

The Pancreas Isn't Laughing; A Case of Severe Acute Pancreatitis Due to Recreational Cannabis Use

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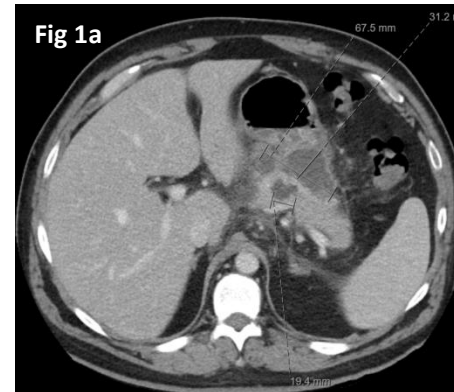
Introduction

- Idiopathic pancreatitis occurs in 15-25% of patients diagnosed with acute pancreatitis (AP)
- AP results in more than \$2 Billion dollars in healthcare costs yearly and remains the most common GI cause of hospitalization
- The most common causes of AP remain alcohol, gallstones, medications, procedures, hypercalcemia and hypertriglyceridemia
- Cannabis induced pancreatitis is a rare phenomenon with rising prevalence which demands awareness

Case Report

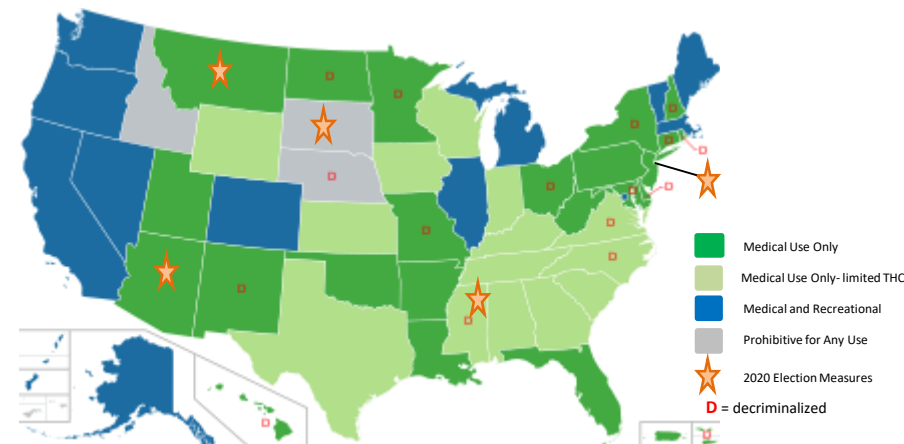
- 54 year-old male patient with daily cannabis use presented with severe abdominal pain, nausea and vomiting. Lipase was elevated to, diffuse abdominal tenderness, and CT findings of severe pancreatitis with pseudocyst and necrosis (figure 1 a-b).
- He admitted to daily marijuana use to treat chronic abdominal pain which only improved with hot water baths.
- The patient denied alcohol use. Abdominal imaging was negative for gallstones and LFTs were normal. Triglycerides were within normal limits on multiple occasions. Pharmacologic drugs were excluded as a cause. He had no recent procedure to explain pancreatitis and no trauma. Calcium levels were normal.
- IgG4 levels were normal and he had no prior history of other autoimmune condition
- He denied family history of pancreatitis. SPINK1, PRSSI and CFTR genes were not tested
- No hereditary cancer syndromes in family, Ca 19-9 WNL
- CT and EUS did not reveal pancreas divisum, mass or other mechanical cause of pancreatitis
- Porta hepatis lymph nodes were biopsied and reactive without malignancy
- His pancreatic inflammation and collections were slow to resolve with continued daily cannabis use

CT findings of complicated pancreatitis



- Figure 1a: complicated pancreatitis with pseudocyst formation with loculations and necrosis
- Figure 1b: diffuse edema of the pancreas with fat stranding near head of pancreas and thickened duodenum

Legality of Cannabis Use in the United States-2020



Discussion

- Cannabis Induced pancreatitis was first described in 2004
- Most report mild illness that improves with fluids and supportive care
- THC and CBD can bind to CB1 and CB2 receptors which are both expressed in the pancreas. Mouse models show agonism of these receptors results in pancreatitis
- Cessation of marijuana use prevents further cases of acute pancreatitis
- Our patient presented with severe, necrotizing pancreatitis with pseudocyst formation and slow resolution with continued cannabis use
- Given the increased prevalence of marijuana use and continued popularity and legality, marijuana should be considered as a cause of AP
- Screening for marijuana with social history and toxicology should be considered on all patients presenting with AP as the incidence is likely to increase over time
- This case also shows that severe, complicated pancreatitis may occur as a result
- Chemical dependency treatment should be offered as recurrence of AP is unlikely if cannabis is no longer used
- In this case, with thorough evaluation, and no clear cause of his pancreatitis, cannabis should be strongly considered as the cause of this "idiopathic pancreatitis"

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NSTEMI Secondary to Acute Covid-19 Infection

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Introduction

- COVID-19 or SARS CoV-2 has caused a global pandemic.
- Transmission is primarily person to person through respiratory droplets.
- Data from multiple countries suggest that 13-19% of cases are hospitalized and 3-5% will need intensive care unit admission.¹
- SARS CoV-2 is a new virus that appears to increase exacerbation of COPD, Asthma, obesity, sickle cell disease, chronic kidney disease, and cancer.
- COVID-19 causes uncontrolled inflammatory responses with specific infection through the ACE 2 receptors leading to thromboembolism and acute cardiac injury.²

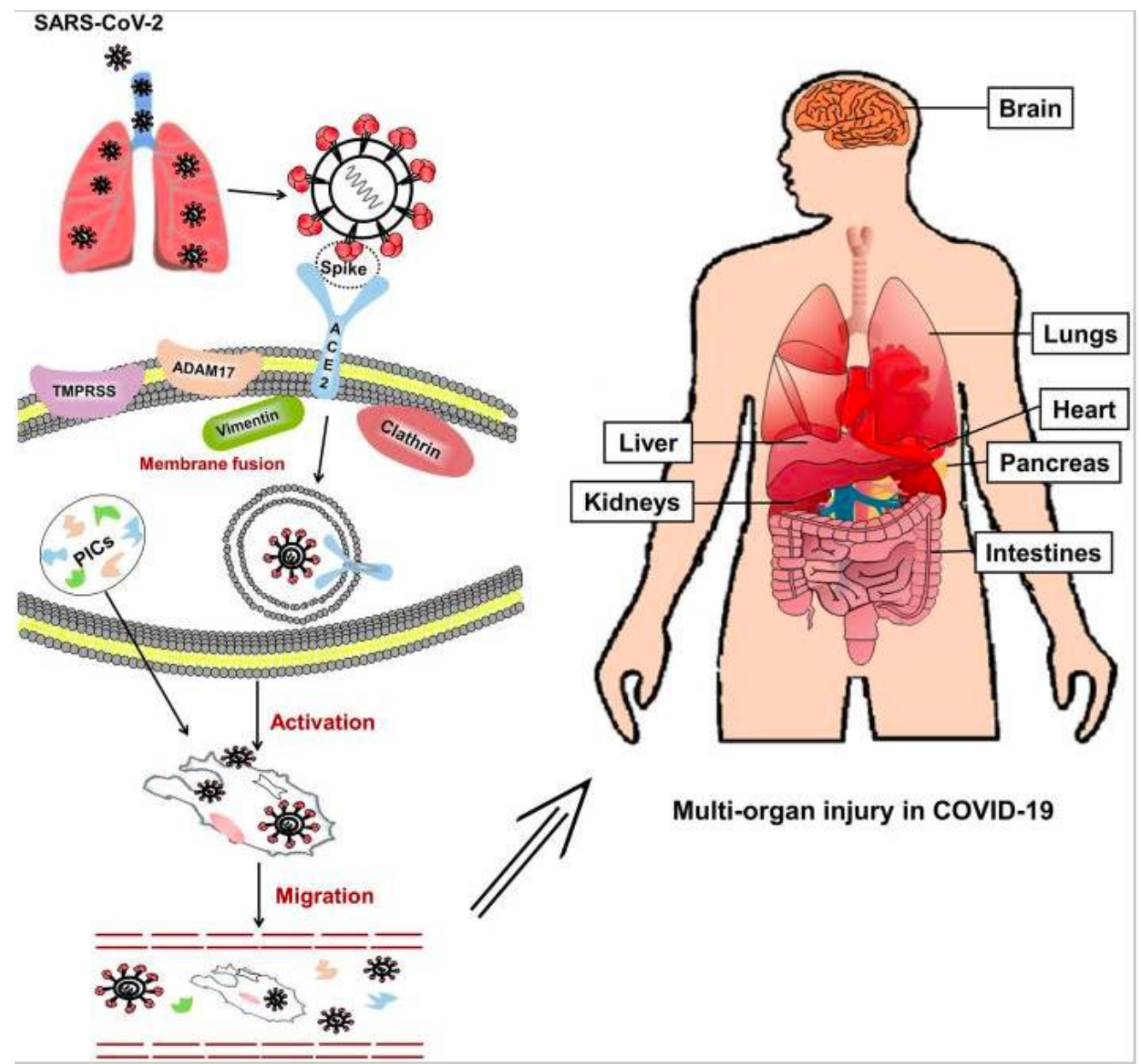
Case

- 54-year-old male
- PMH included hypertension, hyperlipidemia, obesity
- Presented to the ED with left sided chest pain
- Had viral syndrome of fever, chills, SOB 17 days prior to presentation.
- COVID positive 14 days prior to admission
- Chest Pain radiated to arm and jaw
- Started on nitro paste and IV heparin gtt
- Troponins trended 0.008, 0.142, 1.229, 26.43
- Other labs: BNP 92, platelets 494, glucose 102, CRP 24.1
- Cardiology was consulted due to the NSTEMI
- Coronary Angiogram showed 100% occlusion of obtuse marginal artery from a thrombus, 80% occlusion of posterior descending branch, 80% small thrombus in right posterolateral artery 70% proximal left anterior descending artery
- Patient had aspirational thrombectomy followed by balloon angioplasty of obtuse marginal 1 artery.
- Patient was started on aspirin for 1 month, Plavix daily, metoprolol BID, and Apixaban. He was discharged with cardiology clinic follow up.

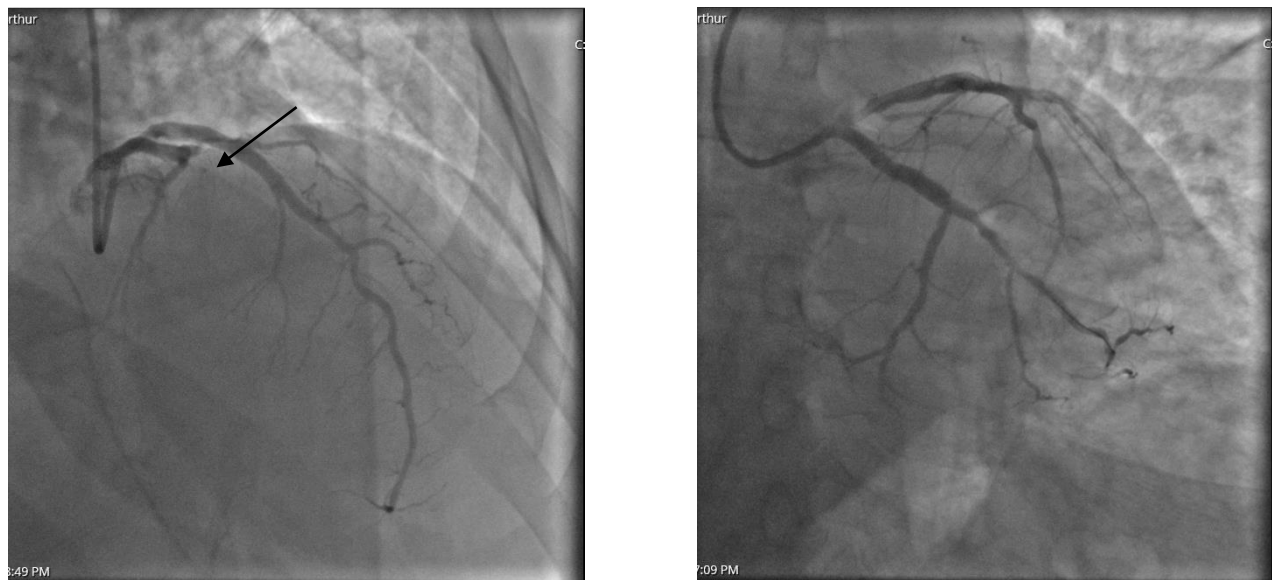
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Lab Values	534	1905	1603	1312
Troponin	26.423	1.229	0.142	0.008
BNP				92
D-Dimer			<.27	
Lipase				20
Glycated Hemoglobin				5.9



Ni W, Yang X, Yang D, et al. Role of angiotensin-converting enzyme 2 (ACE2) in COVID-19. *Crit Care*. 2020;24(1):422. Published 2020 Jul 13. doi:10.1186/s13054-020-03120-0



Discussion

- Mechanism of acute myocardial injury caused by SARS CoV-2 infection might be related to ACE2. ACE2 is widely expressed in the cardiovascular system.³
- In evaluation of 184 patient with SARS CoV-2 that were admitted to the ICU with pneumonia 31% had thrombotic events. ⁴ Venous thromboembolic events were the most common at 27% and majority were pulmonary embolism. ⁴
- The SARS-CoV-2 infection can cause inflammation cascade and the inflammatory reaction can initiate coagulation and reduce the natural anticoagulation mechanism of fibrinolysis system damage. ⁵ ACE2 is expressed in alveolar epithelial cells, arterial endothelial cells, small intestinal epithelial cells as well as immune tissues.⁵
- Ischemic stroke was noted at a seven-fold increased in large vessels in patients under the age of 50. ⁶

Conclusions

- Acute COVID-19 can lead to NSTEMI due to thrombosis within the coronary arteries.
- It appears that patients who have prior cardiac risk factor such as diabetes, HTN, hyperlipidemia Or are elderly have increased risk of cardiac complication with infection from SARS-CoV-2.
- This case exemplifies the need for heightened awareness of acute cardiac injury and thromboembolism in younger patients who have been infected with COVID-19 or SARS CoV-2.



Pulmonary histoplasmosis mimicking primary lung cancer with liver metastasis

Emmanuel Fohle MD, MPH, Rishi Seth, MD, Abhishek Matta, MD

Learning objectives

- Histoplasmosis can mimic lung malignancy on images which can lead to delay in appropriate treatment
- In case of disseminated infection, high degree of suspicion towards any immunosuppressive condition should be entertained and investigated

Introduction

Histoplasmosis is caused by histoplasma capsulatum, a dimorphic fungus

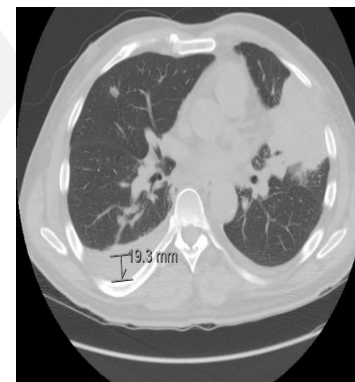
- It is found in fertile moist soil contaminated by bird and bat excreta
- In US- mostly in Ohio and Mississippi River Valleys
- 3 forms of clinical manifestation:
 - Acute pulmonary histoplasmosis
 - Chronic pulmonary histoplasmosis
 - Disseminated histoplasmosis
- Can present with fevers, night sweats, weight loss, hepatosplenomegaly, cutaneous lesions, can involve any organ

Case Presentation

- Pt ID:** 58 y/o Caucasian male
- CC:** 2-3 wks. of progressive fevers, night sweats and productive cough
- VS:** Temp 37 C, HR 101, BP 128/50, RR 18, SpO2 on room air
- Exam:** well developed and nourished, no respiratory distress
- PMH:** Rosacea, chronic lower back pain
- Sc Hx:** 20 pack year smoking, 4 beers/week
- Notable initial labs:** WBC 1.8, (lymphocyte 0.3), CRP 219, Procalcitonin 0.33

Further Evaluation and Interval History

- CXR:** left lobe infiltrate and 1 cm nodule-like density (figure 1)
- CT chest:** 7.7 x6.5 cm mass in left lobe concerning for primary malignancy, hypodense hepatic masses and splenomegaly (figure 2)
- Viral:** HIV, Hep B, Covid-19 → negative
- Bacterial:** mycobacterium, legionella, strep pneumonia, MRSA swab → negative
- BAL and cytology:** patent airway, no malignancy, (+) cytology for fungal organisms
- Liver biopsy:** (+) budding yeast fungal organisms
- Fungal serology:** Blasto, cocci, aspergillus → negative, (+) **Histo antigen**
- Treatment:** Amphotericin B, latter switched to itraconazole due worsening kidney function
- Interval h/o:** persistent fevers, pancytopenic
- Bone marrow:** hairy cell leukemia involving ~50% bone marrow
- Flow cytometry:** monoclonal B lymphoid population, positive CD19, CD20, CD11c, CD25
- Treatment:** Cladribine, Rituximab, Levofloxacin, Acyclovir, Bactrim



Discussion

- Histoplasmosis caused by H capsulatum, more common in endemic area (Ohio, Mississippi River Valleys)
- Can be self limiting disease in immunocompetent patients
- In immunocompromised, it may present primarily as a pulmonary syndrome (cough/hemoptysis, dyspnea) +/- systemic symptoms (fevers, chills, night sweats)
- Pulmonary histoplasmosis can mimic other non infection/infections with symptoms and image findings such as primary lung malignancy, hairy cell leukemia, lymphoma, TB
- Delay in diagnosis can often delay therapy which can be fatal
- Due to similarity with primary lung cancer, patient can be subject to unnecessary biopsy

Conclusion

- Histoplasmosis can mimic primary lung disease on images
- In healthy patient with disseminated histoplasmosis, an attempt should be made to identify an underlying cause of immunosuppression such as malignancy, AIDS, medications

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A rare case of spontaneous splenic rupture in cate-bite induced tularemia

Emmanuel Fohle MD,MPH, Bradley Smith, MD

Learning objectives

- Even though not a common cat zoonoses, tularemia should be suspected in a patient with spontaneous splenic rupture in the setting of cat bite

Introduction

- Tularemia is a zoonotic disease induced by *F tularensis*, a gram negative
- Infection occurs by contact with infected animal, arthropod vectors, inhalation of contaminated dust, food and water
- 6 forms of manifestation
 - Ulceroglandular
 - Oculoglandular
 - Oropharyngeal
 - Pneumonic
 - Typhoidal
 - Intestinal

Case Presentation

- Pt ID: 42 y/o Caucasian male
- CC: Severe abdominal and back pain and near syncope
 - 1 month prior he was in ED for fever after a kitten scratch in left thumb.→sent home with Augmentin
 - 1 wk later, returned with malaise, swelling under left armpit→ sent home with Bactrim
 - Few days later, returned with abdominal and back pain with near syncope→sent to us
- VS: Temp 36 C, HR 97, BP 156/94, RR 31, SpO2 on room air
- Exam: abdominal tenderness, back pain, left thumb healing ulcer, left axillary lymphadenopathy
- PMH: DM, HTN, umbilical hernia, carpal tunnel
- Sc Hx: smokes 0.5ppd
- Notable initial labs: WBC 19.9, Hgb 9.1, CRP 177, LDH 348

Further Evaluation, Interval History and Treatment

- CT abdo: splenomegaly and subscapular splenic rupture and large hematoma (figure 1a, 1b)
- Treatment: Splenic angiogram with embolization, Rabie and tetanus shots
- Blood culture: negative
- Left thumb culture: negative
- Bacteria: babesia, bartonella, brucella, TB→ negative
- Kitten investigation: succumbed to illness, no rabies, no necropsy
- Viral: HIV, EBV- negative
- Fungal serology: Blasto, cocci, aspergillus, Histo →negative
- Treatment: Doxycycline and unasyn
- Interval h/o: persistent fevers, leukocytosis
- TEE: not vegetation
- Treatment: Unasyn, azithromycin and rifampin
- Bacteria: **Francisella serology 1:2560**
- Treatment: Doxycycline



Figure 1a



Figure 1b

Discussion

- Tularemia is a zoonotic disease induced by *F tularensis*, a gram negative, infection via contact with infected animal, arthropod vectors, inhalation of contaminated dust, food and water
- In the US, there are around 57 millions of domestic cats living in one third of all household
- There are an estimated 400,000 cat bites each year with 66,000 hospital emergency visit each year
- Tularemia from cat scratches or bites is rare and accounts for less than 2% of all cases of tularemia
- 6 main categories of splenic rupture: neoplastic, infectious, inflammatory/noninfectious, drug induced, mechanical and normal spleen
 - Infectious cause: EBV, HIV, Plasmodia, Salmonella, Bartonella, Dengue and much more
 - In cat bite- bartonella is well studied and documented in case reports
 - Cat induced tularemia splenic rupture documented in 1946

Conclusion

- This case adds another infectious agent that be associated with spontaneous splenic rupture. In patient who presents with abdominal and back pain in setting of cat bite and splenic rupture, clinicians should include *F tularensis* in the workup

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Diabetes Insipidus, a Rare Complication of Sarcoidosis.

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Background

- Sarcoidosis is a systemic granulomatous disease with multiorgan involvement. ¹
- The classic presentation of fever, painful erythematous subcutaneous nodules of the lower extremities and bilateral hilar lymphadenopathy, a triad coined “Lofgren's syndrome” generally has a good prognosis. ^{1,2}
- Neurological involvement and more specifically neuroendocrine involvement, though rare, has historically been associated with poor prognosis. ³

Case

A 48-year-old female with a past medical history of sarcoidosis presented to an outpatient endocrinology clinic as a referral for polyuria and polydipsia. On presentation she reported 8 months of excessive thirst and increased urination. She was drinking approximately 10-15 glasses of water a day and urinating every 2 hours. She also reported waking 2 to 5 times each night to urinate. As a result, she was referred to endocrinology. Workup revealed a normal TSH at 2.01, a slightly elevated calcium at 10.8 with a PTH of 45 (normal 14-95 ng/dL), low 25-hydroxy vitamin D of 24 (normal 30-80 ng/mL) with a high normal 1,25 dihydroxy vitamin D of 60 (normal 18-78 pg/mL). She underwent a water deprivation test as seen in table 1. After 8.5 hours administration of 1 mg subcutaneous DDAVP, urine osmolality increased to 442 mOsm/kg confirming the diagnosis of complete central DI. An MRI sella was performed which showed pituitary stalk thickening as well as a 2.6 mm microadenoma suggestive of sarcoidosis involvement. Pituitary function was unremarkable except for a slightly elevated prolactin of 22.2 ng/mL. She was started on intranasal DDAVP and prednisone 50 mg daily for the treatment of neurosarcoidosis. She was subsequently transitioned to azathioprine for long-term treatment as she was unable to tolerate steroids due to weight gain and fluid retention.

Figure 1

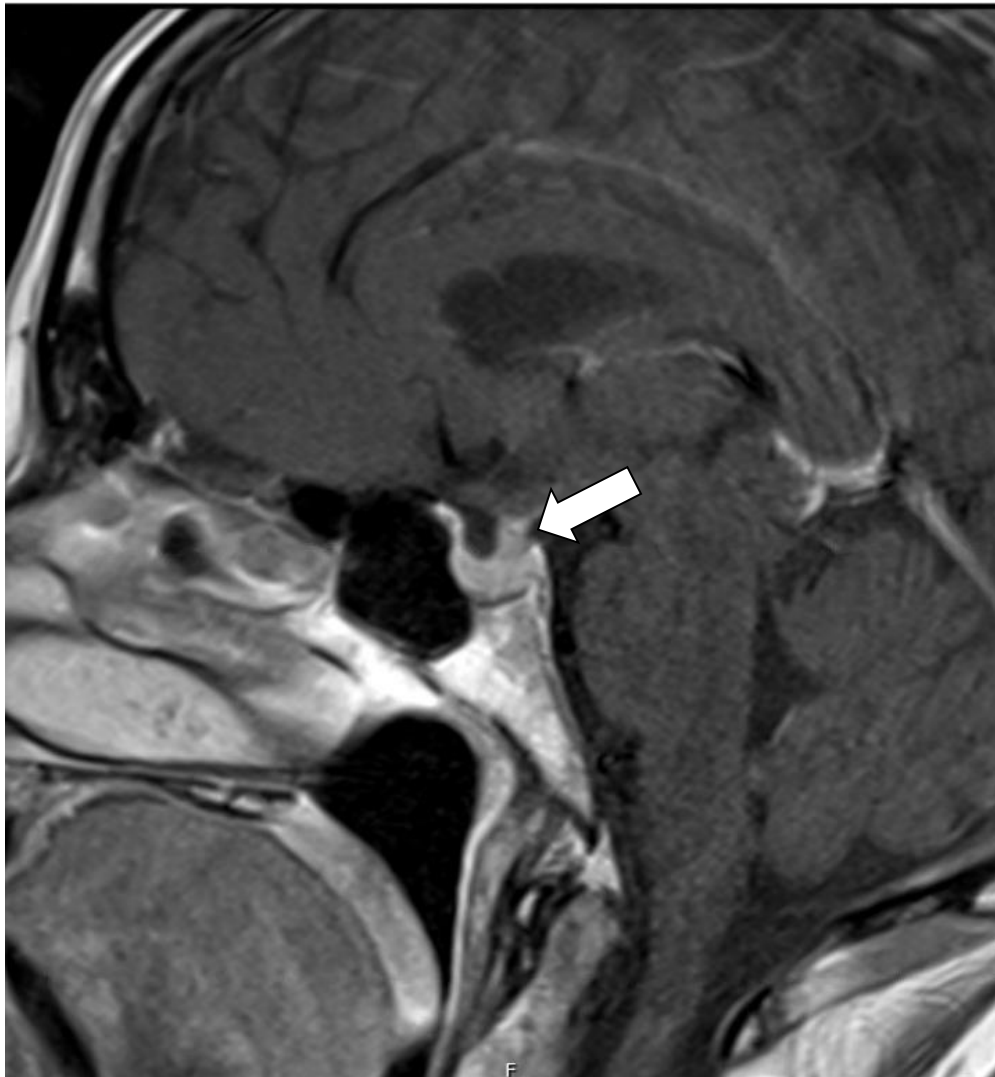


Figure 2



Table 1

Time in hours (Approximately)	Serum Osmolality 280-305 mOsm/kg	Serum Sodium 135-145 meq/L	Urine Osmolality 390-1,000 mOsm/kg
4.5	296	144	134 (L)
6.5	302	145	134 (L)
8.5	303	148 (H)	170 (L)
9.5			217 (L)
10.5			378 (L)
11.5			442

Discussion

- Neuroendocrine involvement of sarcoidosis is exceedingly rare with only 0.5-1% of patients diagnosed with sarcoidosis presenting with endocrine involvement. ⁴
- Water deprivation testing should be used to differentiate between central DI, nephrogenic DI or primary polydipsia in patients who present with polydipsia.
- As seen in this case, sarcoidosis can cause hypercalcemia by increasing the conversion of 5-hydroxy vitamin D into active 1,25 dihydroxy vitamin D. ²
- Corticosteroids are the mainstay in treatment of sarcoidosis and may even result in the restoration of endocrine function if there is neuroendocrine involvement; however, hormone replacement may be needed. ⁴
- Alternative treatments such as azathioprine or other immunosuppressive therapies can be used when corticosteroids have either been inadequate to control the disease or have caused significant side effects. ²

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Myasthenia Gravis and Myocarditis: A Deadly Aftermath of Nivolumab Treatment

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Introduction

Nivolumab is a immunoglobulin G4 for monoclonal antibody which is a promising new immunotherapy for many cancers such as metastatic melanoma, non-small cell lung cancer, and renal cell carcinoma [1]. This is the case of a patient who presented with new onset myasthenic crisis and myocarditis after he was treated with 2 doses Nivolumab.

Case presentation

An 87-year-old male presented to the ER with sudden onset diplopia, drooping eyelids, dysphagia, dysarthria, shortness of breath and fatigue that started after he was treated with 2 doses of Nivolumab (immunotherapy) for malignant melanoma. At admission, he was found to be in hypercapnic respiratory failure, had dysarthria and ptosis. Labs revealed that he had a creatinine kinase of 2348, troponin of 9.7.

Management

Patient was started on IV fluids and was placed on BiPAP and started on pyridostigmine as his symptoms were consistent with new onset myasthenia gravis. Treatment was later augmented with high-dose steroids and IVIG. There was minimal improvement in his symptoms with this treatment. Telemetry monitoring showed variable AV block and tachy-arrhythmias.

Unfortunately, despite all aggressive measures, patient's respiratory status declined overnight and he passed away. Serologic studies showed that acetylcholinesterase inhibitor was negative but anti-striational antibodies were positive which is consistent with immune mediated myasthenia gravis

Discussion

- Nivolumab works as a checkpoint inhibitor by binding to programmed cell death (PD-1) receptor to block programmed death ligand-1 and programmed death ligand-2 (PD-L2) from binding T-cells and prevent them from being inactivated [1].
- Myasthenia gravis and myocarditis are less known side effects of Nivolumab.
- Immune checkpoint inhibitors unbalance the immune system and generate dysimmune toxicities called as immune-related adverse events.
- Few such case reports have been published, some of these cases are negative for acetylcholine receptor antibodies and diagnosis was made clinically.
- It is postulated that anti-striational antibodies react with epitopes of muscle protein titin which is in the skeletal and cardiac sarcomere unit.
- Management includes Corticosteroid administration and discontinuation of immune checkpoint inhibitors as the core treatments, and other options also include intravenous immunoglobulin, cyclosporine A, cyclophosphamide, infliximab, mycophenolate mofetil, and plasmapheresis. Recently, cases of immune-related myocarditis that were treated with alemtuzumab or abatacept have also been reported. [2,3]

- Mechanism of myasthenia gravis secondary to PD 1 inhibitor treatment is unclear and further data is needed to establish true incidence.

Conclusion

- This case portrays myasthenia gravis as an under-recognized side effect of an otherwise very effective immunotherapy agent for treatment of multiple cancers.
- This presentation also underscores the importance of provider awareness, early recognition and prompt response to this potentially fatal adverse event.

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Flecainide Toxicity Mimicking Ventricular Tachycardia

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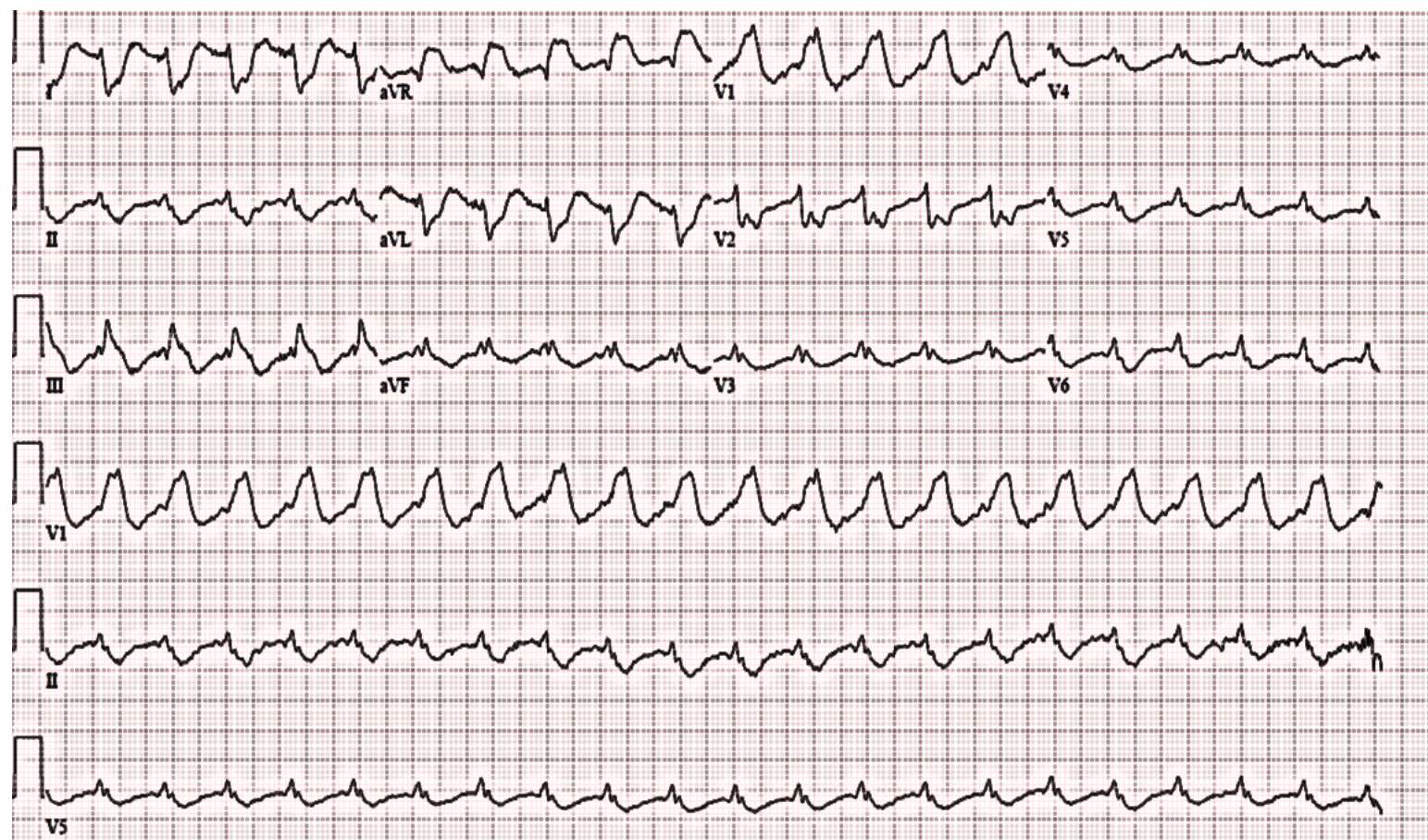
Introduction

Flecainide is a Class 1C antiarrhythmic drug used in the treatment of atrial fibrillation/flutter, paroxysmal supraventricular tachycardia and ventricular arrhythmias. It has a narrow therapeutic index which is between 0.2-1 µg/ mL. Here, we present the case of a patient who presented with wide complex tachycardia which is actually organized atrial flutter with wide QRS due to Flecainide toxicity.

Case presentation

A 69-year-old female with history of paroxysmal atrial fibrillation on flecainide for 6 years presented to the hospital with 2 weeks of shortness of breath, dizziness, palpitations and generalized weakness. Her symptoms developed after she was started on Fluoxetine for depression. On examination she is tachycardic in 130s and hypotensive with systolic blood pressure in 80s.

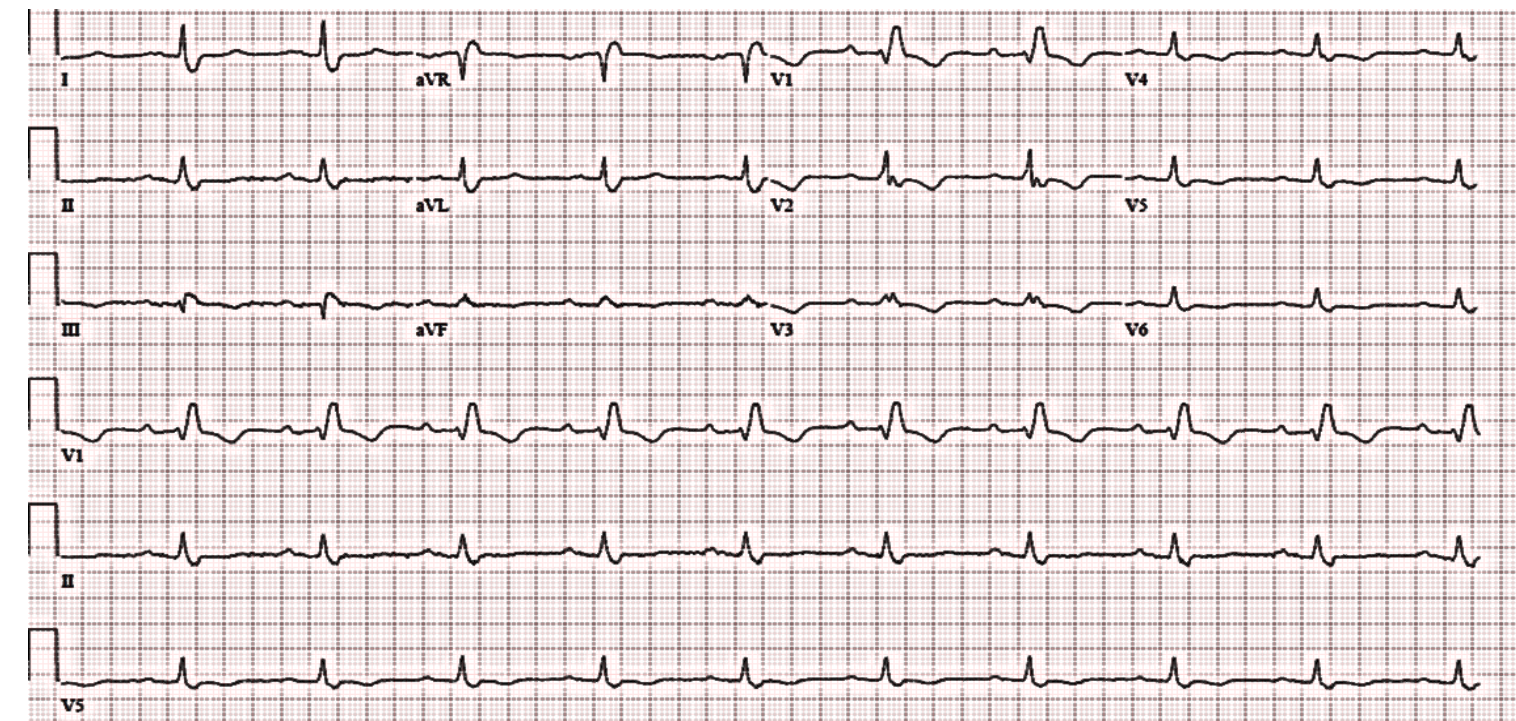
EKG:



Wide complex QRS tachycardia, HR: 120 bpm, QTc: 599 ms

Management

Cardiology was emergently consulted. Her ventricular tachycardia was actually organized atrial flutter from flecainide toxicity. It was hypothesized that Prozac decreased the clearance of flecainide, causing toxicity. Patient underwent treatment with sodium bicarbonate infusion to keep blood pH 7.5. Serum Flecainide level was later found to be elevated at 1.2 which is above the therapeutic index. She continued to have wide complex tachycardia and eventually underwent synchronized cardioversion. She converted to normal sinus rhythm as shown and was then discharged home. Flecainide was permanently discontinued.



Resolution of wide complex tachycardia after synchronized cardioversion

Discussion

While thought to be safe in a patient without underlying ischemic cardiomyopathy, Flecainide carries pro-arrhythmic characteristics and the potential for inducing ventricular tachycardia.

Diagnosis of flecainide toxicity can be difficult as the flecainide serum level may take days to result and there are no pathognomonic clinical signs.

The mainstay of medical therapy is high-dose sodium bicarbonate to offset the cardiotoxic effects of the drug by inducing a high-dose sodium load along with serum alkalinization. Though the molecular mechanism underlying drug pH and drug-sodium concentration is not completely understood, it is thought that increases in both sodium ion concentration and pH prevent flecainide binding to sodium channel receptors by competitive inhibition and electrostatic repulsion. Alkalinization also facilitates flecainide dissociation from the sodium channel binding site.

Additional medical therapy includes intravenous fat emulsion that is thought to sequester the lipophilic drug. Overdrive pacing can also be helpful if needed.

This case highlights the importance of prompt recognition of the wide QRS tachycardia as organized flutter due to flecainide toxicity rather than ventricular tachycardia in patients on flecainide.

It also emphasizes the need for heightened vigilance when prescribing new medications to patients on flecainide to prevent drug interactions.

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