, trace proteinuria, fractional urea excretion below 35%, and urine osmolality below 350 mosmol/kg. Renal ultrasound was unremarkable. Despite fluid resuscitation, the patient remained oliguric with progressive renal failure with creatinine peaking at 8.20 mg/dL. A noncontrast abdominal CT revealed extensive edematous changes in the proximal upper thighs bilaterally extending to the lower pelvis, suspicious for DVT involving the IVC and iliac veins. Doppler ultrasound confirmed venous thrombosis both proximal and distal to the IVC filter. After initiating intravenous heparin, intraoperative venogram showed thrombosis proximal to the IVC filter to the level of right ovarian vein and at the origin of right renal vein and left renal vein. Tissue plasminogen activator was injected into both renal veins with subsequent Angiojet mechanical thrombectomy successfully restoring patency. The patient was discharged on coumadin therapy, and required hemodialysis therapy for one month after which her renal function returned to baseline.

Current literature estimates filter thrombosis varies from 2.7% to 28%, typically lowest with the Greenfield filter. To date, there is no data estimating the incidence of renal failure secondary to bilateral renal vein thrombosis in permanent IVC filters, though limited case reports involving both suprarenal and infrarenal filters noted a similar constellation of lower extremity edema, back pain, proteinuria and hematuria. Though ilio caval thrombosis extending to renal veins is a rare complication of IVC filters, our case demonstrates the need for providers to maintain a high index of suspicion when diagnosing this unique cause of renal failure for which outcomes can range from reversible to fatal. As shown, CT imaging and doppler ultrasound may both adequately achieve diagnosis, with combination anticoagulation and thrombectomy proving effective therapy.

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Title: Valacyclovir induced neurotoxicity in a Multiple Myeloma patient with Herpes Zoster

Introduction:

Valacyclovir is a prodrug of acyclovir. Valacyclovir is an effective agent for treatment of herpes viral infection. Headache is the most common neurological adverse effect observed with use of Valacyclovir. Signs and symptoms of Valacyclovir induced neurotoxicity are variable including dizziness, irritability, ataxia, tremor, myoclonus and seizures. Symptoms usually begin within three days of initiation of therapy and resolve within five days of discontinuation. Risk of Valacyclovir induced neurotoxicity is increased in older patients and patients with renal failure. Results of brain imaging and CSF analysis are negative. Most common abnormality observed on EEG is generalized slowing of brain activity.

Case: A 66 year old woman with end stage renal disease on hemodialysis, Immunoglobulin G Lambda Multiple myeloma currently on chemotherapy was noted to have a rash on the left arm and right side of her back consistent with herpes zoster. She was treated with Valacyclovir 1 gram three times a day (dose was not adjusted for renal failure). After taking three doses she became confused, started experiencing visual hallucinations, and was admitted for altered mental status. Varicella Zoster meningoencephalitis and aseptic meningitis secondary to CNS carcinomatosis were considered as top differentials along with Valacyclovir induced neurotoxicity. Patient declined lumbar puncture. Valacyclovir was held and she was continued on usual hemodialysis sessions. Her symptoms significantly improved after a single session of hemodialysis with cessation of hallucinations and agitation. She improved further after second session of hemodialysis, became alert and oriented to time, place, and person. Her antiviral medication was changed to oral acyclovir considering her creatinine clearance which she tolerated without any neurological adverse effects.

Discussion: Valacyclovir has been used more frequently compared to acyclovir in recent years for treatment of herpes infection because of increased oral efficacy. Cases of Valacyclovir induced neurotoxicity have been reported in the past most commonly when Valacyclovir was prescribed for dermatological herpes zoster. Pharmacokinetics of Valacyclovir are altered in patients with renal failure. Levels of the drug in plasma do not correlate with the neurological symptoms experienced. The risk of neurotoxicity is significantly enhanced if Valacyclovir dose adjustment is not done for renal insufficiency. Chronic or acute renal failure preceded Valacyclovir neurotoxicity in few other reported cases. Valacyclovir induced neurotoxicity can be effectively managed by hemodialysis. Early recognition is the most effective tool for management of Valacyclovir induced neurotoxicity.

Learning points:

-Neuropsychiatric symptoms may accompany use of Valacyclovir.

Clinicians should have a high suspicion for Valacyclovir induced neurotoxicity in patients with renal insufficiency.

**New York Chapter ACP
Annual Scientific Meeting**

**Resident/Fellow
Research**

Resident/Fellow Research

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Institution: Interfaith Medical Center

Title: A Zombie phenomenon and variable presentations of K2 Intoxication: A Community Hospital Experience

Objective: To investigate the variable presentations of K2 intoxication and its associations.

Introduction: K2 is synthetic marijuana also known as Spice/Mojo/Zombie drug which is available as incense or herbal remedies. Although K2 was initially developed for medicinal purpose, it is becoming increasingly popular as an abused substance in United States mainly among young people. Center for Disease control and Prevention reported 3 deaths from 2010 to 2015 because of K2 intoxication. On July 12, 2016, a mass intoxication of 33 persons occurred in Brooklyn, New York which was described as a “Zombie movie scene” by the press because of the appearance of the intoxicated persons. K2 being relatively new drug of abuse, not much is known about its presentation. Wide variation of clinical presentation and concomitant use of other drugs makes the diagnosis even more difficult. There are very few studies investigating on the adverse effect, variation of presentation and outcome. We sought to analyze such determinants.

Method: We retrospectively reviewed cases admitted to our hospital throughout a year period (2013-2014) with self-reported or EMS reported K2 intoxication. Data was analyzed using the SPSS software.

Results: A total of 51 cases were reviewed who were brought to ER with the chief complaints of K2 intoxication. 88% of them were male and 98% of these patients were cigarette smoker and 41 % were alcohol abuser. Most common presentations were related to CNS that includes change in mental status (76 %), confusion (57%), psychosis (31%), agitation (29 %) and seizure (14%). 22% of these cases necessitated ICU care. 33% of patients had urine toxicology positive for other drugs. Men had higher propensity for agitation or confusion whereas psychosis was more common in female patients.

Discussion: Synthetic cannabinoids appear to be 4 to 5 times more potent and appear to have more pronounced and severe cardiovascular adverse effects than traditional marijuana. Because of its harmful effects on health and high potential for abuse, K2 is currently listed as class- 1 Controlled substance in United States. The active compound in K2 has been constantly changed to ward off the quality control and security oversight; so it may not be detected in a screening test. Thus, clinicians should keep a high index of suspicion for patients who present with signs and symptoms suggestive of drug use even though routine drug screening is negative. Furthermore, clinical manifestations that are commonly reported might be altered by adulteration with other toxic compounds and concomitant use of other drugs making diagnosis challenging. Although many of these symptoms are transient and resolved spontaneously, in more than one fifth of cases, symptoms were severe enough that needed ICU admission as seen in our study.

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Title: Impending death of autopsy: Analysis of resident's attitude towards autopsy at a tertiary care center.

Introduction: Autopsy was once considered the gold standard for medical diagnosis. Recent data from United States National Center for Health Statistics (NCHS) has shown that autopsy rate of all deaths decreased from 19.1% in 1972 to 8.3% in 2003. A number of reasons for this decline have been postulated, including improvements in diagnostic technology, fear of litigation and removal of defined minimum autopsy rate standards. Resident physician attitudes towards requesting an autopsy may also play a major role in the declining rate of autopsies in recent years. The purpose of this study was to elicit the attitudes of residents from various departments at a tertiary care center for decline in autopsy rates so that barriers for this valuable teaching tool can be addressed.

Methods: A voluntary 20-question survey regarding resident perceptions towards autopsy was distributed through email to 269 residents from Internal Medicine (IM), Surgery, Emergency Medicine, Family Medicine and Neurology.

Results: 155 of 269 residents responded to the survey with majority responses from IM. Among residents who responded, 84% believed that autopsies improved management of subsequent patients and revealed medical conditions that affect the health of surviving relatives whereas 1.4% felt that autopsies did not improve management. 71% residents agreed that autopsies were an effective use of health care resources with <2% believing that they were not. When asked to identify barriers for not obtaining an autopsy, lack of proper training on how to request an autopsy (66%) was the most common reason cited. Other common reasons include the unpleasantness of requesting an autopsy from families (54%), perceived religious/moral beliefs of families (56%), advanced age of patients (59%) and a perceived obvious cause of death (56%). Interestingly majority of residents (54%) believed that concern for malpractice litigation was not a huge barrier as compared to other studies. Although autopsy was considered a useful tool by residents, 92% reviewed <3 autopsies in their training so far. Furthermore, 80% of residents from the above specialties did not have any formal education on how to communicate with family members while requesting an autopsy and about 83% were not aware of the steps involved in an autopsy.

Conclusion: From our multi-departmental single institution survey, it is evident that residents report educational value in obtaining autopsies; however several barriers, including inadequate training in how to have this difficult conversation with grieving families and inadequate information about autopsy procedures remain. A possible solution to this could be implementation of autopsy orientation program along with monthly interdisciplinary case conferences that include autopsy reports to “revive” this dying educational tool.

Resident/Fellow Research

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Title: INCIDENCE, PREDICTORS AND OUTCOMES OF URINARY TRACT INFECTIONS AMONG PATIENTS WITH UROLOGIC CANCERS: A NATIONWIDE STUDY

Background: Urologic cancers are associated with structural and functional abnormalities predisposing patients to complicated urinary tract infections (UTIs). There is a general paucity of information addressing outcomes in this setting. The aim of the study was to assess the impact of UTI on the frequency of emergency department (ED) visits, hospitalizations, association with urologic procedures, mortality and other outcomes among men with urological cancers.

Methods: This was a retrospective cohort study of patients with prostate, bladder and renal or ureteral cancers who presented to the emergency departments (ED) in the USA in 2013 using the Nationwide Emergency Department Sample (NEDS) database. Inclusion criteria were a principal or secondary diagnosis of prostate, bladder, and renal or other urologic (renal pelvis, ureteral or urethral) cancers and male sex. Patients with a principal diagnosis of UTI were identified. The primary outcome was incidence of UTI. Secondary outcomes were: admission to ICU and length of hospital stay (LOS). The effect of exposure to urologic procedures was also assessed. Diagnoses were identified using the appropriate ICD9-CM codes. Proportions were compared using Fisher's exact test. Multivariate regression analysis was used to adjust for confounders.

Results: Among 246,297 ED visits of patients with urologic cancers, 10,532 (4.3%) had a diagnosis of UTI (prostate and bladder cancers were the 2 largest groups constituting 94% of patients), of whom 5,865 (55.69%) were admitted to the hospital and 106 (1%) died. The incidence of UTI was highest among patients with bladder cancer (7.4%) compared with patients with prostate cancer (4%, $p < 0.01$) and renal or other urologic cancers (2.1%, $p < 0.01$).

Following a urologic procedure, the incidence of UTI during hospitalization increased for all three study groups: bladder cancer (OR 2.28, $p < 0.001$), prostate cancer (OR 3.34, $p < 0.001$), and renal or other urologic cancers (OR 4.76, $p = 0.009$); however, patients with bladder cancer had a longer LOS when compared with patients with prostate cancer (1.07 adjusted mean additional days, 95% CI 0.08 - 2.07, $p = 0.03$). Among patients who did not undergo a urologic procedure, the incidence of UTI was two-fold higher for patients with bladder cancer compared with prostate and renal or other urologic cancers (adjusted OR=2.04, 95% CI 1.83 - 2.27, $p < 0.001$); while the likelihood of ICU admission after a UTI was higher among patients with bladder cancer compared with prostate cancer (adjusted OR=11.95, 95% CI 3.34 - 42.83, $p < 0.001$).

Conclusions: Among urologic cancers, bladder cancer is the most frequently associated with complicated urinary tract infections requiring ED visits. In the setting of a UTI, bladder cancer in men is associated with a higher chance of ICU admission and longest LOS when compared with prostate cancer. Further studies are needed to understand the anatomical, physiologic and pathogenic reasons behind these associations.

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Institution: New York Medical College Metropolitan Hospital Center

Title: Association of QTc Prolongation and Serum Alcohol Levels in Healthy Subjects in an Urban Hospital

Background:

Prolonged QTc interval has been associated with life-threatening cardiac arrhythmias and is an independent risk factor for sudden cardiac death. Moderate alcohol use may decrease risk of heart disease and all-cause mortality. However, the incidence of sudden cardiac death has also been demonstrated to be enormous among heavy alcohol consumers. There is little evidence on the association between elevated blood alcohol levels and QTc interval prolongation. In this study, we investigated the association of detectable serum alcohol levels with QTc prolongation.

Methods: This was a single-site retrospective study of all consecutive 560 patients admitted for management of alcohol dependence between October 2015 through September 2016. Subjects with electrolytes derangements, QT-prolonging medications, positive urine toxicology for recreational drugs, thyroid disease, cardiac arrhythmias, history of myocardial infarction or structural heart disease were excluded. Patients with unreadable electrocardiograms were not included. Our final study population consisted of 201 otherwise healthy patients who had normal electrocardiograms and blood alcohol levels drawn on presentation in the Emergency Department. We collected age, gender, basic metabolic panel, serum alcohol level, heart rate and corrected QT (QTc) generated on the standard 12-lead electrocardiogram machine. Serum alcohol level was detectable if ≥ 3 mg/dl and undetectable if < 3 mg/dl. QTc was prolonged if > 450 ms in males and > 470 ms in females. We categorized the final cohort by gender and compared patients in each gender group with detectable blood alcohol levels vs. undetectable alcohol levels using independent sample student t test at 95% confidence interval. The groups (detectable vs. undetectable) were also analyzed using logistic regression adjusting for sex differences.

Results: The final cohort consisted of 175 males (mean age 48, STD 12) and 26 females (mean age 44, STD 15). Serum alcohol level was detectable in 60% of male patients ($n = 105$, mean alcohol level 174 \pm 117; 110) and 58% of female patients ($n = 15$, mean alcohol level 195 \pm 177; 61). The male group with detectable serum alcohol had statistically significant and higher mean QTc of 449 ms (STD 30) compared to male group with undetectable serum alcohol who had mean QTc 439 ms (STD 34) $p = 0.042$. Among the female group, there was no statistically significant difference between mean QTc in patients with detectable serum alcohol level and undetectable alcohol level ($p = 0.24$). Detectable alcohol group showed a significant QTc prolongation when compared to the undetectable alcohol group ($p = 0.026$, OR = 1.01) in logistic regressions adjusted for sex variable.

Conclusion: Male patients with alcohol dependence and detectable serum alcohol levels admitted to hospital are more likely to have QTc prolongation. Female subjects with detectable alcohol levels did not demonstrate QTc prolongation. Concurrent use of QT-prolonging medications should be avoided in male population with alcohol dependence as it may predispose to cardiac arrhythmias.

Resident/Fellow Research

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Institution: Jacobi Medical Center/ Albert Einstein College of Medicine

Title: ASSOCIATION OF HOSPITAL TEACHING STATUS WITH IN-HOSPITAL OUTCOMES OF ST-ELEVATION MYOCARDIAL INFARCTION

Background: Admission to a teaching hospital has been considered to offer survival benefit for multiple common, high severity conditions. The influence of hospital teaching status on outcomes of ST-elevation myocardial infarction (STEMI) has not been well investigated.

Methods: We queried the 2010-2012 National Inpatient Sample (NIS) databases to identify all patients aged ≥ 18 years hospitalized with the principal diagnosis of STEMI. Multivariate logistic regression was used to compare treatment and in-hospital mortality of STEMI between patients admitted to teaching hospitals (TH; teaching hospitals defined in NIS as hospitals that have residency training programs, belong to council of teaching hospitals, or have a ratio of $<4:1$ beds to resident physicians) versus those admitted to non-teaching hospitals (non-TH). The influence of TH status on length of stay (LOS) and average hospital charges was also examined.

Results: Of 365,769 patients with STEMI, 46.7% were admitted to TH and 53.3% were admitted to non-TH. Compared to patients admitted to non-TH, those admitted to TH were more likely to undergo angiography (87.9% vs 84.4%; adjusted OR 1.18, 95% CI 1.15-1.21, $p<0.001$) and receive revascularization with thrombolysis (3.4% vs. 2.8%; adjusted OR 1.41, 95% CI 1.35-1.47, $p<0.001$), percutaneous coronary intervention (PCI) (78.3% vs. 73.9%; adjusted OR 1.14, 95% CI 1.12-1.16, $p<0.001$) or coronary artery bypass grafting (CABG) (3.5% vs. 3.1%; adjusted OR 1.13, 95% CI 1.09-1.18, $p<0.001$). After adjustment for demographics, other hospital characteristics (region, bed size, location), and baseline comorbid conditions, TH status was associated with 9% lower odds of in-hospital mortality (adjusted OR 0.91, 95% CI 0.89-0.94, $p<0.001$). However, this residual association was entirely explained by differences in revascularization rates between the 2 groups and after additional adjustment for revascularization, there was no difference in in-hospital mortality between STEMI patients admitted to teaching versus non-teaching hospitals (adjusted OR 0.99, 95% CI 0.96-1.02, $p: 0.99$).

Conclusions: STEMI patients admitted to TH have lower in-hospital mortality compared to those admitted to non-TH. This was completely accounted for by higher rates of revascularization with thrombolysis, PCI or CABG in TH.

**New York Chapter ACP
Annual Scientific Meeting**

**Resident/Fellow /Medical Student
Quality**

Resident/Medical Student Quality

<p>Author: Aishwarya Bhardwaj, MD Additional Authors: Huy Phan, Shane Varghese, Mohamed Abdel-Aal Ahmed, Smita Bakhai Institution: University at Buffalo, Internal Medicine</p> <p>Title: Guideline Directed Medical Therapy for Heart Failure Optimization in a Primary Care Setting</p> <p>Purpose: At the Internal Medicine Clinic (IMC) in Erie County Medical Center (ECMC), roughly 15% of the patient population has a diagnosis of heart failure (HF). Due to lack of a consistent methodology for classifying HF in clinic electronic medical record (EMR), many of these patients are not on guideline directed medical therapy (GDMT) based on ACC/AHA/HFSA 2013 guidelines. This, in turn, has a significant negative impact on patients' quality of life (QOL), increased mortality and number of avoidable hospitalizations, which pose a huge burden on limited hospital and national healthcare resources. The aims of this Quality Improvement (QI) project are to appropriately specify type of cardiac dysfunction based on ejection fraction (EF) and increase the use of GDMT by 10% from baseline within 12 months in patients aged 40-75 years with a diagnosis of HF at ECMC IMC.</p> <p>Methods: To design this QI project, we employed Institute of Medicine's STEEEP model (Safe, Timely, Effective, Efficient, Equitable, Patient-Centered). We used root cause-analysis to identify system-, provider- and patient-based barriers in providing optimal GDMT to our patients with HF. In order to implement various interventions and overcome barriers toward improvement of care, we have used the Plan Do Study Act (PDSA) model. Furthermore, we identified a family of measures, as adopted from the Institute for Healthcare Improvement, namely, outcome, process, and balance measures. Data analysis is performed using monthly run charts.</p> <p>Results: Initial chart review revealed that less than 5% of eligible clinic patients with a diagnosis of HF are explicitly classified based on their EF and are on appropriate GDMT based on type of cardiac dysfunction. In September 2016, 26% of patients with HF were classified based on the type of cardiac dysfunction. After two PDSA cycles and 6 months into initiation of the project, in February 2017, 78% of patients were appropriately classified.</p> <p>Conclusions: So far, two PDSA cycles have been implemented comprising 1) resident education reviewing ACC/AHA/HFSA 2013 guidelines 2) collaborating with the clinic Information Technology department to create a database for HF classification and to keep track of ECHO reports. Next PDSA cycle will focus on creation of pamphlets which will serve as reminders for providers and patients for optimization of HF therapy. In the last 6 months, there has been a clear trend toward rise in appropriate HF classification and respective treatment for our clinic patients. In the next 6 months, our overarching goal will be to further augment GDMT through use of these diagnostic categories.</p>	<p>Author: Tarundeep Grewal Additional Authors: Zaid Shakir, Sarah Sethi, and Olivia Onwodi Institution: Kingsbrook Jewish Medical Center</p> <p>Title: Make Social Media Great Again</p> <p>Introduction: Social media, which began merely as a networking tool to stay connected with family, friends and colleagues has recently become a large platform for educational purposes as well. The quick adaptation of social media within the medical field has also provided a platform for learning and teaching opportunities for the healthcare professionals. Medical residents and students gain access to the most current information, guidelines and events through the use of social media.</p> <p>Methods: A 33 question written survey was presented to 3rd and 4th year medical students, medical residents and registered nurses at 3 major hospitals within the NYC area. The survey was completed over a one-week period and the data was then analyzed.</p> <p>Results: Of the 193 participants surveyed in this study (113 MS3, 42 Medical Residents, 28 MS4, 7 Nurses), demographics ranged from ages 25-29 (55.2%), female (54.2%), Asian (44.8%), and US IMG (59.6%). Of the total participants, 97.4% stated they utilized social media on a daily basis. A large sum viewed the role of social media as a positive impact within the medical community (62.2%), and nearly four out of five of the participants were under the age of 30 (76%). A linear trend was observed that signified a correlation between age and the use of social media, proving that as age increases the use of social media decreases (RR 0.88). Four out of five of the participants who agreed that social media provides a positive influence on their daily life (80.3%) say they have used it as a learning tool, while almost nine out of ten (88%) use it as a means to stay up to date with the most current information, events, journals and guidelines. The top three social media platforms used by participants included YouTube (71.3%), Facebook (51.1%) and Instagram (29.8%). More than half of the participants (65.4%) have also used social media to benefit their patients, which included posts on valuable medical information such as health tips, articles, resources and links to studies.</p> <p>Conclusion: With the rapid rise in social media and its adaptation into the medical field, there are still opposing views on how it has impacted medicine. Majority of the participants in our study agree that the use of social media in medicine has provided a positive impact to their medical career. A negative correlation however was also observed among participants in whom medical professionals of older age are less likely to use social media in the field. Overall, with regards to the majority of the participants in our study, it is clear that the integration of social media within the medical community has become a fundamental platform for accessing current information as well as an essential learning tool.</p>
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Resident/Medical Student Quality

Author: Fatima Hosain, MBA

Additional Authors: Josephine Lee

Institution: Albany Medical College Internal Medicine

Title: LIKELIHOOD OF PHYSICIAN RENEWAL OF CHRONICALLY PRESCRIBED CONTROLLED SUBSTANCES BASED ON URINE DRUG TEST RESULTS

LIKELIHOOD OF PHYSICIAN RENEWAL OF CHRONICALLY PRESCRIBED CONTROLLED SUBSTANCES BASED ON URINE DRUG TEST RESULTS

Fatima Hosain MBA (ACP Member), Josephine Lee MD Albany Medical Center, Albany, NY

Background: In response to increasing nationwide misuse of controlled medications, physicians have adopted protocols utilizing urine drug testing (UDT) to manage their patients' drug use; however, the effect of UDT results on physician prescribing habits has not been well established.

Purpose: This study examined changes in physician prescribing behavior within a midsize internal medicine practice based on the UDT results for patients being chronically prescribed controlled medications.

Methods: We conducted a retrospective electronic health record chart review comparing prescription renewals for patients with consistent versus "inconsistent" UDTs, defined as a) specimens with prescribed medications not detected, b) specimens with non-prescribed medications detected (opioids, benzodiazepines), or c) specimens with illicit substances (cocaine, heroin, marijuana) detected.

Results: Inconsistent UDTs comprised 44% of the total UDTs (n=493). Reasons for inconsistency included marijuana (15%), opioids/benzodiazepines (13%), prescribed medication not detected (11%), and heroin/cocaine (5%).

There was a significant difference in the prescription renewal rates of patients with consistent (96%) versus inconsistent (67%) UDTs. Patients found with no prescribed drug in their system had a 56% renewal rate for that drug. UDT results that were positive for marijuana, non-prescribed opioids, or non-prescribed benzodiazepines had much higher renewal rates (79%, 73%, and 83% respectively) than those positive for illicit substances such as heroin and/or cocaine (7%).

Conclusions: Although patients with inconsistent UDT results had their prescriptions for controlled medications renewed less frequently than those with consistent UDT results, the numerically high renewal rates associated with inconsistent UDTs suggest that physicians may be too lenient when renewing patient prescriptions. This is especially apparent in the high renewal rates for UDTs positive for marijuana, opioids, and benzodiazepines, as well as those with the prescribed medication not detected. Overall, the results of this project indicate that physicians can play a central role in curbing the growing problem of medication misuse among patients through tools such as urine drug testing.

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Institution: Interfaith Medical Center

Title: CLINICAL DOCUMENTATION IMPROVEMENT A PROBLEM AND SOLUTIONS- INTERNAL MEDICINE RESIDENTS PERSPECTIVE SURVEY

Objective: To determine the internal medicine residents' perceptions of the problems related to clinical documentations and possible solutions to the problems.

Introduction: Clinical documentation plays a critical role in patient care as it helps in coordination between interdisciplinary team members involved in care, to avoid mistakes, duplication of investigations and create an appropriate plan of care. We sought to understand the perspective of medical residents regarding time spent on clinical documentation, challenges to improvement and possible solutions.

Method: A cross-sectional online survey questionnaire was provided to all internal medicine residents at our institution. Residents voluntarily completed the survey. Questions were structured to elicit their perceptions of current practices, challenges and solutions.

Result: A total 87 residents (PGY1-33, PGY2-23 and PGY3-31) responded to the survey, 67% of them spent 15 to 30 minutes on each note and less than 15 minutes seeing each patient at bedside (53%). They spent more than 2 hours on daily progress notes (63%) whereas less than a total of 2 hours at bedside of all their assigned patients (62%). There were no significant differences in these findings on the residents' post-graduate year level. Half of the respondents (49%) always copied and pasted their notes but nearly all participants (95%) edited their notes properly. Some of the perceived challenges to good documentation included the overly detailed nature of progress notes required for regulatory, billing and legal purpose (67%), burn out or stress (72%), and extra time spent on other clerical documentations (84%), ICD coding (55%).

Most residents responded that shortening the length of clinical documentation (77%) and upgrading EMR system (80%) were possible solutions. Other solutions to improve quality of clinical documentation were using a checklist to prevent propagation of outdated or inaccurate information in the patient chart (79%), upgrading EMR system (80%) and more editing and feedback from faculties and senior residents (75%).

Discussion: Survey demonstrated that residents spent large portion of their time in documenting daily progress notes and in contrast, spent less time in direct patient care. Most residents copy and paste documents from previous notes which could pose a risk to document integrity. But almost all respondents spent time to edit notes. They perceive that disabling copy paste function will not help clinical documentation. They believe in a relatively new idea that using checklist will help in better documentation. Although clinical documentation improvement is crucial, no published articles to date have systematically reviewed strategies to improve clinical documentation. Further studies on interventions are needed.

Resident/Medical Student Quality

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Institution: Wectchester Medical Center

Title: KNOWLEDGE, ATTITUDE AND PRACTICE SURVEY REGARDING PERTUSSIS VACCINATION AMONG TRANSPLANT HEALTH CARE WORKERS- SINGLE CENTER STUDY

KNOWLEDGE, ATTITUDE AND PRACTICE SURVEY REGARDING PERTUSSIS VACCINATION AMONG TRANSPLANT HEALTH CARE WORKERS- SINGLE CENTER STUDY

George Jolly , M.D. (ACP member), Lakshmi Asritha Gollapudi, M.D., Abhishek Goyal, M.D, MPH, Kaushik Kar, M.D., Abhay Dhand, M.D. Department of Medicine, Transplant Infectious Diseases, Westchester Medical Center,NY

Introduction:

Pertussis is a vaccine-preventable respiratory infection caused by Bordetella pertussis. Since the 1980s, there has been a steady increase in the number of reported cases of pertussis in the United States. Advisory Committee on Immunization Practices (ACIP) recommends that all Health Care Workers (HCWs) who have not received or are unsure of the status of their pertussis vaccination , should receive a dose of Tdap as soon as feasible. Pregnant women including pregnant HCWs should be re-vaccinated during each pregnancy. There is scant data regarding pertussis vaccination status of HCWs and compliance with current ACIP recommendations.

Methods: A survey questionnaire was created and validated to assess the knowledge, attitude and practices regarding Pertussis vaccination. The survey was conducted among all the health care workers (HCWs) in a Transplant Center at a tertiary care suburban New York hospital. This paper based survey questionnaire was distributed to all the HCWs in September 2016.

Results: A total of 139 transplant healthcare workers (HCWs) were surveyed with a response rate of 100%. Only 53/139 (39%) of the respondents had received Pertussis vaccination in last 3 years. 55/139 (40%) of respondents were aware of the current recommendations regarding Pertussis vaccination. Factors associated with increased likelihood of receipt of vaccination were HCWs who have direct patient contact (p: 0.024, OR: 5.0), HCWs who have been pregnant or had a pregnant spouse (p: 0.012, OR: 6.3) and in HCWs who were aware of the current vaccination guidelines (p: 0.0061, OR: 3.5). Of the HCWs who received vaccination, 62% received it at their primary care clinic, 26% at employee health clinic and 12% during their hospitalizations. HCWs who did not receive the pertussis vaccination cited concerns about the safety of the vaccine (55%), their physicians not recommending it (14 %) and lack of awareness of current guidelines (14 %).

Conclusion:

Rate of vaccination and knowledge regarding the current guidelines of pertussis vaccination remains inadequate among HCWs. Based on these results, the rates of vaccination among HCWs could be improved by sustained education regarding the knowledge of current guidelines for pertussis vaccination and safety and tolerability of vaccination. HCWs should also be educated about the potential risks of spread of pertussis via healthcare workers especially in at risk patients including neonates and immune-compromised hosts. A visit by HCWs to a primary care physician, pediatrician, employee health services and obstetrician can all be utilized to improve the rate of vaccination.

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Title: ASSESSMENT OF PATIENT HANDOFFS BEFORE AND AFTER AN INSTITUTION-WIDE IMPLEMENTATION OF THE I-PASS BUNDLE: A SINGLE CENTER'S EXPERIENCE

BACKGROUND The I-PASS study, a multi-center pediatric communication and patient safety trial, found that implementation of a bundled intervention to improve communication during patient handoffs correlated with a 30% reduction in preventable adverse events. I-PASS stands for Illness Severity, Patient Summary, Situational Awareness/Contingency Planning, and Synthesis by Receiver. Winthrop-University Hospital (WUH) underwent a hospital-wide implementation of the I-PASS bundle to standardize handoffs across primary mid-level provider teams.

OBJECTIVE Within 2 years, the improvement project aims for 100% adoption of the I-PASS handoff system by all 18 primary mid-level provider services that use the hospital electronic health record system. This report focuses on quantifying adoption of I-PASS template 4-6 months post-implementation and measuring the hospital-wide impact of the initiative on measures of handoff quality.

METHODS We conducted a prospective intervention study of the handoff improvement program measuring documentation of illness severity and contingency planning one month prior and 4-6 months after implementation. More than 400 WUH mid-level providers underwent standardized training that included TeamSTEPPS education, a 1-hour verbal handoff simulation, and a 20-minute demonstration of the electronic I-PASS template. An impartial reviewer collected data from at least five distinct sign-out sessions before implementation and five after for the 18 primary mid-level provider services. I-PASS adoption was measured by the proportion of individual patient-level handoffs within each service that have electronic I-PASS templates.

RESULTS Prior to the intervention, only Pediatrics had adopted I-PASS. Afterwards, 16 of 18 services had some level of I-PASS adoption. CCU (57/57 patients) and MICU (64/64 patients) had 100% adoption. The remaining services are Medicine-Resident (98.93%, 277/280), Medicine-Telemetry (97.24%, 211/217), Pediatrics (96.81%, 91/94), TCV-Floor (95.56%, 43/45), TCV-SICU (91.89%, 34/37), Cath/EP (87.5%, 28/32), MFM/GYN/GYN-ONC (75.24%, 79/105), Neurosciences (65.38%, 51/78), Orthopedics (60.00%, 69/115), SICU (41.3%, 19/46), PICU(23.53%, 4/17), Trauma (16.67%, 5/30), General Surgery (13.04%, 33/253), and Wound Care (11.43%, 8/70). Two services did not adopt the I-PASS template: Urology (0/21) and OB (0/148). In our hospital-wide data set, 7.24% (140/1933) of patients compared with 42.77% (796/1861) had documentation of illness severity pre- and post-implementation. Contingency planning was documented in 8.17% (158/1933) of patients pre-implementation compared with 33.05% (615/1861) 4-6 months post-implementation.

CONCLUSION/DISCUSSION WUH's efforts to implement a standardized handoff system showed disparate rates of adoption among the services. The hospital-wide handoff improvement program has already effected significant change in important measures of handoff quality.

Resident/Medical Student Quality

<p>Author: Brian McNichols Additional Authors: Matthew Gorgone, William Novak, Alec O'Connor, Donald Bordley Institution: University of Rochester</p> <p>Title: THE MEDICAL PROCEDURE TEAM: IMPROVING TIME TO PARACENTESIS</p> <p>THE MEDICAL PROCEDURE TEAM: IMPROVING TIME TO PARACENTESIS Brian McNichols, MD, Matthew Gorgone, DO, William Novak, MD, Alec O'Connor, MD, MPH, Donald Bordley, MD. University of Rochester, Rochester, NY.</p> <p>Introduction: Timely access to diagnostic paracentesis has been associated with a decrease in mortality. The current use of Interventional Radiology (IR) to perform many inpatient procedures often results in patients waiting until the following day for non-emergent cases. Many of these procedures could be performed safely at the bedside by Internal Medicine residents if they had sufficient experience and schedule availability. A novel system to facilitate common procedures at the bedside could decrease the length of time patients wait for procedures and streamline access to Interventional Radiology for more complicated cases. We examined the efficiency of this model in performance of paracentesis on our inpatient wards.</p> <p>Methods: Between November 2015 and May 2016, active outreach to hospitalists, subspecialists, and advance practice providers highlighted a clear need for more timely access to common inpatient procedures. In May 2016, a team of residents developed a new resident procedure coordinator role that would triage, organize, and supervise resident inpatient bedside procedures. Procedures included paracentesis, central line placement, thoracentesis, ultrasound guided peripheral IV, and lumbar puncture. Residents performed this coordinator role primarily while on elective or ambulatory rotations as an additional responsibility. The resident procedure team availability was advertised widely to URMC inpatient providers through weekly emails and self-promotion among residents.</p> <p>Results: During the first seven months of the project (June 2016 – December 2016), the resident procedure team was able to perform 123 bedside paracentesis procedures. Overall, the team performed 86% within 12 hours of the request compared to 26% by the interventional radiology department ($p < 0.00001$) over the same 7 month period. The total number of procedures was similar between both groups including 123 by the resident procedure team and 121 by the IR department.</p> <p>Conclusion: Use of a designated resident procedure team and coordinator has proven an effective means to increase the timeliness of diagnostic paracentesis in an inpatient setting. We hope that this model will facilitate other inpatient procedures in the future.</p>	<p>Author: Neha Naik, MD Additional Authors: Michael E. Tofano, MD (ACP member); David Galinkin, MD (ACP member); Phillip Nizza, DO (ACP member); Richard Poveromo, LMSW; Nirupa Ramji-Singh, RPA-C; Maricelle O. Monteagudu-Chu, PharmD; Lois McCarthy, RN; Liza M. Dominioni, C-TAGME. Institution: SUNY at Stony Brook/John T. Mather Memorial Hospital</p> <p>Title: AN INTERDISCIPLINARY APPROACH TO IMPROVING CARE AND REDUCING LENGTH OF STAY IN PATIENTS WITH CELLULITIS</p> <p>Purpose of Study: A community teaching hospital of 248 beds was in variance (outside the expected norm) to New York (state) and Long Island Health Network (regional) benchmarks on average length of stay and readmission rate for patients admitted with cellulitis over a six quarter period. An interdisciplinary approach was utilized to develop and implement an evidenced based customized program to address the variance.</p> <p>Methods: An interdisciplinary team of approximately 10 voluntary members consisting of Medicine, Nursing, Social Work, Pharmacy, Infectious Diseases and Information Technology (IT) was initiated by Hospital Medicine to address the variance. The team, in collaboration, developed and implemented a customized program for 260 patients over a 12 month period (October 2015 to September 2016). The aim was to decrease the average length of stay and readmission of patients diagnosed with cellulitis. The program consisted of a monitoring group which received a daily report from the hospital's IT department on cellulitis cases based on the Diagnosis-Related Group (DRG) of 602-603. Some members were assigned to attend daily interdisciplinary rounds on resident-teaching services to highlight the variance patients and develop an action plan for disposition with the resident and supervising physician. Formal resident education on early recognition, diagnosis and treatment of cellulitis was given to all internal medicine residents. For each patient, infectious diseases was consulted for appropriate utilization of antibiotics. The hospital's Antibiotic Stewardship team monitored the need for de-escalation of antibiotics. In addition, patients that required a longer intravenous antibiotic duration or wound care were identified and transitioned earlier to skilled care in the community. The team met every month to monitor the progress of this program.</p> <p>Results: The average length of stay for patients with cellulitis decreased from 6.31 days to 4.85 days, which was below both state and regional benchmarks. In addition, the readmission rate for cellulitis decreased by 23.2%. This had resulted in a substantial cost savings to the institution.</p> <p>Conclusions: Interdisciplinary collaboration is essential for developing and implementing customized programs to address identified variances in length of stay and readmission rate related to cellulitis. The interdisciplinary approach utilized here is replicable and applicable across multiple medical diagnoses and serves as an exemplar for improving patient outcomes and reducing costs to the health care institution.</p>
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<p>Author: Sonal Paul, MD Additional Authors: Christina Bortz MD, Sonal Paul MD, Sofya Kostanyan MD FACP, Tzipora Schwartz RN MSN PCCN Institution: NY Presbyterian Brooklyn Methodist Hospital</p> <p>Title: Safety Rounders as an Effective Mechanism to Prevent Inpatient Falls.</p> <p>INTRODUCTION Inpatient falls lead to fractures, serious injuries, and increased length of stay. Among falls resulting in serious injury, nearly 63% result in patient death. Therefore, it is no surprise that hospitals may suffer from lawsuits and withholding of reimbursement for complications related to falls, which average \$14,000 per patient. Safety rounders (SRNAs) were piloted on six units at New York Methodist Hospital in May 2016 to provide purposeful hourly rounding in an effort to decrease inpatient falls. This study was performed to determine whether the introduction of SRNAs decreased the overall incidence of falls.</p> <p>METHOD The objective of the SRNA is to intervene on four factors (â€œ4 Pâ€™sâ€) that commonly lead to falls - Assessing patientâ€™s pain, need for position change, ensuring possessions are within reach, and assisting with transport to the toilet (potty). SRNAs ensure call bells are nearby, bed alarms are functioning, and floors free of clutter. A retrospective cohort study was performed to compare the incidence of falls on six floors. Each floor was composed of patients from various age groups, comorbidities, and medication regimens. Incidence rates of falls were calculated over an 8 month period and divided into groups before and after the initiation of SRNAs (January through September). May was excluded from the study as SRNAs started at different times throughout this month. The likelihood of falls in the two groups were used to calculate relative risk reduction.</p> <p>RESULTS A total of 6,959 patients were admitted to SRNA floors during the study. 3,579 patients admitted from January through April were not exposed to safety rounding (control group). 3,478 patients admitted from June through September were exposed (experimental group). The rate of falls decreased from 2.7% (98/3579) to 1.8% (63/3478) after the implementation of SRNAs. The relative risk reduction was 33% (p=0.01).</p> <p>CONCLUSION The introduction of SRNAs significantly reduced the risk of inpatient falls. Results reveal the importance and effectiveness of designating specific staff members to minimize high-risk factors that lead to falls. The success of SRNAs comes from aligning known risk factors with patient care interventions. Hospitals would benefit from a safety and financial perspective by appointing safety rounders.</p> <p>REFERENCES Inouye, SK, CJ Brown, and ME Tinetti. "Medicare Nonpayment, Hospital Falls, and Unintended Consequences." New England Journal of Medicine 360.23 (2009): 2390-393. Web. Bagian, James P., and Frank Federico. "A Complimentary Publication of The Joint Commission." Sentinel Event Alert 55 (2015): n. pag. Joint Commission. The Joint Commission, 28 Sept. 2015. Web. 29 Nov. 2016. <http://www.jointcommission.org/assets/1/18/SEA_55.pdf></p>	<p>Author: Abhinav Rohatgi, MD, ACP member Additional Authors: Peter Hong MD, Vlad Radulescu MD, Jyoti Sharma MD, Jennifer Chee MD, Brandon Yim MD, Mark Sullivan MD, Zachary Fleischer MD, Elixabeth Sullivan MD, Rachel Wong MD Institution: Stony Brook University</p> <p>Title: OH SNAPPS: AN UNCONTROLLED BEFORE AND AFTER STUDY TO IMPROVE VALUE-ADDED PATIENT VISIT TIME IN AN ACADEMIC RESIDENCY PROGRAM PRIMARY CARE CLINIC.</p> <p>Patients are often dissatisfied with the amount of time they spend waiting during a primary care clinic visit. Delayed care is harmful to patients with acute medical issues that need to be addressed. In addition, fewer patients can be scheduled during the work day as a result of prolonged non value added time. At the Stony Brook University Resident Clinic, there is an additional step that contributes to patient wait time, because as part of a teaching facility, the residents must be precepted by attendings, in order to create a safe assessment and plan. Objective: To reduce non-value-added patient wait times by using a healthcare maintenance template and adapting the SNAPPS presentation model during resident presentations, and educating attendings on how to be a "one minute preceptor." SNAPPS is a learner-centered model for case presentations to the preceptor consisting of six steps: Summarize the history and findings; Narrow the differential; Analyze the differential by comparing and contrasting the possibilities; Ask the preceptor questions; Plan management; and select an issue for self-directed learning. "One-minute Preceptor" is a workshop for preceptors which helps them practice the following skills for precepting: Get a commitment. Probe for supporting evidence. Teach general rules. Reinforce what was right. Correct mistakes.</p> <p>Project Design/Methods: This is an uncontrolled before and after study. In the pre-intervention phase, we used Patient Flow Analysis to measure the time spent in each component of a patient visit. We identified the resident-preceptor presentation as a key rate-limiting step in the primary care clinic workflow. In order to reduce time spent in the resident-preceptor interaction, residents used SNAPPS. To reduce time spent discussing routine screening, we created a health-care maintenance template that could easily be filled and updated on future visits. Simultaneously, attendings attended a workshop to learn "one-minute preceptor" skills to maximize teaching. These interventions were implemented over a two-month period to allow time for healthcare workers to practice these methods, after which post-intervention data was collected.</p> <p>Results/Conclusion: We measured the time duration of the resident-preceptor presentation before and after our interventions. Prior to the interventions, this interaction took an average of 14.85min. After the intervention, it improved to an average of 10.75min. The goal of this QI project was to enhance the resident training process by increasing clinic efficiency by improving value-added patient visit time while maintaining quality patient care. Based on the data collected, there was an improvement in the time spent during the resident-preceptor interaction. The SNAPPS model as well as the one-minute preceptor are effective tools in defining inefficiencies and improving patient flow in our residency outpatient clinic. Future research can be aimed towards determining if there was an overall increase in patient satisfaction, without compromising patient care.</p>
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Title: PATIENT CENTERED MEDICAL HOME TEAM (PCMH) TEAM COLLABORATION FACILITATES A PROGRAM TO DECREASE MISUSE OF CONTROLLED SUBSTANCES IN AN ACADEMIC PRIMARY CARE PRACTICE

Purpose: The PCMH team decided to measure the effectiveness of an enhanced program to decrease chronically prescribed controlled substance misuse in our midsize academic primary care practice.

Methods: The PCMH team, which included attending physicians, residents, nursing staff and a social worker, designed and initiated a quality improvement project. We used an opioid risk tool, a uniform controlled substance agreement (CSA) and random urine drug tests (UDTs) at least once per year with increased patient care follow up visits. Medical residents in the PCMH team researched and agreed upon a standard CSA for practice-wide use. The team changed laboratories performing UDTs by immunoassay to another laboratory using liquid chromatography mass spectrometry testing (LC-MS/MS). This allowed for more sensitive and specific measurements and detection of key metabolites. The overall protocol design required all patients receiving chronically prescribed controlled substances to review and sign the CSA and to undergo random UDTs. We used the Electronic Health Record (Touchworks) to identify patients actively prescribed opioids and benzodiazepines in the 3 months prior to and the 3 months immediately following implementation of the new protocol.

Results: During the 3 month period prior to the initiation of the protocol, we identified 970 patients actively prescribed chronic opioids and benzodiazepines. 309 CSAs (32%) had been signed and 61 UDTs (6%) had been performed. During the 3 month period following the initiation of the protocol, we identified 551 patients actively prescribed chronic opioids and benzodiazepines. 432 (78%) new agreements had been signed and 466 UDTs (85%) had been performed by LC-MS/MS.

Conclusions: PCMH facilitated design and initiation of a new controlled substance protocol. Medical residents were able to assist in the protocol design and participated with attending physicians, nursing and social work staff. The implementation of this PCMH initiated protocol resulted in a large improvement in the percentage of CSAs and random UDTs in an academic primary care practice. This program will aid in facilitating the appropriate use of opioids and benzodiazepines, decrease diversion of prescription drugs into the illegal market, and help reduce illicit drug use. The decrease in the total number of patients receiving chronically controlled substance prescriptions after the initiation of the new protocol can be further investigated.