To Friends of the Department of Medicine

Our program continues to train the best and brightest residents in the country. As we complete another challenging academic year marred by the ongoing fight against gun violence in Philadelphia and across the United States, the opioid pandemic and the ever present threat of another COVID-19 surge our residents have continued to rise to every challenge and thrive as physicians. During the three years we are fortunate to have them at our program, it is my distinct pleasure to watch them grow into confident, dedicated, compassionate clinicians. The residents are not just outstanding clinicians but excel in all aspects of medicine including research, humanities and medical education.

This will be my last year writing to all of you as the Program Director. As I move on to new career challenges, I leave Jefferson with hope and gratitude for the future of medicine. The exceptional altruism and dedication portrayed by our colleagues is unmatched. Despite the challenges, they have treated every patient with grace and poise working alongside faculty to support the Jefferson mission: We Improve Lives. In the midst of the chaos, our residents have still completed research, quality improvement projects and contributed to the humanities. This publication is just one example of the passion, dedication and creativity our residents continue to provide to the Jefferson Community.

This journal, now in its 23rd edition, continues to exemplify the perseverance, inquisitiveness and talent of our Internal Medicine residents. Congratulations to the Editors and all of the residents who contributed to another amazing edition of the Forum. I hope you will enjoy reading it!

Emily Stewart, MD, FACP

Associate Professor of Medicine

Program Director Internal Medicine Residency
FROM THE EDITORS

Dear Students, Residents, Fellows, Faculty, and Friends of the Forum,

It is our honor to present the 23rd Annual edition of The Medicine Forum to the Jefferson community. With all the uncertainties and stressors that exist both inside and outside of the hospital walls, it is truly remarkable to see the quality of contributions by our authors. From the multitude of case reports to the collection of artworks, our residents have generated an array of unique and interesting work. We hope that this edition will continue to honor the tradition of The Medicine Forum, which is the celebration of scholarly activity among physicians in training at our institution.

Sincerely from the Editorial Board,

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Abdul Kazi, MD
Matthew DiMeglio, DO, MBA

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To the Residents,

The four of us have had the unique opportunity to work alongside you and watch you grow throughout the last three years. During an extremely turbulent time at Jefferson, in the city of Philadelphia, and the larger healthcare landscape you all have been the constant presence that has kept the Jefferson IM residency culture alive.

Jefferson residents are: Compassionate, intelligent, dedicated, fun, and resilient. But most of all, Jefferson residents look out for each other and have each other’s backs. This is the culture that brings people to our program and the culture that makes residency training which would otherwise be grueling into a manageable, and at times, fun experience. You all have embodied this culture, and we as well as the residency program are so grateful to each and every one of you.

While it is sad that we are now parting ways, we are so proud to have called you all colleagues during these crazy years, and we could not imagine a better group of people to have gone through this with than you. It’s been an honor and a privilege to be your Chieves this year. We cannot wait to work alongside you and see all that you will accomplish in your careers. Cheers to you all!

Sincerely,

Cristina Angelo, MD
Katie Duffey, MD
Evan Nardone, MD
Michelle Perkons, MD
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 Challenges of Managing Giant Cell Myocarditis: A Case Report on the Mechanical Support Perspective

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INTRODUCTION

Giant cell myocarditis is a rare and fatal disease which may result in heart failure, complete heart block, or ventricular arrhythmias. We describe a patient who previously had been discharged from our institution with a left ventricular assist device and immunosuppressive therapy for management of his giant cell myocarditis. His subsequent course was complicated by further deterioration of heart function which required multiple mechanical circulatory support devices. He successfully received a heart transplant which later had recurrence of giant cell myocarditis. This case highlights the challenges of left and right sided mechanical assist devices in managing giant cell myocarditis.

CASE PRESENTATION

AG is a 33-year-old Hispanic man with a history of heart failure with reduced ejection fraction secondary to giant cell myocarditis implanted with a HeartMate3 left ventricular assist device (LVAD) who presented after experiencing low flow alarms. The patient presented with a three-week history of diffuse abdominal pain and low-flow alarms on his LVAD. Device parameters were as follows: flow of 3.3 L/min, speed of 5500 rpm, PI of 4.2, and Power of 3.9 Watts. Echocardiography was significant for right ventricular dilation and hypertrophy and severely decreased right ventricular function. CT abdomen/pelvis showed hepatic venous congestion and a thick-walled descending colon. There were new findings of fluid overload with new pleural effusions, ascites, and anasarca. The patient was admitted due to worsening right heart failure and increasing inotrope requirements. He also tested positive for SARS COV-2 on admission PCR testing despite being asymptomatic and afebrile.

Right heart catheterization showed RA pressure of 27mmHg, PA 34/23, PCWP 23 with CI of 1.57, CO of 2.87, PVR of 1.04, and SVR of 2033. Milrinone dose was increased and dobutamine was added. The patient continued to clinically deteriorate so an Impella RP was placed One day after placement of the device, AG had darkening of his urine, elevated LDH, and decreased hemoglobin and platelet count, consistent with device-induced hemolysis. A subsequent right IJ angioplasty was performed to allow for placement of a Protek Duo. The patients undocumented immigration status had in the past resulted in limited opportunities for transplantation, however acquisition of health insurance since then allowed for the patient to be a candidate for an orthotopic heart transplant.

DISCUSSION & KEY POINTS

Giant cell myocarditis (GCM) is a rare and fatal disease resulting in significant mortality due to heart failure, complete heart block, and ventricular arrhythmias. Patients with GCM often present at middle-age in fulminant heart failure or arrhythmias, and early diagnosis is critical for management. Even for patients diagnosed with GCM during their lifetimes and managed with immunosuppressive therapy, transplant-free survival at 5-years is only 48%. While the pathophysiology of GCM is not entirely understood, it is characterized by T-lymphocyte myocardial inflammation that can be diagnosed on gold-standard endomyocardial biopsy. Diffuse myocardial inflammatory infiltrates with multinucleated giant cells can also be targeted for biopsy with cardiac MRI assistance. Imaging findings are consistent with myocardial fibrosis; there is myocardial strain and late-gadolinium enhancement visible at affected areas of the myocardium.
Immunosuppression of T-mediated inflammation with prednisone, azathioprine, and cyclosporine can mitigate the disease and improve survival. Patients should also be optimized with goal-directed medical therapy for heart failure, but unstable hemodynamic presentation can limit the extent of medical management. Despite immunosuppression and medical management, mortality is high and serious ventricular tachycardias continue to be the single most prominent reason for death in patients with GCM. Immunosuppression and ICD placement are mainstays of GCM therapy, but this report focuses on mechanical circulatory support (MCS) devices and their role in GCM management.

There are a variety of options for MCS when managing GCM. These options can be temporary or durable, but destination therapy is limited to orthotopic heart transplant. The decision for types of MCS can be influenced by the patient’s clinical status, degree of cardiac dysfunction, institutional preferences, and patient’s listing status for transplant. Because of the rare and fulminant presentation of GCM, it is common for patients to be severely symptomatic prior to biopsy-proven diagnosis. In one case series of seven GCM patients the time interval from referral to device placement or transplant ranged from 2 days to 4 months, and GCM was not diagnosed in any patient prior to intervention. There is favorable evidence for VA-ECMO as the first temporary MCS option. In addition to ECMO, intra-aortic balloon pumps (IABP) or other trademarked devices are feasible as temporary bridge-therapy. More durable MCS options include LVAD, BiVAD, or total artificial heart (TAH) implants.

In the case of AG, the patient had a previous admission with right ventricular dysfunction requiring a Protek Duo RVAD. The Protek Duo Cannula has been shown to be a safe and effective option for short-term Bi-Ventricular support in conjunction with a secondary LV device. This is a dual lumen cannula device percutaneously inserted via the internal jugular vein and positioned to have the inflow lumen in the right atrium and the outflow lumen positioned in the main pulmonary artery. A disadvantage of short-term percutaneous RV assist devices is the possibility of venous scarring resulting in difficulty of device re-insertion. For AG, venous angioplasty was a safe and effective technique to allow for Protek Duo re-insertion in a patient with unfavorable anatomy who has failed Impella RP placement.

After hemodynamic stabilization, AG was able to successfully receive an OHT. His clinical course was then complicated by recurrence of GCM. Studies have shown that approximately 12% of patients can have GCM recurrence after transplant despite continuing immunosuppressive therapy. The rarity, severity, and acuity of giant cell myocarditis results in limited ability to conduct randomized control trials. Despite these barriers to establishing better evidence-based management, the use of MCS, and immunosuppressive therapy are still cornerstones of therapy as bridge-therapy to cardiac transplantation.

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Another Case of Takotsubo Syndrome: Excluded by the Presence of Significant Coronary Artery Disease, or Caused by Significant Coronary Artery Disease?

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INTRODUCTION

Takotsubo syndrome (TTS) is a reversible condition of abnormal myocardial contraction that was first given this name in Japan by Dr. Sato in 1991.1 The name comes from the Japanese word for “octopus trap,” which has a similar shape to that of the left ventricle on ventriculography during Takotsubo syndrome. It is also known as broken heart syndrome, stress-induced cardiomyopathy, or apical ballooning syndrome. The first descriptions of this phenomenon date as far back as the 1960s.2-3

TTS typically presents with symptoms and clinical signs suggestive of acute coronary syndrome (ACS). It may include ST segment elevations on electrocardiogram (ECG) characteristic of acute ischemia even though the syndrome is not caused by direct myocardial ischemia. On echocardiography, TTS is usually characterized by segmental wall motion abnormalities (SWMA) with hyperdynamic contraction of the left ventricular basal walls and akinesis of the apical walls. This results in the “apical ballooning” and is notably not in the distribution of typical coronary artery anatomy.4 Traditionally, the diagnosis of TTS involves the aforementioned findings and coronary angiography showing no obstructive coronary artery disease (CAD).5 We present here a case of an acute lateral ST-elevation myocardial infarction (STEMI) with subsequent cardiogenic shock due to TTS.

CASE PRESENTATION

History of Present Illness

A 68-year-old Caucasian female smoker without significant cardiac history presented to the emergency department of a suburban hospital with three days of progressive abdominal pain, initially thought to be a recurrence of her peptic ulcer disease. She described the pain as sharp and constant without radiation. She denied other symptoms such as dyspnea, chest pain, or palpitations. Physical exam at that time was unremarkable. Chest x-ray was did not show pulmonary edema or consolidation. Labs at the outside facility were notable for elevated troponin I at 3.18 ng/mL and lactate at 4.3 mmol/L. Her ECG showed ST elevations and terminal T wave inversions in I, aVL, and V4-V6 consistent with a lateral STEMI (Figure 1). The patient was transferred to our facility for emergent cardiac catheterization and cardiac intensive care.

Figure 1: ECG on presentation. 1-2 mm ST-segment elevations and T wave inversions in V4-V6, consistent with a lateral STEMI.
Hospital Course

Upon arrival at our facility, the patient underwent coronary angiography, which revealed the culprit lesion was a 100% occlusion of the second obtuse marginal branch of the left circumflex artery. The left main, left anterior descending, and right coronary arteries were found to have only minimal luminal irregularities. A drug-eluting stent was successfully placed and resulted in 0% residual stenosis. Notably, LV end diastolic pressure was elevated at 34 mmHg. She was then transferred to the cardiac intensive care unit for further management. Physical exam was notable for bilateral crackles on lung auscultation. Repeated chest x-ray done five hours from initial image showed new bilateral hazy opacities, consistent with pulmonary edema. She was given intravenous (IV) tirofiban, oral dual anti-platelet therapy, high intensity statin, and IV furosemide. Beta-blocker and angiotensin converting enzyme inhibitor (ACEi) therapies were not initiated due to acute heart failure with mild hypotension (systemic blood pressure 100s/60s).

The patient continued to have intermittent chest discomfort and nausea, without evidence of worsening myocardial ischemia. Serial ECGs showed resolution of ST segment elevations and persistent T-wave inversions. However, repeated lactate was higher than previous and peaked at 6.5 mmol/L. Liver enzymes also increased triggering suspicion for shock liver. Post-intervention transthoracic echocardiogram (TTE) demonstrated a severely reduced left ventricular ejection fraction (LVEF) of 34%, with SWMAs, apical akinesis, and ballooning of the LV apex. Notably, as seen in some cases of TTS, there was no left ventricular outflow tract (LVOT) obstruction or increased intracavitary gradient due to hyperkinesis of the basal segments in this patient. These TTE findings were significantly disproportionate to the relatively small territory of SWMAs expected from her lateral STEMI and more consistent visually and clinically with TTS.

The patient required inotropic support with IV dobutamine in the setting of worsening end organ perfusion from cardiogenic shock. She was also continued on regular IV diuretics for volume status optimization. Status of her cardiogenic shock was not monitored with invasive hemodynamic monitoring, but end organ function was trended closely with serial lactate levels, liver enzymes, and creatinine. On the fifth day of hospitalization, she was successfully weaned off of dobutamine and was transferred from the intensive care unit to the general telemetry floor. TTE on this day showed recovery of LVEF to 60%, with focal inferolateral and anterolateral wall hypokinesis, and there was resolution of the periapical wall motion abnormalities.

DISCUSSION

The diagnosis of TTS is typically made with signs and symptoms of ACS, regional wall motion abnormalities on echocardiography, and the absence of an obstructive culprit lesion on coronary angiography. Bybee et al. proposed the Mayo criteria, which mandate that there is no angiographic evidence of obstructive coronary disease or plaque rupture.5 Other similar criteria have been proposed, and all include the exclusion of obstructive coronary disease and acute coronary thrombus.5,7

The pathophysiology of TTS remains poorly understood, though there have been several proposed mechanisms and theories. The most widely accepted explanation is that increased systemic catecholamine levels lead to decreased LV function through microvascular spasm causing transient ischemia, as well as direct cardiotoxicity.4,6 Emerging data also suggests that there is a role for endothelial dysfunction and estrogen deficiency, resulting in epicardial and/or microvascular spasm.8 This could partially explain the higher prevalence of TTS in post-menopausal women.

More recently, however, cases of concurrent TTS and MI have been described, suggesting that the co-existence of the two are not exclusionary, as once thought.9,10 Adding to this small collection, we describe a case in which apical LV dysfunction was significantly disproportionate to the area of lateral infarction. Ultimately, resolution of the SWMAs supports the diagnosis of TTS. Some recent reports also suggest that transient apical LV wall thickening due to myocardial edema is a characteristic feature in the subacute recovery phase of TTS. This was not appreciated in the recovery echocardiogram obtained in this patient, but T2 weighted cardiac magnetic resonance imaging (MRI) may be a more sensitive modality to assess this.11

Our case demonstrates that an acute MI can be associated with and in this case, cause a stress response that leads to TTS. TTS and acute MI are not mutually exclusive and in certain cases should not be classified as such. If a patient with a seemingly small infarct develops cardiogenic shock, TTS should be considered in the differential diagnosis and can typically be discerned with TTE and managed expectantly.
REFERENCES


Cardiac Amyloidosis: A Known Disease with an Unknown Presentation

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ABSTRACT

Cardiac amyloidosis is an increasingly recognized entity that causes significant morbidity and mortality. Transthyretin amyloidosis (ATTR) is present in about 16% of patients with severe aortic stenosis and up to 17% of patients with heart failure with preserved ejection fraction9,10. Though the screening test of choice, echocardiography is not highly sensitive or specific, and it should not be relied upon to rule out cardiac amyloidosis, especially if clinical suspicion is high.

We present a case of a 58-year-old woman with a history of bilateral carpal tunnel syndrome who presented with paresthesia and syncope. Extensive workup for neurologic, infectious, and malignant etiologies was negative. EKG was remarkable for low voltage. Transthoracic echocardiogram (TTE) was not suggestive of infiltrative disease. Subsequent cardiac MRI demonstrated diffuse biventricular late gadolinium enhancement and technetium 99M pyrophosphate scan revealed diffuse (3+) uptake, which was in stark contrast to the TTE. The diagnosis of ATTR amyloidosis allowed for prompt initiation of treatment in this patient.

Syncope is an uncommon presentation of cardiac amyloidosis. Such significant cardiac burden of disease without appreciable changes on TTE or clinical heart failure, demonstrates the importance of clinical vigilance and thorough workup when suspicion for amyloidosis is high, particularly if characteristic signs and symptoms consistent with systemic disease are present, as timely treatment can significantly reduce the morbidity and mortality of this disease.

CASE PRESENTATION

A 58-year-old female of Irish descent with a history of bilateral carpal tunnel syndrome and bilateral lower extremity paresthesias presented to the cardiology office with complaint of recurrent syncope. She had undergone an extensive evaluation by her primary care doctor and a neurologist. With negative workup, but continued episodes of syncope, she was referred to cardiology.

Her EKG was significant for low voltage and poor R-wave progression (Figure 1). Her review of systems revealed a 15-pound unintentional weight loss over the previous year, in addition to her paresthesias. Her family history was significant for a paternal grandfather who died of cardiac causes of unknown etiology. She had a normal stress echo 4 months prior to evaluation. Labs were unremarkable aside from a mildly elevated ferritin of 207 ng/mL (reference range 15-150 ng/mL).

A transthoracic echo showed normal left ventricular size and systolic function. Concentric remodeling but no left ventricular hypertrophy was present and there was borderline left atrial enlargement. Although there were technical limitations that precluded accurate assessment of global longitudinal strain, the overall strain was reduced but was not consistent with any diagnostic pattern. Assessment of diastolic function revealed a mildly reduced medial annular velocity of 6 cm/s but the lateral annular velocity was normal measuring 11 cm/s and the E/E ′ was normal.
Due to the wide range of symptoms that ATTR can cause in different organ systems, its diagnosis is not straightforward and often delayed by years. Outside of the heart, amyloidosis can present with progressive symptoms in the nervous systems, eyes, kidneys, and gastrointestinal tract. The most common initial symptoms of ATTR-FAP are sensory-motor, such as peripheral neuropathy and carpal tunnel syndrome, and autonomic, such as GI motility disturbances and orthostatic hypotension. These manifestations can pre-date the diagnosis of amyloidosis by years.

Although there are characteristic findings on EKG such as low voltage and high-grade AV block, these are often not present and is usually a late manifestation of cardiac disease. In fact, less than 40% of patients with biopsy-proven ATTR have low voltage on EKG. The first screening tool for diagnosis is most commonly a transthoracic echocardiogram (TTE). There are several characteristic TTE findings associated with cardiac amyloidosis. The most referenced phenotype is characterized by septal and left ventricular hypertrophy, usually with diastolic dysfunction and preserved to low ejection fraction. It is important to note that these characteristics are not specific to amyloidosis. Hypertrophic or hypertensive cardiomyopathy can have overlapping echocardiographic features. However, there have been some established studies showing more unique differentiating features. For example, two-dimensional speckle-tracking to show a relatively
sensitive and specific “apical sparing” longitudinal strain pattern is unique to amyloid cardiomyopathy\textsuperscript{5,6}.

If echocardiogram findings are suggestive of, or the clinical suspicion is high enough for cardiac amyloidosis, the next steps in workup include ruling out AL amyloidosis and in some cases obtaining a cardiac MRI. Once AL is ruled out, bone tracer cardiac scintigraphy is done to assess for ATTR. Endomyocardial biopsy is only needed if the scintigraphy grade is not sufficient to be diagnostic. Once the diagnosis of ATTR is made, genetic testing is used to identify hereditary ATTR\textsuperscript{6}. This is particularly important because family members can be screened.

The treatment of cardiac amyloidosis is a continuously evolving field with the most recent developments being disease-modifying agents such as TTR stabilizers, silencers, and disruptors. Liver transplantation is an option to stop the progression of disease though it does not reverse already existing amyloid infiltration and related symptoms and is becoming less common as medical therapies improve\textsuperscript{7,8}.

In this case, we demonstrate how cardiac amyloidosis can have a varied presentation, requiring prompt recognition and diagnosis. Although TTE is the first screening test of choice, its imperfect sensitivity and specificity do not allow it to be used to rule out a diagnosis of CA. One must maintain a high index of suspicion and pursue further testing especially in situations where other organ systems are already involved\textsuperscript{4}. Our patient had a history concerning for CA given her peripheral neuropathy, bilateral carpal tunnel syndrome, and syncopal episodes. She had low voltage on EKG but her TTE was not suggestive of infiltrative disease.

The patient’s TTE was a stark contrast to the cardiac MRI and PYP scan, which showed extensive late enhancement and diffuse 3+ uptake throughout the myocardium, respectively. Such significant cardiac burden of disease without appreciable changes on TTE further demonstrates the importance of clinical vigilance and thorough workup when suspicion for amyloidosis is high.

Early detection is especially important given the progressive nature of ATTR and the fact that there are therapies with multiple different mechanisms of action available to halt the progression of this disease. These medications are most effective at minimizing the impact of the disease when initiated early, as the compounding effects of this disease over time are not reversible with pharmacologic therapy. Furthermore, cardiac involvement, when present, is the principal determinant of survival\textsuperscript{4}. Our patient’s diagnosis was fortunately confirmed before she had any significant heart failure symptoms, and she was initiated on treatment with tafamidis and patisiran early in her disease course.

ATTR has previously been considered a rare diagnosis. However, recent studies suggest that ATTR is present in about 16% of patients with severe aortic stenosis and up to 17% of patients with HFpEF\textsuperscript{9,10}. This represents a significant percentage of patients with these two fairly common conditions, making accurate and early diagnosis even more crucial, especially given that pharmacologic therapies are most effective when initiated prior to the onset of significant cardiac involvement.

In conclusion, this case serves to emphasize the variation of clinical presentation in ATTR and specifically the limited sensitivity of echocardiography in diagnosis. It remains of high importance to consider and act early when there is clinical suspicion for amyloidosis in patients with a compatible history.

REFERENCES

A Case of Patent Foramen Ovale as a Cause of Persistent Hypoxia

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INTRODUCTION

Patent foramen ovale (PFO) is a congenital cardiac variant caused by failure of the closure of a passage in the atrial septum. It is quite common, occurring in as much as 27% of the population based on autopsy studies. Most cases of PFOs are incidentally discovered or found during work-up of cryptogenic strokes as a potential cause of the stroke. New research is being conducted on the role PFOs play in hypoxia from intracardiac right-to-left shunting, including in patients with co-existent cardiovascular and pulmonary disease.

CASE PRESENTATION

A 52-year-old man with a past medical history of heart failure with reduced ejection fraction (EF 15%) from non-ischemic cardiomyopathy now status post single chamber ICD placement, atrial flutter, and chronic kidney disease presented for worsening hypoxia and bilateral lower extremity edema. Patient acknowledged worsening dyspnea on exertion, orthopnea, and lower extremity edema over the four weeks prior to admission. Other review of systems was negative. Vital signs upon presentation were significant for low blood pressures (90s/50s), tachypnea to mid-20s, and SaO2 88% on 6 L nasal cannula oxygen. Physical exam was significant for elevated JVP, bilateral pitting lower extremity edema, bibasilar crackles up to mid lung fields bilaterally, clubbing of fingernails, and a holosystolic 3/6 murmur. Labs were significant for an acute kidney injury with a creatinine of 2.06 (from baseline 1.7) and proBNP of 7444. Initial right heart catheterization showed elevated filling pressures (RA 15 and PCWP 22). Patient was aggressively diuresed with a furosemide drip with no improvement in hypoxia. Increasing the patient’s nasal cannula from 6 L to higher levels also did not improve the hypoxia.

DIFFERENTIAL DIAGNOSIS

The differential could include hypoxia secondary to an exacerbation of the patient’s heart failure and pneumonia. However, the patient’s hypoxia did not improve with aggressive diuresis, and the patient did not have any clinical symptoms of pneumonia. Work-up was initiated for patent foramen ovale as a cause of persistent hypoxia. Due to a concern for a shunt, right heart catheterization was repeated with a wedge saturation that did show some degree of shunting. A positional ABG showed a PO2 of 111 mmHg while lying and 72 mmHg while sitting upright. A TEE at that time showed a 6 mm PFO with significant R to L shunt. At this point, work-up was started for possible closure of PFO.

OUTCOME AND FOLLOW-UP

Due to a difficult social situation, including poor family/social support and uninsured status, decision was made for patient to complete work-up for PFO closure after discharge with social work’s assistance and follow-up at structural heart clinic. Patient is being followed in clinic after discharge for further evaluation regarding closure of PFO.

DISCUSSION

This case demonstrates how PFOs can be a contributing cause of hypoxia in patients with underlying cardiovascular disease. Patients with significant right to left shunting from PFOs usually present with hypoxia that does not improve with increasing oxygen requirement. Other symptoms include platypnea and orthodeoxia, both of which our patient also demonstrated. Diagnosis of a PFO with shunting can be accomplished via a TEE with bubble study. After diagnosis, treatment depends on weighing the risks of cardiac surgery with the benefits of improvement in hypoxia and quality of life. Research is still being conducted on the definitive benefits of surgical repairs of PFOs in patients with hypoxia. The patient in this case continues to follow with structural heart after discharge and is being evaluated for surgical repair of his PFO, considering the severity of his shunt symptoms, in hope to improve his quality of life.
KEY POINTS

Patent foramen ovales (PFOs) can be a cause of persistent hypoxia in patients when there is significant right to left shunting. Research is still being conducted on the value of closure of PFOs as a treatment for persistent hypoxia.

REFERENCES


Shocked But Not Surprised: The Philly Cardioversion

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INTRODUCTION

Neuromuscular incapacitating devices, colloquially known as ‘tasers’, are typically used by police and security personnel as a non-lethal way to subdue combative assailants. Unfortunately, there are times in the hospital when patients can become assailants, thus potentially necessitating the use of tasers to ensure the safety of staff and other patients. Tasers come in several varieties. However, those typically used by law enforcement have a 50,000-V capacity and deliver 0.36 - 1.76 Joules of energy per pulse, at a rate of ~20 pulses per second, via two barbed projections1. This leads to incapacitation of the assailant via the induction of fused muscle contractions that preclude coordinated neuromuscular inputs, thus inducing a near-tetanic state 2. In medicine, we often use electricity in a coordinated manner to convert dangerous cardiac arrhythmias back to normal sinus rhythm. In this case, we discuss how a patient who was admitted to the hospital in a sustained arrhythmia, became an assailant. A taser was used to subdue him, and we will examine how this theoretically may have impacted his arrhythmia.

CASE

A patient with a past medical history of advanced heart failure with reduced ejection fraction of 10% secondary to methamphetamine use was admitted to the medical intensive care unit for severe COVID-19 infection resulting in hypoxic respiratory failure requiring intubation. His acute respiratory condition stabilized, and he was liberated from the ventilator. His oxygen status continued to improve as he was weaned to 2L nasal cannula, and he was transferred to a general medicine telemetry service.

His hospital course was further complicated by suspected in-hospital use of methamphetamines resulting in atrial fibrillation with rapid ventricular response with rates in the 150s. The patient reported feeling "bad", and he was noted to be diaphoretic and agitated. He was started on a digoxin infusion and given intravenous diuresis. As he experienced subjective improvement with down trending heart rates, he asked to leave the hospital against medical advice (AMA). On examination of the patient, he was unable to express the risks of leaving the hospital and foregoing additional medical interventions. With psychiatry input, patient was deemed to lack capacity to leave AMA.

Patient then became angered, decannulated his intravenous lines, removed his telemetry strips, and attempted to leave the hospital room. He became very aggressive with threats of violence to the medical staff, so security was contacted. His condition visibly appeared to deteriorate as he was in clear extremis, diaphoretic, and weak. Even his screaming became more tired and hoarse. Finally, after an hour of negotiation, the patient lunged at the provider, requiring the security officer to react by activating his taser and delivering a shock. He was then injected with 5mg of intramuscular haloperidol and placed in four-point restraints. The team was hopeful that the delivery of electricity to his chest may have restored sinus rhythm, but the patient remained in atrial fibrillation when placed back on telemetry.

DISCUSSION

It has been previously posited that taser devices pose minimal cardiac threat because of the depth of tissue that must be penetrated to affect the heart, the relative intensity of electrical energy required to activate cardiac muscle as opposed to skeletal muscle, and the different electric pulse widths required to activate cardiac as opposed to skeletal muscle2. There has been extensive original research assessing the cardiac and even neurologic risk associated with taser devices since their development, and this has also been reviewed in the literature1.

Interestingly, it does appear that taser shocks can influence cardiac activity, as was demonstrated in a previous report outlining a pacemaker interrogation that revealed the induction of rapid ventricular conduction in a patient following a taser shock4. Additionally, we were able to locate one case report of atrial fibrillation induced in a patient with no history of atrial fibrillation, normal electrolytes, and a structurally normal heart following the application of a taser shock5. In the case of our patient, there was a fleeting hope among the care team that the taser shock could have
successfully restored sinus rhythm via electrical cardioversion, but this was revealed to not be the case after he was back on telemetry after being subdued.

There are many reasons why this might be, but it seems reasonable that inherent differences among different brands of tasers may affect the potential of each unit to influence myocardial conduction. Further, various areas on the human body where this shock could be applied, it stands to reason that there are many potential physiologic effects of a taser shock that will not be experienced. Additionally, the actual energy delivery per taser shock is quite low, at around an average of 1 J per pulse among different units, and it is difficult to quantify what additive effect the many shocks delivered per second may have on myocardial conduction. Research by Richard et al reports only a 22% success rate with 50 J in external cardioversion in atrial fibrillation, which is significantly higher than the average J delivered per shock from a taser.

In a patient with severe cardiomyopathy, even a coordinated shock from a dedicated defibrillator may not be successful in restoring sinus rhythm, so it is not entirely surprising that a taser-mediated shock, which we fondly refer to as a “Philly Cardioversion”, was unable to restore sinus rhythm in this patient.

Although the authors would certainly prefer that no additional humans undergo a taser shock, further observation of the incidence of the resolution of arrhythmias in the setting of taser shocks will continue to elucidate the potential of these enforcement tools to influence cardiac activity. Since tasers are designed to provide a non-lethal shock, their mechanics would ideally have very low risk myocardial involvement.

REFERENCES


“Just Surgeons” – Heping Sheng, MD
A Confounding Case of Acute Hepatitis A

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INTRODUCTION

Hepatitis A (HAV) is a picornavirus transmitted via fecal-oral route that disproportionately affects homeless persons, men who have sex with men, and individuals who use intravenous drugs.¹ Acute HAV typically presents with nausea, vomiting, abdominal pain, and fever. It is most commonly self-limited but can progress to fulminant hepatic failure in less than 1% of cases.² The following case is a unique presentation of acute HAV infection requiring diagnostic dexterity and critical thinking.

CASE DESCRIPTION

A 28-year-old male with a past medical history of opioid use disorder and chronic hepatitis C (HCV) without baseline evidence of cirrhosis who presented with several days of fevers and lethargy. He denied abdominal pain, nausea, vomiting, and diarrhea. The patient was febrile to 103.4 F and tachycardic to 145 bpm. Initial labs were notable for WBC 4.3 B/L, HgB 11.0 g/dL, Plt 143 B/L, AST 136 IU/L, ALT 193 IU/L, and HCV PCR 7000 IU/L. He received broad spectrum antibiotics, fluid resuscitation, and was admitted to general medicine. Given his high-risk demographic, the HAV vaccine was administered for primary prophylaxis.

Over the next 24 hours, fevers persisted despite antibiotics. Blood cultures remained negative. An MRI spine performed for back and neck pain was negative for infection. The patient’s hepatic function panel peaked at: ALP 270 IU/L, AST 2047 IU/L, ALT 2196 IU/L, total bilirubin 6.3 IU/L, direct bilirubin 5.8 IU/L with associated INR 1.15, PT 13.5 sec, PTT 38 sec (Figure 1). Comprehensive infectious workup (Table 1) came back negative other than a positive HAV IgM antibody. Since the clinical interpretation of positive HAV IgM antibody was unclear in the setting of recent vaccination, an HAV viral PCR was ordered and returned positive, confirming the diagnosis of acute hepatitis A.
The diagnosis of acute HAV was established when HAV presentation.

Finally, acute HIV and tick-borne illnesses were of concern due to active IVDU and homelessness. However, his HIV, ehrlichia, and lyme antibodies were negative. The diagnosis of acute HAV was established when HAV RNA PCR returned and confirmed the positive IgM was due to acute HAV infection rather than vaccination.

In order to avoid confusion and ensure correct diagnosis, this clinical case highlights the importance of checking HAV IgM in anyone presenting with potential infection prior to vaccination against HAV.

REFERENCES


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DISCUSSION & CONCLUSIONS

The etiology of our patient’s acute illness was challenging to diagnose due to several confounding variables. On hospital day 1, he received a single antigen inactivated HAV vaccine. On hospital day 7, his HAV IgM was positive. We hypothesized that the positive HAV IgM antibody represented either acute HAV infection or an antibody response to recent immunization. Evidence suggests that anti-HAV IgM is detected 2-3 weeks after administration of the single dose inactivated HAV vaccine. It would be unusual for the patient to develop anti-HAV IgM in the 7 day period between vaccination and testing.

The patient’s history of HCV also complicated the diagnosis. He received past treatment for HCV, but admission labs were indicative of treatment failure or reinfection. Ultimately, HCV viral load remained stable, reducing concern for acute HCV as the cause of his presentation.

Finally, acute HIV and tick-borne illnesses were of concern due to active IVDU and homelessness. However, his HIV, ehrlichia, and lyme antibodies were negative. The diagnosis of acute HAV was established when HAV
A Case of Acute Pancreatitis Associated with Empagliflozin

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ABSTRACT

Sodium-glucose cotransporter 2 (SGLT2) inhibitors are being prescribed increasingly more often for type 2 diabetes mellitus as well as heart failure. They have not typically been associated with acute pancreatitis, but there has been a steady flow of case reports implicating them in acute pancreatitis over the years since they were initially approved. Here, we present the case of an 82-year-old woman with a past medical history of T2DM, COPD, hyperlipidemia, a remote stroke, peripheral arterial disease, and remote breast cancer now with recurrent localized breast cancer on treatment with ademaciclib and letrozole who presented to the emergency department with abdominal pain, weakness, decreased oral intake, and nausea and vomiting. These symptoms started two weeks after the initiation of the SGLT-2 inhibitor empagliflozin for her T2DM. Initial labs were notable for sodium of 129, glucose of 409, a normal anion gap, beta hydroxybutyrate of 4.6, serum creatinine of 0.92, calcium of 9.8, total bilirubin of 3.0 with direct bilirubin 2.6, alkaline phosphatase of 773, AST of 330, ALT of 446, lipase of 1,159, triglycerides of 237, and leukocyte count of 4.9. Following admission, CT and MRCP demonstrated pancreatitis with no intrahepatic or extrahepatic ductal dilation, gallstones cholelithiasis, or other obvious etiology of her presentation. Her symptoms improved with supportive care following the discontinuation of her SGLT-2 inhibitor and she was discharged to inpatient rehab shortly after presentation. This case highlights the importance of keeping the uncommon diagnosis of SGLT-2 inhibitor associated pancreatitis in mind in patients who present with acute pancreatitis.

INTRODUCTION

According to the Centers for Disease Control, the prevalence of type 2 diabetes mellitus (T2DM) in 2020 was nearly 10.5%1. Given its overall burden and the range of complications that can arise from long-term T2DM, it makes sense that there continue to be emerging therapies to treat it. One of the drug classes approved within the last decade, sodium-glucose co-transporter 2 (SGLT-2) inhibitors, function by reducing the reabsorption of glucose at the level of the renal tubule, thus causing lower blood glucose levels through therapeutic glucosuria. It has been increasingly prescribed for T2DM and was recently approved for the treatment of heart failure with reduced ejection fraction even in the absence of T2DM. Pancreatitis, which has been associated with some classes of T2DM medications, is an uncommon side effect of SGLT-2 inhibitors. Still, there have been a few reports of SGLT-2 inhibitors induced pancreatitis since their approval, and it is important to keep this side effect in mind when prescribing these drugs for the first time or when treating someone for acute pancreatitis in the setting of recent initiation of an SGLT-2 inhibitor2-4.

Here, we present the case of an 82-year-old woman who presented with subacute abdominal pain caused by pancreatitis 30 days after initiation of the SGLT-2 inhibitor empagliflozin in the setting of poorly controlled diabetes.

CASE REPORT

Our patient is an 82-year-old woman who presented to the emergency department in January of 2021 with almost 2 weeks of abdominal pain with radiation to her back, weakness, and decreased oral intake. She had also been experiencing nausea and vomiting for 3-4 days prior to admission. Her significant past medical history included poorly controlled T2DM with recent Hgb-A1C of 11.1%, COPD, a remote stroke, hyperlipidemia, peripheral artery disease, and two previous episodes of breast cancer in 1994 and 2015 now with recurrence. Her medications included metformin, fluticasone/salmeterol, tiotropium, albuterol, rosuvastatin, clopidogrel, lorazepam, abemaciclib and letrozole. She was initiated on empagliflozin to optimize the treatment of her T2DM on at the start of December of 2020. Additionally, she was recently diagnosed with recurrent estrogen receptor positive, progesterone receptor positive, human epidermal growth factor receptor-2 negative inflammatory breast cancer of the right breast in November of 2020, at which time she was initiated on abemaciclib and letrozole.
On presentation, initial vital signs included a temperature of 96.6°F, pulse of 83 beats per minute, blood pressure of 156/75 mmHg, respiratory rate of 18, and oxygen saturation of 96% on room air. Physical examination was significant for lethargy, dry mucous membranes, diffuse abdominal tenderness without rebound or guarding, and a firm right breast mass with dimpling of the overlying skin. Initial labs were notable for a sodium of 129, bicarbonate of 15 (baseline 18-20) with a normal anion gap, serum creatinine of 0.92, glucose of 409, calcium of 9.8, total bilirubin of 3.0 with direct bilirubin 2.6, alkaline phosphatase of 773, AST of 330, ALT of 446, beta hydroxybutyrate of 4.6, lactate of 1.4, lipase of 1,159, triglycerides of 237, leukocyte count of 4.9, and hemoglobin of 11.3. CT of the abdomen and pelvis without contrast demonstrated peripancreatic fat stranding and edema consistent with acute uncomplicated pancreatitis. Notably, it also showed a normal liver, normal bile ducts without dilation, and a normal gallbladder without gallstones. She had held most of her medications on her own prior to presentation due to a concern that they could be related to her symptoms. The following day, ultrasound of the abdomen confirmed absence of cholelithiasis and choledocholithiasis with normal caliber intrahepatic and extrahepatic biliary ducts including the common bile duct as well as a normal gallbladder without stones or edema. It also showed signs of acute pancreatitis, though this was better imaged on the CT scan from the prior day. Due to concern for an occult obstructive lesion in the biliary system in the setting of persistently high ALP, AST, ALT, and bilirubin in the setting of malignancy, she was sent for magnetic resonance imaging with MRCP, which demonstrated no intrahepatic or extrahepatic biliary ductal dilatation and again confirmed no cholelithiasis or choledocholithiasis. It also visualized a 0.7 cm intraductal papillary mucinous neoplasm, which was slightly increased in size since the last time it was imaged in 2012. At this point, her symptoms had improved significantly since her SGLT-2 inhibitor was held, but her liver function tests (LFTs) were persistently elevated. As such, she underwent esophagogastroduodenoscopy with endoscopic ultrasound, which showed diffuse thickening of common hepatic duct and common bile duct walls without dilation or stones. In consultation with GI, it was determined that her persistently elevated LFTs were in the setting of acute pancreatitis, and they were expected to trend down over the next days to weeks. The patient continued to improve symptomatically, and her LFTs did trend down before her eventual discharge to inpatient rehabilitation.

**DISCUSSION**

SGLT-2 inhibitors have been implicated in drug-induced pancreatitis in a few case reports. A recent review aggregated case reports of SGLT2-associated pancreatitis and found that four reports were associated with canagliflozin, two with empagliflozin, and one with dapagliflozin. Since that paper was published, another case report for empagliflozin-induced pancreatitis has been published. Our case report is the fourth that we were able to find for empagliflozin. In the previous review, the mean time interval from initiation of the SGLT-2 inhibitor to diagnosis of pancreatitis was 39 days, which falls in line with the time interval observed in this case – 30 days.

Our patient presented with clinical, laboratory and imaging findings of acute uncomplicated pancreatitis. In the absence of any of the other classic causes of pancreatitis (including alcoholism, gallstones, obstructive mass lesion in the setting of malignancy, and hypertriglyceridemia) and the recent initiation of an SGLT-2 inhibitor, we believe drug-induced pancreatitis to be the most likely cause of her presentation. Letrozole and abemaciclib were also recently initiated, but these are less likely to be causes of pancreatitis. We were not able to find any case reports of pancreatitis associated with these two agents, and in fact, letrozole has been prescribed as an alternative to tamoxifen in a number of cases where tamoxifen was implicated in pancreatitis. The mechanism for the development of pancreatitis is unknown with regards to this drug class, which has only been associated with pancreatitis in a few case reports. It is becoming more apparent that SGLT-2 inhibitors have broader in vivo actions than just SGLT-2 inhibition in renal tubular cells, and it is possible that some of these effects are responsible for the development of pancreatitis. The range of conditions for which SGLT-2 inhibitors are approved is expanding, such that they will be prescribed increasingly more commonly over the next years, which could lend further insight into the extent of their physiologic actions.

This case report highlights the importance of keeping the rather uncommon diagnosis of SGLT-2-induced pancreatitis in mind when treating a patient with acute pancreatitis, as timely discontinuation of the SGLT-2 inhibitor caused improvement of symptoms in all case reports that we reviewed.
REFERENCES


“Not fragile like a flower, fragile like a bomb” – Heping Sheng, MD
A Case Report of Methemoglobinemia and Hemolytic Anemia in the Setting of COVID-19 Pneumonia and G6PD Deficiency

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INTRODUCTION

It is well known that hereditary or acquired methemoglobinemia can cause hypoxia due to the oxidation of heme, which impairs its ability to offload oxygen (Figures 1 & 2), and that acquired methemoglobinemia is most often caused by exposure to drugs and toxins that oxidize hemoglobin to methemoglobin, directly or indirectly1. Recently, a few case reports have highlighted methemoglobinemia in patients with COVID-19 pneumonia. Some of these reports were due to treatment with hydroxychloroquine and others from unidentifiable causes2-4. We present a case in which a patient with COVID-19 pneumonia was diagnosed with methemoglobinemia and acute hemolysis from G6PD deficiency in the setting of worsening hypoxia after receiving treatment with dexamethasone, remdesivir, and high-dose vitamin C.

CASE PRESENTATION

A 32-year-old African American male with no significant past medical history presented to the emergency room with exertional shortness of breath and non-productive cough for 6 days. He was febrile to 103°F and had an oxygen saturation of 91% on room air. He had no evidence of crackles or wheezes on lung auscultation, but his chest x-ray showed bilateral patchy opacities and a COVID-19 nasopharyngeal swab was positive. He was admitted and started on Remdesivir and dexamethasone 6 mg/day. On hospital day (HD) 2, he enrolled in a COVID high-dose vitamin C trial where he was randomized to the treatment arm and received 34g of vitamin C (0.3g/kg) IV on HD 2 and 68g (0.6g/kg) IV on HD 3. He was removed from the trial on HD 4 for worsening hypoxia. On HD 5, his oxygen saturation was 80% on 15L of oxygen. Despite his low oxygen saturation, he had no shortness of breath or increased work of breathing. An arterial blood gas while on 15L showed: pH 7.39, PaCO2 43 mmHg, PaO2 110 mmHg, measured O2 saturation 88%, and calculated O2 saturation 99%. He was subsequently transferred to the intensive care unit for closer monitoring.

Due to the discrepancy between his pulse oximeter (SpO2) and arterial oxygen saturation (PaO2), a co-oximetry panel was sent. This revealed a mildly elevated methemoglobin level of 8.1%. Notably, his hemoglobin had declined from 13.7 on admission to 6.8 g/dL by HD 8, and his creatinine climbed to 3.6 mg/dL from 1.0 mg/dL. In addition to methemoglobinemia, he was found to have a non-immune hemolysis, and review of his peripheral blood smear showed bite and blister cells with the presence of Heinz bodies. A serum G6PD level obtained at the time of active hemolysis was appropriately low at 3.5 U/g Hg. Hematology recommended treatment of his methemoglobinemia with 1.5g of vitamin C every 8 hours for a total of 4 days, which improved his methemoglobin level to 4%. Methylene blue was not used given his diagnosis of G6PD deficiency.

Figure 1: Hemoglobin-Methemoglobin Redox Mechanism

Figure 2: Oxygen–Dissociation Curve
and his methemoglobin level was only mildly elevated. Methylene blue is considered in patients with moderate to severe elevations in methemoglobin (>20-30%). By HD 8, he was maintaining 95-100% oxygen saturation on room air, and his hemoglobin eventually recovered to 12 g/dL one month later without additional treatment (see Figure 3 for complete timeline of events).

**DISCUSSION**

Undoubtedly, this patient’s hypoxia from COVID-19 pneumonia was exacerbated by methemoglobinemia and acute hemolysis from underlying G6PD deficiency, but the cause of the methemoglobinemia remains unclear.

Since he was only exposed to three medications (remdesivir, dexamethasone, and high-dose vitamin C), there is concern that one of these pharmacotherapies acted as an oxidative stressor inducing both methemoglobinemia and hemolytic anemia. While physiologic doses of vitamin C act as a reducing agent, there are case reports that suggest supraphysiologic doses of vitamin C (30g or more) can act as an oxidizing agent. This mechanism is due to the production of hydrogen peroxide as a byproduct of ascorbic acid in high serum concentrations cycling between its ionized and radical forms, allowing hydrogen peroxide to oxidize hemoglobin and cause lipid peroxidation of the cell membrane leading to intravascular hemolysis.

It is worth noting that there is another case report similar to ours (but without the use of Vitamin C) where no known inducer of methemoglobinemia was identified and the authors discussed the possibility of COVID-19 acting as an oxidative stressor itself like that of other viral infections that produce reactive oxygen and nitrogen species. Thus, it is also possible that the viral insult may have contributed to the development of this patient’s methemoglobinemia.

Despite the unknown etiology of our patient’s methemoglobinemia, this case emphasizes the importance of keeping a broad differential and recognizing the discrepancy between the patient’s SpO2, PaO2, and clinical exam. It is imperative to recognize this abnormality and obtain an arterial co-oximetry panel once erroneous causes of this discordance are ruled out such as: poor probe positioning, hypothermia, and acrylic/painted nails. It also has important implications for the management of COVID-19, especially when considering treatments known to cause methemoglobinemia or trigger hemolysis in G6PD deficiency, like hydroxychloroquine and supraphysiologic doses of vitamin C.

**KEY POINTS**

- It is important to keep a broad differential for hypoxia in the setting of COVID-19 pneumonia and recognize discrepancies between SpO2, PaO2, and the clinical exam.
- It is imperative to recognize this abnormality and obtain an arterial co-oximetry panel once erroneous causes of discordance are ruled out such as: poor probe positioning, hypothermia, and acrylic/painted nails.
- Although vitamin C is a treatment for mild methemoglobinemia due to its reduction potential, it has been reported to be an oxidizing agent at supraphysiologic doses (>30 grams).

**REFERENCES**

INTRODUCTION

Tumor lysis syndrome (TLS) is an oncologic emergency that is caused by electrolyte derangements from the lysis of malignant tumor cells. The syndrome consists of several laboratory abnormalities including hyperuricemia, hyperkalemia, hyperphosphatemia, and hypocalcemia. When these lab findings are associated with end-organ damage such as acute renal failure, seizures, or cardiac dysrhythmias amongst others, it is known as clinical TLS. TLS is more commonly associated with hematological malignancies given their tendency of rapid cellular turnover. The most common culprits include acute lymphocytic leukemia and Burkitt’s lymphoma. It is, however, quite rare for TLS to occur secondary to a solid malignancy. In fact, only 74 cases of solid-tumor TLS have been reported between 1977-2011. Furthermore, in case of solid tumors, they are almost always related to administration of cytotoxic chemotherapy leading to rapid cell death. Therefore, the case described here of spontaneous TLS leading to atrial flutter in an 89-year-old female with large pelvic mass is a rare presentation.
CASE REPORT

An 89-year-old female arrived at the ED on the recommendation of her outpatient provider after a lab test showed hyperkalemia. For a few months before this, she had been noticing fatigue, bloating, and increased abdominal girth. She was referred to gynecologic oncology about a month prior to her presentation who performed an ultrasound which showed a very large complex multiloculated, complex, cystic mass that measured 24 x 13 x 28 cm overtaking the entire pelvic and abdominal region which prompted the decision to carry out an exploratory laparotomy. The preoperative lab work was significant for a potassium level of 6.0, for which she was asked to seek care immediately.

At presentation to the emergency department, her heart rate was 145, blood pressure 127/79, respiratory rate 20, and oxygen saturation of 99%. She was uncomfortable with diffuse wheezing, profound abdominal distention, and 2+ bilateral lower edema. An EKG carried out at the time exhibited atrial flutter with a right bundle branch block. Prior EKGs had been normal sinus rhythm and the patient denied any history of rhythm disorders. Initial labs were significant for potassium 5.7, BUN 105, creatinine 4.23, calcium 8.0, LDH 526, urate 15.7, and phosphate of 8.2. She was given diltiazem, which decreased her rate to the low 100’s. CT abdomen and pelvic mass showed that the mass was significantly displacing the abdominal structures including vasculature, but no hydronephrosis was present (Figure 1). She was admitted to the inpatient service for further management.

Patient’s hyperkalemia was temporized with administration of insulin and dextrose along with albuterol and multiple doses of calcium gluconate. Upon admission, she was started on bicarbonate drip at 80ml/hr and was dosed several times with intravenous Lasix. However, her urine output remained poor with only 200 ml over the course of the first 24 hours of admission. Her acute renal failure was likely precipitated by a combination of TLS and mass effect. Given the significant elevation in her uric acid level 3 mg IV rasburicase was also administered on admission and was redosed 24 hours later. Despite the above interventions, the patient’s electrolytes abnormalities and her renal function worsened. Dialysis was considered as a possible management option and nephrology was consulted, however, given the overall poor prognosis the decision was made to abort this effort in discussion with the family. At this point her mental status acutely worsened, and she became lethargic, only intermittently opening her eyes during discussions. Goals of care discussions were initiated with her family, who ultimately decided on comfort measures following which she was discharged to inpatient hospice.

DISCUSSION

Tumor lysis syndrome is one of the true oncologic emergencies precipitated by the release of intracellular components of malignant cells into the bloodstream often following initiation of chemotherapy. TLS is usually defined by the Cairo and Bishop’s Classification System which requires at least two of the following metabolic derangement -- potassium ≥6 mmol/L or 25% increase from baseline; phosphorus ≥4.6 mg/dL or 25% increase from baseline; calcium ≤7.0 mg/dL or 25% decrease from baseline; uric acid ≥8.0 mg/dL or 25% increase from baseline--- for the diagnosis to be made. If the above-mentioned criteria is fulfilled, then the process is characterized as laboratory TLS. If any end organ damage is present in addition to the electrolyte abnormalities, including but not limited to acute kidney injury and cardiac dysrhythmias, then diagnosis of clinical TLS can be made. Our patient made the criteria for both laboratory and clinical TLS given her increased potassium, phosphate, uric acid along with acute kidney injury as evidenced by increased creatinine and oliguria along with new onset atrial flutter.

Given TLS is a consequence of rapid destruction of malignant cells, it is often associated with rapidly proliferating and chemosensitive hematological malignancies. Although they more commonly occur following chemotherapy, TLS can also develop in absence of any intervention. In such cases it is known as spontaneous TLS. This is often observed with particularly aggressive hematological cancers including acute lymphocytic leukemia, Burkitt’s lymphoma, Diffuse large B-Cell lymphoma amongst others. Although the exact reason for this is not clear, TLS is quite rare in solid tumors and cases of spontaneous solid tumor TLS are even more scarce. As stated previously, only 74 cases of solid-tumor TLS have been reported between 1977-2011 with only a fraction of them being spontaneous. Therefore, the presentation of severe TLS in this patient with gynecologic tumor in absence of chemotherapy is quite unique. It is likely this was precipitated by the heavy tumor bulk in the context of other risk factors including advanced age and overall poor health of the patient.

Another interesting part of this presentation is the development of new onset atrial flutter in the setting of hyperkalemia caused by her TLS. The ECG changes
associated with hyperkalemia have been well-described and include peaking of T waves, P-wave flattening, and QRS complex widening. Arrhythmias less commonly seen include atrial fibrillation and even asystole or ventricular fibrillation in severe cases. Atrial flutter, as seen in our patient, rarely occurs during hyperkalemia with only two previously described cases in our literature search7. In this patient’s case, her risk for arrhythmia was likely increased by the presumed rapid onset of hyperkalemia along with her concurrent hypocalcemia, which can potentiate the effects of hyperkalemia.

Treatment of TLS is geared towards addressing the electrolytes derangements. The mainstay of treatment remains fluid administration with close monitoring of urine output. The rate of fluids is adjusted to maintain the urine output between 80 to 100 ml/hr2,8. If that level of output is not reached, diuretics are utilized to increase urine flow. Fluid administration ensures increased renal blood flow with subsequent increase in GFR and upregulation of elimination of potassium, phosphorous, and uric acid. In situations where, appropriate urinary output cannot be achieved, as might be the case in patients with chronic kidney disease, dialysis is indicated. Other management options are geared towards specific electrolyte derangement. Rasburicase, which upregulates transformation of uric acid into allantoin which is highly soluble in water thus facilitating urinary excretion is used to treat acute rise in uric acid. Allopurinol, a xanthine oxidase inhibitor, which block conversion of xanthine to uric acid can be used concurrently to prevent accumulation of uric acid2,8. Similarly, while hyperkalemia is more definitively addressed through urinary excretion or dialysis, intravascular potassium levels can be temporized through administration of insulin. Calcium, which is often depleted due to binding by phosphate is repleted through of addition of calcium gluconate.

CONCLUSION

Tumor lysis syndrome (TLS) is an oncologic emergency that is caused by electrolyte derangements from the lysis of malignant tumor cells resulting in laboratory abnormalities including hyperuricemia, hyperkalemia, hyperphosphatemia, and hypocalcemia. It is often associated with rapidly proliferating and chemosensitive hematological malignancies often following cytotoxic treatment. The case presented in this paper where TLS occurred not only with solid tumor but spontaneously in absence of any interventions is a rare presentation.

REFERENCES

“Healing” – Heping Sheng, MD
Pulmonary Metastases of Basal Cell Adenocarcinoma Presenting as Hemoptysis

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INTRODUCTION

Basal Cell Adenocarcinoma (BCAC) is a rare malignancy, only accounting for approximately 2% of all salivary neoplasms¹. Considered the malignant counterpart of basal cell adenoma, it most commonly presents at 60 years of age without gender predilection. Sites of involvement frequently includes the parotid gland, but sites in the minor salivary glands, nasopharynx, buccal mucosa, and tongue have also been reported²,³. Often regarded as an indolent malignancy, BCAC can occasionally cause invasive disease and infrequently, metastatic disease². Among all solid tumors, endobronchial metastases is quite a rare occurrence, contributing to approximately 4% of endobronchial biopsies⁴. Most common sites of metastases in BCAC include cervical lymph nodes with sparse reports of pulmonary, hepatic and cutaneous involvement⁵. We present a case of endobronchial metastases from BCAC of the base of the tongue.
**DISCUSSION & KEY POINTS**

The management of BCAC, as with all salivary gland neoplasms, is particularly challenging due to the phenotypic heterogeneity and low clinical incidence. The most common strategy includes surgical resection with adjuvant radiotherapy. While distant metastases is an uncommon recurrence, local recurrence is quite common despite tumor-free surgical margins with rates of upward to 50%\(^2\). Chemotherapy has been found to be of limited use, predominately in the setting of recurrent or metastatic disease. Currently, there are no National Comprehensive Care Network (NCCN) recommendations for specific regimens. The most common agents include cisplatin, doxorubicin with either 5-fluorouracil or cyclophosphamide. However, no regimen has produced significant and reproducible improvements in overall survival or disease-free survival\(^6\).

This case demonstrates several unique features. This patient did not experience local recurrence, but rather distant metastasis to a novel location. Also, this patient featured an exceptionally high proliferation index, indicating that BCAC has the potential for clinical heterogeneity. Finally, this case highlights the need for further research into the role of imaging surveillance during remission.

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**CASE PRESENTATION & OUTCOME**

A 76-year-old male with a past medical history of BCAC who underwent right tongue base excision with bilateral neck dissection followed by adjuvant radiotherapy in 2018 and prostate cancer treated with radiotherapy who presented with a one-day history of hemoptysis. Two weeks prior to initial presentation, he underwent a CT-guided lung biopsy for a right upper lobe nodule that was nondiagnostic. There was concern that his hemoptysis was related to his recent procedure, so he was transferred to the academic medical center for further evaluation.

On presentation, the patient felt well overall and had not experienced any additional occurrences of hemoptysis. Vital signs were stable, and physical examination was significant for decreased breath sounds in the right lower lung field. CT chest with contrast was performed and revealed an enlarging mediastinal and right infra-hilar lymph node causing severe stenosis of the bronchus intermedius, right middle, and right lower lobe bronchi. Findings also included direct invasion with an intraluminal thrombus of the right superior pulmonic vein (Figures 1A, 1B).

On hospital day 2, patient underwent bronchoscopy that revealed extensive endobronchial tumor involvement of the bronchus intermedius resulting in approximately 80% luminal compromise (Figure 1C). Tumor debulking was performed as well as tracheal stent placement (Figure 1D). Patient was discharged following the procedure, and an outpatient PET scan confirmed malignant involvement (Figure 1E). Pathology of the endobronchial mass revealed a poorly differentiated basaloid epithelial neoplasm that was morphologically similar the prior tongue biopsy. Of note, histology revealed an exceptionally high proliferation index (Ki67 > 90%). Comprehensive genomic profiling did not reveal any potential targeted therapies. After discussion with the multidisciplinary tumor board, the decision was made to pursue palliative radiotherapy followed by pembrolizumab monotherapy. The patient expired at home approximately two months after initial diagnosis.
Internal Medicine Residents’ Experience with using Handheld Ultrasound Machines in Point-of-Care Ultrasonography

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Point-of-care ultrasonography (POCUS) is defined as the acquisition and interpretation of ultrasonographic images generated by the clinician at the bedside. The advent of handheld machines has increased access and practical application of ultrasound technology in internal medicine training and medical education. The most common system involves a single portable ultrasound probe that connects to a smartphone or tablet, and storage of images are stored via cloud-based technology. We discuss our experience with POCUS using handheld ultrasound machines in the Thomas Jefferson University Hospital academic setting.

There are several benefits to having a handheld ultrasound machine as an Internal medicine resident. Immediate access to ultrasound technology has advantages in many settings, but especially in emergent settings such as rapid response alerts with critically ill patients in emergent situations. Specifically, POCUS imaging lung views (B-lines, pleural effusions, lung sliding, hepatization), global heart function, IVC collapsibility, vascular access are very helpful. In comparison, the process of moving the department ultrasound machine to the scene of a rapid response alert can take up to 10 minutes, assuming a machine is available. The clinical applications of POCUS as an adjunct to the physical exam and auscultation with a stethoscope have been emphasized by national groups; POCUS integration into diagnostic pathways allows for immediate visualization and results while avoiding radiation and high costs. There is an added benefit of increasing face to face time and direct patient care. The ability to store imaging on cloud services, which is a common service for the handheld ultrasound machines, also provides an opportunity for educational review and comparison with reference images.

An example of the use of POCUS as an Internal medicine resident: I responded to a rapid response alert to the dialysis unit, which is a setting in the hospital that lacks many of the resources of the wards or critical care units. The alert was called for a 50 year old male in respiratory distress. He was admitted for hyperkalemia and he has a past medical history of end stage renal disease requiring thrice weekly dialysis as well as compensated heart failure. This was his first dialysis session after being admitted for hyperkalemia. The patient was feeling very short of breath and anxious. Chest x-ray was ordered but would take 10 minutes to arrive. The initial impression of the responders was that given the medical history, the patient was volume overloaded and the respiratory distress could be from flash pulmonary edema. On my exam, ultrasound of the lungs demonstrated only A-lines and normal lung sliding, with no B-lines or pleural effusions. In addition to the poor air movement and lack of significant rales on auscultation, the differential changed and favored bronchospasm. Treatment was started with albuterol-ipratropium. Later, chest XR confirmed the POCUS findings. Soon, the patient also started breaking out in an urticarial rash and continued shortness of breath with wheezing. Dialysis was stopped and he was treated with epinephrine, intravenous steroids, and antihistamines. Urgent labs and vascular access were also obtained by ultrasound guided venous access. Later we found that the patient was having an allergy to the dialysis filter, causing an anaphylactic reaction. The use of POCUS enabled immediate diagnostic information that changed our differential and prompted different treatment. Especially with the finding that the dialysis filter had been the reason for decompensation, if the original working diagnosis of flash pulmonary edema and volume overload had led to increasing the ultrafiltration rate on dialysis, then the patient could have further decompensated.

There are also barriers and disadvantages to use of handheld ultrasound machines in the inpatient setting. Unless trained, residents should not rely on POCUS in clinical decision making; it should only be used for educational purposes. What you see on an educational scan can prompt ordering “formal” imaging though.
Owning the machine alone doesn’t replace an ultrasound tech, radiologist, and specialist experience. The opportunity to receive formal mentorship can be limited by faculty availability and access to the machines. The cost of the machine can also be prohibitive for individuals to purchase, ranging from $2000 to $5000.

The following clinical images serve as an example of resident utilization of POCUS devices: cardiac imaging of heart failure secondary to chronic hypertension (Figure A), abdominal imaging of large volume ascites in alcoholic cirrhosis (Figure B), retroperitoneal ultrasound of hydronephrosis in a patient with acute renal failure (Figure C), and vascular imaging revealing thrombus in proximal brachial vein of right upper arm using biplane imaging (simultaneous longitudinal and transverse views) (Figure D).
George Washington suddenly fell ill with a sore throat and labored breathing at his estate in 1799. Initial management consisted of a “mixture of molasses, vinegar and butter,” that was followed by “sage, tea and vinegar.” With no signs of clinical improvement, his doctors were called to his bedside. As was standard medical care at the time and thought to be beneficial in various afflictions, he was “bleed” more than 2L of blood in an attempt to restore his good health. The three doctors overseeing the bleeding process noticed the General become weaker despite their best efforts. His breathing became more labored and he passed shortly after his treatment.

Since 1799 medicine has evolved. Treatments, like “bleeding” or bloodletting, were researched for effectiveness and weighed against potential harms. Finding generally poor outcomes associated with the treatment, the practice was abandoned and is no longer used in modern medicine. In fact, many treatment methodologies that were once accepted by the medical community as the standard of care even a few decades ago, have fallen out of favor today in the wake of ongoing research and discovery.

As a modern physicians, we have the benefit of hindsight, an ever expanding knowledge base and a library of publications in every specialty to inform our practice today. The medical community has documented epidemics through incidence spikes recorded in collected data, thus allowing research to meet the communal need for intervention. There is one epidemic, however, for which research is stifled. In the particular case, modern interventions are largely agreed upon to ensure patient safety, but are blocked from practice. Movement on this particular public health ailment has been halted; modern medicine and lawful intercession cannot move forward. This unique plague is the gun violence epidemic.

We have seen rates of gun violence escalate nationally at remarkable rates. Unlike other epidemics, data gathering is difficult largely due to an amendment to a spending bill that has restricted the Centers for Disease Control from funding research on gun deaths and injuries since 1996. Physicians agree that the rise in gun violence should be viewed as a national epidemic. Organized medical groups, such as the American College of Physicians, have put out position papers urging for research, legislative change, and for gun violence to formally be labeled and managed as an epidemic. Unlike generations before us who have observed outcomes, collected data, researched, analyzed, and adjusted practice to be evidence based as to minimize harm, as a country we have instead chosen to turn to a 250 year old document to be our absolute guide on our practice.

General Washington’s cause of death has been extensively reviewed in the literature for centuries. Publications use the evidence recorded at the time and weighed that against the growth in understanding of the modern medical community. Advancements in strategies to better diagnose, predict outcomes and treat have shifted our understanding of the underlying pathology from what those bedside doctors had available to them two centuries ago. It is now believed that Washington suffered from acute epiglottitis and that the management of bloodletting hastened Washington’s death significantly.

Despite constraints on collecting data, the science around gun violence while limited, has persisted. We see study after study informing the public that gun violence is increasing. We have evidence supported models for intervention strategies. Research has shown us that age limits, assault rifle bans, and universal background checks could be manages to manage this epidemic. Why do we follow evidence based guidelines in all other aspects of medical and epidemic interventions except for the issue of gun violence? Why is this the pathology that we refer back to the practices of 250 years ago?

Washington’s contemporary and namesake of our hospital and university, Thomas Jefferson, was among the authors of the very document we use to dictate our modern management of gun violence: The United States Constitution. Yet even he would disagree with this strategy of rejecting modern advancements and evolving practices in the name of the tradition and loyalty to the constitution. In 1789, Thomas Jefferson wrote to his friend James Madison on the question of whether one generation of men has the right to preside over another.
He wrote:

“I suppose [this] to be self-evident, ‘that the earth belongs in usufruct to the living’: that the dead have neither powers nor rights over it...On similar ground it may be proved that no society can make a perpetual constitution, or even a perpetual law. The earth belongs always to the living generation.” (Jefferson, 1789)

Jefferson advocates for renewed reflection, research, and change with each new generation. Similar to medicine in which the field and guidelines are always changing with new generations of scientists and assumed new understandings, so too should we update our societal practices and laws with the growing body of knowledge.

If Washington presented to a modern hospital today we would have been diagnosed with acute epiglottitis with radiographic imaging, and treated with appropriate antibiotics. The management for his fatal pathology has evolved from bloodletting. How much more blood needs to be shed as a result of the gun violence epidemic until we acknowledge our current laws are causing harm. It is time to change. We can no longer let the laws and practices of the dead rule over the living.

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