NON-ALCOHOLIC BEER: A UNIQUE CASE OF ACUTE ALCOHOLIC HEPATITIS
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LEARNING OBJECTIVE 1:
Recognize that patients who are to abstain from alcohol should also abstain from non-alcoholic beer.

CASE:
Acute alcoholic hepatitis (AAH) can develop in patients with chronic alcohol abuse or recent heavy intake. Patients who go untreated with severe acute alcoholic hepatitis (defined as Maddrey discriminant function (DF) >32) have mortality rates as high as 25-35% within one month. Patients substitute non-alcoholic beer (NAB) for alcohol-containing beer, however, NAB is a misnomer as it also contains alcohol. Most brands contain 0.5-1% alcohol by volume (ABV) compared to regular beer that contains approximately 4% ABV. We describe a patient with severe AAH after drinking NAB. 69-year-old woman presents with worsening abdominal pain and fullness, confusion and jaundice for the past week. She has a history of alcohol abuse but stopped drinking alcohol three years ago because she developed AAH. Since then she has been consuming 7-8 NABs per day and denies any other alcohol consumption (corroborated by her supportive family). Physical exam revealed icteric sclera, jaundiced skin and 3+ pitting edema. Labs revealed a creatinine of 4.1mg/dL, ALT of 33u/L, AST of 107u/L, total bilirubin of 23.6mg/dL, albumin of 2.6g/dL, platelet count of 208 and INR of 1.8. CMV IgM, HSV IgM, hepatitis A, B, C serologies and EBV PCR were all negative. ASMA, ANA and AMA were also negative. Abdominal MRI without contrast did not show signs of liver cirrhosis, splenomegaly or varices, however, it did show ascites and a fatty liver. Within four days, her liver function worsened and she had a DF of 39. Given her anuric acute renal failure, she was started on pentoxifylline, prednisone and treated with octreotide, midodrine and albumin for hepatorenal syndrome. Ultimately she required a week of hemodialysis. Subsequently, her total bilirubin started trending down and eventually her kidney function improved as well. Two weeks later, the patient was back to her baseline at which point her creatinine was 0.8mg/dL, INR was 1.3 and total bilirubin was 6.4 mg/dL. Given her MELD score of 39, she was evaluated for a liver transplant. She underwent extensive psychosocial evaluation of her alcohol consumption by a transplant psychiatrist, social worker, and transplant hepatologist. These specialists all agreed that she was honest about her consumption of only NAB. Ultimately no transplant was required as her liver function continued to improve.

DISCUSSION:
The patient was drinking eight NABs everyday for three years. With 0.5-1% ABV per NAB, her total alcohol consumption per day exceeded the amount consumed from one regular beer. The 2010 dietary guidelines for alcohol consumption recommend drinking no more than 1 drink per day for women. If not treated appropriately, AAH carries a high morbidity and mortality. AAH in association with low alcohol content beer has previously not been described in the literature. Patients with cirrhosis, on medications that should be avoided with alcohol, or with chronic liver disease are advised not to drink alcohol. These patients may gravitate towards NAB without realizing these drinks still contain alcohol. It is imperative to educate physicians and patients about the existence of alcohol in NAB. The amount of NAB that patients can consume remains unclear, and it is our recommendations that they avoid all NAB.
CHLORPROMAZINE-INDUCED HEPATITIS
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LEARNING OBJECTIVE 1:
Diagnose drug-induced hepatitis with significant clinical signs and symptoms, imaging, and procedures (ie biopsy)

LEARNING OBJECTIVE 2:
Recognize adverse effects of medications commonly prescribed

CASE:
A 34yo woman with a history of bipolar disorder and attention-deficit hyperactivity disorder presented with a one-month history of nausea and vomiting of non-bloody non-bilious emesis, and anorexia. Three days prior to presentation, the patient developed severe right upper quadrant abdominal pain. On presentation to the emergency department, the patient was afebrile and other vital signs were stable, but she was noted to be severely jaundiced with scleral icterus and abdominal exam revealed right upper quadrant that was tender to palpation without rebound or involuntary guarding. HIDA scan indicated possible common bile duct dilation and abdominal ultrasound revealed a contracted gallbladder with possible non-shadowing stones. Computed tomography of the abdomen was unrevealing for acute processes and ERCP revealed no obstructive etiologies. Serum anti-mitochondrial antibody, anti-smooth muscle antibody was negative, and ceruloplasmin were all either negative or within normal limits, ruling out primary biliary cirrhosis, autoimmune hepatitis and Wilson's disease, respectively. Anti-nuclear antibody was positive but with only a 1:400 titer. The patient subsequently underwent liver biopsy, which revealed significant cholestasis with marked eosinophilia, inflammation and bile duct edema. These findings were felt to be most consistent with drug-induced hepatitis. The team conducted a detailed review of the medications the patient had taken in the months prior to presentation, revealing metoclopramide 10mg, clonazepam 0.1mg three times daily, lithium 300mg daily, methylphenidate 36mg daily, chlorpromazine 25mg three times daily (of which the patient was advised to take two at a time), ondansetron 4mg every eight hours as needed. Her lithium dose had been increased a week prior to admission to the above dose with the intention to taper clonazepam. The patient also developed a diffuse pruritic urticarial type rash with eosinophilia several days after presentation.

DISCUSSION:
Drug-induced hepatitis occurs in 1 out of every 1000 to 100,000 patients and is more common in women (1,2). The clinical manifestations of most drug-induced hepatitis resemble viral hepatitis with malaise, jaundice and transaminitis. However, different pharmacological entities have specific patterns of injury (i.e. hepatocellular, cholestasis, autoimmune, fibrosis, etc). In cholestatic injury, there are four histological types: pure (canalicular, bland or noninflammatory), cholestatic, ductopenic, and sclerosing cholangitis (7). Symptoms typically develop within 1-6 weeks of initial ingestion of the medication and may continue evolving even after the agent is withdrawn (1,8). Some drugs have a strong allergic component, causing fever, rash, lymphadenopathy, and hepatic injury. Although chlorpromazine is typically associated with cholestasis, it is possible that a mild form of this "reactive metabolite syndrome" was the source of this patient's pruritic rash as this developed several days after the patient began having other signs of hepatic injury (3,4). Chlorpromazine-induced hepatitis occurs in 0.2-2% of patients (8), with 80-90% of cases developing within the first four weeks (5). The mechanism by which chlorpromazine decreases canalicular function and bile flow has been hypothesized to be due to poor genetic sulfoxidation of free radicals and hydrocarbons making affected patients more susceptible to cholestasis (9). Light microscopy shows cholestasis and a predominance of mononuclear and eosinophilic cells, although hepatocellular injury and granuloma formation is possible as well (6). In the case of our patient, liver functions tests and bilirubin trended down with removal of the offending agent to near-normal levels approximately 42 days after initial presentation, and her abdominal pain gradually dissipated as well. The hypersensitivity or reactive metabolite syndrome improved with triamcinolone cream within 24 hours of treatment. The etiology of her emesis and remains unclear, however her remaining symptoms are gradually resolving and thought to be due to chronic hepatitis from the inciting injury.
ATRIAL FIBRILLATION CAUSED BY INdwELLING PErIPHERALLY INSERTED CENTRAL CATHETER (PICC)

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LEARNING OBJECTIVE 1:
Identify indwelling Peripherally inserted Central Catheters (PICC) as a cause of new onset arrhythmias

LEARNING OBJECTIVE 2:
Evaluate and successfully treat PICC associated arrhythmias

CASE:
A 22-year-old female with no previous cardiac disease was admitted for treatment of meningitis. A right-sided PICC line was placed in arm for intravenous access and antibiotic administration. Chest radiographs had showed the PICC tip at the aorto-caval junction. A day later, she complained of palpitations. These started within an hour of PICC placement. Symptoms were worse with lying flat, arm movements and deep breaths. Electrocardiogram (EKG) showed atrial fibrillation (AF) with atrial and ventricular rate of 210/min and 78/min respectively. Previous EKGS at admission and dating back 6 months showed sinus rhythm. The PICC was thought to be the likely cause of AF given the positional variation of symptoms. The PICC withdrawn 1cm from its prior location. Immediately after this, she returned to normal sinus rhythm. Placement at the distal third of the Superior Vena Cava (SVC) was confirmed on chest radiograph. Thyroid function tests, complete blood count, electrolytes and echocardiogram were all within normal limits. She was asymptomatic and maintained sinus rhythm until discharge.

DISCUSSION:
Peripherally Inserted Central Catheters (PICC) are commonplace in patients requiring long-term intravenous therapy in both inpatient and ambulatory settings but arrhythmias are an infrequent complication of indwelling PICC lines. While transient arrhythmias during PICC placement may occur, these resolve within minutes of insertion. Arrhythmias associated with indwelling PICC may be of atrial or ventricular origin and are caused by irritation of the myocardial wall by the tip of the PICC. Blind PICC placement at the bedside is a cost effective alternative to placement with direct radiologic guidance. However blindly inserted PICCs are less successful than those placed under direct vision (84% vs 100%). Blindly placed PICCs are also more likely to migrate irritating the myocardium. Even uneventful and well placed PICCs, may still be complicated by arrhythmias due to PICC migration. Additionally, changes in body posture for example arm abduction, deep respiration and lying supine may vary the position of the PICC resulting in intermittent positional arrhythmias. Repositioning the PICC is often all that is needed for treatment of these arrhythmias; in our patient, the PICC was withdrawn from the aorto-caval junction into the distal third of the SVC for relief. More proximal PICCs that is proximal SVC increase the risk of PICC associated thrombosis while more distal placement may lead to cardiac complications including arrhythmias, cardiac perforation and tamponade. Cardioversion is rarely needed. About 0.9% of the patients with central catheter associated arrhythmias eventually require cardioversion. In conclusion, the evaluation of new arrhythmias in patients with a PICC line should include assessment of the effect of postural changes on the PICC and the arrhythmias.
A CASE OF FEVER AND ABDOMINAL PAIN IN A YOUNGER GENTLEMAN

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LEARNING OBJECTIVE 1:
To recognize the clinical manifestations and pathophysiology of Ehlers-Danlos syndrome Type IV in the setting of an acute vascular presentation

LEARNING OBJECTIVE 2:
To review evidence-based management for Ehlers-Danlos syndrome Type IV

CASE:
A 31 yr old gentleman without significant previous medical history presented with severe abdominal pain that started suddenly in his right and left upper abdomen. On arrival to the emergency room, the gentleman was noted to have several episodes of bilious emesis and multiple episodes of brown, watery diarrhea. A mild leukocytosis with left shift was observed along with a fever of 101.5 F and stable hemodynamics. Concerns were raised for diverticulitis or colitis and a CT abdomen/pelvis was performed demonstrating a left common and external iliac dissection (with evidence of prior extravasation without active leak) as well as bilateral renal infarcts, a small L common iliac aneurysm (1.7cm) and a question of bilateral renal artery aneurysms. A vascular surgery consultation was placed and a CT head/neck/chest was performed without additional concerning findings. Due to concerns for the presence of possible renal artery aneurysms, anti-coagulation was initiated with intravenous heparin. Renal artery duplex was unhelpful and a renal angiogram was planned but delayed by emergent cases. On hospital day (HD) 2, additional low grade fevers were also noted. Blood cultures drawn on admission remained negative. On HD 3, after additional questioning, the patient remembered further medical history including prior bilateral inguinal herniorrhaphies in a setting of playing sports as an adolescent and multiple bilateral spontaneous shoulder dislocations. A focused exam revealed very pale skin, periorbital hyperpigmentation, talipes equinovarus and exaggerated joint laxity. Concerns for connective tissue disease were raised. Rheumatology was consulted on HD 3 and recommended a genetics consultation to rule out connective tissue disease. Renal angiogram performance was then scheduled for early afternoon the following day (HD 4). On HD 4, the genetics fellow saw the patient just prior to the patient's transfer to the operating room for renal angiogram performance. After a discussion with the genetics attending, the genetics fellow contacted the primary team with a likely diagnosis of Ehlers-Danlos syndrome Type IV and a request for expedited cancellation of the renal angiogram, a procedure that could result in massive dissection/obliteration of arterial vasculature in an Ehlers-Danlos syndrome Type IV patient. The procedure was cancelled just moments prior to the start of the procedure. Collagen vascular disease markers were sent for testing as a part of the genetics work-up. All rheumatologic and hypercoagulability testing returned negative. An angiotensin-converting enzyme inhibitor was initiated as standard therapy. Genetics testing later returned positive for Ehlers-Danlos syndrome Type IV.

DISCUSSION:
Ehlers-Danlos syndrome Type IV is a rare inherited connective tissue disorder (one of six types of Ehlers-Danlos syndromes) caused by a mutation in type III procollagen (Col3A1). It may be seen in 1/25,000 people. The diagnosis is made by identifying clinical criteria and testing positive for the type II procollagen genetic mutation. The clinical features include a characteristic narrow facies with periorbital hyperpigmentation, pale skin with visible subcutaneous vessels, easy bruisability and history of arterial/digestive tract rupture. Obstetric complications may also be seen in women. Arterial complications including aneurysm rupture and dissection as well as gastrointestinal rupture can be fatal and occur in 80% of these patients prior to reaching age 40. While there is no active reversal of this condition, medical management with optimal blood pressure control via an angiotensin-converting enzyme inhibitor with anti-TGF properties, anticoagulation (indicated in the setting of aneurysm formation) and avoidance of contact sports/strenuous activity is generally recommended. Although surgery may be performed in a setting of trauma, invasive procedures should typically be avoided. Taking a careful history and understanding the underlying clinical pathophysiology of Ehlers-Danlos syndrome Type IV as well as standard management and therapy are integral to delivering evidence-based care and avoiding catastrophic events in this population.