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Annual Scientific Meeting

E- Poster Presentations

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Thursday, Sept. 30, 2021

New York Chapter
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Medical Student Clinical Vignette

Medical Student Clinical Vignette

Edsel Embry

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When Posterior Reversible Encephalopathy Syndrome Gets the PRES-idential Treatment

INTRODUCTION: Posterior reversible encephalopathy syndrome (PRES), previously known as Reversible Posterior Leukoencephalopathy Syndrome (RPLS) was first reported by Hinchey et al in 1996. Since this initial study, there has been a lack of randomized controlled studies regarding PRES. Our case report presents an early diagnosis followed by prompt treatment that is associated with up to 90% improvement.

CASE PRESENTATION: A 39-year of age obese female presented for headache, nausea and photophobia with blood pressure (BP) 187/103 at triage. Neurological and funduscopic exams were benign, along with unremarkable findings on laboratory work up. Head CT revealed increased hypoattenuation involving the subcortical white matter of parietal and occipital lobes. Per neurology consult, optimum BP control and MRI of brain were recommended. Patient continued to improve, and her BP was decreased at goal. She was discharged with counseling for lifestyle modification, along with close follow up to outpatient primary care and neurology. Based on the clinical and radiological findings, a diagnosis of PRES was made.

DISCUSSION: Epidemiological data on PRES is currently limited. The true incidence and mechanism of PRES are yet not completely understood. Incidence shows that women are more affected than men, and all age groups can be affected. A proposed mechanism hypothesizes involvement of auto-regulatory ability of cerebral vasculature, which results in vasogenic edema and a compromised blood-brain barrier. The majority of cerebral vasculature can typically manage blood pressures up to 150-160 mmHg. However, an exception to this lies in the parieto-occipital lobes, where little sympathetic innervation exists in the posterior fossa. As a result, parieto-occipital regions are more vulnerable to hyperperfusion, as observed in the CT findings of our patient. Common associated conditions of PRES include hypertension, sepsis, eclampsia, autoimmune disorders, renal failure, electrolyte imbalances and exposure to immunosuppressants. Clinical manifestations of PRES are variable. The most common manifestation of PRES is encephalopathy (80%), followed by seizures (75%), headaches (50%), visual disturbances (33%), focal neurological deficits and status epilepticus (both 15%). CT and MRI are typically done in workup, which reveals posterior cerebral edema, although variations do occur. Management of PRES aims to control BP, treat any underlying co-morbidity, and discontinue any offending medication. Prognosis is typically benign and takes days to weeks for recovery, so long as management is not delayed. Given the prompt diagnosis and treatment of our patient, her improvement reflects the typical prognosis of PRES.

CONCLUSION: PRES can be easily misdiagnosed due to its numerous etiologies, variable manifestations, and complicated imaging findings. PRES typically results in reversal of symptoms. However, a delay in diagnosis leads to irreversible neuronal cell death, progressive cerebral edema or intracerebral hemorrhage. Given the limited data on PRES, this case report aims to enhance future considerations for prompt management of PRES.

Medical Student Clinical Vignette

Nolberto Jaramillo

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Management of Refractory Symptomatic Premature Ventricular Contractions In A Patient With Louis-Dietz Syndrome Type 3, Ehlers-Danlos Syndrome, and Systemic Lupus Erythematosus

Introduction

Louis-Dietz syndrome (LDS) Type 3 is an extremely rare disorder caused by an autosomal-dominant mutation in SMAD-3, resulting in aberrant expression of TGF-Î² pathway proteins. LDS Type 3 typically manifests as aortic aneurysms and early-onset osteoarthritis, however other dermatologic, cardiovascular, and skeletal abnormalities have been reported. We present a case of highly symptomatic and drug-refractory premature ventricular contractions (PVCs) in a patient with LDS and other connective tissue disorders that emphasizes the importance of evaluating and treating PVCs in these patients.

Case Description

A 51-year-old woman was referred to the cardiology clinic for palpitations, syncope, chest pain, and shortness of breath during the COVID-19 pandemic. She was previously successfully treated for congestive heart failure and had a history of cardiomyopathy (CM), patent foramen ovale, atrial septal aneurysm, myocarditis, and pericarditis. She also has a pertinent medical history of hypermobile Ehlers-Danlos syndrome (hEDS), Raynaudâ€™s syndrome, Arnold-Chiari malformation, and systemic lupus erythematosus (SLE). Her family and social history were remarkable for a daughter with SLE. Following a normal cardiopulmonary exam, evaluation with an ECG and Holter monitor showed normal sinus rhythm with unifocal premature ventricular contractions (PVCs) that correlated with her symptoms. She was originally managed with metoprolol however it did not produce a therapeutic effect. A subsequent electrophysiology study (EPS) demonstrated frequent PVCs, for which mexiletine was prescribed. Since this medication was not available at the patientâ€™s pharmacy, verapamil was prescribed instead. The patient remained highly symptomatic with PVCs and underwent ablation with magnetic navigation using the Stereotaxis system (St. Louis, MO). A left ventricular septal focus was identified and successfully ablated with radiofrequency energy. Following the procedure, she was less symptomatic and was diagnosed with a right internal carotid artery aneurysm. This prompted genetic testing which demonstrated a SMAD-3 mutation and the diagnosis of LDS Type 3 was made.

Discussion

This case highlights the importance of the evaluation and treatment of refractory symptomatic PVCs. Due to their frequency, the PVCs may have also contributed to the patientâ€™s cardiomyopathy. Additionally, LDS Type 3, hEDS, and SLE all can have cardiac manifestations that may have had a synergistic effect on this patient's cardiac condition. LDS Type 3 has been associated with cardiac arrhythmias thus further understanding of the role of SMAD-3 in these patients is required. Basic studies in a mouse model have demonstrated that SMAD-3 dysregulation may lead to fibrotic changes in the heart that potentially explains an increase in arrhythmias in this patient. Further studies are necessary to determine the true interplay between these connective tissue disorders and arrhythmogenesis.

Medical Student Clinical Vignette

Jacqueline Nikakis

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THE MANAGEMENT AND SCREENING OF FAMILIAL ARRHYTHMOGENIC CARDIOMYOPATHY

Introduction:
Arrhythmogenic cardiomyopathy (ACM) is a hereditary condition in which fibrofatty tissue replaces the myocardium, predominantly of the right ventricle. ACM is associated with ventricular tachycardia (VT) and sudden cardiac death (SCD). Gene variants are only identifiable in about 50% of individuals diagnosed with ACM and the disease has a 0.02 - 0.05% global prevalence.

Case Description:
A 55-year-old woman was referred for evaluation of rapid palpitations, and recurrent syncope and presyncope. She felt as though her chest was â€œhaving a seizure.â€ Her past medical history included hypermobile Ehlers-Danlos syndrome, and spontaneous carotid artery dissection. Her family history was remarkable for myocardial infarction in her father and cardiac disorders - including SCD - in many non-first-degree relatives.

Her ECG revealed sinus rhythm with a right bundle branch block, and left anterior fascicular block. The patient underwent an electrophysiology (EP) study in February 2020, during which atrial fibrillation and two beats of AV nodal reentrant tachycardia were induced. No ablation was performed. An implantable loop recorder (ILR) was implanted, and subsequently revealed a 27-beat run of VT. Due to the COVID-19 pandemic, a wearable defibrillator was provided during lockdown. Additionally, a cardiac MRI demonstrated localized right ventricular apical free wall akinesia and mild right ventricular enlargement, meeting the criteria for ACM. Due to her symptomatic VT and ACM, her ILR was explanted and replaced with an implantable cardioverter defibrillator. One year following the procedure, the patient had no episodes of VT, but has had recurrent supraventricular tachycardia (SVT) consistent with atrial flutter, and an ablation is being considered.

The patient tested negative on the Invitae Arrhythmogenic Cardiomyopathy Panel for identifiable gene variants. Despite no variants, her three daughters were screened by our cardiac clinic. They underwent ILR implantation in August 2020 as a result of an rSRâ€™ pattern seen on their ECGs. One is asymptomatic, another has had episodes of SVT, and the last has had episodes of nonsustained ventricular tachycardia (NSVT).

Discussion:
This case demonstrates that during a pandemic wearable defibrillators can be used to manage ventricular arrhythmias while under lockdown, thereby aiding in the treatment of ACM by protecting against possible SCD. This case also illustrates that patients who test negative for gene mutations associated with ACM can still pass down the condition to their children. Although our patient tested negative for known gene variants, more are still being discovered today. Two of her three daughters were found to have symptomatic arrhythmias, one of whom has had episodes of rapid NSVT and has been referred for a cardiac MRI to rule out ACM. Close follow-up and management with the cardiac team will ensure safe and effective management of this potentially lethal, but treatable condition.

Medical Student Clinical Vignette

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Procoagulant effects of Moringa Oleifera inducing Pulmonary Embolism

Introduction: Pulmonary embolisms have been associated with factors that impact hyper- coagulable states, endothelial damage, or prolonged immobilization. In this study, we aim to investigate the relationship between Moringa Oleifera and its effect on the clotting cascade and resultant pulmonary embolisms. Case presentation: A 63 year old female with a past medical history of hypertension and type 2 diabetes presented with two weeks of progressive shortness of breath and chest pain. On Emergency Room presentation, she was mildly tachypneic and tachycardic, saturating 94% on room air with no significant findings on physical exam. The patient traveled from Mexico to New York one day prior to admission; however, symptoms began a week before travel. Initial blood work was significant for high D-dimer. POCUS did not reveal heart strain. Doppler ultrasound of bilateral lower extremities was negative for deep vein thrombosis. A CT angiogram scan of the chest was positive for submassive pulmonary embolism. Further workup was negative for hyper-coagulable state or autoimmunity. Malignancy as a risk factor was ruled out given prior normal mammogram, colonoscopy, and intravaginal ultrasound. During the interview, the patient mentioned she had been taking the herb, Moringa, daily for the past five months to better control her cholesterol levels. Discussion: Moringa has been utilized for its various benefits. In one study, Moringa leaves have been found to lower blood pressure and cholesterol, and are also reported to possess hepatoprotective and antibacterial activity. This case report highlights the possible association between Moringa and thromboembolic events. Moringa has been found to have proteolytic activity on fibrinogen. Fibrinogen is converted to fibrin by thrombin to promote clotting. Fibrinogen subunits contain alpha, beta, and gamma polypeptide chains. Moringa hydrolyzes the alpha and beta chains of fibrinogen, promoting the formation of fibrin rings after the addition of CaCl2 to human citrated plasma, and reduces the recalcification time in clot formation by activating the factors or by precipitating the co-factors involved in the blood coagulation cascade. This observed procoagulant effect could result in complications such as clot formation, however may be masked by the beneficial uses of Moringa. As herbal therapies remain popular, there is a growing need for research in the benefits as well as complications of such remedies. Interim history: The patient was started on therapeutic anticoagulation. After three days of treatment, stated relief of symptoms. She was instructed to stop taking Moringa and discharged home with close follow-up. Conclusion: Moringa is a traditional herb broadly used in Mexico and Central America for different therapeutic benefits but lacks support in clinical studies. It has a pro- coagulation effect that can lead to pulmonary embolism, and its use should be limited.

Medical Student Clinical Vignette

Jeremy Santarelli

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ANAPHYLACTIC URTICARIAL REACTION TO LORATADINE: A CASE REPORT

Introduction:

H1- Antihistamines are a group of medications widely used to treat various allergic diseases. Even mild adverse reactions to this class of medications are considered rare. <1% of patients have reporting anaphylaxis and urticarial reactions resulting in little research and literature to be found on the subject.

Case: A 29-year-old female denying any past medical history presented to the emergency department with difficulty breathing, dysphagia, nausea with vomiting, and a generalized rash beginning a few hours after taking a generic Loratadine tablet for seasonal allergies. The patient states she usually takes brand name Claritin and denies any reaction like this in the past as well as any other known allergies. On admission the patient was alert and oriented x 3 and in mild distress. She was tachycardic and tachypneic with an O2 saturation of 95% on room air. Physical exam revealed a patent airway with no stridor and diffuse erythematous blanching hives. Chest X-ray was unremarkable. A diagnosis of anaphylactic urticaria was given and the patient was treated with 0.3mg intramuscular epinephrin, 10mg Decadron IV, 20mg Pepcid IV and was observed. The patient quickly returned to baseline and the urticaria resolved completely. However, it soon returned with associated pruritis requiring 50mg Benadryl IV and inpatient admission for prolonged observation. Solumedrol 40mg BID and Benadryl 50mg BID were started and the Allergist was consulted. Thus, concluding the reaction was most likely from the new generic loratadine, and recommending Fluticasone nasal spray BID, Benadryl PRN and abstinence from Claritin and its generics. The patient continues to follow her PCP and an outpatient allergist for further workup and allergy testing.

Discussion:

H1-antihistamines are commonly used to treat allergies by directly inhibiting histamine from binding to H1 receptors, preventing allergic symptoms. Due to the operant MOA of these drugs, anaphylaxis and urticaria must be considered very rare and detailed reports are limited. The exact MOA of these adverse reactions remains unclear due to the variability in presentation, non-standardized testing, and lack of overall research into the subject. Some reports have used skin prick, skin patch, and oral challenges to attempt to confirm reactions with little to no success due to the high variability of outcomes from patient to patient.

Conclusion:

This case is to inform clinicians of the possibility that H1- antihistamines routinely used to treat allergic reactions have the potential to be a causative agent themselves. It can easily be overlooked or mistaken for the primary pathology being treated therefore it is imperative to educate treatment providers to prevent misdiagnosis and unintended outcomes.

Leana Wang

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INDICATION FOR SUBCUTANEOUS IMPLANTABLE CARDIOVERTER DEFIBRILLATORS IN A CASE OF FAMILIAL SUDDEN CARDIAC DEATH

Introduction

Familial sudden cardiac death (SCD) has been attributed to conditions such as long QT syndrome, Brugada syndrome, arrhythmogenic cardiomyopathy, and hypertrophic cardiomyopathy (HCM). It is imperative to recognize these conditions and manage them with the appropriate procedures and devices, such as cardiac catheter ablation and implantable defibrillators, to prevent SCD.

Case Description

A 29-year-old man presented with a chief complaint of palpitations and presyncope during weightlifting. His past medical history was remarkable for rapid palpitations and recurrent syncope. His palpitations were terminated by the Valsalva maneuver, with all but one syncopal episode preceded by palpitations, which may indicate paroxysmal supraventricular tachycardia. Family history was significant for SCD in his 30-year-old brother (autopsy revealed cardiomyopathy, and genetic testing showed a TNNI3K mutation of unknown significance) and his 50-year-old uncle. Physical examination showed elevated blood pressure and a normal cardiopulmonary examination. ECG showed sinus rhythm, extreme axis with rSRâ€™ pattern in V1, ST segment elevation in V1 to V3, and left atrial enlargement.

The patient was referred to the emergency department at Mount Sinai Morningside Hospital and underwent a cardiac MRI and an electrophysiology (EP) study. The cardiac MRI detected asymmetric septal hypertrophy with the width of the basal septum measuring 2.2 cm. The EP study demonstrated easily inducible AV nodal reentrant tachycardia (AVNRT), nonsustained ventricular tachycardia (NSVT), and hemodynamically unstable polymorphic ventricular tachycardia. AVNRT was successfully ablated using radiofrequency energy applied to the slow pathway of the AV node. A subcutaneous implantable cardioverter defibrillator (S-ICD; Emblem® MRI, Boston Scientific, Marlborough, MA) was subsequently implanted. This patient met class IIa recommendations for S-ICD implantation, based on the current HCM guidelines.

Discussion

A complete evaluation would be necessary to determine the presence of multiple arrhythmias, even if ventricular tachycardia was presumed to be the primary arrhythmia. As discussed, this patientâ€™s AVNRT was treated with ablation, so an S-ICD was chosen as a means of SCD prevention since it is preferred in younger patients who do not require pacing therapies.

It is also essential to discern the signs and symptoms that may be precursors of SCD in patients with HCM. HCM is an autosomal dominant disorder of sarcomere proteins that increases risk of SCD, especially in adolescents and young adults. With a prevalence of 0.16 - 0.29% in the adult population, performing a genetic analysis for possible SCD-associated mutations, such as in TNNI3K, may help with the identification and prevention of familial SCD.

Medical Student Clinical Vignette

Caroline Weiss

Joseph Wayne, MD

Albany Medical College

A Case of Vertebral Osteomyelitis Caused by E. Coli Complicated by Multiple Soft Tissue and Muscle Abscesses

Vertebral osteomyelitis accounts for approximately 3-5% of all cases of osteomyelitis diagnosed in the United States each year1. Of these cases of vertebral osteomyelitis, Staphylococcus aureus is the most common cause of infection (32-67%), followed by Escherichia coli, usually from a urinary source2. We present a patient diagnosed with Escherichia coli vertebral osteomyelitis three weeks after hospitalization for urosepsis.

The patient is a previously healthy 59-year-old female admitted at an outside hospital (OSH) three weeks prior to presentation with back pain and recurrent falls and was found to have a UTI and sepsis with blood cultures (+) for Escherichia coli. X-rays of her spine at the initial hospitalization showed degenerative changes. She was also diagnosed with Type 2 Diabetes mellitus during her initial admission and started on metformin and insulin. She was treated with IV ceftriaxone for six days and discharged on oral amoxicillin clavulanate twice daily for seven days. Three weeks later, she again presented to the OSH with progressive low back pain and new right leg weakness. CT-spine showed endplate disruption of the L2 vertebral body, and paravertebral soft tissue thickening/stranding at L1-2. The patient was transferred to Albany Medical Center.

On arrival, patient was alert and oriented, but uncomfortable. Vital signs were T: 36.2, BP 176/91, heart rate 100, respirations 18, and POx 97% on 2L O2 via nasal cannula. Lungs were clear and heart exam showed a regular rhythm with no murmurs. Upper extremity strength, sensation, and range of motion were normal. Lower extremity sensation was normal bilaterally, with 2/5 strength on the right and ability to lift against gravity on the left. Labs were WBC 10,200 with 82% segs, ESR 96 MM/hr and CRP (122.1 mg/L). Her urinalysis showed 1+ protein, trace leukocyte esterase, 2+ urine hemoglobin and 1+ ketones.

MRI of the spine and pelvis showed STIR hyperintensity and postcontrast enhancement at the L1-L2 disc space, a right paraspinal soft tissue abscess, and a left iliopsoas abscess. Blood and urine cultures were negative. The right paraspinal abscess at the level of the right crus the diaphragm was drained, and culture yielded pansensitive Escherichia coli.

The patient was given IV cefepime (1g every 6 hours) for one day then IV ceftriaxone (2g every 24 hours) for 6 weeks. Pain and lower extremity weakness improved by discharge. One month later she reported decreased back pain and the ability to walk utilizing a walker.

It is important to consider the diagnosis of vertebral osteomyelitis in patients presenting with E. Coli bacteremia, sepsis and back pain to prevent severe complications such as multiple abscesses due to the continued spread of infection, as was seen in this patient.

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Medical Student Research

Medical Student Research

Roshni Kalkur

John M. Lamar, Ph.D.

Albany Medical College

The Role of TEADs1-4 in YAP/TAZ-Mediated Metastatic Melanoma

Melanoma is the deadliest form of skin cancer due to its tendency to metastasize. While the 10-year survival rate prior to metastasis is 95%, it plummets to just 24% following metastasis. To improve survival of metastatic melanoma patients, we must identify pathways that can be targeted to inhibit the growth/survival of metastatic cells. Yes-associated protein (YAP) and transcriptional co-activator with PDZ-binding motif (TAZ) are transcriptional coactivators that promote migration, invasion, primary tumor growth, and metastasis in a variety of cancers including melanoma. Previous studies demonstrate that YAP and TAZ play a causal role in tumor formation, tumor progression, and metastasis making them potential therapeutic targets. However, targeting YAP and TAZ directly may cause adverse side effects due to their diverse role in essential cellular processes. YAP and TAZ drive most pro-cancer effects through their interaction with four TEA-domain (TEAD) proteins.

Therefore, we hypothesize that targeting specific TEAD transcription factors that are upregulated in melanoma is a more refined approach to inhibit the pro-cancer effects of YAP/TAZ. First, we asked which TEADs were expressed in several metastatic melanoma cell lines using qPCR and Western blotting. Our results indicate that melanoma cell lines typically overexpress at least one of the four TEAD proteins in relation to the others. TEAD1 and TEAD2 are more often highly expressed than TEAD3 and TEAD4. Cell lines stably expressing shRNAs targeting each of the four TEADs were generated. Lysate and RNA were collected to confirm knockdown, and YAP/TAZ activity was tested using a Dual Luciferase Reporter Assay that measures YAP/TAZ transcriptional activity. The results indicate that TEAD1 knockdown significantly impairs YAP/TAZ activity. Preliminary data shows that the knockdown of other TEADs also decreases YAP/TAZ activity, but to a lesser extent than TEAD1 knockdown. Future efforts will be focused on performing in vitro migration, invasion, and proliferation assays and in vivo studies to further elucidate the therapeutic potential of TEAD knockdowns in preventing YAP/TAZ-mediated melanoma metastasis.

Jordan Mendelson

Andrew Moawad, MD; Lauren Tesoriero, DO; Marc Wilkenfeld, MD

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Determining the Efficacy of Various Governmental Interventions in Stemming the Spread of SARS-CoV-2 in Seven Locations

Introduction: Since the beginning of the Covid-19 pandemic, the efficacy of various governmental interventions in stemming the spread of SARS-CoV-2 has been the source of much public debate. Through identification of those specific governmental interventions which have been most effective in stemming the spread of SARS-CoV-2, this research has the potential to help guide future governmental responses to the Covid-19 pandemic.

Objective: To determine the efficacy of various governmental interventions in stemming the spread of SARS-CoV-2.

Methods: Data pertaining to the number of daily cases of Covid-19 between February or March and August 15th, 2020 were collected from New York, Texas, Florida, Louisiana, California, Italy, and Sweden. We plotted these datasets as epidemiological curves and superimposed various governmental interventions onto them. We also superimposed May 25th onto the domestic curves to reflect mass gatherings following the death of George Floyd and to determine their effect on the spread of SARS-CoV-2.

Results: In New York, Louisiana, Texas, Florida, and Italy, various governmental interventions appear to have had meaningful impact on the number of daily cases recorded and prevention of future outbreaks. California seemingly did not gain much benefit from these interventions. Sweden, despite no governmental intervention, experienced only a transient surge in daily cases.

Conclusions: Governmental interventions were largely effective in stemming the spread of SARS-CoV-2. Stay-at-home orders proved efficacious in decreasing daily cases quickly during ongoing outbreaks. Social distancing measures and mask mandates, meanwhile, proved to be effective in preventing subsequent outbreaks, at lesser economic and psychological costs.

Lynna Zhong

Nia-Simone Woods and Carla Boutin-Foster, MD, MPH

SUNY Downstate Medical Center

I want to feel good about myself: Motivations for Weight Loss Among Black Women Enrolled in a Pilot Weight Loss Intervention in Brooklyn, NY.

Background/Objectives: Compared to other racial and ethnic groups, Black women (59.6%) have the highest prevalence of obesity among the racial and sex groups. In Central Brooklyn, obesity affects 1 in 3 residents, and over half of the adults seen at SUNY Downstate and its affiliated sites are overweight. Understanding motivations for weight loss is paramount to tailoring interventions and reducing health disparities.

Methods: Participants who self-described as overweight or obese and who were enrolled in a pilot weight loss intervention were recruited. Individual qualitative interviews were integrated into baseline assessments of Afro-Caribbean and African American women enrolled in a pilot weight loss intervention. Participants were asked to describe their motivations for wanting to lose weight. A codebook was drafted, and in-vivo responses were independently coded into concepts and categories by two trained coders. Discrepancies were discussed until a consensus was reached, and the codebook was refined. Data was coded using Cloud Atlas.ti software.

Results: A total of 36 women were recruited, their average age was 51 +14, and BMI was 36 +5.52. Motivations for loss weight were categorized as: health-related reasons (93 quotations), appearance-related reasons (46 quotations), socioeconomic impact (4 quotations), and quality of life (36 quotations). Among this cohort of Black women, motivations for weight loss were multifactorial and emanated from a desire to improve health, self-perception, and family interactions. Participants noted obesityâ€™s association with their comorbid health conditions and identified their weight as a barrier to participating in recreational activities with their family.

Conclusions/Implications: Understanding individual motivations for weight loss has implications for tailoring future weight loss interventions and can inform patient-centered counseling in diverse, historically disadvantaged, urban patient populations such as those SUNY Downstate serves in Flatbush, Brooklyn.

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Resident / Fellow Clinical Vignette

Resident/Fellow Clinical Vignette

Sameer Acharya MBBS

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Cayuga Medical Center

A Case of Waldenstrom Macroglobulinemia Presenting with Minimal Change Disease

INTRODUCTION

Waldenstrom macroglobulinemia (WM) is a rare lymphoproliferative condition manifesting typically with 10% lymphoplasmacytic infiltrates in bone marrow and IgM monoclonal protein >1 g/dl in serum, linked genetically with somatic mutations in the myeloid differentiation primary response 88 gene. Renal manifestations of WM are very uncommon with a cumulative incidence of 5.1% in 15 years. Here, however, we present a case of an 87-year-old male with refractory edema initially thought to be from acute heart failure, then found to have minimal change disease (MCD) due to WM which progressed to acute kidney injury requiring hemodialysis, who recovered successfully after rituximab therapy.

CASE PRESENTATION

An 87-year-old male with history of hypertension presented to the emergency room with worsening anasarca. He was initially assessed to have heart failure with preserved ejection fraction. Serum creatinine was 1.87 mg/dl (baseline 1.1 mg/dl). Urinalysis showed 3+ proteinuria. He was treated with escalating doses of diuretics and was discharged on bumetanide and carvedilol.

Despite the administered diuretics, over the course of two months he experienced refractory anasarca, worsening renal function (serum creatinine climbed to 4.74 mg/dl) and proteinuria totaling 8 gm per day. He was referred to nephrology, and a renal biopsy was performed. The biopsy results showed features of MCD and atypical IgM-Kappa positive B-cell infiltrate with plasmacytoid differentiation, suggestive of secondary MCD. Atypical B-cell infiltrates prompted a hematology-oncology consultation, at which time a bone marrow biopsy was performed. The marrow biopsy results revealed lymphoplasmacytic lymphoma cell infiltration in an interstitial pattern suggestive of WM. He was treated with Prednisone 40 mg daily and four cycles of rituximab 375 mg/m2. During the course of illness, he required four weeks of hemodialysis. Gradually, his kidney function did improve: nephrosis resolved, proteinuria dropped from 8 gm/day to <0.2 g/day, and serum albumin normalized. Ultimately, the anasarca resolved as well.

DISCUSSION

MCD in children is commonly a primary condition, while in adults, it is often secondary. The atypical B cells infiltrate on kidney biopsy was the clue which led to the diagnosis of secondary MCD due to WM. Both MCD and WM respond well to rituximab, fortunately, which was the reason for the good outcome in our case.

Resident/Fellow Clinical Vignette

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Unity Hospital Internal Medicine Residency Program

LETTUCE NOT DO THIS AGAIN

Introduction

Anticholinergic toxicity seen in the ED is usually due to medication overdose. We present a case of anticholinergic toxicity secondary to accidental ingestion of Jimsonweed, Datura stramonium.

Case Description

A 35-year-old woman presented to the ED with a 1-hour history of progressive blurry vision and dizziness culminating with confusion and agitated delirium. An hour prior to onset of symptoms she had ingested some cooked greens from her garden. She subsequently had rapid progression of blurry vision and agitated delirium requiring chemical and physical restraints.

Examination demonstrated dry mucous membranes, tachycardia (HR 150) and hypertension (BP 150/94). She was noted to have warm dry skin, mydriasis, and urinary retention; the rest of her examination was not contributory.

CBC, BMP, and liver enzymes were within normal limits and her serum and urine toxicology screens were negative. EKG revealed sinus tachycardia and a prolonged QTc (HR 150, QTc 517 ms). A non-contrast head CT scan was normal. An NG tube was passed and activated charcoal administered. Samples of the greens she consumed were identified as Jimsonweed, leading to diagnosis of anticholinergic toxicity.

Treatment consisted of IV lorazepam, physostigmine and magnesium sulfate. She did not require intubation. Mental status and tachycardia improved within 12 hours and normalized within 24 hours. She was discharged home 48 hours after admission.

Discussion

This case demonstrates the value of a complete history to identify the source of a toxidrome. Anticholinergic toxicity is diagnosed when a patient presents with symptoms suggestive of antimuscarinic effects in addition to ingestion of a known anticholinergic medication. Commonly implicated drugs include antihistamines, tricyclic antidepressants, antispasmodics and mydriatics. It is important to note that plants can be a source of anticholinergic ingestion; the patient in this case accidentally ingested Jimsonweed (Datura stramonium). Consumption of any part of this flowering nightshade plant has the potential to result in severe anticholinergic toxicity and it has been used as a hallucinogenic drug of abuse. At discharge, she was counselled on the need to properly identify potentially noxious plants in her vegetable garden to prevent a recurrence of this episode.

Resident/Fellow Clinical Vignette

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Don't play with my heart: Malingering arrhythmia in a high-risk patient

Background:

Wide-complex ventricular tachycardia resulting from intentional manipulation of chest leads is an exceptionally rare, but concerning, presentation in a high-risk patient.

Case:

A 41-year old incarcerated male with a past medical history of non-ischemic CM, HOCM s/p AICD placement which was explanted (4 times total, last removed 9 months ago) due to recurrent endocarditis, recurrent atrial fibrillation status post ablation three times, and reported history of VT s/p loop recorder placement with subsequent removal due to endocarditis a month ago, was brought in to the hospital after he developed retrosternal pain associated with diaphoresis and palpitation. During his transfer to the hospital, EMS recording revealed runs of non-sustained VT.

While hospitalized, the patient has numerous such symptomatic episodes associated with wide-complex tachycardia on telemetry tracing. Episodes were not witnessed by health personnel. Multiple EKGs obtained immediately after such episodes failed to show any persistent arrhythmia.

Closer inspection of the telemetry leads showed normal waveforms, which could be marched, buried within the artificially induced non-sustained wide complex VT resembling waveforms. Patient eventually admitted to manipulating the RA and LL leads manually to induce the waveforms.

Decision-making:

Workup for endocarditis was initiated due to compatible symptomology. An echocardiogram showed moderate TR, which was also visualized 4 years ago. Multiple EKGs and Troponin levels obtained during the patient's stay were unremarkable. Blood cultures revealed no growth.

Telemetry strips were analyzed and it was deemed that the apparent wide complex tachycardia waveforms were artificially introduced artifacts. The patient was confronted with the concern and he admitted to manipulating the RA and LL leads in the 3-electrode system manually to induce the perplexing waveforms whilst feigning chest pain.

Conclusion:

This case highlights the importance of recognizing malingering as a possible differential for symptomatic wide-complex VT since the subsequent financial and therapeutic implications for the patient resulting from a misdiagnosis may be considerable.

Resident/Fellow Clinical Vignette

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Uncontrolled Type II Diabetes Management: Insulin versus Other Diabetes Medications Systematic Review and Meta-analysis of Randomized Controlled Trials

Objective: Given the lack of head-to-head systematic reviews and meta-analyses comparing GLP-1 receptor agonists, DDP-4 inhibitors, and SGLT-2 inhibitors to insulin in uncontrolled T2DM management, we conducted this systematic review and meta-analysis to compare the efficacy and safety of these medications to insulin in uncontrolled T2DM management.

Design: A systematic review and network meta-analysis.

Data sources: Cochrane database, PubMed, MEDLINE, ClinicalTrials.gov, and EMBASE were searched from January 2010 to March 2021.

Eligibility criteria: All included trials were randomized clinical trials published in English from January 2010 to January 2021. Included trials compared insulin to one or more of GLP-1 receptor agonists, DDP-4 inhibitors, and/or SGLT-2 inhibitors medications. All included trials had participants with a baseline mean HbA1c of >= 8%. Participants were 18 years or older. All included studies assessed glycemic control following at least 24 weeks of intervention.

Results: Out of 1195 identified studies, nine studies were eligible and included in the analysis (N=3757 participants). The included trials were seven GLP-1 receptor agonist trials and two DPP-4 inhibitors trials. No trials compared SGLT-2 inhibitors to insulin in patients with HbA1c of >=8%. The pooled analysis of included trials showed, in insulin naïve patients with uncontrolled T2DM, GLP-1 receptor agonists and DDP-4 inhibitors led to a significant drop in mean HbA1c (MD=0.32, 95% CI 0.04 to 0.60, P=0.02), weight in Kg (MD=2.81, 95% CI 1.86 to 3.76, P<0.00001), and SBP (MD=3.55, 95% CI 2.17 to 4.92, P<0.00001), with fewer hypoglycemia events (RR=2.16, 95% CI 1.71 to 2.73, P<0.00001) in comparison to insulin at six months. Subgroup's analysis showed GLP-1 receptor agonists not only were more effective than insulin in reducing the mean HbA1c (MD=0.57, 95% CI 0.43 to 0.72, P<0.00001), and weight (MD=0.6, 95% CI 0.46 to 0.74, P=0.0002) but also with fewer hypoglycemic events (RR=1.84, 95% CI 1.55 to 2.19, P<0.00001). Combining GLP-1 receptor agonists and TZD was associated with the most significant reduction in HbA1c (-1%, P < 0.001) compared to insulin. Compared to insulin, DPP-4 inhibitors were less effective in reducing the mean HbA1c (MD=-0.49, 95% CI -0.64 to -0.34, P<0.00001); however, they were associated with more significant weight loss (MD=2.11, 95% CI 1.02 to 3.02, P<0.00001) and fewer hypoglycemic events (RR=3.17, 95% CI 2.56 to 3.93, P<0.00001).

Conclusions: GLP-1 receptor agonists use for poorly controlled type II diabetes patients with Hb1c>=8 % was associated with a significant drop in HbA1c, fewer hypoglycemic events, and more weight loss when compared to insulin.

Resident/Fellow Clinical Vignette

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Utilization of Non-Opioid Analgesics: Assessment of Knowledge & Perception Among Internal Medicine Residents

Introduction

Per National Institute of Drug Abuse, the U.S. peaked at ~70,000 drug overdose deaths in 2017, led by synthetic narcotics. Our county, Suffolk, has the highest opioid overdose death rate in NYS. Prescription diversion and misuse is a risk factor for opioid abuse. There are few studies exploring Clinical Decision Support1 as a method to reduce opioid prescribing. A study2 of postpartum women which used a power plan linked to activity goals, rather than pain goals, demonstrated reduced opioid use. We collaborated with our analgesic prescribing task force to create a multimodal power plan in our EHR system to facilitate inpatient non-opioid analgesic prescribing.

Methods

The powerplan contains cryo/thermotherapy, topical, oral and IV medications categorized by mode of action or type of pain, and consult menu for support services. We surveyed, then taught a 30 minute didactic3 lecture on safe analgesic prescribing practices and introduction to the powerplan, to ~84 residents in the Internal Medicine Residency Program.

Results

42 residents (50%) completed the 19-question anonymous electronic survey. 54% asserted they would be extremely likely to prescribe non-opioid analgesics for severe acute pain. The largest reasons for not educating patients about alternatives is that they would reject the medication, and secondly that there is not enough time. Lack of alternatives and poor provider knowledge were reported as lesser reasons. At least 70% agreed that a power plan would increase their prescribing of non-opioid alternatives. 64% believe that oral Tylenol and NSAIDs are underutilized. 68% believe that patients are unaware of, forget, or are unable to ask for their PRN pain meds frequently or daily. 25% have rarely or never prescribed topical analgesics. Lidocaine and fentanyl were the most commonly prescribed topical agents, with methyl salicylate accounting for only 1%. Ten percent believe that topical NSAIDs are contraindicated in AKI or CKD. 60% over-estimated the amount of topical NSAID that is absorbed into the bloodstream. 42% incorrectly believed that oral NSAID use is contraindicated in anuric ESRD patients. 62% correctly selected that topical NSAIDs are the most effective topical analgesic for muscular pain. 29% correctly selected topical lidocaine as the most effective topical analgesic for superficial pain (including herpes zoster). 12% felt that Prescription Drug Monitoring Program (NYS I-STOP) queries were always documented. 50% felt that patient education was rarely documented upon discharge regarding opioid risks, benefits, safety and disposal. 40% felt that the pain follow-up plan was rarely documented upon discharge.

Conclusion

Most of our resident physicians agreed that a power plan would increase their prescribing of non-opioids. Future studies should assess the effects of resident education and utilization of the power plan on analgesic knowledge, prescribing habits, and pain outcomes.

Resident/Fellow Clinical Vignette

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NYU Internal Medicine

Profound hypercalcemia and leukocytosis in the setting of squamous cell carcinoma

Introduction

Squamous cell carcinomas are the most common malignancy that drive humoral hypercalcemia of malignancy, with parathyroid hormone-related peptide (PTHrP) as the likely causative agent. Volume expansion is the first therapeutic step, followed by calcitonin and bisphosphonates. Denosumab can be used off-label for patients who do not respond to bisphosphonates, as presented in this case. This case also illustrates the potential for profound and persistent steroid-induced leukocytosis in patients on chronic steroid therapy.

Case Presentation

A 58 year old woman with a history of tobacco use presented in March 2019 with chest CT imaging demonstrating a right middle lobe lung mass. Biopsy demonstrated squamous cell carcinoma (SCC) with 100% PDL1 positivity, and she was started on immunotherapy with the anti-PD-1 inhibitor pembrolizumab.

In April 2020, she developed skin rash that was attributed to the pembrolizumab and she was started on high-dose prednisone 60mg daily in June 2020. WBC went from 17.2 on 6/4/2020 to 36.2 on 6/25/2020 to 61.0 on 7/24/2020. She developed bilateral lower extremity weakness and was admitted in August 2020. She had developed Cushing's syndrome and myopathy so a steroid taper was initiated. Despite tapering to 5mg prednisone, WBC was 83.2 on discharge on 8/26/2020. Infectious workup was negative, and evaluation for new malignancy with flow cytometry was also negative. The etiology of the leukocytosis was deemed to be most likely steroid-induced.

During that same admission, she was noted to have asymptomatic hypercalcemia to a peak of 15.8 on 8/11, corrected for hypoalbuminemia (1.86 ionized). PTHrP was markedly elevated at 36.7, concerning for metastasis of SCC. Bone scan did not demonstrate new lesions. IV fluids and calcitonin were administered with minimal improvement. Pamidronate initially produced an improvement, but calcium began to uptrend in the subsequent week to despite a second dose of pamidronate. After interdisciplinary discussion, denosumab was administered with good response.

Discussion

This clinical vignette illustrates two important learning points. Firstly, it demonstrates the potential of denosumab to correct hypercalcemia of malignancy in cases of limited response to bisphosphonates. The most common causes of hypercalcemia in the setting of malignancy include humoral hypercalcemia of malignancy mediated by parathyroid hormone-related peptide, osteolytic cytokine production, and excess 1,25-dihydroxy vitamin D production. Denosumab is a monoclonal antibody to the receptor activator of nuclear factor ÎºB-ligand (RANKL), that can be used for hypercalcemia in cases refractory to bisphosphonates.

Secondly, this case shows the profound leukocytosis that can be caused by prolonged prednisone administration and its persistence despite steroid taper. Limited data are available on when this leukocytosis resolves after steroid discontinuation, but it typically reaches maximal value after two weeks of therapy and is dose-dependent. These general patterns were not reflected in this patient's case.

Resident/Fellow Clinical Vignette

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NYU Internal Medicine

ITP in the setting of quiescent ulcerative colitis

Introduction

Inflammatory bowel diseases, namely Crohn's disease and ulcerative colitis (UC), are systemic illnesses with many extraintestinal symptoms. Immune thrombocytopenia (ITP) has been rarely associated with inflammatory bowel diseases, and typically occurs in the setting of a flare. Here we present a unique case of severe ITP in the setting of quiescent UC.

Case Presentation

A 56 year old man with UC not on treatment presented from clinic after routine blood work showed a platelet count of 4 x 109 L from a baseline of 190 x 109 L one year prior. He denied any oral or rectal bleeding. He noted that when his ulcerative colitis flares he has hematochezia, but the last instance was 4 years prior. He had self-discontinued mesalamine long before the admission.

Repeat labs on admission confirmed the severely low platelet count of 4 x 109 L, with a normal hemoglobin and WBC count. ADAMSTS13 activity was within normal limits, LDH was elevated at 721 U/L (normal 100-250 IU/L), HIV was negative, Hepatitis C antibody was nonreactive, and rheumatoid factor and ANA serologies were negative. He had no recent heparin or antibiotic exposures. A CT abdomen/pelvis showed no splenomegaly and no adenopathy to raise concern for lymphoma. Hematology was consulted and suspected ITP in the context of UC, and he was started on dexamethasone and IVIG 1 g/kg/day for 2 days. His platelet count improved to 51 and he was discharged home with outpatient follow-up and resumed mesalamine treatment.

Discussion

The association between ITP and UC is reported to have an estimated prevalence of 0.5%. This patient case thus reflects a rare but important complication of UC. In ITP, thrombocytopenia results both from immune-mediated platelet destruction due to autoantibodies and decreased platelet production due to cross-reactivity of antiplatelet antibodies with megakaryocytes. Eighty percent of ITP cases are considered primary, without an obvious initiating or underlying cause. Twenty percent of ITP cases are secondary to an underlying disease or drug exposure, including autoimmune disease (as in this case), infection (eg, hepatitis C, HIV, and Helicobacter pylori), malignancy, or medications (eg, heparin-induced thrombocytopenia and antibiotics). ITP is a diagnosis of exclusion, and initial workup includes medication review, peripheral blood smear, testing for HIV and hepatitis C, evaluation for splenomegaly, and consideration of bone marrow biopsy, quantitative Ig levels, and H pylori testing (with the latter two being weak recommendations). There is insufficient evidence to support routine testing for antiplatelet antibodies, antiphospholipid antibodies, antinuclear antibodies, antithyroid antibodies and thyroid function testing, thrombopoietin levels, serum complement, or acute and persistent infections other than hepatitis B, hepatitis C, and HIV.

Resident/Fellow Clinical Vignette

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Leiomyosarcoma Manifesting as Heart Failure

Leiomyosarcoma (LMS) is a tumor of smooth muscle tissue that arises from the uterine wall and has poor prognosis. Treatment of leiomyosarcoma is usually guided by histology and staging, but even with intervention, due to the aggressive course, many patients do not survive. Early intervention can abate symptoms and increase the odds of survival.

A 68-year old African American female presented to her primary care physician for fatigue, shortness of breath, and dizziness. Nine months prior to her presenting symptoms, she was diagnosed with a right common femoral vein DVT that extended to the popliteal vein. Due to her current symptoms she underwent an outpatient echocardiogram where she was found to have a right atrial mass measuring 44x31mm that was filling the entire atrium and obstructing blood flow to the right ventricle. She was then sent to the emergency room for urgent evaluation.

On exam, patient had a large palpable mass in the periumbilical region. CT was found to have extensive metastatic disease detailed as follows: large retroperitoneal tumor invading the IVC and extending into the right atrium which was associated with tumor emboli within the right lower lobe of the pulmonary arterial branches, hepatic vein thrombus, in addition to pulmonary and hepatic masses, and right hydronephrosis. She underwent Interventional radiology biopsy of the abdominal mass and was diagnosed with LMS. It was determined that she would not be a candidate for surgery of the tumor invading her right atrium as there was extensive metastatic disease. She was then referred to the oncology team where she underwent two cycles of Doxorubicin. Four weeks after her second cycle, she began to have worsening lower extremity edema and shortness of breath, unresponsive to Lasix treatment and was admitted for further evaluation. She was found to be in acute renal failure requiring nephrostomy tube. The family declined further intervention due to decline in clinical status. She became encephalopathic and the decision was made to provide comfort care. Two weeks after admission to the hospital, she died.

LMS typically has an asymptomatic presentation and may be incidentally discovered, unless there is mass effect impacting an organ. LMS has three subtypes: somatic tissue, cutaneous/subcutaneous, or vascular origin. It is usually retroperitoneal and can arise from the Inferior Vena Cava, as it is the most common tumor of the IVC, or any small vessel with unilateral or bilateral lower extremity swelling. Once in the IVC, the tumor can grow into the heart causing heart failure. Treatment is guided based on location and stage, targeted with debulking surgery, chemotherapy, radiation, or hormonal therapy. Thorough evaluation of a post-menopausal woman presenting with signs/symptoms of potential underlying malignancy including imaging may decrease morbidity and mortality.

Resident/Fellow Clinical Vignette

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INFECTIOUS MONONUCLEOSIS PRESENTING AS SUSPECTED APPENDICITIS

INTRODUCTION

Acute appendicitis is one of the most common abdominal emergencies and is frequently diagnosed in adolescents and young adults. However, when this diagnosis is questionable and does not fully explain a patient's symptoms, other etiologies should be sought. We describe a case of a young transgender female presenting with predominantly gastrointestinal symptoms, found to have underlying infectious mononucleosis.

CASE DESCRIPTION

A 21-year-old transgender female (on estrogen therapy), with asthma, ADHD, and depression presented with nausea, vomiting, and generalized abdominal pain and dyspnea for approximately 4 days. Further history revealed the symptoms first began with a sensation of postnasal drip, followed by intermittent fevers, chills, sweats, and generalized malaise. The patient denied any recent travel or sick contacts. Vitals were all within normal limits on admission. Exam was notable for mild guarding in all 4 quadrants of the abdomen, which was otherwise nondistended with no rebound tenderness, as well as scattered anterior cervical lymphadenopathy. Labs showed WBC 6.9, Hgb 12.0, platelets 156, AST 172, ALT 310, Tbili 1.0, and alkaline phosphatase 332. HIV screen, UA, urine toxicology, and alcohol level were all unremarkable. CT chest/abdomen/pelvis showed no pulmonary embolism but noted a mildly dilated appendix concerning for possible early appendicitis, as well as mildly enlarged mesenteric nodes in the right lower quadrant of the abdomen, multiple mildly enlarged perirectal nodes, and splenomegaly. Surgery was consulted for concern for appendicitis, and the patient was briefly on IV ceftriaxone and metronidazole for empiric coverage. Further workup revealed a positive heterophile antibody test, and the patient was diagnosed with infectious mononucleosis. Symptoms improved rapidly by the following day with supportive therapy alone; the patient did not require any surgical intervention and was discharged home with plan for outpatient primary care follow up.

DISCUSSION

The patient's presenting symptoms and imaging were initially most concerning for acute appendicitis. However, the initial respiratory symptoms and generalized malaise combined with cervical lymphadenopathy and enlarged spleen were early clues pointing towards infectious mononucleosis. While acute appendicitis has been noted in infectious mononucleosis, this is an exceedingly rare complication, and appendicitis alone did not fully explain the rest of the patient's symptoms, exam, labs, and imaging. Maintaining a broad differential early on to help rule out common diagnoses can hasten a diagnosis while minimizing unnecessary interventions, complications, and prolonged hospital stays.

Resident/Fellow Clinical Vignette

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A rare case of Evans Syndrome following following first dose of COVID Moderna Vaccine

Introduction:

The Moderna Vaccine, an mRNA-1273 COVID-19 vaccine, is a lipid-nanoparticle encapsulated mRNA vaccine developed with advanced technology (1) which in clinical trials showed a 94.1% efficacy at preventing Covid-19 illness and only transient local and systemic reactions (2). Following an EUA there has been more widespread vaccinations and we now report a unique case of a young woman with immune mediated thrombocytopenia and Coombs positive hemolytic anemia which was diagnosed as secondary Evans syndrome, within three weeks of her first Moderna vaccine dose.

Case presentation:

A 24-year-old female with no significant past medical history presented to the ED with lower extremity petechiae and ecchymoses first noted 3 days after her first dose of Moderna vaccine 3 weeks prior. She denied any recent infection, sick contact or use of any new medications and was hemodynamically stable. Physical exam was otherwise unremarkable. Her labs were significant for hemoglobin of 11.5 with normal MCV (no urinary/GI bleeding per patient), corrected reticulocyte count of 1.2%, platelets 8K, WBC 3.8. Further lab work showed lactate dehydrogenase (LDH) of 467, haptoglobin <20, positive Coombs test (warm agglutinin IgG) and normal LFTs/INR/PT and PTT. Peripheral smear showed medium sized platelets with no schistocytes. Hepatitis and Covid-19 PCR was negative. Initial diagnosis of immune thrombocytopenic purpura and autoimmune hemolytic anemia was made. Rheumatological workup revealed positive ANA and anti dsDNA antibodies. She improved remarkably following the administration of oral prednisone (1mg/kg) and intravenous immunoglobulins. Upon discharge her platelets were 69,000 and increased to 247,000 after 2 months.

Discussion:

Evans syndrome is an autoimmune, extremely rare disorder characterized by either simultaneous or successive development of autoimmune hemolytic anemia and immune thrombocytopenia and/or immune neutropenia in primary (idiopathic) or secondary (associated with an underlying disorder) (3), (4). After inoculation of the vaccine, mRNA is transcribed by ribosomes into SARS-COV2 spike (S) glycoprotein and the spiked protein are displayed by the antigen presenting cells leading to T and B-cells mediated immunity(5). As of February 2021, greater than twenty case reports of patients with thrombocytopenia following vaccination have been reported (6). Most of the cases regarding Covid-vaccine induced thrombocytopenia are seen within 6 weeks. There have been cases of Covid-19 leading to Evans syndrome (7), but to our knowledge this is the first case of Covid-vaccine induced Secondary Evans syndrome. Use of steroids and intravenous immunoglobulin remain the mainstay of treatment.

Conclusion:

This manuscript highlights the importance of immune mediated response to Covid vaccine resulting in autoimmunity that was timely diagnosed and promptly managed with good outcomes.

Resident/Fellow Clinical Vignette

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ANTI-MAG ANTIBODY ASSOCIATED POLYNEUROPATHY TREATED EFFECTIVELY WITH IBRUTINIB

Introduction:

Polyneuropathy associated with anti-myelin associated glycoprotein (MAG) antibodies can lead to severe ataxia and sensory deficits. The current standard of treatment with Rituximab has been effective in only 50% of patients, and often loses its effectiveness with subsequent infusions. We present the case of a patient with peripheral neuropathy with elevated anti-MAG antibody levels, effectively treated with Ibrutinib.

Case presentation:
72 year-old-male evaluated for untreated non-hodgkin's lymphoma diagnosed 8 years prior. He was diagnosed with lymphoproliferative disorder (LPD) after a bone marrow biopsy. He was also found to have high levels of monoclonal IgM. During this presentation, he endorsed worsening neuropathy in his feet and an unsteady gait. Complete blood count, metabolic panel, vitamin B12 and folate were within normal range. Serum IgM level was >3300 (40-230 mg/dl). Bone marrow biopsy and aspirate showed 46% lymphocytes and 4%plasma cells, positive: CD19/20/25 and negative: CD5/10/23, BCL6 and Cyclin D1, favoring low grade LPD/marginal zone lymphoma (MZL). Full body computed tomography (CT) scan was negative for adenopathy and splenomegaly. Anti-MAG antibody levels were 1:102,400 (Ref: â‰¤1:1600 mg/dL). Although low grade LPD/MZL does not require treatment, considering his debilitating neuropathy it was decided to initiate treatment with oral Ibrutinib. Over 3 months he reported significant overall improvement and his IgM levels decreased to 1389 mg/dl and anti-MAG antibody levels are still pending.

Discussion:
Anti-myelin associated glycoprotein (MAG) antibodies cause neuropathy secondary to sensorimotor demyelination leading to progressively worsening and disabling ataxia and sensory deficits. These antibodies are often associated with IgM monoclonal gammopathy, monoclonal gammopathy of undetermined significance (MGUS) and Waldenstrom macroglobulinemia. The current standard of treatment is Rituximab which suppresses antibody production and induces immunoregulatory T-cells, however, the treatment course is prolonged requiring frequent re-dosing and only effective in 50% of patients.

In this case we used Ibrutinib, a potent Bruton kinase inhibitor, causing downregulation of the B-cell receptor pathway leading to decreased B-cell growth, proliferation and survival. Ibrutinib is widely used in the management of B-cell lymphomas and has also shown effectiveness against anti-MAG antibody associated neuropathy in previous smaller studies. Our patient reported significant improvement in symptoms and decrease in IgM levels following treatment.

Conclusion:
Ibrutinib has been used to treat certain B-cell lymphomas, showing promising results in the symptomatic treatment of anti-MAG neuropathy in previous smaller studies. Although the correlation between anti-MAG antibody titers and the degree of neuropathy and IgM levels is not proven, the concurrent improvement of symptoms and lowering of IgM titers is certainly reassuring, evidencing the need for large-scale studies to further evaluate this association and compare Ibrutinib with the standard of treatment Rituximab.

Resident/Fellow Clinical Vignette

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Multisystem Inflammatory Syndrome in Children Mimicking Gastrointestinal Disease: Two Case Reports of Well-defined Terminal Ileitis in Adolescent Patients Diagnosed with MIS-C

Introduction

Acute terminal ileitis is often associated with appendicitis or inflammatory bowel disease. It has also been reported as a finding in children with Multisystem Inflammatory Syndrome in Children (MIS-C). MIS-C is diagnosed with recent exposure to SARS-CoV-2 who are less than 21 years of age, have elevated inflammatory markers, and require hospitalization for symptoms involving at least two organ systems. We report two 18-year-old males with terminal ileitis diagnosed of MIS-C.

Case Description/Methods

Patient A without past medical history presented with fever, nausea, emesis, and abdominal pain for 2 days. A family member was recently diagnosed with COVID-19. Vital signs showed tachycardia. Labs showed increased inflammatory markers, troponin 0.164 ng/mL, proBNP 26,562 pg/mL. Exam showed right lower quadrant (RLQ) tenderness. Abdominal CT showed terminal ileal wall thickening with surrounding fat stranding. A SARS-CoV-2 PCR was negative. GI was consulted in concern for Crohn's Disease. Other common GI infectious and rheumatologic labs were negative. Empiric antibiotics initiated. Dyspnea worsened. An echocardiogram showed acute heart failure. Pulmonary embolism was ruled out. SARS-CoV-2 serology was positive. Steroids were initiated for MIS-C. Transferred to PICU at a tertiary care center. Progressed to respiratory failure requiring intubation and Pressors. IV IG, Anakinra, and steroid were given. Extubated on day 3 of PICU and was discharged on day 8 of the hospitalization.

Patient B with no past medical history working as a real estate agent presented the same day as Patient A with similar symptoms. Vital signs, exams, imaging, and labs showed similar findings. Patient B did not have heart failure. A SARS-CoV-2 PCR was negative, but serology was positive. GI service was consulted. Similar workups were done with the same findings. Dyspnea worsened. Transfer to the same PICU. Same treatments were provided. Discharged on day 8.

Discussion

Delay in MIS-C diagnosis and treatment may lead to rapid decompensation. Our aim is to add to the body of literature to increase awareness of terminal ileitis as a finding associated with MIS-C. Pediatric COVID-19 patients present with fewer GI symptoms. In MIS-C, up to 92% of patients report GI symptoms. We suspect that terminal ileitis as a manifestation of MIS-C is due to the higher density of ileal Peyer's patches. Its detection should alert clinicians for early Intensive Care involvement while ruling out common etiologies of terminal ileitis.

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Carotid Sinus Syndrome presenting with Mobitz Type 1 AV Block and 2:1 Conduction in a patient with Head and Neck cancerâ€¨

Carotid sinus syndrome (CSS) is often the cause of potentially fatal presentations such as bradycardia, presyncope or syncope. Here, we describe a patient with squamous cell carcinoma of the right parotid gland who developed a unique presentation of CSS and second degree Mobitz type 1 heart block. â€¨â€¨

An 83 year-old male with medical history most significant for first degree atrioventricular (AV) block and squamous cell carcinoma of the right parotid gland post chemo and radiation therapy presented with lightheadedness and poor oral intake for 3 weeks. He denied nausea, diaphoresis, shortness or breath, chest pain, dizziness or hearing loss. He attributed his poor oral intake to loss of taste from radiation therapy. On examination, he was afebrile and hemodynamically stable with no abnormal heart sounds. Hyperpigmented skin with brown drainage and several areas of partial thickness tissue loss was visible on the right side of his neck. These skin changes were attributed to his radiation treatments.

â€¨â€¨Electrocardiogram showed a rate of 20 with Mobitz Type 1 AV block and 2:1 AV conduction (Wenckebach phenomenon) Echocardiogram showed an EF of 55-60%, indeterminate diastolic function and no regional wall motion abnormalities. All electrolytes were within normal limits. Troponins were negative. TSH was low to 0.35 however free T3 and free T4 were normal. The patient was not administered medications affecting his conduction system. He denied smoking history. As other etiologies were ruled out, his bradycardia was attributed to an exaggeration of the baroreceptor reflex.â€¨â€¨

The patient’s treatment plan was focused on providing adequate nutrition through PEG tube placement. Repeat EKG did not show evidence of the Mobitz Type 1 AV Block. Patient remained asymptomatic with no widening of the QRS complexes and was not a candidate for cardiac pacing. â€¨â€¨

Most patients with carotid sinus hypersensitivity(CSH) are asymptomatic however occasionally the baroreceptor reflex arc may transmit a strong efferent vagal discharge resulting in marked sinus bradycardia, sinus arrest or impaired AV conduction. In patients with head and neck cancer, it may be caused by mechanical pressure to the carotid sinus or post-irradiation damage to the receptor milieu. If these reflexes produce symptoms such as lightheadedness, dizziness or syncope the condition is termed as CSS. There is not much documented literature on head and neck cancer manifesting as CSS and the incidence of AV block in CSS is even lower (4.9 to 16.7%) as reported in many case series. Therefore we conclude that unexplained abrupt episodes of bradycardia and intermittent heart block should alert the physician of the possibility of carotid sinus compression by a neck mass in patients with head and neck cancer and should be explored further.

Resident/Fellow Clinical Vignette

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Neurosyphilis presenting as Guillan Barre Syndrome

Neurosyphilis characterizes a broad presentation of symptoms that may occur at any time following inoculation with Treponema pallidum. Unlike the more defined early, late, and latent stages, it does not follow a strict pattern. Our patient, a 51 year old Male, with a past medical history of HTN, HLD, DM, Raynaud's and Covid-19 (4/2020), presented to the ER complaining of 3 weeks bilateral progressive numbness and weakness beginning in his lower extremities, and ascending towards his pelvis, with recent involvement of his upper extremities and the appearance of a maculopapular rash on his flanks. No history of recent personality changes, or ocular complaints. Of note, Patient received his 2nd Moderna vaccine 1 week prior to the appearance of symptoms, and 4 weeks prior to admission. Prior to his admission, he had sought treatment elsewhere and was prescribed steroids and muscle relaxants without effect. Additionally, a recent Lumbar MRI was negative. Clinical findings of ascending weakness subsequent to vaccination prompted an initial clinical diagnosis of Guillain Barre, and the patient was transferred to the ICU for respiratory monitoring. Patient afebrile. RPR was taken in light of the maculopapular rash, HIV serology ordered, Infectious Disease, Neurology and PM&R were consulted. Lumbar Puncture, EMG, MRI Brain/Spine obtained. Prompt treatment with IVIG was started, as well as Doxycycline and Ceftriaxone with marked improvement further suggesting a diagnosis of GBS. MRI brain/spine showed no acute change. RPR returned reactive 1:64, and CSF VDRL was reactive 1:2, with negative HIV. Additional LP findings showed glucose 120, total protein 148, WBC 73, Lymphocytes 86, Neutrophils 9, Monocytes 5, RBC 9. EMG showed asymmetric demyelinating > Axonal sensorimotor polyneuropathy without myopathy. CSF Leukocytosis in conjunction with the serum RPR and positive CSF VDRL confirmed the diagnosis of neurosyphilis. IV Penicillin G was started for treatment of Neurosyphilis, and IVIG was continued. Patient's physical and neurologic symptoms continued to improve and he was discharged from the ICU after receiving 5 doses of IVIG and receiving IV Penicillin G with further outpatient completion of a total 14 days high dose PCN G. Syphilis is often referred to as the great mimicker, and this case is no different. A thorough history & physical and expedient laboratory work made this diagnosis of Neurosyphilis possible, especially in light of early symptoms resemblant of Guillain Barre Syndrome. As is always the case, it is of the utmost importance to be methodical in your approach and cast a wide net when creating a differential list.

Resident/Fellow Clinical Vignette

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POTENTIAL NOVEL LIVER INJURY DEVELOPED FOURTEEN WEEKS AFTER RECOVERY FROM CRITICAL COURSE OF COVID-19

Liver injury is a common condition caused by acute COVID-19. In this case, liver injury developed a few months after recovery from critical course of COVID-19.

A 40-year-old female with alcohol use disorder was hospitalized for acute hypoxemic respiratory failure due to COVID-19 pneumonia. Her condition was severe and required intubation, hemodialysis, and vasopressor support. On admission, the patient's liver enzymes were normal except mildly elevated alkaline phosphate (ALP). After intubation, the labs worsened to ALP 336, aspartate aminotransferase (AST) 8860, alanine aminotransferase (ALT) 2368 and bilirubin 0.7. A liver ultrasound showed distended gallbladder with stones and sludge without dilation of the common bile duct. She was extubated on day 30. On day 49, the patient had a cardiac arrest, and return of spontaneous circulation was reached in five minutes. She received chlorpromazine and valproic acid for fourteen days each during the hospital stay. She was discharged on day 68 with tracheostomy collar with ALP 1144, AST 75, ALT 72 and bilirubin 0.9.

Fourteen weeks later, the patient was sent to the hospital for two weeks of jaundice. She had occasional vomiting, dark urine, light grayish-greenish stool, dysuria and fever, for which she was prescribed nitrofurantoin. On admission, the labs showed elevated total and direct bilirubin 6.9 and 6.2 respectively, ALP >2330, AST 168, ALT 102, and gamma-glutamyl transferase (GGT) 1028. Repeat liver ultrasound showed mild gallbladder wall thickening, and an enlarged heterogeneous liver with new multiple hyper-echoic foci. Magnetic resonance cholangiopancreatography showed hepatomegaly and indeterminate left hepatic lesions without cholelithiasis, choledocholithiasis, or biliary ductal dilation.

Anti-mitochondrial antibodies and anti-liver-kidney microsomal antibody were negative, anti-smooth muscle antibodies was mildly positive. Hepatitis panel, HIV, HSV PCR, Epstein-Barr Virus IgM were negative. CT-guided liver biopsy showed moderate-to-severe pericentral bile-stained xanthomatous macrophages and occasional neutrophils without the feature of primary biliary cholangitis, primary sclerosing cholangitis or extra-hepatic biliary obstruction. The report noted there is a possibility of post-covid condition. The patient was empirically started on ursodiol, and liver enzymes slowly trended down.

This case presentation suggests a potential novel post-COVID-19 liver injury. Drug-induced liver injury (DILI) is the main differential diagnosis, however this case doesn't fit the typical presentations. DILI from nitrofurantoin usually presents with fever and rash, and resolves after the cessation of medication. Cholorpromazine commonly causes DILI within five weeks and an elevation of liver enzymes above three times the upper normal limit is uncommon. DILI from valproic acid typically doesn't induce GGT elevation. Given that this case differs from typical DILI, it is important to consider COVID-19 as a potential cause of long-term liver injury.

Resident/Fellow Clinical Vignette

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Use of digital health tools for health promotion among individuals with rheumatologic diseases in the United States

Background: Rheumatic diseases are chronic and progressive. They cause damage to the musculoskeletal system, leading to disability which can significantly impact quality of life. Positive effects of physical activity, physical training and dietary modification have been scientifically proven for many chronic diseases and should be incorporated into the treatment of inflammatory rheumatic diseases. Scarce data exist on the use of digital health tools (DHTs) for health promotion in rheumatologic diseases. Here, we explored the predictors of DHT ownership and usage among individuals with rheumatic disease.

Methods: We utilized patient data from the Health Information National Trends Survey fifth edition (HINTS 5) cycles 1 (collected 01/25/2017 to 05/05/2017) and 2 (collected 01/26/2018 to 05/02/2018). We categorized the survey respondents into two groups: rheumatologic disease (RD) and no rheumatologic disease (NRD) based on self-report in the survey.

We compared the groups based on sociodemographic factors, ownership of a DHT (smartphone, tablet or health application), and usage of DHTs (tracking health, making health decisions, or discussing with health providers). Additionally, the RD group was categorized into users of health applications and non-users of health applications. These groups were compared based on sociodemographic characteristics and adoption of positive health behaviors (smoking cessation, meeting diet and exercise guidelines). Chi-square and logistic regression were used for analysis.

Results: We identified 6,657 individuals, of which 1,493 (22.4%) reported prior diagnosis of a rheumatologic disease. Compared to the NRD group, a greater proportion of the RD group were female (60.9% vs 48.0%), White (70.3% vs 63.8%), 65 years or older (41.8% vs 12.2%), had an educational level below college (53.7% vs 36.5%), had annual household income <$20,000 (27.7% vs 14.5%), and had rural residence (18.3% vs 12.4%). The RD group was more likely to use smartphones or tablets to discuss with health providers [OR 1.34, 95% CI (1.08, 1.67); p=0.010]. There was no difference in health behavior change by health application use status within the RD group (all p>0.05). In the RD group, multivariable logistic regression revealed that males [OR 0.56, 95% CI (0.40, 0.79); p=0.001] and older individuals [OR 0.48, 95% CI (0.34, 0.68); p<0.0001] were less likely to own mobile health applications, while individuals with annual household income >$75,000 [OR 2.56, 95% CI (1.34, 4.89); p=0.005] were more likely to own mobile health applications.

Conclusions: Our study indicates that, in a nationally representative sample, individuals with rheumatic disease were more likely to use digital applications. Gender, age, and income level were identified as significant, independent predictors of ownership and usage of digital health applications. With increasing costs of treating chronic inflammatory rheumatic conditions, imminent physician shortages, and increasing awareness of digital presence in our patients, DHTs may help bridge gaps in health care delivery and accessibility.

Resident/Fellow Clinical Vignette

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LABORATORY CLUES OF COEXISTENT COAGULOPATHIES: A PATIENT WITH LIVER CIRRHOSIS AND ACQUIRED HEMOPHILIA A

Introduction:
Liver cirrhosis is a known cause of coagulopathy. This can pose challenges to diagnose other concurrent coagulation defects.

Case:
44-year-old female with past medical history of alcoholic cirrhosis presented to the hospital for abdominal distention. She underwent paracentesis which was complicated by prolonged bleeding from the needle site, extensive diffuse ecchymosis, and bleeding from multiple intravenous sites. Pertinent laboratory results showed partial thromboplastin time (PTT) 115 seconds, prothrombin time (PT)/international normalized ratio (INR) 20.6/2.17, D-dimer 13.33 ug/mL, and fibrinogen 128 mg/dL. The mixing study showed partial correction of PTT. Factor assays showed Factor V 37 U/dL, factor VII 19 U/dL and Factor VIII (FVIII) <1%. These findings were attributed to disseminated intravascular coagulation and liver dysfunction. Partial correction on mixing study was attributed to elevated D-dimer. She was supported with transfusions and subsequently discharged.

Two days after discharge, she presented with headaches, nausea, and vomiting. Physical examination was unremarkable except for extensive ecchymosis. Laboratory results were unchanged from before. Computed tomography of the head showed multiple subdural hematomas of varying age without midline shift. She was supported with fresh frozen plasma, cryoprecipitate, and packed red cells transfusions. This time due to worsening bleeding, undetectable FVIII levels, and mixing study results, concern for acquired FVIII inhibitor was raised. Bethesda assay for FVIII inhibitor came positive at 17.2 BU. She received FVIII inhibitor bypass activity (FEIBA), prednisone, and rituximab. Unfortunately, her hospital course was complicated by hepatic encephalopathy, hepato-renal syndrome, and unresolving coagulopathy. Subsequently, she was transitioned to comfort care.

Discussion:
PT and INR observed in cirrhotic patients often occurs with a normal or near-normal PTT. This is usually due to FVIII upregulation. VWF is also increased.

Acquired Hemophilia A is characterized by the development of autoantibodies against FVIII. This condition is rare and has potential to cause life-threatening bleeding with a mortality rate of up to 20%. Therefore, it is important to be familiar with the diagnosis. In about half of the cases, it is associated with an underlying condition, mainly post-partum, autoimmune disease, malignancies, and drug use. The diagnosis is suspected with prolonged PTT, failure to correct PTT in a 1:1 mixing study, and low FVIII. High FVIII inhibitor on Bethesda assay provide confirmation.

The treatment goal in acquired FVIII inhibitor involves bleeding control and elimination of the inhibitor. In severe bleeding, FEIBA or recombinant FVIII can be used. The inhibitor is eliminated with steroids and immunosuppressive agents (such as cyclophosphamide or rituximab).

Conclusion: Although prolonged PTT can be seen in patients with severe liver disease. It is usually normal or near normal. When present, we need to rule out other coagulopathies.

Resident/Fellow Clinical Vignette

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MYOPERICARDITIS MASQUERADING ANGINA IN A PATIENT WITH SEVERE LEFT MAIN CORONARY ARTERY DISEASE

Introduction:
Left main coronary artery disease (LMCAD) has a higher mortality than other types of coronary artery disease (CAD) and should not be missed. Myopericarditis is a clinical diagnosis. Its main differential is acute coronary syndrome (ACS), however cardiac catheterization is not always required. We present a case of myopericarditis masking the diagnosis and delaying the management of LMCAD.

Case Presentation:
85-year-old male previous smoker with history of peripheral artery disease, carotid artery stenosis, dyslipidemia, and family history of CAD presented with sharp chest pain which radiated to the back and shoulders, aggravated by inspiration and alleviated by leaning forward, without relation to exercise and non-reproducible on palpation. Physical exam was remarkable for pericardial friction rub, tachycardia, irregular heart rate, and new oxygen requirement.
Electrocardiogram (ECG) showed new-onset atrial fibrillation (AF) with rapid ventricular response and diffuse ST segment elevations in anterolateral and septal leads. Intravenous diltiazem, metoprolol, and apixaban were started.
Troponin T level peaked at 0.22 ng/mL. Echocardiogram was unremarkable without pericardial effusion. Myopericarditis was diagnosed. He was discharged on colchicine, ibuprofen, carvedilol, and apixaban.

One month later, the chest pain recurred, however was now associated with pedal edema and dyspnea on exertion. Echocardiogram now revealed grade II diastolic dysfunction. His symptoms were attributed to diastolic heart failure and recurrence of myopericarditis. Furosemide and Lisinopril were added to his medication regimen.

On follow-up, he endorsed dyspnea and chest pain now only on exertion, no longer alleviated by leaning forward. Nuclear stress test was done and significant for marked inferolateral ischemia. Subsequent cardiac catheterization showed LMCAD, and he underwent coronary artery bypass grafting.

Discussion:
Myopericarditis typically presents as pleuritic chest pain in a patient with two or more of the following: pericardial friction rub, characteristic ECG changes or pericardial effusion. With troponin elevation and without new ventricular function impairment on imaging.

Complications, though rare, include: recurrence 13%, residual left ventricular dysfunction 3.5%, and cardiac tamponade 0.9%. ACS is the main differential diagnosis of myopericarditis. In our case, the ECG and chest pain characteristics were highly suggestive of myopericarditis, thus further workup was not pursued.

CAD is present in 35% of patients with pericarditis. In myopericarditis, dyspnea is usually associated with pericardial effusion. In our case, it was likely an anginal equivalent as no effusion was seen on echocardiogram. Dyspnea conveys worse prognosis even in otherwise asymptomatic patients and should prompt special consideration.Given this patient's cardiac risk factors, he should have been initially evaluated with cardiac catheterization, as he was later found to have severe LMCAD. Ischemic workup is vital in patients with myopericarditis who possess CAD risk factors.

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HYPONATREMIA IN AN ALCOHOLIC? IMPENDING OSMOTIC DEMYELINATION SYNDROME

Introduction

Osmotic demyelination syndrome (ODS) is an iatrogenic syndrome that can be caused by rapid correction of hyponatremia. The objective of this paper is to present and discuss a case of ODS secondary to rapid correction of hyponatremia in a chronic alcohol user, and the proper management in the hopes to prevent future occurrences.

Case

A 25 year old male patient with a history of chronic alcohol abuse presented after a pre-syncopal episode, and was found to have hyponatremia with sodium of 115. He declined admission and left against medical advice. He presented again two days later following a syncopal event, and was found to have severe hyponatremia with sodium of 101. His last alcohol intake was 1 day prior to presentation. Patient was admitted to ICU, and was started on hypertonic saline (3%) with a target sodium correction rate of 6-8 mEq in 24 hours. However during the course of treatment, the sodium started to correct rapidly. Hypertonic saline was switched to normal saline, and then changed to 5% dextrose solution. Desmopressin was also used to decrease the rate of correction. Despite best efforts of the medical team, hyponatremia corrected by 16 mEq/L in 24 hours. With continued use of dextrose and desmopressin, sodium was finally lowered to 112 mEq/L on day 2 of hospitalization. Later that same day, patient developed alcohol withdrawal seizures and required intubation for a total of 5 days. Following extubation, patient was interactive but was not at his baseline. Approximately 2-3 days following extubation, he developed worsening encephalopathy. Initial CT brain and MRI brain were unrevealing. Multiple EEGs did not show any epileptiform activities. Repeat MRI 6 days later showed evidence of ODS. Patient received 5 sessions of plasmapheresis with minimal improvement. He subsequently underwent tracheostomy and PEG placement. Palliative care was consulted and discussions are ongoing regarding the goals of care.

Discussion

ODS is a rare complication reported after rapid correction of hyponatremia. Chronic alcohol use has also been reported as a risk factor for lowering threshold of demyelination. Current guidelines recommend close monitoring of serum sodium with correction goal of 6-8 mEq/L in 24 hours. Neurologic manifestations are typically delayed for 2-6 days and can present with paraparesis, disorientation, lethargy, etc. In severe cases, ODS can cause patients to be in a locked-in state. Various preventive measures include preemptively using desmopressin from the start of the treatment in patients who are at increased risk, reactively using dextrose water and desmopressin for rapid trajectory of correction, and promptly re-lowering sodium levels in patients who have already exceeded the correction limit. In our patient, the last two strategies failed in preventing the overcorrection of hyponatremia leading to devastating results.

Resident/Fellow Clinical Vignette

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Pembrolizumab induced Neuromuscular Disease and Respiratory Failure

Pembrolizumab induced Neuromuscular Disease and Respiratory Failure

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Introduction:

Immune checkpoint inhibitors have dramatically changed treatment of advanced cancers with poor prognosis in the past decade. Despite favorable oncological outcomes with checkpoint inhibitors, they are associated with various immune related adverse effects, some are potentially life threatening. Here, we report a case of Pembrolizumab induced neuromuscular disease and respiratory failure in a patient with renal cell carcinoma.

Case Presentation:

A 75 years old ADL independent male presented to the emergency room with progressive weakness and shortness of breath for 1 month. He was started on PD-1 inhibitor Pembrolizumab for metastatic renal cell carcinoma 3 months ago, and completed 2 cycles with the last dose 5 weeks prior to presentation. He described increasing difficulty moving around, and he started a walker in the past 1 week. He also endorsed dyspnea at rest and worse on exertion, choking on both liquid and solid food. His initial physical examination revealed tachycardia and tachypnea at rest, left eye ptosis with positive curtain sign, reduced neck flexion, grade four proximal muscle weakness of all limbs, +1 reflexes. Labs showed creatinine kinase of 1096 IU/L, negative acetylcholine receptor (AChR) antibodies and MuSK antibodies. His repetitive nerve stimulation test didn't show any decremental decrease. He required intensive care monitoring with his bulbar weakness, and was eventually intubated for respiratory failure with negative inspiratory force 10 cm of H2O. He received intravenous immunoglobulin, pyridostigmine, steroids for 5 days, slight improvement was noticed but still required ventilation. He eventually weaned off the ventilator successfully after receiving plasma exchange in another facility, but his bulbar palsy remained for which he required a jejunostomy tube on discharge.

Discussion:

With the wide use of checkpoint inhibitors in various cancers, it's imperative for an internist to be acquainted with their complications, especially severe adverse effects such as devastating neuromuscular diseases and endocrinopathies. Checkpoint inhibitor induced neuromuscular disease, as demonstrated in this case, its clinical presentation is similar to Myasthenia Gravis, but they may have negative AchR antibody, negative nerve stimulation test. This probably represents a new disease entity that is different from classical myasthenia gravis, but the exact pathophysiology, clinical features, optimal treatments are still not well described so far.

Checkpoint inhibitor induced neuromuscular disease can be severe and life threatening leading to respiratory failure. Therefore, internists must have high level suspicion of neuromuscular disease in patients with weakness after recent checkpoint inhibitor therapy. Early recognition and treatment will be crucial in improving outcomes.

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Myeloid neoplasm masquerading as hypereosinophilia and sweet's syndrome

Introduction:
Hypereosinophilia can be reactive to a range of infectious or immunological conditions or a primary myeloproliferative disorder and can be associated with serious complications and organ-damage. Sweet syndrome is characterized by painful, papular, and nodular skin lesions, and it has been linked to underlying malignancies. In this case, we present a patient diagnosed with a cross-over myelodysplasia and myeloproliferative neoplasm with associated sweet's syndrome.

Case Presentation:
A 72-year-old male presented with 6-month history of a painful maculopapular rash along with night sweats, fevers, and weight loss. He was treated multiple times with antibiotics and steroids with no improvement. A skin biopsy 3 months prior to diagnosis demonstrated neutrophilic dermatosis consistent with sweet's syndrome. Laboratory studies a week before diagnosis showed hemoglobin 7.1 g/dl, WBC 12.9 x103/uL, 30% eosinophils, absolute eosinophil count 3x109/L, and platelet count 160 x103/uL. Extensive infectious and immunological work up was negative. CT scan which revealed splenomegaly with 2.3cm mass. Bone marrow biopsy showed 100% hypercellularity, trilineage atypia, increased eosinophils to 43% (normal 1-5%) and 3-4% blasts positive for CD34 and CD117. FISH studies detected loss of PDGFRB signal, cytogenetics revealed a complex karyotype. He was diagnosed with a cross-over myelodysplasia and myeloproliferative neoplasm with peripheral eosinophilia and associated sweet's syndrome. Given his complex karyotype, anemia, and subsequent revised international prognostic index, his myelodysplasia was considered very high-risk. The patient is now planned for allogenic hematopoietic stem cell transplant.

Discussion:
Hematological disorders are associated with several paraneoplastic syndromes including sweet's syndrome (SS), also known as acute febrile neutrophilic dermatosis. The literature describes SS occurring most commonly with AML but can also be seen with other disorders like MDS and rarely in solid malignancies. The distinction between SS and infection or immune-medicated rash can be challenging as it requires histopathologic evaluation and is usually mistreated as cellulitis.

Hypereosinophilia is defined as persistent eosinophil count of at least 1.5x109/L. It can be idiopathic or associated with allergic, rheumatologic, infectious, or neoplastic conditions. Clonal hypereosinophilia is most frequently associated with chronic myeloid neoplasms such as myeloproliferative neoplasm (MPN) or overlapping myelodysplastic/myeloproliferative neoplasms (MDS/MPN), and more rarely with acute myeloid leukemia (AML). Hypereosinophilia related to hematological malignancies has been linked to gene rearrangements involving platelet derived growth factor receptor alpha (PDGFRA), PDGFRB, FGFR1, and JAK2.

Patients with documented rearrangements or mutations in PDGFRB are treated with imatinib, which is a potent kinase inhibitor. However, patients with high-risk MDS/MPN with associated eosinophilia are typically treated as MDS and should undergo allogenic stem cell transplantation if eligible.

Conclusion:
Both hypereosinophilia and sweets syndrome have been linked to myeloid neoplasms. Early recognition of either phenomenon as a paraneoplastic syndrome is important for early diagnosis and treatment.

Resident/Fellow Clinical Vignette

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A Case of Parsonage- Turner Syndrome Post Johnson and Johnson COVID-19 Vaccination

Introduction:

Parsonage- Turner syndrome or Neuralgic amyotrophy is an inflammatory disorder affecting the brachial plexus. While the exact pathophysiology is unknown, immunological, infectious, mechanical and genetic processes have been implicated, as it has been noted in some cases to be preceded by infections, surgery, trauma, immunizations etc. It is characterized by acute to subacute onset of pain from the neck to hand, muscle atrophy and motor deficit. Sensory symptoms like hypoesthesia and paraesthesias may be present in some cases. Diagnosis is clinical, though blood tests, imaging and electro-diagnostic studies may help rule out other causes. The course is usually self-limited, and recovery can take around 6 months- 2 years, with some patients having residual deficits.

Case description:

A 75 year old male with a history of metastatic rectal cancer presented with right shoulder and arm pain, with inability to abduct his right arm. The pain was sub-acute in onset, severe in intensity, and would impair his sleep. There was no history of recent trauma or infections. He had received the Johnson & Johnson COVID-19 vaccine on his right deltoid 3 weeks prior to the onset of these symptoms. He had 0/5 strength for shoulder abduction on his right arm, with intact sensation and reflexes. Blood work was unremarkable, and MRI of his right shoulder revealed edema and fatty atrophy involving the supraspinatus and infraspinatus muscles, indicating suprascapular neuropathy. No identifiable lesion at the suprascapular notch was visualized. Patient was referred to physical therapy, and experienced significant pain relief after ultrasound guided suprascapular and axillary nerve block and opioid analgesics.

Discussion:

In the reported case, patient developed the symptoms 3 weeks after receiving the vaccine.

His clinical presentation and MRI findings were suggestive of Parsonage- Turner syndrome.

While there are many hypotheses regarding the iteology of this condition, immune mediated pathophysiology is the most strongly supported, as infection and immunization precedes the development of symptoms in 20-52% and 15% cases respectively. Tetanus and influenza immunization has been associated with the condition according to previous literature. There have been several case reports of Parsonage- Turner syndrome associated with COVID infection. Pfizer COVID-19 vaccine has also been associated with this condition according to 2 case reports. This case maybe the first reported case of Parsonage- Turner syndrome post J & J vaccine.

Resident/Fellow Clinical Vignette

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A Case of Pulmonary Embolism in a Patient on the Human Chorionic Gonadotropin Diet

Introduction: The Human Chorionic Gonadotropin (HCG) diet consists of extreme calorie restriction to 500 kcal/day and regular HCG intake via intramuscular (IM) injection or sublingual lozenges. It has been advertised as a mode of weight loss since the 1950s by increasing metabolic consumption and fat redistribution. Studies thus far show no statistically significant difference between HCG diet and calorie restriction alone for weight loss; rather the HCG therapy can lead to serious adverse effects including thromboembolism. We report a case of pulmonary embolism (PE) in a patient on the HCG diet.

Case: A 41-year-old woman presented with palpitations, chest pain, and shortness of breath of three days duration exacerbated by climbing stairs. Wells score was 4.5. D-dimer was elevated to 3057. Computed tomography angiogram revealed extensive bilateral pulmonary emboli with mild right heart strain. History and physical examination were negative for any risk factors for thromboembolism, with the exception of being on the HCG diet for one month. She was receiving intramuscular injections of HCG while on a low carbohydrate, low calorie diet. She was a non-smoker, had no history of travel or surgery, had no oral contraceptive use, had no family history of thrombosis or cancer, and was not pregnant. She was negative for COVID-19 virus. Patient stated she had lost 29 pounds since initiation of HCG treatment. Therapy for PE was started with intravenous heparin and patient was admitted to MICU. The Naranjo probability scale determines the initiation of the HCG diet as a probable cause of the PE.

Discussion: HCG is a hormone produced by trophoblastic cells in the uterus, and is used clinically as a marker for pregnancy and related disorders. The HCG diet was first publicized in the 1950s and is still utilized today. Several studies have shown that there is no significant weight loss associated with HCG administration. Though some patients experience weight loss, this may be due to severe calorie restriction. Furthermore, HCG therapy has been associated with adverse effects including thromboembolism. The theorized mechanism is that HCG stimulates the ovaries, which release hormones and vasoactive substances leading to an increase in vascular permeability, hemoconcentration, and a hypercoagulable state. Despite lack of FDA approval, HCG products are available over the counter and are prescribed as an intramuscular adjunct to calorie restriction by physicians. While largely disproven, several books have been published on this weight loss strategy and it remains prevalent today.

Conclusion: The HCG diet is a fad weight loss strategy that has little efficacy and has the potential risk of causing life-threatening thromboembolism. This case report is important in spreading awareness about this diet and its complications among healthcare providers.

Resident/Fellow Clinical Vignette

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PERIANAL ABSCESS DUE TO PASTEURELLA MULTOCIDA

Introduction:

Pasteurella multocida is a non-motile gram-negative facultative anaerobic coccobacillus that resides in the respiratory tract of cats and dogs. The most common infections caused by it are local infections like cellulitis or abscess, which can cause deeper skin and soft tissue infections leading to deep tissue infections, septic arthritis, and osteomyelitis. Here, we present a rare case of perineal cellulitis and abscess secondary to Pasteurella multocida with no apparent animal bite or scratch.

Case report:

A 63-year-old female with a past medical history of COPD presented to the emergency department complaining of 3 days of perianal pain. The pain was excruciating, continuous, associated with an inflamed lump that worsened with sitting. It was accompanied by a fever of 101 at home and chills. She denied any drainage from the perineal area and could not recall a traumatic event. The patient admitted having dogs and cats at home but denied any bite or scratch from them. Physical exam revealed perirectal cellulitis and abscess. Computed tomography of the pelvis demonstrated left gluteal skin thickening and underlying fascial stranding extending deep into the perineum. At admission, the patient showed leukocytosis with normal range vital signs. Incision and drainage of the abscess was done, and wound culture showed Pasteurella multocida; blood cultures had no growth. The patient was started on intravenous aztreonam and oral doxycycline. Five days after the I&D, her pain got controlled, and she did not present any further complications, so she was discharged on the same treatment for 14 days.

Discussion:

Perianal abscess is a common disorder caused by infection from Escherichia coli, Proteus Vulgaris, Staphylococcus aureus, Streptococcus species, Bacteroides, and Peptostreptococcus. There are no documented cases of gluteal abscess caused by Pasteurella Multocida. Infection by P. Multocida is transmitted via direct or indirect contact with dogs, cats, and other felines. There have been cases reported of P. Multocida abscesses in different locations and systemic infections without a known animal bite or scratch. It is more common in immunocompromised humans with chronic diseases (e.g., cirrhosis, diabetes, malignancies, COPD like our patient).

Diagnosis of P multocida infection is made by isolation of the organism from the wound, sputum, blood culture, bronchoalveolar lavage, pleural fluid, ascitic fluid, or cerebrospinal fluid. Treatment of perianal abscess is incision & drainage with or without antibiotics. Antibiotic therapy for P. multocida is with Penicillin; if penicillin allergy is noted, as our patient, doxycycline and fluoroquinolones can be used. In conclusion, this case emphasizes that our patient had a rapid onset and spread of the infection. Also, we have to keep in mind that Pasturella infection can occur without traumatic animal bite contact.

Resident/Fellow Clinical Vignette

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Pfizer BioNTech COVID-19 vaccine and Guillain-Barre Syndrome

Introduction:

Guillain-Barre Syndrome (GBS) is an autoimmune disorder of the peripheral nervous system. Infections and vaccines are causes of GBS. The pathogenesis is molecular mimicry against the myelin or axons of peripheral nerves. We present a case of GBS secondary to the PfizerBioNTech COVID-19 vaccine.

Case:

An 85-year-old man with PMH of HTN, right Bell's palsy received the Pfizer BioNTech COVID-19 vaccinations on 1/21/2021 and on 2/11/21. He presented with numbness and tingling of hands and feet and weakness in his legs, where walking was â€œlike trying to walk on spongesâ€ starting early April. He denied infectious symptoms. An outpatient workup was done. MRI brain revealed no acute infarct, white matter disease, age related ventricular changes. MRI C-spine demonstrated only degenerative disc disease. EMG showed severe sensorimotor axonal and demyelinating peripheral neuropathy. As the symptoms progressed, he was admitted for further management. In the hospital, neuro exam was significant for no visual field deficits, 5/5 strength in upper and 4/5 strength in lower extremities, decreased sensation to touch and pain in hands and legs below the knees, and absent patellar and ankle reflexes bilaterally. Lab data included A1C 5.0, B12 309, TSH 1.8, VDRL neg, Lyme antibody neg, ANA neg, HSV neg. Lumbar puncture showed WBC 0, protein 111, Cryptococcal antigen neg, EBV neg, CSF Culture neg. MRI L-spine showed no definitive leptomeningeal enhancement and no significant spinal stenosis. Patient was treated with IVIG 0.4 g/kg for 5 days with improvement of paresthesias and weakness and was discharged home.

Discussion:

This is one of a few cases of presumed Covid vaccine induced GBS. The diagnosis was made based on his symptoms, EMG and lumbar puncture, six weeks after the second dose of the PfizerBioNTech COVID-19 vaccine. Historically, the 1976 influenza vaccine was stopped due to increased risk of GBS, and a meta-analysis showed that the 2009 Influenza vaccines had an increased risk of 1.6 excess cases of GBS per million vaccinations. Infection with influenza has a greater likelihood of causing GBS than the vaccine, with a relative incidence of 7.35 of GBS within 90 days of infection. There are also emerging reports of SARS-CoV2 infection causing GBS. Current CDC guidelines list a history of GBS less than 6 weeks after a previous dose of influenza vaccine as a precaution, not a contraindication, to getting a yearly influenza vaccine. History of GBS is not a deterrent for the Covid vaccine as there have not been enough cases to warrant such a precaution.

Conclusion:

While the Covid vaccine may represent a solution to the pandemic, it is associated with the development of GBS. Adverse effects should be monitored to ensure that the benefits outweigh the risks of this treatment.

Resident/Fellow Clinical Vignette

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PERICARDIAL DECOMPRESSION SYNDROME: A RARE BUT DANGEROUS COMPLICATION OF PERICARDIAL DRAINAGE

Pericardial decompression syndrome is defined as a paradoxical worsening in hemodynamics and development of pulmonary edema leading to right ventricle, left ventricular, or biventricular dysfunction. It is a rare complication of patients with cardiac tamponade who undergo surgical or percutaneous drainage. Though the true incidence is unclear, it is estimated to be around 5% with an increased risk in patients with malignancy, history of radiation therapy, and connective tissue disorders.

A 57 year old female with a past medical history of multiple myeloma, previously treated with radiation therapy, presented with persistent cough and was found to have a large pleural effusion. Further evaluation with echocardiogram revealed a large pericardial effusion with tamponade physiology. The patient was taken for an emergent pericardial window and 1.2 liters of bloody fluid was drained. Post-operative echocardiogram showed wide open tricuspid regurgitation with acutely decreased right ventricular systolic function and the patient was noted to be in decompensated heart failure. Right heart catheterization was done and showed only mildly elevated filling pressures. Patient was suspected to have pericardial decompression syndrome and was diuresed with near full recovery in right ventricular systolic function by six days after the procedure. Cytology of the pericardial fluid favored a reactive etiology. A rheumatologic workup was sent and the patient was found to be positive for ANA and SS-A with suspicion of possible cryoglobulinemia. Further workup is pending though ultimately the cause of the patient's pericardial effusion is likely her underlying multiple myeloma.

Though its exact pathophysiology of pericardial decompression syndrome remains unclear, one proposed mechanism is the sudden increase in venous return after rapid removal of fluid causing expansion of the right ventricle which was previously compressed during tamponade. This often occurs at the expense of the left ventricle resulting in reduced effective cardiac output, left ventricular and right ventricular dysfunction, and pulmonary edema. Prevention can be practiced by planning staged pericardiocentesis or placing a drain and slowly removing fluid over a longer period of time, allowing for more progressive cardiac compensation. Treatment is supportive but requires close monitoring as patients may require aggressive heart failure therapy with diuretics, vasopressors, and inotropic support. Though pericardiocentesis can be a life saving measure in patients with cardiac tamponade, caution should be taken when fluid is being removed as sudden decompressions of large effusions can lead to a life-altering complication such as PDS.

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Neutropenic fever with unknown etiology, a rare presentation of Hemophagocytic Lymphohistiocytosis

Background

Hemophagocytic lymphohistiocytosis (HLH) is a rare syndrome involving an aggressive immune hyperactivation state with cytokine release that causes excessive systemic inflammation and tissue destruction. HLH presents as a febrile illness with signs and symptoms imitating a viral infection. This condition usually develops in the setting of genetic mutations or is sporadically triggered by infectious, inflammatory or neoplastic causes. The most common trigger in genetically predisposed as well as sporadic cases is infection, usually viral. Prompt diagnosis and treatment is critical as HLH is a life-threatening syndrome with extremely high mortality.

Case Presentation

This is a case of a healthy 32-year-old male with no medical history who presented with flu-like symptoms of fevers, chills and night sweats. Symptoms initially started as a sore throat, nausea and congestion, however, progressed to weakness, fatigue, malaise with fevers in the last two days prior to presentation. He has no significant family history or previous sexual and social history; no risk factors at work identified. On admission, he was neutropenic and febrile with T 101.7F and a WBC count of 1.1 with ANC 0. Respiratory NAAT panel was negative for any RSV, Influenza A and B. Hepatitis panel, PCR for EBV, CMV, parvovirus, HIV, tickborne and anaplasma were all negative. Blood, urine and sputum cultures showed no growth. Peripheral smear at the time showed marked leukopenia and neutropenia with predominantly small lymphocytes with 25-30% reactive lymphocytes. A bone marrow biopsy was done. Patient was hospitalized at our facility for 12 days, managed with empiric antibiotics and neupogen in the interim. He acutely deteriorated on day 10, developing multiorgan failure with diffuse skin manifestations of a maculopapular rash; labs at this time showed elevated liver enzymes, unexplained renal dysfunction, cytopenia, elevated ferritin, LDH, fibrinogen and triglyceride levels. Cytogenetic testing and Chromosomal karyotype analysis were normal. T-cell clonality by PCR was positive for a monoclonal T-cell receptor gamma gene rearrangement. Bone marrow biopsy results showed rare histiocytes with hemophagocytosis concerning for HLH.

Patient was transferred to another facility for higher level of care, where the Hematology team did additional studies of serum immunofixation which showed an abnormal band in the gamma region of an M spike of 0.13; urine immunofixation did not show any evidence of paraprotein. He was treated with a pulsed dose of 5 days of IV decadron 20mg, then changed to IV solumedrol, after which his labs showed improvement in ferritin, fibrinogen, LDH, AST/ALT, triglycerides levels. Patient was discharged with a prolonged course of decadron therapy with eventual taper; he is currently doing well and has returned to work.

Conclusion:

Neutropenic fever without any infectious or neoplastic etiology and familial history, triggering a hyperactive systemic inflammatory syndrome is an unusual presentation of HLH.

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Increased Propofol Dose Requirements and Daily Marijuana Use: A Case Report

According to the National Institute on Drug Abuse, â€œin 2018, more than 11.8 million young adults reported marijuana use in the past year.â€ Marijuana use is becoming increasingly more frequent due to recreational and medical legalization across many states. Patients are told to abstain from Marijuana at least twenty-four hours prior to anesthesia to decrease the risk of complications. Such complications may include airway sensitivity, severe hypotension, and tachycardia. According to recent research, increased Propofol boluses may be required to achieve anesthesia sedation in daily marijuana users. Delayed awakening from anesthesia, respiratory depression, and hypotension are potential complications of higher Propofol requirements.

To achieve sedation with Propofol in adults, the drug should be administered in an initial bolus of 1.5-2.5 mg/kg. Our patient weighed 89.8 kg and admits to daily multi-marijuana use. He denies marijuana use 24 hours prior to anesthesia onset. At the start of anesthesia our patient received Midazolam 6 mg IV. Seven minutes later, anesthesia induction began with Fetanyl 100 mcg IV. Five minutes following, a Propofol 200 mg IV bolus was administered, followed five minutes later by an additional Propofol 50 mg IV bolus. The patient continued to show signs of movement despite the additional Propofol bolus. Subsequently, he required paralysis with Rocuronium 100 mg IV and an additional Propofol 50 mg IV to achieve complete sedation.

Our patient required more than the recommended Propofol induction dose; therefore, our clinical findings provide further supporting evidence that larger boluses of Propofol may be required to reach sedation in daily users of Marijuana. This finding carries clinical significance, since larger doses of Propofol may put a patient at increased risk of drug complications mentioned previously.

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SOLELY LUPUS NEPHRITIS

Introduction:

Systemic Lupus Erythematosus (SLE) is a heterogenous autoimmune disease with an array of clinical/serologic manifestations that can involve nearly any organ. Arthritis, arthralgias and skin/mucous membrane lesions are one of earliest manifestations of SLE in >90% of patients which subsequently leads to rheumatologic workup for further evaluation. Over the course of the disease, lupus nephritis usually develops in patients with SLE. However, it rarely occurs as the only presenting sign leading to diagnosis of SLE. We discuss an atypical case of SLE diagnosed with the sole presentation of nephritis.

Case Presentation:

A 30-year-old Hispanic female with past medical history of hypothyroidism and diet-controlled hyperlipidemia was sent to the emergency department by her PMD for blood pressure of 182/108. She complained of a bilateral suboccipital headache and lower extremity edema for the past week. The rest of review of systems was negative. She reported having elevated blood pressures at previous clinic visits, however, was being monitored by her PMD and not on any antihypertensive medications. Patients only home medication was levothyroxine and she denied any over the counter supplement use. Physical examination was significant for 2+ bilateral lower extremity edema extending up to the thighs. Laboratory results were significant for creatinine of 4.0mg/dL (patient reported previously normal renal function), urine protein/creatinine ratio of 7.6, and microscopic hematuria on urinalysis. Nephrology performed a renal biopsy showing diffuse proliferative, sclerosing and membranous glomerulonephritis suggestive of lupus nephritis. Further workup revealed positive ANA (1:340, speckled pattern), anti-ds DNA ab, anti-Smith ab, RNP, SS-A, and MPO. Serum complements (C3 and C4) were low. Anti-GBM ab, SPEP, HIV and Hepatitis B and C serologies were negative. Rheumatology treated patient with pulse dose steroids and Rituximab infusions. Patient was also treated with antihypertensives throughout the hospital course with continued clinical improvement of renal function and blood pressure.

Discussion:

This case with high suspicion for glomerulonephritis that led to renal biopsy highlights that lupus nephritis can be a presenting sign of lupus. SLE is an autoimmune disease with chronic inflammation which can affect virtually any organ and is commonly clinically diagnosed by early manifestations such as rash and joint/muscle pain. Lupus nephritis develops in approximately 50% of patients with SLE and is known to significantly increase morbidity and mortality of SLE patients. Renal manifestations are more common in Hispanic and African American populations and likely to present earlier and progress to ESRD in these populations. Clinicians should be aware that lupus nephritis can be the first sign of SLE and monitor these patients' renal function and urinalysis closely.

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A CASE OF ACUTE RESPIRATORY DISTRESS SYNDROME DUE TO NON-COVID-19 CORONAVIRUS OC43 AND VAPING ASSOCIATED LUNG INJURY

Acute Respiratory Failure has become familiar due to the COVID-19 pandemic. Prior to the pandemic, E-cigarette or vaping use similarly contributed to a rapid rise in the development of lung injuries (EVALI). Significant overlap exists between the COVID-19 pandemic and EVALI epidemic. Both SARS-COV-2 infection and vaping related lung injury are associated with varying degrees of lung injury, from mild hypoxia to acute respiratory distress syndrome (ARDS). Both also share radiological findings of diffuse bilateral lung infiltrates. Here, we present a case of a 44-year-old male with cough and diffuse myalgias found to have acute hypoxic respiratory failure secondary to Coronavirus OC43 infection and e-cigarette vaping induced hypersensitivity pneumonitis.

A 44-year-old gentleman presented to the hospital with worsening cough, vomiting, diarrhea, and diffuse myalgias. Medical history includes hyperlipidemia, gastrointestinal reflux disease, opioid dependence, hepatitis A, giardiasis and recent Helicobacter pylori infection for which he completed triple therapy days prior to presentation. Social history significant for vaping nicotine and marijuana. Sick contacts included his son with cold symptoms and his daughter who had COVID-19 three weeks prior. He received his second dose of COVID-19 Moderna vaccine one week before admission.

On presentation, vitals showed temperature 98.8F, BP 146/88, RR 20, HR 108, and SpO2 91% on room air. Physical exam notable for respiratory distress with accessory muscle use. Lab work revealed WBC 17.2 K/cmm with left shift, Procal 4.53 ng/mL, ESR 50mm/hr, CRP 296.9mg/L. Legionella and pneumococcal urinary antigens were negative. Stool and blood cultures were negative. Influenza, RSV and two separate SARS-COV-2 PCR tests were negative. SARS CoV-2 IgG titers showed negative anti-N (nucleocapsid) antibodies and positive spike IgG and IgM antibodies. Sputum gram-stain showed <1 PMN per low power field. Respiratory viral panel was positive for Coronavirus OC43. Chest computed tomography (CT) showed bilateral multifocal ground glass opacities with patchy consolidations. He was empirically treated with antibiotics for pneumonia and high dose steroids for suspected EVALI.

The patient subsequently developed worsening respiratory failure requiring intubation. He was transferred to the medical intensive care unit for ARDS due to non-COVID-19 Coronavirus OC43 infection and EVALI. His PaO2/FiO2 ratio was 74. He was sent to a tertiary hospital for ECMO evaluation but deemed not a candidate due to improvement. Hospital course was later complicated by disseminated intravascular coagulation and multiple venous thromboembolisms. Months after, he continued to require supplemental oxygen and suffered a stroke despite being on anticoagulation.

Infection with other strains of coronavirus can result in a COVID-19-like clinical presentation for those with underlying respiratory conditions like EVALI. Clinical suspicion should remain high and RVP should be ordered with SARS-COV-2 testing. Further studies are needed to assess the relationship between the coronavirus family, COVID-19/COVID-19-like disease and long-term effects.

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Cardiac Tamponade in K-RAS Mutant Colorectal Adenocarcinoma: A Case Report

Malignant pericardial effusion is a common clinical disorder seen in cancer patients. Metastasis to the heart from colorectal cancer, however, is exceedingly rare. We present the case of pericardial metastasis in a patient with KRAS-mutated colon adenocarcinoma who presented with a pericardial effusion and cardiac tamponade.

A 69-year-old Hispanic male with a medical history of Hepatitis C, Diffuse Large B-Cell Lymphoma diagnosed in 2011 currently in remission, and Stage IV Colon Adenocarcinoma diagnosed in 2013 s/p right hemicolectomy, prior treatments including FOLFOX, FOLFIRI and Avastin and palliative radiation therapy presented to the Emergency Department with gross hematuria for 3 consecutive days. On arrival, vitals were significant for tachycardia and hypotension. On physical examination, he appeared euvolemic, without jugular venous distension or muffled heart sounds, lungs were clear to auscultation and there was no lower extremity edema. Laboratory workup showed a hemoglobin of 13.0 g/dL, creatinine of 0.78 mg/dL, alanine aminotransferase of 57IU/L, aspartate aminotransferase of 86 IU/L, alkaline phosphatase of 136 IU/L, NT-pro-B type natriuretic peptide (NT-proBNP) of 179 pg/mL, troponin of <0.01 ng/mL and a carcinoembryonic antigen (CEA) of 487.1 ng/mL. Computerized Tomography (CT) of the chest, abdomen and pelvis revealed unchanged pulmonary metastases and peritoneal carcinomatosis with small volume ascites, an unchanged soft tissue mass in the pelvis and an increased moderate to large pericardial effusion. A transthoracic echocardiogram (TTE) confirmed a large circumferential pericardial effusion with tamponade physiology. Cardiothoracic surgery was consulted for a pericardial window placement. Serologic and pericardial fluid testing for infectious pathogens were negative. Fluid cytology showed a fluid protein level of 6.0 g/dL, glucose of 54 mg/dL and lactate dehydrogenase (LDH) of 1203 IU/L. Pericardial biopsy revealed immunohistochemical stains positive for CDX2, cytokeratin 20 (CK20) protein and cytokeratin 7 (CK7) protein, consistent with metastatic colon adenocarcinoma.

A primary tumor of gastrointestinal origin involving the pericardium is rare. Further, there are very few instances documenting primary colorectal cancer as the cause of cardiac tamponade. Proposed mechanisms of metastasis include hematogenous spread, lymphatic spread, transvenous extension and direct extension. While CDX2 is a sensitive and specific marker for GI tract carcinomas, only 20% of colon cancers have positive expression of both CK7 and CK20. This uncommon phenotype may be correlated with atypical sites of metastasis such as the pericardium and further studies are needed to assess the clinical course and prognosis in such patients. The patient continued therapy with FOLFIRI and Avastin and has had resolution of his pleural effusions and ascites and near resolution of his pericardial effusion. These findings support the possible benefit of cytotoxic chemotherapy in patients with symptomatic metastatic cardiac tumors. Ultimately, further studies will be necessary before a standardized treatment approach can be established for cardiac metastasis from colorectal cancer.

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A Rare Case of Neuro-Behcet's in a 50-Year-Old Man: A Diagnostic Challenge

Introduction: Behcet’s syndrome is a systemic vasculitis that is classically characterized by recurrent mucocutaneous ulcers. Neurologic involvement of Behcet's syndrome, also termed Neuro-Behcet's, is a relatively rare presentation that can masquerade as other neurological conditions.

Case Presentation: A 50-year-old African American man with Behcet's disease diagnosed at the age of 26 after having recurrent oral and genital ulcers and a positive pathergy test, and has since been on daily prednisone, presented with a five-day history of progressively worsening left-sided weakness and numbness, left-sided facial droop, and dysarthria. He had no oral or genital ulcerations on presentation, but reported increasingly frequent flare-ups in the past two years with orogenital ulcerations, fatigue, and arthralgia with morning stiffness. Neurologic examinations showed marked left-sided ptosis and left arm weakness, without other neurological deficits. He was initially thought to have a stroke based on CT findings of infarct involving left-basal ganglia, left-cerebral peduncle, and left midbrain. CT angiography showed right cavernous internal carotid artery calcified atherosclerosis with no conclusive evidence of vasculitis. However, MRI showed Fluid-Attenuated Inversion Recovery (FLAIR) signal abnormalities involving the left-basal ganglia, internal capsule, thalamus and left midbrain, and pons, which were more suggestive of neuro-Behcet's. He had significant improvement clinically after three days of pulse-dose steroids that was later transitioned to prednisone-tapering regimen. Repeat MRI three months later showed near-complete resolution of previously seen signal abnormalities.

Discussion: This case not only highlights the importance of recognizing neuro-Behcet's as a differential diagnosis in patients presenting with stroke-like symptoms, but also the remarkable clinical and radiographic improvement seen after treatment with corticosteroids in neuro-Behcet's.

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JAK2V617F Positive Essential Thrombocytosis After 40 Years of Treatment for Immune Thrombocytopenia

Objective:

Immune thrombocytopenic purpura (ITP) is an autoimmune condition and management with immunosuppressants, and splenectomy is often curative. We present the case of a patient who developed thrombocytosis secondary to JAK2 V617F mutation, 40 years after undergoing treatment for ITP.

Case:

89-year-old male with hypothyroidism was referred to Hematology clinic for evaluation of thrombocytosis with platelet count of 472 K/mm3 (Ref: 140'375). At age 47, he had been evaluated for thrombocytopenia and spontaneous bruising over limbs. Existing documents reveal that he was diagnosed with ITP and failed treatment with 6-week prednisone therapy requiring splenectomy followed by treatment with Azathioprine and prednisone daily for 10 years until his platelet counts stabilized between 150-200 K/mm3. He followed up with primary care every 6 months with repeat blood counts. Over the next 40 years, he was diagnosed with rheumatoid arthritis (RA) treated with long-term steroids and atrial fibrillation on rivaroxaban and metoprolol.

During a routine follow up appointment he was noted to have mild anemia, hemoglobin 11.5 g/dl (Ref: 13.5-17), elevated MCV 101 fl (Ref: 83-98) and platelet count 472 K/mm3. He denied any episodes of bleeding/bruising, headache, dizziness, changes in vision, numbness in extremities, weight loss, change in medications or familial history of hematologic disorders. Peripheral smear revealed normochromic and normocytic red blood cells with Howell-Jolly bodies and thrombocytosis with normal platelet morphology. The elevated MCV was attributed to hypothyroidism and daily alcohol use. It was considered that thrombocytosis was a sequela of splenectomy, however, presentation after 40 years was atypical. He underwent work up for possible myeloproliferative disorders which revealed positive JAK2 V617F mutation, negative BCR/ABL mutation and erythropoietin levels 8.1 mIU/ml (Ref: 2.6-18.5), making myelodysplastic syndrome and chronic myeloid leukemia (CML) less likely and essential thrombocytosis (ET) the most plausible explanation. Cytoreductive therapy with hydroxyurea was deferred as the patient was asymptomatic and receiving anticoagulation with rivaroxaban. Bone marrow biopsy was not performed as per patient's request. He continues to follow with Hematology for monitoring of ET every 3 months and platelet counts remain stable between 450-470 K/mm3.

Discussion:

Thrombocytosis after splenectomy is a well-known phenomenon due to decreased platelet sequestration, however, it is unusual for thrombocytosis to develop after an interval of 40 years. The development of ET and JAK2 mutation in this case could be secondary to prolonged treatment with immunosuppressants such as Azathioprine and steroids or the concurrent presence of autoimmune conditions such as RA, however, the association is not well-established. We also speculate that detection of JAK2 mutation at the time of diagnosis can affect prognosis of ITP. Further large-scale studies are required to evaluate the effect of positive JAK2 mutation and use of immunosuppressives on evolution of ITP.

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When the Cure is the Culprit

Introduction:

Three common considerations in identifying a source of fever are infections, connective tissue disorders, and malignancy. A small incidence (3 to 5%) of adverse drug reactions will also have fever as its sole presentation. This includes certain antimicrobials, which are a mainstay treatment in bacterial infections. We present a case of persistent fevers in a patient with an extensive history of rheumatologic diseases undergoing treatment for infection.

Case report:

The following is a case of a 59-year-old African-American female with complaints of shortness of breath, nausea, and weakness. This was associated with episodes of productive cough, fever, chills, and sweats (for 30 days). She is known to have an extensive past medical history of alcohol use disorder, gout, asthma, bronchitis, chronic sinusitis, and a remote history of granulomatosis with polyangiitis. On initial evaluation, she was noted to be hypertensive and tachypneic with adequate saturations in room air. She initially had mild tremors, while the remainder of her examination was unremarkable.

Laboratory work-up showed normal WBC, macrocytic anemia, thrombocytosis and transaminitis. Blood and sputum cultures grew Staphylococcus aureus. She was started on vancomycin, and later she was switched to Oxacillin after final sensitivities showed MSSA. An echocardiogram ruled out valvular vegetations. In search for a source of the bacteremia, a maxillo-facial CT was pursued, but findings were negative overall as were previous ENT workups. Her clinical course was complicated with pneumonitis which required a course of steroid coverage. As her status improved, the patient then re-developed fevers, chills, and leukocytosis after steroid withdrawal. Antibiotic coverage was broadened empirically with Zosyn and later with Ertapenem after urine cultures tested positive. Despite antimicrobial coverage, her fevers persisted. Inflammatory markers were noted to be elevated. An extensive work-up to identify a source of fever was pursued which included procalcitonin testing, repeat imaging and cultures, WBC tagging, and an extensive rheumatologic work-up due to her past history ‘ all of which were unrevealing. A slow development of eosinophilia was then observed in her CBC trend during her battery of workups. A consideration of a potential drug fever was considered, with a trial of antibiotic withdrawal later resolving her febrile episodes and eosinophilia.

Discussion:

This case illustrates the potential of how anchoring biases based on positive blood cultures and history of granulomatosis with polyangiitis may lead to potentially unnecessary workups and delays in diagnosis. As it is important to rule out life-threatening causes of the patient’s fevers such as sepsis, a thoughtful review of the patient’s clinical course and laboratory trend would have identified the drug-related fever at an earlier time. Vigilance on the potential adverse effects of medications, especially those known to cause drug fever, is imperative in ensuring its timely identification and management.

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Inhalation Injury After a House Fire

Many house fires occur annually with a large percentage of victims experiencing burns and inhalation injury due to prolonged exposure. In our patient's case with severe inhalation injury, systemic steroids proved to facilitate earlier healing, decreased airway edema, and successful extubation within a short duration of time.

Clinical Course:

A 56 year-old female with no significant past medical history was brought in as a level 1 trauma for 25% total body surface area (TBSA) burns and suspected inhalation injury after a house fire. In ED, the patient was intubated and was treated with duonebs and Patient had 2nd degree burns with 25% TBSA. Erythema was noted in both eyes and there was visible soot present in the nasopharynx and oral cavity. Patient was placed on IV methylprednisolone 40 mg q12hrs for 7 days, flonase, duonebs, IV protonix, and initiated on empiric antibiotics with cefazolin and vancomycin.

Labs showed WBC 26.89 K/mm3. Initial ABG on FiO2 50% ventilator support revealed a pH 7.30, PCO2 42.2 mmHg, PO2 122 mmHg, bicarb 20 mmol/L with O2 saturation of 99%. Initial carboxyhemoglobin was 22.5%, methemoglobin was 4.9%. COVID19 testing was negative. Chest x-ray revealed bilateral pulmonary edema. Bronchoscopy was performed twice for therapeutic lavage and revealed dark soot deposits in the nasopharynx, oropharynx and bilateral tracheobronchial trees. A third bronchoscopy showed decrease in edema and clearing of soot on admission day 6. The patient was successfully extubated on admission day 7; no post-extubation stridor was observed.

Discussion:

Steroids can be useful in the prevention of further destruction from the host's immune response and is an area of interest given increased risk of infection in the setting of burns. Clinical models performed on rats exposed to smoke showed no clinical significance. Yet, when exposed to a medium-dose steroid regimen (4 mg/kg), steroid use had a significant effect on both inflammation and overall mortality. This suggests the possibility of a therapeutic effect of use of steroids. Other literature reviews have suggested that steroids may decrease edema in the airways, which led to a decreased re-intubation rate. We believe implementation of IV methylprednisolone 40 mg every 12 hrs for 7 days led to moderate improvement of airway edema & successful extubation in our patient.

Conclusion:

we suggest re-evaluation of steroids in the management of inhalation injury with severe airway edema. It is our belief that the use of systemic steroids facilitated earlier healing, decreased airway edema, and successful extubation within a short duration of time. Currently, no consensus exists for both the diagnosis and/or grading of inhalation injury and the treatment varies worldwide.

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Rare case of Cerebral Infarction 25 days after IVIG therapy for Common Variable Immune Deficiency (CVID)

Case: A 59 year old Caucasian female with significant history of PVCs, CVID on IVIG therapy (last dose 25 days ago), and depression presenting to the ED for altered mental status. The patients husband had found his wife at the kitchen table staring blankly at the wall; non-verbal and called EMS. The patient's husband told of a long history of depression; stable on Zoloft for many years. Vitals were BP 114/82mmHg, HR 96 bpm, Temp. 98.3F, oxygen saturation of 97% and RR of 16 breaths/min. Physical examination was completely benign with no focal deficits; non-verbal able to follow both simple and complex commands. ECG showed normal sinus rhythm. Non-contrast CT head and CTA of the head and neck was completely unremarkable. Psychiatry recommended a trial of ativan for catatonia. The patient subsequently improved; however, never returned to her baseline. She suffered word finding difficulty, and memory lapses. Brain MRI obtained 5 days later showed bilateral basal ganglia and left cerebellum subacute infarcts. Subsequent testing ruled out causes such as vasculopathic, embolic, thrombophilic, and inflammatory etiologies.

Discussion: Stroke is a rare complication of IVIG infusions occurring typically within 2-10 days after infusion. There has been only one other case report of stroke occurring 25 days later. Serious reactions occur in 2 to 6 % of patients. Risk factors include smoking, CAD, diabetes, hypertension, dyslipidemia, older age, and estrogen use. For our patient risk factors were older age and elevated LDL; although her ASCVD risk was 3.6%. Lipoproteins have a direct effect on blood viscosity; LDL causes red cell aggregation, whereas HDL has antiatherothrombotic properties. IVIG can increase the viscosity of plasma and whole blood both in vivo and in vitro. The dose should not exceed 500mg /kg in a single day and infusion at a rate no more than 50mg/kg/hr. Our patient met these requirements; yet an adverse event occurred. As similarly with the previously published case by Chang, et al. both patients had normal cerebral and extracerebral vessels.

Conclusion: Although published only once before, we postulate that the stroke occurring 25 days after the patient's last infusion, given normal cerebral vasculature and absence of thrombosis or emboli, suggests a hyperviscosity vs. vasospastic event. Our case proposes that predisposition to adverse effects persists over a longer period and may result in vascular complications. Given the effectiveness of subcutaneous immunoglobulin therapy for CVID; more patients might benefit if switched to the subcutaneous route.

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Moderna COVID-19 vaccine induced anaphylaxis or is it?

Anaphylaxis following COVID-19 vaccines has been a topic of debate. Anaphylaxis is a challenging diagnosis as the differential includes approximately 40 other diseases.

A 41 year old woman presented for a suspected anaphylactic reaction to the second dose of the Moderna mRNA vaccine. Within minutes of receiving the second dose of the vaccine she started to feel throat swelling, generalized pruritis, and dyspnea. Patient self-reported a similar but less severe reaction to the first dose. She received Methylprednisolone 125mg IVPB, Benadryl 50mg IVP, and epinephrine 0.3mg IM in the emergency department. She was admitted to the ICU for close observation; while overnight she began to complain of severe anxiety, dyspnea, and began to desaturate. She was subsequently intubated; however anesthesia found her airway intact without any visible edema during the intubation process. She was extubated 2 days later. After extubation she remained anxious and was started on Xanax 0.25mg daily; without improvement a psychiatric evaluation was placed. She was diagnosed with adjustment disorder and given counseling. Four days after admission the patient was downgraded to medicine; Methylprednisolone was restarted at 40mg every 12 hrs. for continued symptoms. The patient had stridor on exam on the second day of the medicine service and improved after intramuscular epinephrine was administered. Allergy consult was placed that same day and a serum tryptase level was sent; which was negative. Allergy recommended stopping Methylprednisolone as it contains polyethylene glycol and could possibly be the allergen; as also present in the Moderna vaccine. They recommended continuing H1 and H2 blockers for at least 1 week. ENT performed a laryngoscopy and vocal cords had no visualized abnormalities; however they noted poor inspiratory reserve and global weakness. Concerned for a neuromuscular disorder; neurology evaluated the patient. Impression was that the recurrent laryngeal nerve could have been damaged; however MRI of the head and neck; as well as a CT of the soft tissue of the neck revealed no abnormalities. An EMG and acetylcholine receptor antibodies were found to be negative. With neuromuscular and physiologic abnormalities ruled out; again psychiatry consultation was obtained and the patient was diagnosed with possible conversion disorder vs adjustment disorder.

Conversion disorder or functional neurologic disorder (FND) can be caused by a variety of physical/ or emotional events including vaccinations. This case outlines the importance of not only educating the public on conversion disorder; but also possibly the over-reporting of anaphylaxis reactions in vaccinated individuals. Educating the public and addressing these cases directly should be of absolute priority to ensure continued public compliance in the vaccination process.

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A rare presentation of an autosomal dominant disorder; Dysphagia and hiccups in Von-Hippel Lindau Disease.

Introduction:

Von Hippel Lindau (VHL) disease is a rare autosomal dominant syndrome caused by mutation in VHL gene. Patients with VHL may be affected by tumors most commonly hemangioblastomas that may be located in the brainstem, retina, or spinal cord. Onset of disease may be from childhood to adulthood. This case reports a patient with a non-specific constellation of symptoms before a potentially life-threatening tumor was discovered.

Case Presentation:

A 21-year-old female presented with a several week history of hiccups, progressive dysphagia to solids, and bilateral flank pain. On further questioning, patient admitted to worsening bilateral upper limb weakness immediately prior to the onset of her dysphagia. She had been previously seen in multiple clinics and ER for neck and back pain and treated for muscle spasms. On a prior admission, she presented with hypertensive urgency and back pain prompting CT of the abdomen which showed polycystic kidneys. CT neck showed a mass at the level of C5-C7 with cord compression. Was transferred for neurosurgical evaluation and successfully underwent C5-C7 laminectomy, resection of the intramedullary tumor and C5-C7 laminoplasty with improved mobility and function status post surgery. Pathology analysis revealed a WHO grade 1 hemangioblastoma. Given prior history of polycystic kidney disease and its well-recognized association with von Hippel-Lindau disease, a diagnosis of VHL disease was made with further outpatient genetic testing for confirmation.

Discussion:

Von Hippel-Lindau disease is a heritable, multisystem syndrome that manifests with varying degrees and complexity of tumors. Males and females are equally affected. As almost 20% of cases are de novo, there are no strongly correlated risk factors known to date. Symptoms from tumor formation are dependent on tumor size, growth, rate and presence of peritumoral cysts. This patient's manifestation of disease included bilateral upper limb weakness along with bulbar symptoms which are among the least common symptoms produced by hemangioblastomas in VHL. Most common presenting symptoms are headaches, gait ataxia and dysmetria. Despite their benign pathology, these tumors result in further damage to the spinal cord, leading to decrease in motor, sensory, bulbar function, quadriplegia and even death. It is vital for providers, especially those in an outpatient setting without immediate access to imaging, to maintain high suspicion given the constellation of clinical symptoms presented above. Though VHL disease is a rare disorder and thus infrequently encountered, a missed diagnosis could be detrimental and contribute to higher morbidity as early recognition and treatment may be life-saving.

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CONVULSIVE SYNCOPE IN AN AVID RUNNER WITH PRESERVED LEFT VENTRICULAR SYSTOLIC FUNCTION AND PREVIOUSLY UNDIAGNOSED SEVERE AORTIC STENOSIS

Introduction: Aortic stenosis (AS) is the most common form of valvular heart disease among older adults in the United States. It is associated with increased risk of sudden cardiac death. While syncope is considered a classic symptom of severe AS, it is rarely seen in clinical practice. Only about 3% of patients with severe AS present with syncope. Furthermore, while studies indicate that presentation of some forms of heart disease may be misdiagnosed as seizures, AS has not been described as presenting with seizure-like activity. We report a case of severe AS presenting with syncope initially suspected to be seizures.

Case Presentation: Patient is a 67-year-old male avid runner with history of hypertension and seizure disorder (last seizure two years earlier, not on anticonvulsants). After he had gone on his routine daily run, he was found unconscious with abnormal movements suspicious for seizure-like activity. He was emergently intubated in the field for airway protection. Upon arrival in the ER, examination was remarkable for a systolic murmur loudest at the right sternal border. Trauma work-up, including non-contrast CT head, revealed no acute pathology. Laboratory evaluation showed elevated troponin of 1.28 ng/mL. No acute ischemic changes were seen on EKG. Continuous video-EEG monitoring showed no evidence of epileptiform activity. Transthoracic echocardiogram showed preserved left ventricular systolic function (ejection fraction >55%) with severe AS (severe calcification, valve area of 0.5 cm2, peak velocity of 4.1 m/s, mean gradient of 42 mmHg) and normal-sized cardiac chambers. He was successfully extubated on Day 2 and denied any prior episodes of syncope, exertional dyspnea, or angina. Cardiac catheterization on Day 2 revealed mild nonobstructive CAD. After a multidisciplinary heart team meeting and discussion with the patient, transcatheter aortic valve replacement (TAVR) was deemed to be the most appropriate treatment approach. The patient underwent TAVR with no complications. Echocardiogram on post-procedure Day 1 showed no paravalvular regurgitation. On post-procedure Day 2, he was discharged home, with scheduled outpatient follow-up.

Discussion: Of the classic symptoms of severe AS, exertional dyspnea is the most common and presents earlier, while only 1 in 10 cases of symptomatic severe AS present with syncope. Our patient had been asymptomatic and tolerating daily high-intensity exercise prior to his syncopal episode. Studies report a propensity to misdiagnose abnormal movements following syncope as seizures. Despite a past medical history of seizure disorder, continuous video-EEG monitoring did not corroborate seizures in our patient. Studies indicate that abnormal movements following a syncopal episode ‘ termed convulsive syncope ‘ are induced by cerebral hypoxia. To ensure accurate diagnosis, careful history and physical examination, including auscultation for murmurs, should be performed in patients presenting with transient loss of consciousness, and echocardiography should be considered to evaluate for severe AS.

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Images in medicine: A central, submassive pulmonary embolism presenting with clot-in-transit

Clot-in-transit, or thrombus-in-transit, is an uncommon presentation associated with pulmonary emboli that carries with it a high mortality1. Current literature suggests several approaches to management including open surgical thrombectomy, catheter-directed mechanical thrombectomy, catheter-directed thrombolysis, systemic thrombolysis, or continued anticoagulation2,3. With the advent and routine use of point-of-care ultrasonography (POCUS) in clinical evaluation, early diagnosis and management of clots-in-transit is made possible. Here, we describe a case of a 73-year-old man presenting with syncope who was subsequently found to have a high-risk submassive pulmonary embolus with multiple clots-in-transit on POCUS. The patient had several poor prognostic markers for pulmonary embolism including clinical history presenting with syncope, having a positive shock index (heart rate greater than systolic blood pressure) in absence of overt hypotension, positive cardiac markers on labs, and imaging evidence of right heart strain. After expert consultation, multidisciplinary discussion, and confirmatory echocardiography ruling out mimickers, the decision was to treat the patient with half-dose systemic thrombolysis. He was successfully treated with 50mg of intravenous alteplase, and intravenous unfractionated heparin without complication, and subsequently discharged on systemic anticoagulation with apixaban. Included are echocardiographic videos detailing the resolution of his intracardiac thrombi and improvement in right ventricular function.

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ISCHEMIC STROKE ATTRIBUTABLE TO USE OF SELECTIVE ANDROGEN RECEPTOR MODULATORS IN A YOUNG ADULT

Introduction: Individuals younger than 45 years account for about 10-15% of ischemic stroke cases. Prevalent stroke risk factors in young individuals include congenital heart diseases, hematologic and metabolic disorders, and substance use. We present the case of a patient with ischemic stroke associated with use of selective androgen receptor modulators (SARMs).

Case Description: A 33-year-old male steelworker with nicotine dependence presented with one-day history of worsening headache, dysphagia, diplopia, left facial numbness and left sided weakness. He could not stand due to severe vertigo and was averse to eye-opening. For the preceding three weeks, he had been weightlifting at a gym and consuming two tablets daily of an over-the-counter bodybuilding supplement containing the following SARMs: ostarine, cardarine, and andarine. Family history was negative for stroke and hypercoagulability. Vital signs showed hypertension (179/95 mmHg) and tachypnea (22 breaths/min). Examination revealed left-sided pronator drift, decreased left-sided pinprick and fine touch sensation, dysmetria, and gait ataxia. Initial non-contrast CT head was unrevealing, while CT angiogram of the head and neck showed left vertebral artery occlusion. MRI brain was contraindicated due to presence of an embedded steel shrapnel in patient's cheek. He had presented outside the therapeutic window for thrombolytic therapy; therefore, medical management was recommended. Laboratory tests, including chemistry panel, toxicology screen, HBA1c, thyroid function test, Vitamin B12 level, homocysteine level, and hypercoagulable workup were unremarkable, except for low HDL cholesterol (33 mg/dL). Repeat CT head showed small evolving infarcts in inferomedial left cerebellum, confirming the diagnosis of ischemic stroke. There was no evidence of intracardiac shunt or any other abnormalities on transesophageal echocardiogram. Cardiac monitoring did not reveal any arrhythmia. Treatment included aspirin, high-intensity statin, meclizine, physical and occupational therapy, with improvement in his neurological deficits. He was advised to stop SARMs and cigarette smoking and discharged to an acute rehabilitation facility on Day 5, with plans for outpatient follow-up.

Discussion: Use of cocaine, methamphetamine, heroin, marijuana, androgenic anabolic steroids, and SARMs increase the risk of stroke by inducing tissue hypoxia, vascular injury and spasm, and cardiac arrhythmias. Moreover, cigarette smokers have a two-to-fourfold increase in stroke risk compared to nonsmokers. Our patient likely developed ischemic stroke from a synergistic effect of SARM and cigarette smoking. Although, potential clinical applications of SARMs include treatment of prostate cancer, hypogonadism, osteoporosis, and cachexia, SARMs have not been approved by the FDA, in view of increased risk for stroke, heart attacks, and liver damage. SARMs are increasingly becoming popular among bodybuilders due to their musculoskeletal anabolic effect and reduced androgenic properties. More scientific data is required to establish the safety and efficacy of SARM supplements along with a collaborative effort between pharmaceuticals, clinicians, and policymakers to increase awareness.

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Think the Unthinkable, A Rare Case of Infective Endocarditis of Multiple Native Valves with Polymicrobial Non-HACEK Gram-Negative Organisms Leading to Septic Shock and Heart Failure.

Introduction: Infective endocarditis is associated with profound morbidity and mortality risk. Subacute bacterial endocarditis is most commonly caused by gram positive organisms. Gram negative infective endocarditis is extremly rare. Furthermore, the majority of patients with subacute bacterial endocarditis do not develop heart failure or septic shock. This is a case of polymicrobial gram negative endocarditis in a patient with mitral and aortic valve vegetations leading to septic shock and heart failure.

Case Presentation: 84 year old man with a history of coronary artery disease, heart failure with preserved ejection fraction, chronic obstructive pulmonary disease, hypothyroidism, and a prior pancreaticoduodenectomy for pancreatic adenocarcinoma who presented to his outpatient cardiologist with complaints of several weeks of progressive dyspnea and subjective fevers. He was ill appearing, hypotensive and had tachycardia on initial presentation, and was immediately sent to the emergency room.

In the emergency department, he was afebrile with a blood pressure of 73/37 mmHg and a heart rate of 102 beats per minute. He had elevated jugular venous pressure, bilateral rales, and holosystolic and early diastolic murmur. Blood cultures drawn resulted with gram negative rods in less than 24 hours. He was initially admitted to the intensive care unit and started on broad spectrum antibiotics, he was also started on vasopressors for septic shock.

Multiple blood cultures subsequently grew out Klebsiella pneumoniae and Escherichia coli, both of which were sensitive to ceftriaxone. Patient's antibiotics were subsequently switched to ceftriaxone. A transthoracic echocardiogram showed echogenicity on the mitral and tricuspid valves along with new onset of moderate mitral and tricuspid regurgitation. A transesophageal echocardiogram was performed, which showed vegetations on the mitral and aortic valves along with moderate mitral regurgitation and minimal aortic regurgitation along with reduced ejection fraction.

His hemodynamics improved and blood cultures cleared with antimicrobial therapy and the decision was made to continue with conservative management. The patient had a peripherally inserted central catheter placed and was continued on intravenous ceftriaxone for 6 weeks with discharge and outpatient follow up arranged.

Discussion: The most frequent organisms involved in subacute infective endocarditis are gram positive organisms. Endocarditis due to gram negative bacteria is infrequent. Gram negative infective endocarditis typically caused by Haemophilus, Aggregatibacter, Cardiobacterium, Eikenella and Kingella (HACEK species). Endocarditis caused by gram negative non-HACEK species is extremely rare. Furthermore, polymicrobial non-HACEK infective endocarditis involving multiple native valves is even rarer. Infective endocarditis with gram negative organisms carries higher morbidity and mortalirty, hence early detection and treatment is paramount of clinical importance.

Conclusion: This case illustrates the importance of considering infective endocarditis as a potential cause of gram-negative septicemia. Early echocardiography and targeted antibiotic treatment in such patients is pivotal for appropriate treatment and consideration of surgical intervention in select cases.

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Salicylate toxicity from ingestion of wintergreen oil

Introduction

Salicylates are commonly used for their analgesic, antipyretic, anti-inflammatory and antiplatelet properties. Acute salicylate poisoning is a common overdose resulting in high morbidity and mortality. We are presenting a case of insidious onset salicylate toxicity from ingestion of wintergreen oil, which has 98%methyl salicylate. Among all the salicylates, methyl salicylate is more toxic.

Case report

A 59 year old female presented with complaints of abdominal pain for 3 days duration. Abdominal pain was diffuse throughout the abdomen, worsening gradually for 3 days, associated with nausea, vomiting and fatigue. On examination, patient was resting comfortably on room air. No significant findings on physical examination. Labs revealed high anion gap metabolic acidosis with bicarb of 19 meq /dl, salicylate levels of 41.2mg/dl. Acetaminophen and alcohol levels were negative. urine drug screen was negative. Patient denied taking aspirin, NSAIDs. On further questioning, patient stated that she added 2 drops of wintergreen oil in her tea and her smoothies every morning for about 8months.She stated that adding wintergreen oil was refreshing in the morning. She has also been taking calcium, magnesium, vitamin B and D. No other over the counter supplements or topical analgesics. she also added that she has experienced abdominal pain and nausea intermittently over the past 5 months but she did not seek medical attention. As the pain worsened, she presented to the hospital for this visit. On admission, patient was started on bicarb drip, IV fluids and supportive therapy with close monitoring. Bicarb drip was discontinued the next day as the acidosis improved and salicylate levels decreased. Patient was discharged on day3 as she was back to her baseline. Patient was given a list of substances that have salicylates in them and was advised to avoid them.

Discussion

Methyl salicylate (oil of wintergreen) has more salicylate than other salicylates; 5 ml methyl salicylate is equivalent to 7000mg of salicylate .Salicylates are commonly found in wide variety of substances including aspirin, topical analgesics, medicated oils, mouthwash solutions, as flavoring agent in chewing gums, mints and candies. Clinical features of salicylate toxicity include tachypnea, tachycardia, fever, hypoglycemia, metabolic acidosis progressing to altered mental status and seizures. Usually, doses greater than 500 mg/kg of salicylate can result in severe hyperpnea, coma, and occasionally seizures. Treatment depends on severity of toxicity. Correction of acidosis with bicarbonate and alkalinisation of urine with sodium bicarbonate to enhance urinary excretion of salicylate are the mainstay of treatment. As our patient ingested small amounts of wintergreen oil for months, her presentation was not very severe and she improved with bicarb drip. The general population has to be educated about the importance of reading the labels in all the supplements they take to avoid complications

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IATROGENIC IMMUNODEFICIENCY ASSOCIATED LYMPHOPROLIFERATIVE DISORDER IN PATIENTS WITH ACUTE LYMPHOBLASTIC LEUKEMIA:A CASE SERIES

Iatrogenic immune deficiency associated lymphoproliferative disorders (LPDs) with a primary diagnosis of acute lymphoblastic leukemia seen in elderly patients are an extremely rare entity with no formal WHO classification with only 2 other cases reported in the literature to the best of our knowledge. We describe 3 patients aged 15-25 years who developed LPDs after being on maintenance chemotherapy for a period of 10-18 months when they presented with symptoms similar to the ones which led to the initial cancer diagnosis. A repeat 18-FDG PET-CT scan was performed for all 3 of them which then led to repeat biopsies that showed atypical lymphocytes and histiocytes. Immunohistochemistry was performed. All 3 patients were positive for EBV LMP1. For one of our patients, it showed CD30, CD15 positivity suggestive of Hodgkin's lymphoma. For patients two and three it showed CD20 positivity indicative of EBV-associated lymphoid proliferation. Clonality analysis was positive for Ig gene rearrangement. The etiology was thought to be dual in nature with Epstein Barr Virus positivity and the use of Methotrexate. A finding in our study which did not match the literature was the age of these patients. The literature review presented a median age of about 40 to 70 years whereas our patients belonged to a relatively young age group of 15 to 25 years. The etiology for this discrepancy is unclear at present given that limited data available with global literature reporting a total of 71 cases only. In our cohort, these LPDs regressed on withdrawal of immunosuppression. Given the variable prognosis due to morbidity associated with the primary malignancy and chemotherapy which promotes a state of chronic immunosuppression, our cohort is currently under follow-up for recurrence.

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Signet Ring Cell Carcinoma of the Colon, A Rapid & Fatal Progression

SIGNET RING CELL CARCINOMA OF THE COLON: A RAPID AND FATAL PROGRESSION

Introduction: Signet Ring Cell Carcinoma, (SRCC) is a rare morphological variant of colorectal cancer (CRC) which accounts for 1-2% of all cases, and is twice as common in younger adults, age 20-40. It represents one of the most aggressive subtypes with an estimated 5-year survival rate of less than 10%.

Case Description: A 38-year old female with a history of untreated H. pylori and GERD presented with vague epigastric pain for one year associated with nausea, vomiting, altered bowel habits and weight loss. Examination was remarkable for moderate abdominal tenderness without distension. Initial abdominal CT identified a soft tissue mass in the left upper abdomen with paraaortic lymphadenopathy and small amount of fluid in the abdomen and pelvis. Colonoscopy revealed marked edema and mucosal ulceration at the hepatic flexure. Biopsy showed poorly differentiated adenocarcinoma with signet cells features and positive immunohistochemistry for CDX-2, CK 20, and SATB2, favoring a primary GI site. Tumor markers CEA, AFP, CA 19-9 and CA-125 were positive. Over the next 3 weeks, she developed rapidly progressive diffuse lymphedema, shortness of breath, abdominal distention and worsening abdominal pain. Repeat abdominal CT showed severe progression of abdominal ascites with thickening of colonic wall, enlarged mediastinal nodes, metastatic nodules in right lung, a 2.0 x 2.2 cm nodule in the left lobe of liver, peritoneal implants and diffuse omental caking. A multidisciplinary approach was implemented with surgery, interventional radiology, oncology, and palliative care. She was started on capecitabine-oxaliplatin regimen for two cycles, with poor response. Following chemotherapy, she opted for comfort care only. She passed away within ten weeks from initial presentation.

Discussion: Manifestations of SRCC are non-specific. To our knowledge, this case represents one of the most aggressive metastatic progression of SRCC documented, with severe omental caking, widespread metastasis, clinical debilitation in just three weeks, and death within ten weeks from initial presentation. Late presentation leads to diagnostic delay, with metastatic lymph node involvement in most cases at the time of diagnosis. There have been very few studies describing SRCC of the colon in young adults due to its rarity. Right sided presentations are more commonly associated with carcinomatosis, such as in our case. Primary gastric origin should be excluded, which is seen in 96% of cases of SRCC. Prognostic studies of gastric SRCC show that overall survival is determined by age, tumor site and stage at presentation. The poor prognosis may also be attributed different molecular pathways that are affected. Although recent studies have shown an effective role of adjunctive chemotherapy with surgical resection in prolonging survival in patients with SRCC, the mortality remains high. Additional studies of this subtype of CRC are warranted.

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PITUITARY APOPLEXY INDUCED TAKOTSUBO CARDIOMYOPATHY

Introduction: Takotsubo Cardiomyopathy (TTC), defined by transient myocardial dysfunction with patterns of regional hyperkinesis, commonly arises after an emotional or physical stressor and hypokinesis. Here, we report a case of TTC in the setting of pituitary apoplexy.

Case: An 82-year-old Male with a history of hypertension, hyperlipidemia, pituitary adenoma, and complete heart block status post permanent pacemaker was admitted to the emergency department after one week of malaise, nausea, and vomiting. The day prior, he developed a severe frontal pressure headache and intermittent blurry vision. His initial vitals were stable, but he later became lethargic and febrile to 101.6F with nonsustained ventricular tachyarrhythmias. EKG showed deep T-wave inversions in V2-V4 concerning for pathologic central nervous system T-waves. An echocardiogram revealed reduced left ventricular ejection fraction (LVEF) of 20% and akinesis of all mid-myocardial apical segments. Labs included troponin-T 0.56, lactate 5 mmol/L, and electrolyte derangements of magnesium 1.5 mEq/L, phosphorous 1.0 mEq/L, and potassium 3.2 mEq/L. In the cardiac care unit, his pacemaker settings were increased from 60 to 80 bpm due to QT prolongation and torsades de pointes on telemetry. Given his history of pituitary adenoma, an MRI Sella was performed and was notable for pituitary apoplexy and interval gland enlargement. Further labs revealed low cortisol, TSH, and testosterone. Stress dose steroids and levothyroxine were initiated and he underwent transsphenoidal resection. Following surgery and hormone initiation, he remained afebrile and no longer experienced his chief complaints. Repeat EKG and echocardiogram showed T-wave resolution in V2-V4 and LVEF 50-55%, respectively.

Conclusion: Takotsubo cardiomyopathy is caused by stressors, which in this case took the form of hormonal deficiencies linked to panhypopituitarism. Classified as a state in which the pituitary gland cannot make most or all hormones, panhypopituitarism could have contributed to TTC via two proposed pathways. First, in hypothyroid states there is less triiodothyronine directly affecting the transcriptional activation of cardiac enzymes involved with sodium-potassium ATPase pumps and calcium transport at the sarcoplasmic reticulum. Secondly, in adrenal insufficiency, inadequate cortisol levels can result in severe electrolyte disturbances including low phosphate levels. Phosphate depletion is associated with low intracellular supply of ATP, which is essential for myocardial contractility, and may have caused sudden heart failure in the patient. Through glucocorticoid supplementation, chemical gradients are restored to allow for excitation-contraction pathways and modulate the vascular response to beta-agonists. This case illustrates the need for physicians to examine correctable metabolic causes in the workup for TCC that include panhypopituitarism to ensure favorable outcomes.

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Acute fulminant myocarditis associated with COVID-19 Multisystem Inflammatory Syndrome in Adults

Introduction:

Coronavirus disease 19 (COVID-19) is known to be a cause of myocarditis (1), both in the acute phase and as part of the delayed multisystem inflammatory syndrome in adults (MIS-A). Interleukin 6 has been increasingly recognized as the cause. The time to onset of symptoms is variable and can present in patients with a positive test or during the late convalescent stage. Fulminant myocarditis occurs quickly and leads to severe heart failure or circulatory failure, with mortality rates ranging 50-70%. Hence early identification and initiating appropriate treatment is crucial for survival. Here we present a case of fulminant myocarditis in a young patient as a part of COVID-19 MIS-A.

Case presentation:

21-year-old male with no prior medical history presented with five days of fevers, chills, headaches, abdominal pain and myalgia. Social history was significant for high-risk sexual activity without alcohol and drug use. He had a week-long trip to Nigeria. On initial evaluation, he was febrile with a temperature of 102.5° F, heart rate 122 with oxygen saturation of 95% breathing room air, had no lymphadenopathy or mucocutaneous findings. He was started on intravenous fluids for presumed sepsis but developed respiratory distress and chest pain with worsening tachycardia. Troponin trended up. CT pulmonary angiogram was negative. Echocardiogram showed global hypokinesia with severely reduced ventricular systolic function. Patient continued to have chest pain, rising troponin and worsening hypotension despite initiation of norepinephrine and broad-spectrum antibiotics. Given concern for cardiogenic shock, he was started on dobutamine. Right heart catheterization showed bilateral elevated filling pressures and low cardiac output, with cardiac index 2.2 L/min/m2 despite being on inotropic support. Intubation and mechanical ventilation were needed. Infectious workup remained negative with the exception of COVID-19 nucleocapsid antibody indicative of past infection. Infectious disease was consulted, methylprednisolone and intravenous immunoglobulin were started for possible MIS-A. He responded rapidly to treatment and could be extubated. A repeat echocardiogram showed recovery of cardiac function. Patient was discharged with a plan to follow up with eventual cardiac MRI.

Discussion:

MIS-A is still a rare entity but clinically significant usually involving the gastrointestinal tract, kidney and reticuloendothelial system. Few have been reported to have cardiac, hematologic and neurological involvement. It is still unclear whether myocarditis is an indirect complication or direct cardiac manifestation of coronavirus. Patients are too sick to have the diagnosis based on MRI or endomyocardial biopsy (4), or expertise may not be available. They are thus diagnosed when the electrocardiogram and the cardiac biomarkers are available and there is low likelihood of the acute coronary syndrome (5).

We emphasize the importance of multidisciplinary care and early recognition of the possibility of the syndrome and order antibody testing. Glucocorticoid and IVIG proved beneficial in our patient.

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IR Guided Suction Thrombectomy in a 94 Year Female with Pulmonary Embolism at a Community Hospital

Introduction:

Treatment of intermediate risk, submassive pulmonary embolism (PE) in the elderly population requires careful consideration of risks and benefits. The risk of intracranial hemorrhage is thought to increase with age and comorbid conditions limit the use systemic thrombolysis/fibrinolysis. Mechanical interventions such as catheter-directed thrombectomy and cardiothoracic surgery are often not available at rural or community hospitals. We present the case of suction thrombectomy via interventional radiology as a viable solution in a patient with contraindications at a community hospital.

Case Description:

A 94-year-old female presented with shortness of breath secondary to a suspected pulmonary embolism. This was the second PE for the patient in 10 months, which she was previously treated for 6 months of rivaroxiban. She stopped due to theoretical risk of bleeding. On admission, D-Dimer was elevated over 9000, a pathognomonic S1Q3T3 was found on EKG, and echocardiogram demonstrated a severely reduced right ventricular function and severely elevated pulmonary arterial systolic pressure in addition to McConnel's sign. The confirmatory CTA was not able to be done due to declining renal function and ventilation perfusion scan was unavailable. It was decided by family and patient to not use systemic thrombolysis given risks at the age of 94. Patient was admitted to the intensive care unit, started on weight based lovenox, placed on vasopressor norepinephrine and high flow nasal cannula. Despite treatment, kidney function began to decline and oxygen requirements increase. At that point, inotrope dobutamine was added at fixed rate and INOmax inhaled nitric oxide was started through the high flow nasal cannula, which resulted in patient improvement. At that time, the decision was made to pursue IR guided suction thrombectomy. Pre-procedure, the patient was continued on maximum ventilatory nitric oxide to lower pulmonary pressures to adequate levels required for suction thrombectomy. Initial pulmonary arteriogram revealed complete occlusion of the pulmonary artery superior segment of the right upper lobe. Post-procedure, the right upper lobe was revascularized and pulmonary arterial systolic pressure reduced. The patient reported symptomatic improvement and no longer required oxygen supplementation after a few days.

Conclusion:

Large pulmonary emboli require prompt diagnosis and rapid surgical intervention has been shown to improve outcomes. Systemic or catheter directed thrombolysis is typically first line treatment for intermediate and high-risk pulmonary emboli without hemodynamic contraindications. Thrombectomy is also an option for patients with contraindications. However, thrombectomy has been typically reserved for experienced tertiary and quaternary centers by cardiothoracic surgery or interventional pulmonology. Suction thrombectomy is a procedure that can be done by interventional radiology in community settings when hemodynamic instability make transport to experienced centers difficult. Therefore, IR guided suction thrombectomy is a viable option for community hospitals when timely intervention is required.

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Hyperimmune state after second Dose of the Pfizer-BioNTech COVID-19 Vaccine in a patient with pANCA positive vasculitis

Introduction

Microscopic polyangitis is an autoimmune, systemic vasculitis that presents most commonly with glomerulonephritis, and occasional pulmonary involvement later in the disease course. We present the case of a patient with pANCA positive vasculitis with acute renal failure and pulmonary hemorrhage after receiving the COVID-19 vaccine.

Case Presentation

A 75 year old female with a history of untreated pANCA positive vasculitis presented to the emergency department with new onset shortness of breath one week after the second dose of the Pfizer-BioNTech COVID-19 vaccine, and four months after a self-resolved episode COVID-19. Patient at baseline required no supplemental oxygen, reported no orthopnea, reported no exertional dyspnea, and had stable chronic kidney disease. On presentation, patient was in acute respiratory distress and demonstrated signs of acute kidney injury. In addition, the patient had hemoptysis and imaging was consistent with pulmonary hemorrhage. Worsening hypoxemia and deteriorating renal function led to pressure-controlled mechanical ventilation and emergent hemodialysis. Pulse dose methylprednisolone and rituximab were administered and broad-spectrum antibiotics were given for possible infection. Patient was transferred to a teirtary center for plasmapheresis.

Discussion

There have been case reports of COVID-19 infection causing new presentation of ANCA-associated vasculitis and acute exacerbation of existing ANCA-associated vasculitis. SARS-COV-2 has been theorized to promote the release of pro-inflammatory mediators such as IL- 1Î², IL-6, IP-10. TNF, INF-Î³, MIP 1Î± and 1Î², and VEGF among others. The American College of Rheumatology has specialized guidance for COVID-19 vaccination in patients with rheumatic and musculoskeletal disease. For patients on chronic rituximab therapy, the ACR recommends COVID-19 vaccination one month prior to the next scheduled dose of the rituximab cycle and resumption of rituximab 2-4 weeks after the second COVID-19 vaccination dose. Such guidelines are to promote sufficient immune response to vaccination and were based on immune response to the influenza vaccine. The fact that our patient was noncompliant with rituximab therapy, did not have exaggerated immune response to actual COVID-19 infection, and likely had an exacerbated immune response to mRNA vaccination deserves further investigation.

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Atezolizumab-associated toxic epidermal necrolysis

Checkpoint inhibitors have been approved for treatment of various malignant conditions. Rare side effects are increasingly being noted. We present a case of PD-L1 inhibitor-associated diffuse skin eruption.

70-year-old male with metastatic small cell lung cancer, on active treatment with carboplatin, etoposide and atezolizumab for 12 weeks presented to the hospital with 2 days of diffuse blistering skin rash and sloughing predominantly involving the groin and lower extremities. The last cycle of chemoimmunotherapy was administered two weeks prior to presentation. The patient was admitted to the burn intensive care unit and was noted to have severe necrosis of the skin of the groin and underwent debridement. Lab analysis revealed creatinine kinase of 8319 U/L and creatinine of 1.9mg/dl, from a baseline of 1.4 mg/dl. The patient was placed on broad spectrum antibiotics and received aggressive intravenous fluid resuscitation. A punch biopsy of the skin showed necrosis of the epidermis and effacement in predominantly a subepidermal process. There was modest dermal perivascular interstitial inflammation with infiltration of lymphocytes and neutrophils. Given extensive skin involvement of greater than 30% body surface area, a diagnosis of toxic epidermal necrolysis (TEN) was made and was therefore placed on intravenous immunoglobulin and hydrocortisone. The rash resolved over the course of 6 weeks with continued aggressive wound care and withholding of further atezolizumab.

Atezolizumab is a monoclonal antibody that binds to PD-L1 and acts as a checkpoint inhibitor. PD-L1 inhibitors can result in T-cell mediated self-directed cytotoxic inflammatory reactions, including of the skin (1). Most reported cases of checkpoint inhibitor associated TEN have been seen early in the treatment course, with a median time of onset of 3 to 6 weeks (1,2). Our patient's presentation after 12 weeks of therapy suggests that there may be a delayed recruitment of self-directed T cells. If identified early, treatment involves prompt discontinuation of the offending agent (3). Immunomodulation with plasma exchange and steroids may be considered in severe cases (1). Aggressive wound care and management of volume status are critical. Our case demonstrates the importance of maintaining a high index of suspicion for TEN with checkpoint inhibitor use and new onset of a skin rash as prompt discontinuation of the offending agent and aggressive wound care may improve outcomes.

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Resident/Fellow Clinical Vignette

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A UNIQUE CASE OF PERINEPHRIC ABSCESS

Introduction:

Perinephric abscess is defined as an accumulation of suppurative material between renal capsule and Gerota's fascia. The incidence of perinephric abscess is 1-10 per 10,000 hospital admissions and the average size of the perinephric abscess is 4.95cm. We present a rare case of perinephric abscess of size 12×11×13 cm that was also suspicious for malignancy.

Case description:

32 y/o gentle man with no prior medical history presented with increased urine frequency for 2 weeks, fever, chills, nausea, vomiting and left flank pain for 1 day. On examination, vitals were- temperature 102.9F, BP-152/87 and HR-120/min. Patient had a neutrophilic leukocytosis of 23.6×103/ul, elevated creatinine of 1.40mg/dl (baseline of 0.9mg/dl) and urinalysis with positive leukocyte esterase and negative nitrites. Renal CT without contrast showed 12×11×13cm cystic lesion at the upper and mid left kidney with septation superiorly and perinephric fat stranding and surrounding edema. The initial differential diagnosis was- complicated UTI, malignancy and nephrolithiasis. The patient was started on intravenous Piperacillin-Tazobactum, intravenous Promethazine, intravenous Ketorolac and other as needed analgesics and antiemetics in spite of which his symptoms persisted. US guided drainage and pigtail catheter insertion was performed on Day 2 of admission after which patient's condition started to improve. Gram stain of the fluid drained was negative for microbial growth and showed only moderate Wbcs. Cytology was negative for malignancy. Repeat CT Abdomen/Pelvis with contrast revealed an irregularly shaped complex and partially decompressed cyst. Leukocytosis resolved by Day 4 of admission. Intravenous Piperacillin-Tazobactum was continued for 2 weeks and patient was discharged after a 7-day stay. Nine days post discharge CT scan abdomen/pelvis with IV contrast showed reduction in size of the abscess to 4.8×3.8×2.7 cm.

Discussion:

Perinephric abscess often occurs in patients with predisposing conditions such as nephrolithiasis, diabetes mellitus, renal structural abnormalities, pregnancy or UTI. Our patient only had features of cystitis without any predisposing factors. Given the size of the cystic lesion and absence of significant risk factors for UTIs, malignancy was considered. CBC 1 month prior showed Hb-19.7 and RBC- 6.91 × 106/ul, which retrospectively suggest considering an erythropoietin secreting renal malignancy. Fortunately, fluid cytology was negative for malignant cells. The most common causative organism for perinephric abscess is E.coli. This patient's fluid, blood and urine cultures were unremarkable for microbial growth. Literature suggests the mean size of a perinephric abscess is 4.95+/- 2.99 cm (range 1-15 cm). On review of the literature interventional treatment approach results in better clinical outcome and the same was applied to this case.

Resident/Fellow Clinical Vignette

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A Case of Mycobaterium abscessus nodular skin lesions following Lipodissolve injections

Introduction:

Mycobacterium abscessus (MA) is an acid-fast, rapid-growing, non-tuberculous mycobacteria (NTM). Nosocomial infections can be caused by direct inoculation of contaminated substances, with an incubation period as short as one week for rapid growers. We present a case of MA that was part of a small outbreak.

Case:

A 53 year-old healthy female underwent Lipodissolve â„¢ injections (phosphatidyl choline in deoxycholate solution) in both arms. She presented a week later with 15 painful nodular skin lesions at the sites of injection (Figure 1) with no systemic symptoms. She was treated with trimethoprim-sulfamethoxazole for 10 days without improvement. Incisional biopsy showed deep dermal suppuration and inflammation with numerous neutrophils and granulomas (Figure 2). Acid fast culture was positive for MA (Figures 3,4). She was started on moxifloxacin/clarithromycin then referred to infectious diseases. The isolate was sensitive to amikacin and clarithromycin, intermediate to cefoxitin, and resistant to linezolid, imipenem, doxycycline, moxifloxacin, and ciprofloxacin. Other agents tested included bedaquiline(MIC 0.25 mcg/ml) and tigecycline (MIC 0.5 mcg/ml), while tedizolid (MIC 4 mcg/ml), omadacycline(MIC 2.0 mcg/ml) and clofazamine(MIC 1.0 mcg/ml) were considered unlikely to be effective. Her regimen was modified to amikacin, azithromycin, and bedaquiline. Within 2 weeks, most lesions had flattened out. Four more cases of MA skin infections following Lipodissolveâ„¢ injections have been reported from the same practice, within several weeks. The provider denied using single-use vials more than once.

Discussion:

NTM are highly abundant in the environment and resistant to certain antiseptics and sterilizing agents. Outbreaks of MA skin and soft tissue infection (SSTI) in immunocompetent hosts have been reported following cosmetic and surgical procedures such as abdominalplasty[1], liposuction[1-3], tattoo[4, 5], acupuncture[6, 7], and surgical wounds[8]. NTM should be included in the differential diagnoses for subacute or chronic nodular infections, especially if refractory to the conventional antibiotic regimen for SSTI. It is essential to send tissue samples for AFB and fungal smear and culture to establish the diagnosis. It is also crucial to maintain constant communication with microbiology lab as susceptibilities are often not done until requested. MA is inherently resistant to isoniazid, rifampin, ethambutol, and can develop resistance to macrolides due to the presence of the inducible macrolide resistance gene, erm. Thus, empiric therapy for MA can lead to inadequate therapy. In our case, because of the multi-drug resistance pattern, we used bedaquiline as part of the regimen. As both bedaquiline and azithromycin can prolong the QT interval, periodically monitoring electrocardiograms is prudent. Investigation by the FDA and local health department is vital in identifying and potentially limiting outbreaks.

Resident/Fellow Clinical Vignette

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A CASE OF COVID-19 ASSOCIATED WITH COLD AGGLUTININ ANTIBODIES AND PULMONARY EMBOLISM

Introduction: Novel coronavirus disease 2019 (COVID-19) has been associated with hematologic manifestations including disseminated intravascular coagulation, hypercoagulability, and hemolytic anemia. Several case reports have also described the presence of cold agglutinins, which are autoantibodies against RBC antigens that cause hemolysis at less than physiologic temperatures. We present a case of COVID-19 infection in a patient who was incidentally found to have cold agglutinins and subsequently developed pulmonary embolism (PE).

Case: A 49-year-old woman with a history of remote lower extremity DVT presented with fevers, dyspnea, and hypoxia in the setting of recent positive COVID-19 testing nine days prior to admission. She underwent CT chest angiogram, which was negative for PE. She was treated for severe COVID infection with remdesivir and dexamethasone. She was also treated with prophylactic enoxaparin. On the fifth hospital day, CBC with differential showed cold agglutination of red blood cells. Concurrently, her hemoglobin dropped from 12.3 to 9.5 g/dL, but her total bilirubin, haptoglobin, and reticulocyte counts remained normal suggesting absence of significant hemolysis. Direct Coombs testing was positive for C3 and negative for IgG. Cold-agglutinin titer later came back at 1:64. On the sixth hospital day, she developed worsening chest pain and CT angiogram was repeated. This time it showed evidence of bilateral segmental and subsegmental PEs. Her enoxaparin was increased to therapeutic dosing. Follow-up blood work during hospitalization and five months later showed no obvious signs of hemolysis and her hemoglobin eventually stabilized.

Discussion: Cold agglutinin disease (CAD) can cause autoimmune hemolytic anemia (AIHA), which is a rare phenomenon. It can be idiopathic or secondary to an underlying infection, malignancy, or autoimmune disease. Most cold agglutinins are clinically insignificant as they do not cause meaningful hemolysis at body temperature. However, cold agglutinin related AIHA in COVID-19 patients has been correlated to increased mortality in the setting of delayed diagnosis and subsequent multiorgan failure. Cold agglutinins have also been implicated in renal replacement therapy circuit failure, highlighting the importance of identifying them. Our patient presented with a mild case of CAD with no significant hemolysis, but later developed bilateral PEs. Prior case reports have described CAD contributing to thrombotic events in the setting of AIHA. It is unclear if this patient's PE was related to the presence of cold agglutinins, but it brings up several clinical insights.

Conclusion: The incidental presence of cold agglutinins on routine CBC with differential may be an important clue to prompt further laboratory assessment including hemolysis workup. It is also useful to remember that most cold agglutinins are clinically insignificant at body temperature. Furthermore, COVID-19 infection should be considered as an underlying etiology for diseases such as CAD, AIHA, and thromboembolism. Consequently, these patients should be monitored closely for potential complications.

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Lepromatous Leprosy Worsened by Immunosuppressive Treatment of More Common Diseases: A Case Report

Lepromatous leprosy is a chronic granulomatous infectious disease that affects multiple organ systems, and is caused by the slow-growing acid-fast bacillus Mycobacterium leprae. Clinically, lepromatous leprosy causes hypo-pigmented skin lesions associated with sensory deficits, and can spread to involve the eyes, nose, kidneys, testes, and bone. More common causes of similar symptoms with systemic manifestations include chronic inflammatory demyelinating polyradiculopathy, vasculitides, sarcoidosis, and amyloidosis, amongst many others. We report the case of a 63-year-old man with a twelve-year progression of neuropathic symptoms, whose clinical course worsened with immunosuppressive therapies.

A 63-year-old Bangladeshi male with a history of hyperlipidemia and type 2 diabetes, presented to our outpatient clinic after 12 years of progressive numbness and weakness of his hands and feet. He reported his symptoms originally starting in his toes, moving up his legs to his knees, followed by a similar spread of numbness up his fingers to his elbows. Since 2015, the patient received multiple treatments of IVIG over the course four years, without any change in his symptoms. During that time, he completed electromyography and nerve conduction studies, which were consistent with severe chronic sensorimotor polyneuropathy with active denervation. He had a complete rheumatologic workup, MRI brain, PET scan, and lumbar puncture, all of which were unremarkable. He then underwent a skin biopsy to evaluate for possible underlying vasculitis, which was inconclusive. The patient was ultimately started on prednisone and mycophenolate mofetil, after which he presented with new development of cystic and nodular lesions over his body, face, and ears. The lesions were associated with deformation of his fingers and feet. He was sent for sural nerve biopsy which revealed the presence of severe endoneurial and perineurial fibrosis, in addition to chronic inflammation associated with acid fast bacilli, consistent with neuropathy due to lepromatous leprosy. He was subsequently started on dapsone, clofazimine and rifampin with improvement in his symptoms.

Lepromatous Leprosy, while rare, is important to consider in patients who present with slow-progressing polyneuropathy who have had otherwise negative neurologic and rheumatologic workup. This diagnosis is easily disregarded in the United States, as there are only 150-250 new cases annually, however, there are roughly 200,000 new cases per year globally. The lepromatous subtype is the systemic variant of the two major subtypes, which spreads to involve the skin and multiple organ systems, and can cause high disease burden. With the more widespread use of biologic and immunosuppressive therapies, early identification of leprosy is important, as treatment of more common differentials can worsen clinical outcomes.

Resident/Fellow Clinical Vignette

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A rare case of Osimertinib-Induced Cardiomyopathy

Introduction:

Osimertinib is the drug of choice for metastatic non-small cell lung cancer (NSCLC) and it inhibits signaling pathway and epithelial growth factor mutations. Cardiotoxicity including arrthymias, atrial fibrillation, and heart failure has been reported as an extremely rare incidence. We report a rare case of osimertinib induced cardiomyopathy and highlight the importance of close echocardiogram surveillance to monitor for cardiotoxicity.

Case:

78-year-old female with history of lung nodule for the past ten years presented to the ED with dyspnea, ten pound weight loss during the past six months and CT chest showed large left lower lobe lung mass. CT-guided biopsy revealed metastatic NSCLC with positive epithelial growth factor receptor (EGFR) mutation. She was started on osimertinib 40 mg/day. Cardiac echocardiogram, three months prior to starting osimertinib was normal with EF 60% with mild mitral and tricuspid valve regurgitation. The patient had multiple hospitalizations after starting chemotherapy due to orthopnea and exertional dyspnea with hypoxia. During these hospitalizations the patient required bilevel ventilation, broad-spectrum antibiotics for community-acquired pneumonia, diuretics for fluid overload and ICU level care due to critical illness. Repeat echocardiogram six months after starting osimertinib revealed severely reduced EF<25%, severe mitral valve regurgitation and moderate to severe pulmonary hypertension with pulmonary artery pressures> 60mmHg. The patient underwent right and left heart catheterization, and found to have 70% lesion in left anterior descending artery, EF<15% and severe global hypokinesia. There was no angiographically significant disease in left main coronary artery and circumflex right coronary artery. The patient was diagnosed with osmertinib induced cardiomyopathy and was started on sacubitril/valsartan, carvedilol and spironolactone. Osimertinib was stopped immediately due to cardiotoxicity. The patient followed up with cardiology as an outpatient; repeat echocardiogram showed improved cardiac function after stopping osimertinib.

Discussion:

We report a case of osimertinib induced cardiomyopathy. Osimertinib is a third-generation EGFR inhibitor and it is approved for metastatic NSCLC. Osmirtinib induced cardiomyopathy is a rare incidence 3.7%, however, the underlying mechanism of osimertinib-related cardiotoxicity remains unclear and there is little information is available regarding its pathophysiology. (1) The patient in this case had normal cardiac function before starting osimertinib, with subsequent decline in ejection fraction within six months of starting osimertinib. The patient had consistent findings of heart failure on echocardiogram and physical examination. The patient improved after discontinuing osimertinib and continues to follow with cardiology as an outpatient with improvement in overall functionality. As chemotherapeutic agents continue to evolve with better survival outcome, it is important to quantify and identify cardiotoxicity and follow patients very closely with surveillance echocardiogram as per guidelines.

New York Chapter
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Resident / Fellow Research

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Lenox hill Hospital

Attitudes Towards COVID-19 Vaccination in the People Living with HIV (PLWHIV) at an Urban Primary Care Center

Introduction:

With the arrival of a novel vaccine against COVID-19 infection, attitudes have been mixed regarding its public reception. In New York City alone, COVID-19 vaccine hesitancy was estimated to be as high as 42% in January of 2021. For this reason, we aimed to analyze attitudes and COVID-19 vaccine behavior of people living with HIV (PLWHIV), a patient demographic particularly susceptible to COVID-19. We conducted this study at an urban primary care center in New York City that delivers HIV specialized care.

Methods:

A total of 226 patients were contacted by phone or approached during their office visit and asked survey questions with their consent. Analysis was performed to determine if certain factors, like medical comorbidities, race, sex, age, smoking status, sexual preference, highest level of education, and viremic control were predictive of attitudes towards COVID-19 vaccination among PLWHIV. Patients who initially refused the vaccine were provided with additional resources as well as individualized counseling and vaccine behavior was then re-assessed.

Results:

Data from the 226 patients surveyed showed that nearly 75% of patients intend to become vaccinated or have already received at least one dose of the COVID-19 vaccine. Of the patients who refused the vaccine, the largest deciding factor was hesitancy towards the COVID vaccine in particular, and not towards vaccines in general. Only 16% of patients surveyed reported being diagnosed with a natural COVID-19 infection, and nearly half identified as current or prior smokers. The demographics most represented in this data set self-identified as: Black (53%), Hispanic or Latinx (23%), White (17%), Asian or Pacific Islander (2.3%), and Other (4.2%). Finally, and perhaps most compelling, of the patients who were unsure about being vaccinated, nearly one third agreed to the COVID-19 vaccine after receiving additional education about risks and benefits of the vaccine from a clinician.

Discussion:

Overall, compared to the general population, people living with HIV were ultimately more likely to pursue being vaccinated against COVID-19, and further information sharing led to a reduction in vaccine refusal. These results illustrate the importance of clinicians engaging in honest and non-judgmental conversations with people living with HIV about vaccine hesitancy in general, but also especially as it pertains to the COVID-19 vaccine.

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Differences in Healthcare Outcomes Based on Race among Patients Admitted with Acute Myocardial Infarction to Teaching Hospitals: A Nationwide Analysis

We sought to study the differences based on race in outcomes amongst patients with acute myocardial infarction (AMI) admitted to teaching hospitals.

We used the national inpatient sample (NIS) 2016-2018 to identify patients with an AMI (STEMI and NSTEMI) who were admitted to teaching hospitals in the US. Cohorts were identified based on ICD-10 codes and were weighted using an algorithm provided by the NIS which allows for national estimates. End points of mortality, length of stay (LOS), hospital charges, hospital outcomes, and differences in coronary intervention were evaluated and compared among whites vs. non-whites.

A total of weighted 407,741 (31.26%) non-whites and weighted 896,613 (68.74%) whites with AMI in teaching hospitals were evaluated. In non-whites and whites, average age was 63.99 +/-13.59 & 67.65 +/-13.09 and majority were male (60.82% & 64.10%), respectively. Baseline comorbidities were equal among both cohorts; however, non-whites were more likely to have anemia, acute renal failure, and cocaine use disorder. Non-whites were less likely to receive percutaneous coronary intervention (PCI) (47.03% vs. 52.24%) with a longer time to PCI [adjusted mean difference (aMD) 0.11 day] and coronary artery bypass graft (CABG) (aMD 0.62 days; $p<0.05$) after adjusting for variables that may delay coronary procedures. Moreover, non-whites had a lower odd of receiving coronary intervention (aOR 0.60; $p<0.05$). In addition, non-whites had a longer LOS (aMD 0.12 days) and higher hospital charges adjusted for inflation (aMD $5,145.9; $p<0.05$); however, there were no significant differences in mortality [adjusted odds ratio (aOR) 0.98]. Likewise, non-whites were more likely to use Medicaid and have a lower household income based on zip code. In a subgroup analysis of AMI, in attempt to control for socioeconomic determinants of health, among the highest median income quartile based on zip code admitted to teaching hospitals, non-whites continued to have a longer time to PCI (aMD 0.08 day), higher hospital charges (aMD $7692.03), longer LOS (0.29 day), and lower odds or receiving coronary intervention (aOR 0.54; $p<0.05$).

Racial disparities among non-whites and whites in healthcare have been highlighted previously and may exist due to differences in available access to resources. Our study shows that non-whites with AMI who presented to teaching hospitals were more likely to be in the lower median income quartile based on zip code, rely on Medicaid or self-pay, have longer times to PCI or CABG, and have higher hospital charges. Some of these variable differences among whites vs. non-whites remained the case even after analyzing AMI in the highest income quartile in an attempt to control for socioeconomic determinants of health. While overall, the mortality in the two groups was no different, our results still highlight areas for improvement in AMI management.

Christopher Showers

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Patient Perceptions of Telemedicine for Primary Care in Queens, New York City

Background: The Covid-19 pandemic has severely restricted patient access to primary healthcare services [1]. Telemedicine, the use of telecommunications technology to provide healthcare remotely, emerged as a means to connect patient with primary care providers and was quickly adopted by many primary care clinics [2]. Despite rapid adoption, patient perceptions of telemedicine used for primary care in areas with high Covid-19 incidence rates remain unclear.

Objective: To assess perceptions of telemedicine used for primary care among patients at an urban patient-centered medical home in Queens, New York City.

Methods: Following an initial telemedicine primary care encounter performed between 03/01/2020 and 05/29/2020, patients over 18 years of age were contacted via telephone and asked to participate in a brief survey. Survey questions assessed familiarity with telemedicine and perceptions of multiple values using Yes/No and Likert Scale questions. Survey questions are listed in Figure 1.

Results: A total 64 patients (average age 54+16 years; 70% female; 8% White, 27% Asian, 27% Black, 38% Latinx) participated in the survey. Though only 42% of participants were familiar with telemedicine as a healthcare delivery strategy, greater than 90% indicated both that they were able to speak freely about their health concerns and that they would participate in telemedicine again. Greater than 90% of participants felt comfortable or very comfortable with their telemedicine encounter, and greater than 88% believed that the encounter was convenient or very convenient. Greater than 72% indicated that they were not concerned or not at all concerned about privacy during the encounter. Importantly, greater than 85% indicated that they believed that telemedicine protected them from exposure to SARS-CoV-2. Overall, greater than 85% of survey participants indicated that they were satisfied or very satisfied with the telemedicine encounter. Data are summarized in Figure 2 and Figure 3.

Conclusions: These results demonstrate positive opinions of telemedicine used for primary care during the Covid-19 pandemic among patients at an urban patient-centered medical home, and therefore support further use of telemedicine to conduct primary care during periods of restricted access to traditional healthcare services.

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New York Chapter
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Resident / Fellow/Medical Student
Quality- Patient Safety

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IMPROVING WARFARIN SAFETY AMID CLINIC MERGER DURING COVID-19 PANDEMIC

Purpose:

A major gap identified was in warfarin management during the pandemic in a newly merged clinic. The aim of this quality improvement (QI) project is to increase TTR (Time in Therapeutic Range) to 65% from baseline of 50% and to maintain monthly INR completion rates of 80% in patients on warfarin at a newly merged academic, primary care clinic within six months. The COVID-19 Pandemic has impacted INR testing, leading to increased risk of bleeding and clotting.

Methods

A multidisciplinary QI team utilized PDSA method for this QI. There are 66 residents, 7 attending physicians and 25 nursing and ancillary staff in the clinic. This patient population is underserved and increased to 140 from 60 on warfarin after clinic merger. The QI project was implemented from Nov.2020 â€“ April 2021. Root cause analysis was performed to identify barriers to optimal anticoagulation management. Major barriers comprised of knowledge gap in patients and providers, fear of blood draws, lack of POC INR and lack of interoperable electronic health records. The team used various QI tools including stakeholder analysis, process flow map and driverâ€™s diagram to address identified barriers. Outcome measures included percentage of TTR, number of bleeding episode and INR over 5. Process measures comprised of INR completion rates, percentage of patients enrolled in the warfarin database, and providerâ€™s attendance rates in educational sessions. Balancing measures included nursing and provider satisfaction. We completed 5 PDSA cycles. Major components of PDSA cycle included providers, nursing staff and patientsâ€™ education, creation of electronic patient registry, patient agreement, enhancement in electronic note templates and patient outreach. Data analysis was performed by monthly run charts.

Results:

There was a sustainable increase monthly INR completion rates from 72% to 87% within 6 months. There was an initial decline in initial monthly TTR, but it later improved from 53% to 55%. There was also a steady increase in trajectory TTR to 57% after initial decline at the start of the merger. There were no bleeding episodes during the last 4 months. 100% (n=143) of the patients were added to Warfarin database. Residentsâ€™ pre and post-tests demonstrated drastic improvement in knowledge from 57% to 83%.

Conclusions:

Multi-faceted strategies improved safety of warfarin with successive increase in TTR and increased INR completion rates. Engagement of the multidisciplinary team was crucial for the success. Future directions include identification and addressing health care disparity in patients on warfarin and DOACs and integration of POC INR testing and pharmacist in the clinic.

Quality

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Decreasing the Need for CT Pulmonary Angiograms in COVID Positive Patients using D-Dimer Levels

Background

Patients presenting with hypoxia and COVID-19 are at increased risk for pulmonary embolism (PE). Elevated D-dimers are a hallmark feature of severe COVID-19 infection, regardless of presence of PE. Clinicians tend to obtain CT Pulmonary Angiograms (CTPA) in a significant proportion of patients based on these 2 variables alone. We observed that this practice has led to over-utilization of CTPAs which increases resource utilization, hospital costs, risks of contrast-induced nephropathy, and chances of viral spread during transport.

Objective

The specific aim of this project is to decrease over-utilization of CTPAâ€™s in patients admitted with COVID-19 without negatively impacting patient care, with goal of decreasing negative CTPAâ€™s by 25%.

Methods

This project was completed at a community hospital in a Queens, NY. Sixty beds dedicated to COVID-19 patients during project.

The initial intervention was to educate clinicians about elevated rates of negative CTPAs in COVID-19 patients and encourage the use of other clinical factors before ordering CTPA, including Wells Score, D-Dimer levels, and lower extremity doppler results. Nurses and respiratory therapists advised to notify residents of desaturation or tachycardia. Inflammatory markers, including D-Dimer levels, collected every 48 hours. Thromboprophylaxis started on all patients with suspected or confirmed COVID-19. Interdisciplinary communication during daily patient rounds.

During initial intervention, D-dimer values and CTPA results were collected in order to determine a D-dimer value that was sensitive for predicting PE in patients with COVID-19. Patients were excluded if admitted directly to the MICU, younger than 50 y/o, or had a negative COVID-19 swab. For the initial phase of our project, presence of venous duplex results were not taken into account.

Results

A total of 68 patients with COVID-19 infection had a CTPA with a corresponding D-dimer value within 48 hours. The median (min, 25th percentile, 75th percentile, max) D-dimer for the group was 2,026 ng/mL (220; 1,193; 4,730; 106,004). CTPA was positive for PE in 11 (16%) patients.

Cut-point analysis to determine the D-dimer level with maximum sensitivity for predicting PE on CTPA revealed a D-dimer of 1,710 ng/mL provided a sensitivity of 1.00 and a specificity of 0.49, with AUC of 0.75. If one had used a cutoff D-dimer value of 1,710 ng/mL to determine which patients would have had a CTPA, 27 (40%) patients would not have undergone a CTPA, and no PE would have been missed.

Quality

Conclusions

Over-utilization of CTPA has been observed among COVID positive patients with hypoxia regardless of D-dimer levels. We found that a D-Dimer level of 1,710 ng/mL provided 100% sensitivity and 49% specificity in diagnosis. Over the next 60 days, our plan is to validate results in an independent sample at another institution and analyze additional factors that could enhance specificity of our results.