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LBJ Farm and Rehabilitation Center

Over a year in planning, our new farm is the culmination of an inter-department initiative to provide our fellow Houstonians with the information and first steps into healthy habits along with access to fruits and vegetables. There is also space for rehabilitation and recreation for our community members. We hope to integrate our clinics with our farms and assist with exercise programs.

Photo Cred: Tiffany Truong, MD
Finding Coarctation of Aorta in a patient with bicuspid aortic valve who presented with spontaneous Mitral valve chordal rupture.

Jawad Chohan, MD
Syed Humayun Naqvi, MD

Abstract:
Coarctation of the aorta is often associated with other abnormalities that, when not identified early, can progress to severe, sometimes life threatening symptoms. This case demonstrates the sequelae that can occur when the coarctation and its associated abnormalities are not identified and managed early.

Introduction:
Early identification and prompt intervention of potentially life threatening cardiac pathology is crucial to prevent serious complications. In this case, bicuspid valve and aortic coarctation were not diagnosed early, and the delayed diagnoses led to potentially preventable complications that required urgent and invasive cardiothoracic surgery.

Case:
A 37-year-old man with hypertension and hyperlipidemia presented to Memorial Hermann Hospital-TMC with four months of progressive dyspnea on exertion and non-radiating chest pain. He denied any personal or family history of heart disease. On presentation, he was found to have increased work of breathing with a loud holosystolic murmur and jugular venous distention. Initial chest radiographs also demonstrated cardiomegaly and pulmonary edema. Transthoracic echocardiogram (TTE) demonstrated a severely dilated left ventricle with global hypokinesis. Given his clinical presentation, physical exam findings, and echocardigraphic results, the patient was diagnosed with decompensated heart failure. Additionally, there was tethering of the posterior mitral valve leaflet noted on TTE causing moderate mitral regurgitation as well as mild dilation of the ascending aorta to 4.0 cm. Transesophageal echocardiogram (TEE) confirmed the findings as above and also demonstrated a flail anterior mitral leaflet with ruptured chordae. The TEE also revealed a bicuspid aortic valve (BAV) with fusion of the left and right coronary cusps (type 1), focal nodular thickening of the lateral leaflet, and moderate aortic regurgitation. Coronary angiogram showed no evidence of obstructive coronary disease. Subsequent thoracic aortogram revealed coarctation of the aorta distal to the left common carotid artery with post stenotic dilatation. Computed tomography of the chest with contrast confirmed a thoracic aorta coarctation 1.9 cm distal to the origin of the left subclavian artery with a post stenotic aneurysm dilatation of the descending thoracic aorta to 4.5 cm. He underwent a staged surgery for resection of the coarctation and replacement with a Dacron Tube Graft at T6. He was monitored for improvement of the valvular regurgitation to determine the need for additional surgery. The aortic and mitral regurgitation failed to improve following coarctation repair and patient was readmitted for decompensated heart failure. On readmission, both valves were replaced with mechanical prostheses. The patient is now on lifelong anticoagulation for the valves and optimal medical therapy for chronic heart failure.

Discussion:
Coarctation of the aorta is a diffuse aortopathy resulting from cystic changes in the aortic media. If left untreated, coarctation of the aorta can lead to early onset systemic hypertension and can predispose the patient to aneurysms and dissection. It can often coexist with congenital valvular abnormalities such as BAV that has a tendency to become stenotic or develop regurgitation. BAV is also associated with higher rates of infectious vegetations. These complications can ultimately lead to mitral valve abnormalities including chordae tendineae rupture. Valvulopathy can also cause decompensated heart failure as seen in this patient. The morbidity and mortality associated with these numerous conditions is high and often requires prompt surgical intervention. Early detection of coarctation is therefore important in planning for early treatment in at-risk individuals before development of significant complications. While not financially feasible or practical to scan every patient for coarctation, techniques can be employed in the primary care setting to aid in identifying patients at risk. Established methods described in the
literature includes careful auscultation of the left interscapular region for murmurs or bruits suggestive of coarctation or collateral vessels as well as the anterior chest for systolic ejection murmur suggestive of BAV.\textsuperscript{2,5}

**Conclusion:**
Coarctation of the aorta when combined with other cardiac abnormalities has the potential to progress to severe disease if not corrected early. This case highlights the consequences of delayed diagnosis and intervention that can lead to worsening heart function requiring more invasive cardiac procedures. Furthermore, it stresses the importance of implementing effective screening methods in the primary care setting.

**References:**
Phentermine-induced Seizure

Sara Ali, MD

Abstract
The global obesity epidemic has serious health concerns up to and including metabolic syndrome, and joint destruction. While there are several weight loss methods, they can pose significant health concerns as well, especially pharmacological therapy. Here, we present a case of a weight loss medication, phentermine causing a seizure.

Introduction
Obesity, defined as body mass index > 30 kg/m2, is a global epidemic effecting more than one-third of the adults in the United States. Treatment of obesity can include lifestyle modification, pharmacotherapy and surgical intervention. A wide array of pharmacotherapy exists and remains a popular option for many. Given the convenience and increasing popularity of weight-loss drugs, healthcare professions need to be cognizant of adverse effects of the pharmacotherapy. Here, we present a case of phentermine-induced seizure to demonstrate a serious adverse effect of weight loss pharmacotherapy in the obesity epidemic.

Case
A 37-year-old Caucasian female presented to the emergency department after experiencing three witnessed generalized tonic-clonic seizures. The duration of seizure was noted to be two minutes with tongue biting and urinary incontinence. She had no prior history of seizures. Her medical history was significant for obesity and Stiff Person Syndrome. Her only home medication was phentermine. Upon presentation to the emergency room, she had a witnessed tonic-clonic seizure and was given an intravenous dose of lorazepam followed by intravenous levetiracetam. The patient’s vital signs on admission were as follows: temperature of 99.2°F, heart rate of 113 beats per minute, blood pressure of 133/69 mmHg, respiratory rate of 18 breaths per minute, and an oxygen saturation of 99% while breathing ambient air. On physical exam, she was agitated and not following commands. Her pupils were dilated to 6 mm bilaterally. She was able to move all her extremities without focal deficits, 2+ deep tendon reflexes and an absent Babinski sign.

Initial laboratory results demonstrated white blood cell count of 16.6 K/uL with 91% neutrophils, lactic acid of 0.8 mmol/L and creatinine kinase of 6,829 U/L. Her serum glucose level was 53 mg/dL and urinary drug screen positive for amphetamine. Lumbar puncture revealed cerebrospinal fluid (CSF) studies with white blood cell of 5/uL, red blood cell of 5,000/uL, protein of 40 mg/dL, glucose of 69 mg/dL and IgG of 1.82 mg/dL. Magnetic resonance imaging (MRI) of the brain was unremarkable without intracranial abnormalities. An electroencephalogram (EEG) was performed without evidence of abnormal electrical activity. The results of the CSF cultures, enteric cytopathic human orphan viruses, West Nile virus, cytomegalovirus and Epstein Barr which were negative. Initial treatment included aggressive fluid repletion. Over the course of two days, her mental status improved and returned to baseline without repeat episodes of seizures. Patient admitted she had been taking more than the prescribed quantity of phentermine in hopes of losing weight faster. She was unable to quantify how much more or the duration. A presumptive diagnosis of amphetamine-induced seizures with rhabdomyolysis was made. Upon discharge, she was instructed to discontinue phentermine.

Discussion
Phentermine was approved by the FDA in 1959 for a short term (3 months) weight loss medication and has since become one the most prescribed appetite suppressants in the world. The mechanism of action is similar to that of a sympathomimetic with increased release of norepinephrine and dopamine, as well as, blocking reuptake.1,2 Side effects mimic increase of sympathetic effects including headache, elevated blood pressure, heart rate, insomnia, dry mouth, overstimulation of the central nervous system and tremors. In the 1990's, a combination therapy of fenfluramine-phentermine had a known association of lowering seizure threshold as well as epileptic agent. Per literature review, there have been cases of patients with a history of seizures who had spontaneous remittance of
seizure activity while on phentermine and after discontinuation of therapy, remained seizure free without antiepileptic medication.3

Although limited literature exists on long term use of phentermine, the increased noradrenergic levels in the brain seem to be as the mechanism for its proconvulsant activity. It is unclear if phentermine promotes development of seizures or unmasks a predisposition.3 This case demonstrates the importance and benefit of being aware of long-term effects of phentermine. Physicians should be cognizant of the potential side-effects and phentermine should be used cautiously in patients with underlying seizure disorder.

References

Chronic Anovulation and its Consequences

Aysha Chaudhri, MD
Shahla Nader, MD

Abstract

Functional hypothalamic amenorrhea is a cause of secondary amenorrhea not due to an organic etiology. It is caused by low energy availability which can be secondary to decreased caloric intake or excessive energy expenditure. Diagnosis and treatment is critical since serious adverse health effects are associated with a chronic estrogen deficient state. We present a case of functional hypothalamic amenorrhea to demonstrate the complex nature of this disorder and the therapeutic challenges commonly encountered.

Introduction

Secondary amenorrhea is the absence of menses for three months or longer in females who previously had regular menstrual cycles or for six months or longer in females who had irregular menstrual cycles. A common cause of secondary amenorrhea includes hypogonadotropic hypogonadism such as functional hypothalamic amenorrhea (FHA). FHA accounts for 25 to 35% of cases and is a diagnosis of exclusion after organic causes have been ruled out. The most common causes of FHA include low energy availability and stress. In women of reproductive age, effects of secondary amenorrhea are those associated with an estrogen deficient state including decreased bone density, infertility and sexual dysfunction. FHA can potentially also have cardiovascular consequences including development of early coronary artery disease. The Endocrine Society recommends a multidisciplinary approach to treat FHA. The guidelines include correcting the energy imbalance, nutritional support, psychological support, avoiding bisphosphonates to improve bone mineral density, avoiding ovulation induction until BMI and energy balance have normalized, as well as short-term use of transdermal E2 therapy with cyclic oral progestin in women who have not had a return of menses.

Case

The patient is a 42 year-old female initially referred to endocrinology for amenorrhea. Patient’s menarche occurred at age thirteen with monthly menstrual cycles that lasted for five days. She had her first child in 2002 at age twenty-seven. She breast-fed from 2002 to 2006. However, breast feeding was exclusive only for the first eight months. In 2006, the patient adopted three children. The amenorrhea started in 2006 and had been ongoing for ten years. From 2016 to December 2017, the patient had minimal spotting once every six months. The patient presented to our clinic in December 2017 for evaluation of amenorrhea. She had a progestin challenge with no subsequent bleeding indicating that the patient had not had endogenous estrogen exposure. She also had a uterine sonogram which did not show any anatomical abnormalities.

At the time of our evaluation, she had normal libido and denied vaginal dryness, hot flashes, night sweats or galactorrhea. She denied any headaches or visual disturbances. Prior to 2016, she would exercise four to five times per week. From 2016 to 2017, the patient would do aerobic exercise one to two times per week for forty-five minutes each time. The patient was never a marathon or long-distance runner. Her weight history was as follows. Her lowest weight was in 2007 with a BMI of 17 kg/m2 and weight of 125 pounds. Her maximum weight outside of pregnancy was 148 pounds.

On initial presentation, her BMI was 19 kg/m2 and weight was 136 pounds. Her physical exam was not significant for any abnormalities and the patient appeared well nourished. Hormonal work up at initial presentation in 2017 included normal TSH, normal prolactin (3.7 ng/ml), normal FSH (9.9 mIU/ml), low LH (1.88 mIU/ml), and low estradiol (13.9 pg/ml). Pituitary MRI was normal. Due to prolonged amenorrhea and a hypoestrogenic state, a DEXA scan was ordered which showed osteoporosis of the L1 to L4 spine (T score -2.7) and osteopenia of the total hip (T score -1.8) and femoral neck (T score -1.5).
Patient’s clinical and laboratory studies were most consistent with functional hypothalamic amenorrhea. Hormonal replacement was discussed with the patient for bone health. Patient deferred hormonal replacement initially and opted to attempt weight gain to restore normal function of her hypothalamic-pituitary-ovarian (HPO) axis. In January 2018, one month after the initial evaluation, patient’s weight had only increased by three pounds with weight at that time of 140 pounds (Table 1). She was still not having menstrual cycles. Hormonal replacement was again discussed with the patient and she agreed to an estradiol 0.1 mg patch as well as progesterone 200 mg for the first twelve days of each month to maintain uterine health. At her follow up appointment in February 2018, patient admitted that she was not compliant with hormonal therapy and self-discontinued the medication after two weeks of treatment because it caused excessive nausea with some emesis. Although she was offered a lower dose of hormonal treatment, she wanted to attempt weight gain again. At this time she weighed 144 pounds. Patient was told that she would have to gain enough weight to exceed her prior maximum weight of 148 pounds to regain normal function of her HPO axis. By May 2018, patient achieved a weight of 154 pounds and had two menses. Her estrogen and progesterone were checked to determine if patient was ovulating. Labs showed estrogen of 78 pg/ml and progesterone of 14.8 ng/ml in the luteal phase of cycle indicating that patient was ovulatory. Patient’s most recent visit was in July 2018. She continued to have monthly menstrual cycles and progesterone levels again indicated that patient was ovulatory. Given the resolution of her amenorrhea and return of normal ovulation patient was able to achieve pregnancy which is what she desired.

<table>
<thead>
<tr>
<th>Encounter Date</th>
<th>Weight</th>
<th>BMI (kg/m2)</th>
<th>Treatment recommendations</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>December 2017</td>
<td>137 lbs</td>
<td>19.1</td>
<td>Hormonal replacement</td>
<td>Patient deferred treatment, no menses</td>
</tr>
<tr>
<td>January 2018</td>
<td>140 lbs</td>
<td>19.5</td>
<td>Hormonal replacement</td>
<td>Patient not compliant with treatment, no menses</td>
</tr>
<tr>
<td>February 2018</td>
<td>144 lbs</td>
<td>20.1</td>
<td>Hormonal replacement</td>
<td>Patient deferred treatment, no menses</td>
</tr>
<tr>
<td>May 2018</td>
<td>154 lbs</td>
<td>21.5</td>
<td>Maintain weight</td>
<td>Beginning to have menstrual cycles</td>
</tr>
<tr>
<td>July 2018</td>
<td>156 lbs</td>
<td>21.8</td>
<td>Maintain weight</td>
<td>Continued monthly menstrual cycles</td>
</tr>
</tbody>
</table>

Table 1: Patient’s weight, treatment recommendations and outcome are presented in chronological order.

**Discussion**

Functional hypothalamic amenorrhea is a complex phenomenon effecting multiple endocrine axes including the hypothalamic-pituitary-ovarian (HPO) axis. Energy deficit, weight loss and stress are the main causes of FHA. These are all associated with a decrease in gonadotropin-releasing hormone (GnRH) secretion subsequently leading to a
decrease in gonadotropin secretion. These abnormalities lead to reduced estradiol production in the ovary resulting in profound hypoestrogenism which can cause many adverse health effects in women.\textsuperscript{6}

Estrogen and androgens also play a key role in bone health. Bone loss or inability to achieve peak bone mass is the most significant chronic risk factor of FHA. As seen in our patient, long-standing FHA can lead to osteoporosis in young women which increases fracture risk, specifically stress fractures. A low energy state can lead to low bone formation and low bone turnover as well as increased rates of bone resorption.\textsuperscript{2} The International Society for Clinical Densitometry recommends a densitometry study if amenorrhea secondary to low estrogen states has been ongoing for at least 6 months.

Cardiovascular health is also potentially adversely affected. Estrogen deficiency can have negative effects on endothelial function and vascular resistance. Women with FHA may be at higher risk of developing early coronary artery disease. They have more unfavorable metabolic profiles including higher total cholesterol, higher triglycerides and higher low-density lipoproteins as well as impaired glucose tolerance compared to controls.\textsuperscript{3}

Abnormal functioning of the HPO axis leads to anovulation and infertility causing issues with reproductive health. Even after treatment of FHA, there is an increased risk of miscarriage and pregnancy-related complications. It is important to achieve an adequate weight before embarking on pregnancy.\textsuperscript{7}

Conclusion

Functional hypothalamic amenorrhea can be a challenge to diagnose and treat. The Endocrine Society recommends evaluation for functional hypothalamic amenorrhea in women whose menstrual cycle interval is consistently longer than 45 days and in women who present with amenorrhea for 3 months or longer. This diagnosis should only be made after organic or anatomic pathology for amenorrhea has been ruled out. Prompt diagnosis is needed to prevent adverse effects related to prolonged estrogen deprivation. Physicians should work with their patients to establish a treatment plan that is tailored to their patients' needs and goals of care.

References

Rare life threatening presentation of embolic acute myocardial infarction from peripartum cardiomyopathy with left ventricular thrombus.

Farzan Husain, MD  
Ritesh S Patel, MD  
Abhijeet Dhoble, MD

Introduction
Peripartum Cardiomyopathy (PPCM) is a rare, life-threatening condition of pregnancy that is associated with significant morbidity and mortality. A rare, but fatal complication associated with PPCM is thromboembolic events originating from a severely dysfunctional left ventricle (LV). We present a 17-year old female with PPCM and multiple mural thrombi complicated by multiple coronary emboli leading to acute myocardial infarction.

Case
A 17-year old woman with history of pre-eclampsia presented to the emergency department with complaints of increased dyspnea, fatigue, and chest pain within the last 24 hours. She was 8 weeks postpartum and her pregnancy was complicated by pre-eclampsia, which required her to deliver at 37 weeks. Since then, she had been experiencing worsening dyspnea, orthopnea, intermittent lower extremity swelling, and fatigue. She also reported weight gain of 20 lbs, right upper quadrant pain, and decreased appetite. She described her chest pain as intermittent, dull-achy, and right-sided. She did not have any previous medical history or family history significant for cardiac disease. Her presenting vital signs were heart rate of 122, respiratory rate of 20 on two liters oxygen on nasal cannula, blood pressure of 117/85 on dobutamine for ionotropic support. Physical exam revealed a loud tricuspid regurgitation murmur without jugular vein distention or peripheral edema. Electrocardiogram showed Q-waves in the leads I, AVL and >1 mm ST- elevation in leads V2-V5 (Figure 1). Troponin-I was elevated to 11.50 ng/ml. Echocardiogram showed mildly dilated LV with severely reduced systolic function and an ejection fraction of less than 20%. Additionally, multiple thrombi were seen in the LV apex.

Given the electrocardiogram and echocardiogram findings with elevated cardiac markers, an emergent coronary angiogram was performed. On the coronary angiogram, there was a thrombotic occlusion of the large second (D2) and smaller third diagonal branch of the left anterior descending (LAD) Artery. There was also a distal thrombotic occlusion of the right posterior descending artery. Aspiration thrombectomy was performed twice with aspiration of dark red thrombus and restoration of flow to TIMI Grade II and no further angiographic evidence of thrombus. Additionally, 10 mg of intra-coronary intravenous abciximab was administered in the second diagonal branch of the LAD. Repeat images showed resolution of the thrombus in the D2 with TIMI Grade II-III flow restored. However, the combination of intra-coronary anti-platelet therapy and aspiration thrombectomy did not lead to meaningful recovery as expected. Unfortunately, the patient required placement of a left ventricular assist device intended to bridge for a heart transplant. She later successfully underwent orthotopic heart transplant.

Figure 1: Electrocardiogram with Q-waves in lateral leads I, AVL and >1mm ST- elevation in leads V2-V5.
Figure 2: Cath Images (A) Before and (B) after aspiration thrombectomy

Discussion
PPCM is a rare, life-threatening cardiac condition that manifests late in pregnancy or the few months in the postpartum period. The European Society of Cardiology (ESC), defines PPCM as an idiopathic cardiomyopathy with LV systolic dysfunction towards the end of pregnancy or within 5 months following delivery, with no other identifiable cause for heart failure, and left ventricular ejection fraction less than 45% with or without LV dilatation.¹ The incidence of PPCM is estimated to affect 0.025% to 0.03% of live births worldwide with propensity toward women of African descent.² The risk factors for developing PPCM included advanced maternal age, multiparity, multiple gestations, African descent, pre-eclampsia, and gestational hypertension.³ Typically, patients with PPCM present with severely reduced left ventricular function at the time of diagnosis, causing dyspnea, lower extremity edema, and fatigue within the first few months of the postpartum period. Upon extensive literature review, only three case reports of acute myocardial infarction due to coronary emboli in the setting of a LV thrombus and peripartum cardiomyopathy were found. However, there are several case reports describing various thromboemboli to pulmonary and peripheral arteries.⁴⁻⁸ Aspiration thrombectomy was attempted in one case, and intra-coronary antiplatelet therapy was performed in another case. Both interventions led to patient recovery. This case is unique as it is the first case to describe multiple coronary emboli being treated with both aspiration thrombectomy and intra-coronary antiplatelet therapy. Unfortunately for our patient, the combination of intra-corporal antiplatelet therapy and aspiration thrombectomy was unsuccessful in resolving the myocardial infarction. We believe it was most likely due to heavy burden of intra-coronary clots complicated by cardiogenic shock, which limited our patients’ response to the afore-mentioned interventions. Also possible was reperfusion injury, as the patient decompensated after her intervention and subsequently required extracorporeal membrane oxygenation (ECMO). The presence of multiple intracoronary thrombi in the setting of LV thrombi suggests that the LV was the source of thromboemboli, which is similar to previous case reports.⁵⁻⁷ PPCM is associated with higher incidence of thromboembolism with LV thrombus discovered in 10% - 17% of initial echocardiograms. The increased incidence of LV thrombi in PPCM may be due to the known hypercoagulable state during pregnancy,⁹ and an increased risk of hemostasis in a dilated, dysfunctional LV. Anticoagulation should be considered in all patients with PPCM for these reasons, and may be especially indicated for patients with severe LV dysfunction, presence of systemic embolism, and/or known LV thrombus. Our case report further documents another incidence of intracoronary embolism due to multiple LV thrombi. Currently, no specific guidelines or clinical trials exist for long-term management of patients with coronary embolism. The general consensus is to treat coronary emboli with long term anticoagulation. However, coronary emboli causing acute myocardial infarctions (MI) are associated with much
higher mortality than MI due to plaque rupture.\textsuperscript{10} Therefore, more clinical investigations regarding interventions and management of coronary emboli are warranted. It is likely management strategies would differ depending on the etiology of the MI. More specifically, better understanding the incidence of coronary emboli in PPCM may help uncover prevention strategies in patients that are at high risk for PPCM.

References

9. Functional and immunologic protein S levels are decreased during pregnancy. Comp PC, Thurman GR, Welsh J, Esmon CT
Grey Zone

Astrid Grouls, MD

Abstract
This documents my experience caring for an unusual patient, with a reminder that sometimes medicine may push your limits.

Grey Zone

I dodged back to his room after noon conference ended. His room had the perk of being a corner room, but he was too far gone to appreciate the light streaming into the edge of the room, sunbeams out of reach of the hospital bed, shrouded in shadow.

“I just removed his breathing tube a few minutes ago,” his nurse said quietly as I marched in. I just wanted to ensure things were moving smoothly – that he was as comfortable as he could be for whatever time he had left. I didn’t intend to stay longer than necessary.

His chest no longer had the reliable rise and fall it did before. His breath was as ragged as he was – hair matted, skin swollen and bruised, eyes fixed on the ceiling unblinkingly. It was a pitiful sight – one I’d seen before. While I regretted that no family stood vigil next to him, I also knew how painful this may have been for them to see.

He’d arrived with impressive head injuries over a week before. While he was initially stable in the emergency department, he rapidly developed symptoms of lethargy and was found to have a massive hemorrhage. Despite surgical intervention, his injuries were devastating and an ethics committee had determined to stop life-prolonging treatment after a week-long search for both improvement and family.

“We should go ahead and call the chaplain,” I told the nurse. The monitor bleeped with his rising pulse. Eighty, ninety. Minutes to hours, I thought to myself. I’ve always appreciated the grey zones in medicine – that period when nothing is absolute. The end-of-life still felt to me like a grey zone. You can never predict just how long someone will make it.

A guard shuffled from the corner of the room, trying not to get too close. “Is he --?” making a slicing motion at his throat. His avoidance of the word “dead” seemed less jarring to me than the slicing. It felt crude and unnecessary.

“Not yet.”

He retreated back to his couch and his phone. I wondered who he could be texting.

I stood with my patient as his breaths grew more shallow, his heart began to slow. Eighty. Seventy. Fifty. Minutes passed, but it felt longer. The chaplain wandered in as his respiratory rate must have hit just five or so. He took his last breath as the last rites began. As the chaplain proceeded along the familiar words, his heart continued to slow. I wondered if he’d been a religious man. We turned off the monitor as the beeping of the alarms grew more constant.

It wasn’t long until I called time of death.

The figure wandered back again. He had a renewed interest as he’d seen a bit more commotion at bedside. “So that’s it, then?”
I felt anger welling in me, but I couldn’t quite place why. “Yes. Time of death 13:42.” I wanted to chastise him for fiddling on his phone instead of bearing witness to this man dying surrounded by strangers. I didn’t know my patient’s past, but surely we all deserve a moment of recognition when we pass, prisoner or not. This was one aspect of end-of-life that wasn’t grey to me. No one should die alone.

Later I spoke with the medical examiner. I recounted to her the laundry list of injuries documented on arrival – cervical fracture, jaw fracture, orbital fracture – all head and neck. She asked if a cause for the injuries was identified. “Per report on arrival, the patient spat at a guard, who retaliated.”

There was nothing grey about that.
Into the Sun

Marina Ibraheim, Medical Student

Abstract
In Edgar Allen Poe’s piece, *The Masque of the Red Death*, Prospero “bids defiance to contagion” to evade the Red Death. Prospero’s plight, when analyzed alongside a student’s encounter during a medical mission trip in Guatemala, captures the universality of the struggle against Death and its cost.

Into the Sun
The Guatemalan sun was unrelenting. It beat down on everyone—on the children who chased the dogs that ran around the school-turned-clinic, on the mothers who attempted to keep the kids from straying too far, on the old women who gossiped together and fanned themselves while old men shared curt nods of understanding, and on us students who had volunteered to go on this medical mission trip to Guatemala. The Guatemalans weathered the fiery beatings fairly well, but they could tell by looking at our dripping faces, squinting eyes, and our steadily reddening faces that the sun and heat were merciless.

After taking the patients’ vital signs and filling out the relevant information on their one-page “chart” we would chat. Sometimes I asked about the region; sometimes I asked about their families; but most of the time I asked about the weather. They would smile wryly and chuckle, “Yes, it’s always hot here during this time. Yes, *this* hot.” Before I could pry further, the doctor would call for the next patient. We reluctantly parted ways, and I was left to think about the blinding heat.

At one point, an old woman approached. After greeting her, I gathered her vital signs, filled out her “chart,” and handed it back to her. After this, we waited together in silence as I wracked my brain for a conversation starter. I did not want to ask her about the weather. Instead, I wanted to know how she came upon this particular clinic.

“What time did you arrive here?” I asked.

“I arrived at 4 AM. I had to leave my town at 2 AM. It’s cold up there compared to here.”

She had waited in line outside the clinic: both before and once it had opened. She had to wait with me before going to the doctor while the noontime sun scorched us both.

What convinced her to endure the cold, the heat, the agony of walking and waiting for so long? I did not articulate this question—I knew why: She had no choice. Healthcare in Guatemala is virtually inaccessible to the majority; it is a commodity of the wealthy. Consequently, many Guatemalans are forced to latch onto any opportunity to access healthcare. So, they wait. Then they get up early. Then they walk, weathering the beatings of the sun and scuffling against the grass and gravel that gradually mar their well-worn shoes. Then they wait once more. Then they hope that these foreign student volunteers and foreign doctors are knowledgeable enough to help them. Then they pray that the makeshift pharmacy does not run out of the prescription drugs that they need. Then they pray that they do not become sick again since they do not know when they will see a doctor again. And, if they do have the misfortune of falling ill, then they hope that a new group of volunteers will arrive shortly. Then they endure the same litany of events once more.

This is their price to pay in order to stay alive—this is their cost of living.

Is this fair? No. This woman was consigned to a fate of poverty and poor healthcare, a fate seemingly as fixed as the sun’s indiscriminate, unrelenting, and searing rays.
It is tempting to think that Americans are not forced to pay this price, but this is not always the case. Though indeed as a medical student I am extremely fortunate to live and study during a time in which scientists and clinicians have made great progress in treatment of chronic diseases—afflictions that can kill or severely maim people if left untreated—I have seen the cost of such feats, costs that can seem incalculable when a patient has not yet begun to suffer physically. It seems that direly ill patients are confronted with the following choice: Would you rather part with your money or with your life? Is this a reasonable question to ask—after all, can a person part with his money if he never had it at all? The underpinning of these questions is the premise that money, backed by knowledge, can save people from Death. The premise grants us more control than we actually have. We hide behind the guises of money and knowledge hoping that Death will fail to see us, but it always does so eventually. We see this clearly in Poe’s *The Masque of the Red Death.* The characters are brought face to face with the reality that Death follows them regardless of their reluctance to admit it. The horror that the characters experience stems from the fact that they are forced to confront reality as it is, not as they had hoped it would be.

In response to The Red Death ravaging the land and its inhabitants, Prince Prospero refuses to confront Death head on. Instead, he dons a veil that distorts his vision; it convinces him that he can escape Death and prevents him from acknowledging that painful reality. In Prospero’s eyes, Death cannot touch him so long as he remains in his fortified castle. In doing so, Prospero “bids defiance to contagion”, reflecting his arrogance. He falsely assumes that wealth and isolation from those who are ill will protect him from Death. Like the sun, The Red Death does not discriminate, and his “defiance” proves pitiable and useless. Prospero’s arrogance and inability to contemplate Death trap him and his courtiers in a fortified grave, unable to escape.

In Prospero’s defense, contemplation of Death is painful, as it reveals our frailty and our fear of imagining the inevitable. It is infuriating and humiliating to be confronted by that reality. When Prospero finally realizes that The Red Death has infiltrated his castle, Prospero “maddens with rage and the shame of his own momentary cowardice”. The Red Death has made a fool of Prospero: He has shown Prospero that his intelligence and wealth were all for naught; that nothing in the world can save him from the Inevitable. Prospero cannot accept this humiliation, so he decides to fight Death. But this is reality: Prospero must lose.

I do not wish to belittle Prospero for acting in this way, for we do the same. In practicing medicine, patients and doctors attempt to fight the Red Death together despite already knowing the outcome.

But we toil away at this Sisyphean task. We take pill after pill, hoping that contagion will give up the war for conquest over our bodies. We go from doctor to doctor, searching for the one who is knowledgeable and proud enough to save us from—or stave off—the Inevitable. People like the woman in Guatemala travel from village to village, descend from one mountain and ascend another, walk until the soles of their feet are rubbed raw—all in the hopes of fulfilling the impossible task of staying alive for longer than Nature might have otherwise decreed. These actions are our collective attempts at “bidding defiance to contagion” in the same way that Prospero did. Our defiance lasts a lifetime. From birth to death, we are keenly aware of our bleak outcome, but our intractable drive to continue living dictates that we ignore the futility of our precarious situation: that we are on the cusp of death. We blind ourselves to the reality, laugh lightly “at our own nervousness” as the death knell rings on, and hope that readjusting the veil over our own eyes is enough to avoid seeing the fragility and futility and absurdity of our situation, fighting on against an enemy that we have made for ourselves by vainly struggling against it. We are not much different from Prospero—the struggle against death is universal. But the path towards recognizing one’s own limitations is individualistic.

The people in Guatemala whom I met, though thankful for medicine, could see its limits by examining its abilities through the lens of faith. Each time I spoke with them, they thanked God for bringing us there to help. They attributed God to every act; even though human hands and minds worked to bring about reprieve from death for a little while longer, it was God who willed these events into or out of existence in their eyes.
Based on this, one might believe that the Guatemalans lacked the veil that Prospero—and perhaps many Americans—don. But even they cannot remove the veil from their eyes. In fleeting moments along their journey, the sunlight pierces through their veils, allowing them to gain a better glimpse of reality. In these moments, human limitation comes into focus, and in turn, they witness the reality of the struggle. Then the light’s rosy fingertips fade away, leaving them to decide for themselves whether or not the cost they might incur is justifiable.

Perhaps the aim is not to remove the veil. Perhaps the veil protects us from the rays of light that, in their own right, can prove blind and debilitating. Bearing witness to reality, with eyes vulnerable and bare, can become unbearable. Instead, we can pursue the light.

With the veil on, with eyes guided by the light’s rays, the Guatemalans who spoke to me freed themselves from living the rest of their days feeling the rage and humiliation that racked Prospero upon seeing the Red Death. Unlike Prospero, they could see their limitations, and in doing so, they could see their other Sisyphean brothers and sisters toiling away at a problem that wealth can never truly solve.

With the veil on, with eyes guided by the light’s rays, we may attempt to escape from that fortified cage of decadence, falsehoods, and phantasms that we have constructed for ourselves. Behind the veil, as light radiates through each fiber, reality becomes clear enough to reveal our vulnerabilities and renders us aware of how feeble our attempts at elongating life can sometimes be.

With the veil on, with eyes guided by the light’s rays, we can choose for ourselves our own cost of living. We can raise our heads as the warmth radiates through our skin, open our eyes, catch glimpses of glimmering golds and rosy reds as they dance across our eyes, leaving us with nothing but a suggestion of what else might exist beyond that moment. Then we walk until its rays meet us again, sending us running headlong into the sun.
Improving ESAS Documentation Compliance with a Dedicated Patient Care Technician in Palliative Care Clinic

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Abstract

The Edmonton Symptom Assessment Scale (ESAS) is a validated patient-rated symptom assessment scale that is a core quality indicator in palliative care clinics. Documentation of the ESAS is critical to both billing and quality care. At the LBJ Palliative Care clinic, ESAS documentation was noted to be below an acceptable level of at least 90%. The ESAS was administered by patient care technicians (PCT) prior to patients being roomed. After a root cause analysis, we theorized that having the same PCT for daily palliative care clinic rather than a rotating PCT would increase ESAS documentation rates. A dedicated PCT increased ESAS compliance rates from 72% to 94%, with particular improvement on half-day clinics as compared to full-day clinics.

Introduction

The Edmonton Symptom Assessment System (ESAS) is a validated tool in the hospice and palliative care setting that is structured as a 9-item patient-rated symptom visual analogue scale. (Bruera et al, 1991; Dudgeon et al, 1999) Additionally, the ESAS is a part of the Measuring What Matters core 10 quality indicators. This means that it is mandatory in the evaluation and documentation of a palliative care outpatient encounter. (Dy et al, 2015; Richardson and Jones, 2009)

The patient care technician (PCT) rotation at the Harris Health system clinics limits PCT knowledge of individual clinics and their workflow. In the Harris Health palliative care clinic, PCTs are responsible for giving patients the ESAS form, retrieving it after the patient completes it and logging each answer into the EMR. Therefore, the ESAS in some instances may not be logged, even if it is completed by the patient. We hypothesize that we will have improvement compliance with ESAS documentation with a dedicated PCT for the palliative care clinic.

Methods

The process for ESAS documentation was reviewed. Multiple areas were identified as potential causes of poor documentation, including administrative, patient-related, PCT-related and provider-related. The ESAS is a paper provided in English or Spanish to patients presenting for a palliative care appointment, therefore, printed copies must be available in an easily identified location. A writing utensil may have to be provided as well. Any patients not speaking or reading English or Spanish would be unable to complete the form at present, and may require a family member for assistance. Form completion may also be limited by a physical disability, in which case a family member is presently expected to assist. Other than providing the form, the PCT does not actively participate in the completion of the ESAS. Patients may opt not to complete the form. As the form is currently given to patients after vitals are taken and while patients are waiting in the waiting area before being called back to a room, patients who bypass vitals as direct transfers from other clinics may also be missed for ESAS. The PCT must identify where the form is found, ensure sufficient copies for each day, provide a correct language form to each patient, retrieve it when rooming the patient and log it in EPIC. The PCT documents this via dropdown options in the flowsheets tab. The provider must link to the results in their note for appropriate documentation and billing. The ESAS is automatically populated in the note when the provider uses the appropriate palliative care clinic template. If the note is started before a patient is roomed and the PCT logs the ESAS, the provider must refresh the appropriate SmartPhrase in their note. If an ESAS is not populated in the note, the provider is responsible for communicating to the PCT that it is not present. As residents, fellows and medical students are only inconsistency in the clinic, this often falls to the attending.

On review of the workflow, the PCT was deemed central to ESAS documentation, as they are responsible for ensuring the forms are given to and retrieved from patients, and documented in a manner that will allow for
automatic population in notes by providers. For this reason, it was determined that it was essential to have a PCT familiar with ESAS workflow reliably available in the palliative care clinic.

Patient encounters in the Palliative Care Clinic were assessed for completion of ESAS documentation in February 2017. Missing or incomplete ESAS documentation was logged as absent. A permanent PCT was assigned to the clinic and acclimated to the clinic for 3 months. ESAS documentation was reassessed in February 2018 and the data from both time periods was divided into full and half-clinic days. The average lack of complete ESAS was compared before and after PCT presence, standard deviations calculated and data was compared with a 1-tailed paired T-test.

**Results**

In February 2017, 99 patients were seen, of which 72% on any given clinic day had a completed documented ESAS. On the busier full clinic days 70% of clinic notes had a complete ESAS compared to 75% on half clinic days. (Figure 1)

In February 2018, 113 patients were seen, of which 95% on any clinic day had a completed documented ESAS. On the busier full clinic days 92% of clinic notes had a complete ESAS compared to 96% on half clinic days. Comparing 2017 to 2018 data for all clinic days, there is a significant difference in ESAS documentation (Figure 1).

![Figure 1. ESAS Documentation Rates in Feb 2017 vs. Feb 2018](image)

**Discussion**

In this study we found that ESAS documentation increased after our intervention. This is suspected to be related PCT knowledge of clinic workflow, as the same PCT was assigned daily to clinic, rather than a rotation schedule with more than 10 rotating PCTs who inconsistently were or were not assigned to the clinic. While in this situation, it is possible that the selected PCT is simply more motivated to perform well, it is also well-known that workflow awareness is critical to work quality. (Clarke and Haque, 2008) Nurses have likewise identified that insufficient
staffing leads to self-identified poor quality care. (Aiken et al, 2002) It stands to follow that staff requiring repeated retraining by physician leadership in the clinic may be unable to complete the tasks to achieve quality care.

In both 2017 and 2018 there were lower rates of ESAS documentation in the full day clinics as compared to the half day clinics. This is suspected to be related to the higher pace of patient care and appointment volume, though this overall study did not aim to compare compliance with ESAS documentation on full versus half day clinics. This likewise correlates with known impact of staffing ratios on quality of care. (Aiken et al, 2002) This correlates with the observation during data collection that on full clinic days, an additional PCT might be pulled from a busy clinic to assist the permanent PCT. This additional PCT was often unaware of clinic procedures and on such days, ESAS documentation rates were lower due to unfamiliarity with procedures.

There were some limitations to this study, including increased physician awareness of ESAS documentation shortcomings in 2018, possibly resulting in increased vigilance in 2018. Regardless, the same physician ran the clinic in both years, suggesting increased vigilance would be unlikely to account for the 20% increase in documentation. Future areas for evaluation include assessing how improved ESAS documentation impacts other palliative care quality indicators and billing practice.

Conclusion
A dedicated PCT in the palliative care clinic is associated with significantly increased ESAS documentation. Further investigations are necessary to determine if this has impacted patient care or billing practices. Other areas for investigation include impact of a dedicated PCT on the quality measures in other Harris Health clinics, and methods to improve communication of clinic expectations to new or rotating staff.

References