University of Cincinnati Medical Center - Department of Emergency Medicine

Annals of B Pod

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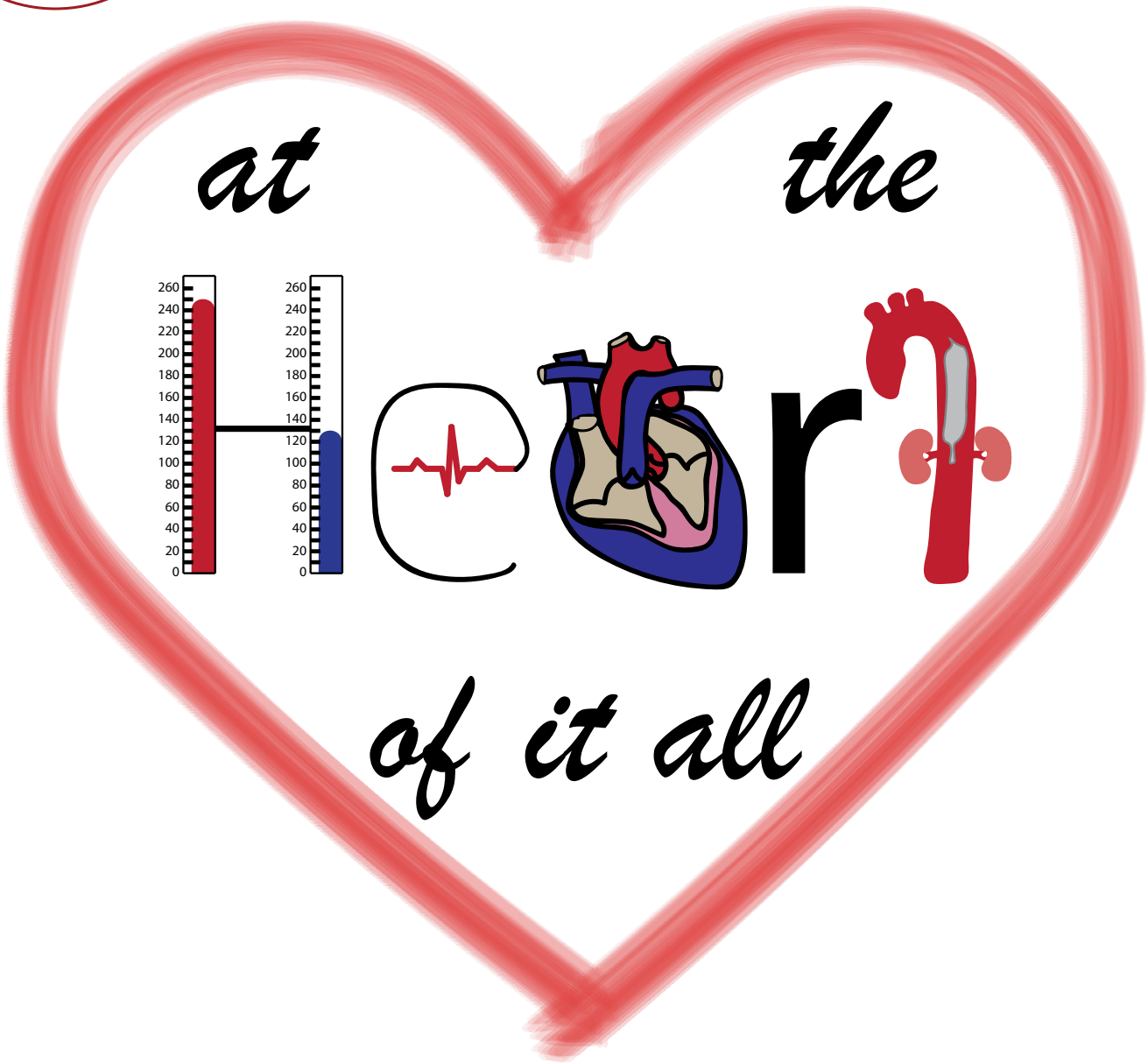

EMERGENCY MEDICINE
ANNALS OF B POD
WINTER 2016

In this Winter 2016 edition of Annals of B Pod, we focus on cardiovascular emergencies and their management in the Emergency Department and beyond.

In our new Air Care column, we write about the prehospital management of patients with profound cardiogenic shock requiring intra-aortic balloon pumps. In our recurring pharmacology column, we discuss the ED management of hypertensive emergency. Back in B Pod, Dr. Scanlon writes about a rare complication of renal failure in a patient with a large uremic pericardial effusion, and in our new procedure piece, we walk through the technical steps required to perform an emergent pericardiocentesis. On the back page, our EKG corner goes over the modified Sgarbossa criteria.

This issue of Annals of B Pod gets back to the heart of it all, highlighting what makes residency so rewarding: interesting pathology, challenging procedures and clinical excellence.

aircare

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Air Care

Intra-Aortic Balloon Pump

The intra-aortic balloon pump (IABP), first developed in the 1960s, is one of the most widely-used cardiac assist devices. Placed in critically-ill patients with cardiogenic shock, it increases coronary blood flow and decreases afterload. Patients with IABPs are frequently transferred to tertiary referral hospitals via helicopter emergency medical services (HEMS) transport. As such, prehospital and ED providers must become comfortable with the management of these patients and be aware of complications from these devices. Two recent Air Care patients highlight this need.

Case #1

The patient is a female in her 60s who presented to an outside hospital with chest pain and was found to have an anterior ST elevation MI (STEMI). She underwent stenting of the left anterior descending (LAD) coronary artery, but continued to have ongoing severe chest pain. Repeat coronary angiography revealed no evidence of in-stent thrombosis, but was notable for poor flow to additional coronary arteries. As such, an IABP was placed secondary to persistent unstable angina. On Air Care arrival, she was alert and hemodynamically stable with a heart rate in the 80s and a blood pressure 120s over 70s.

The patient was transitioned to Air Care's IABP portable console while still on the cath lab table. The IABP was set to trigger based on the electrocardiogram, 1:1 assist ratio and had good capture on the monitor. The patient's diastolic pressure was augmented into the 130s. She tolerated transport well and was taken to the Cardiovascular Intensive Care Unit without any hemodynamic instability. The IABP was removed within 24 hours, as she remained hemodynamically stable and reported improvement in chest pain.

Case #2

The patient is a male in his late 40s who presented to an outside hospital five days after an inferior STEMI. Of note, coronary angiography at that time revealed chronic total occlusion of the right coronary artery. However, revascularization was unsuccessful, and he was treated with medical management. Upon repeat presentation, the patient reported continuing shortness of breath and chest pain since being discharged. A bedside echocardiogram revealed severe mitral regurgitation and thrombus in the left ventricle.

The patient was taken back to the cath lab and found to have papillary muscle rupture. While in the cath lab, the patient suffered a PEA arrest with subsequent return of spontaneous circulation (ROSC). An IABP was then placed and Air Care was called for emergent transport to our hospital for operative repair of the valve. On Air Care arrival, vitals were notable for a blood pressure of 90s/50s with a heart rate in the 90s. The IABP was set to pressure trigger at 1:1 assist ratio. Exam was notable for a holosystolic murmur, thready distal pulses, diffuse crackles in all lung fields with pink, frothy ETT secretions and significant JVD.

As the patient was being loaded into the aircraft, he became bradycardic to the 30s. Push-dose epinephrine was administered with improvement in his heart rate. However, once in the aircraft, the patient went into ventricular tachycardia with thready pulses before quickly proceeding into asystole. CPR and ACLS were initiated during take-off and continued during the flight. Resuscitative efforts continued on arrival in the ED for an additional 25 minutes without ROSC. Cardiology and Cardiothoracic Surgery evaluated the patient at bedside in the ED and agreed with terminating efforts based on down time and prognosis.

Discussion

These cases illustrate two extremes of patients that prehospital HEMS and Emergency Medicine providers may encounter with IABPs. These assist devices are indicated for use in patients with acute cardiogenic shock, failure to wean off cardiopulmonary bypass after cardiothoracic surgery, refractory unstable angina, or ventricular arrhythmias. Although initially developed for use in the perioperative period for coronary artery bypass grafting (CABG), there is increasing use for those patients with acute cardiogenic shock. The pumps are contraindicated in those with aortic dissection or severe aortic regurgitation, and are cautiously used in those with severe peripheral vascular disease or coagulopathies.[1]

The purpose of the IABP is to increase coronary perfusion and myocardial oxygen supply while decreasing myocardial oxygen demand by several mechanisms. A balloon sits in the proximal descending aorta, approximately 1 cm distal to the left subclavian artery, and is triggered to inflate and deflate at different phases of the cardiac cycle (Figure 1).

The balloon is triggered to inflate in early diastole, which causes an increase in the early diastolic blood pressure (DBP), subsequently increasing coronary perfusion. Deflation occurs in early systole, just before the aortic valve opens. This creates a vacuum effect, reducing cardiac afterload by decreasing aortic end-DBP and decreasing myocardial demand. These physiologic alterations serve to benefit both those in acute cardiogenic shock and those with acute myocardial infarction.

The IABP is composed of a catheter with a distal cylindrical balloon sized by patient height. This is connected to a console with a helium tank used to inflate the balloon. Helium is used as it is an inert gas which is easily absorbed into blood in the rare event of balloon rupture. The console is also connected to ECG leads and an arterial pressure line, which are used to trigger the inflation of the balloon at the appropriate time within the cardiac cycle and monitor hemodynamic response. Newer models augment the traditional arterial line with a fiberoptic cable built into the balloon catheter itself, allowing for auto-calibration of timing. Placing the balloon pump in the ECG trigger mode, which is based off the R-wave, marking the beginning of systole, will work for most patients. Newer models automatically calculate the timing of the cardiac cycle and will then inflate at the appropriate time, which is mid T-wave or early diastole.

For all models, there are other trigger modes, including pressure trigger and pacer trigger. Pressure mode may be used for backup when ECG leads are not reading, there is significant artifact or the patient is in arrest. The trigger in this case is the systolic upstroke of the arterial waveform. Pacer mode is used for patients with 100% AV/V pacing where there are no reliable R-waves to trigger the ECG mode. ECG mode will work, however, for the majority of paced patients that have good capture. Regardless of the trigger mode, the arterial waveform is used to determine if the timing with the cardiac cycle is accurate.

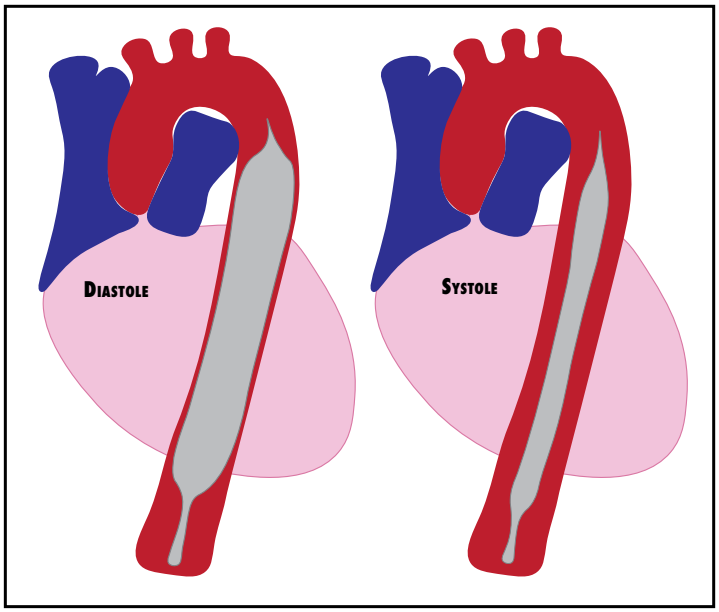

Figure 1. This cartoon shows the balloon of the IABP sitting in the descending aorta. The image on the left shows the balloon inflated during diastole. The image on the right shows the balloon deflated in systole.

In addition to the trigger mode, the operator must also set the assist ratio. Assist ratio is the number of assisted cycles to the number of intrinsic beats. The most common is 1:1 and is efficient up to 120 beats per minute. The 1:2 and 1:3 ratios are most often used for weaning from the IABP in the postoperative period or in extremely tachycardic patients.

Figure 2, on page 12, illustrates the optimized arterial pressure waveform with IABP augmentation.[2] The goal is to have appropriate timing of inflation and deflation with the cardiac cycle in order to most effectively improve hemodynamics.

There are several free resources available with scenarios of poorly timed waveforms and their solutions that may be helpful.[3,6] The newer models, including the Maquet Cardiosave Hybrid™ that is carried on Air Care, are equipped with an "auto-mode" that automatically fills the balloon, performs calibration using fiber-optic cable, selects the most appropriate lead and trigger, and sets the appropriate inflation timing.

While Emergency Medicine physicians must have an understanding of which patients are candidates for urgent IABP placement and how these devices function, it is also essential for those involved in prehospital care and those at quaternary referral centers to know the basics of managing a patient who already has one of these devices in place. The largest study to date of IABP complications found that the most common complications included limb ischemia (2.9%), bleeding from the access site or from aorto-iliac dissection (2.4%), balloon rupture (1.0%) and death attributable to balloon pump (0.05%).[5]

Prior to transport, the HEMS provider should always perform a neurovascular exam and check the access site for hematoma or active bleeding. Any report of sudden change in the quality or degree of pain should raise concern for dissection and the vascular exam should be repeated immediately. If the provider notices blood in the helium line, this is representative of a balloon rupture, which can be life-threatening due to exsanguination through the line. Clamping the line with a Kelly clamp or hemostat can temporize this complication.

Another issue that arises in the emergent care of these patients is represented by the second case above. There are a few pertinent points to be aware of if the patient that experiences cardiac arrest. First, CPR

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Uremic Pericardial Effusion

Taking renal failure to heart

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History of Present Illness

The patient is a male in his late 30s with a past medical history significant for trisomy 21, stage III chronic kidney disease, and hypertension who presents to the Emergency Department with emesis and dark stools. The patient is unable to contribute significantly to his history, but his family relates that two days prior to presentation, the patient experienced two episodes of "coffee ground" emesis according to the patient's home health nurse. Over the next day, the patient subsequently experienced several episodes of melenic stools. His family also notes that he has seemed feverish, more lethargic, and less active than his baseline.

Past Medical History

Trisomy 21
Chronic kidney disease
Gout
Hypertension
Hyperlipidemia

Surgical History

Ventricular septal defect repair
Orchiopexy

Medications

Allopurinol
ASA
Atorvastatin
Bicitra

Vitals

T 37.2 HR 125 BP 183/108 RR 14 SpO2 97% on RA

Physical Exam

Cardiovascular examination is notable for tachycardia with a regular heart rhythm. A grade III/VI systolic murmur is best appreciated over the left parasternal border without audible gallops, murmurs or rubs. His abdominal exam is normal without organomegaly, focal tenderness, or guarding. Digital rectal examination was performed, which revealed guaiac-positive, black stool in the rectal vault. He has evidence of bilateral lower extremity pitting edema. The patient is lethargic but oriented to person, place, time, and purpose with no focal neurological deficits. His speech is clear without aphasia or dysarthria, and his skin is warm and dry without diaphoresis.

Labs

WBC: 10.7 Hgb: 4.9
Na+: 131 K+: 5.7 BUN: 153 Creatinine: 24.63
Fecal occult: Guaiac +

Imaging

Chest x-ray: Cardiomegaly with patchy consolidation in the right lower lobe concerning for pneumonia.

Cardiac ultrasound: Evidence of moderate to large pericardial effusion (Figure 1)

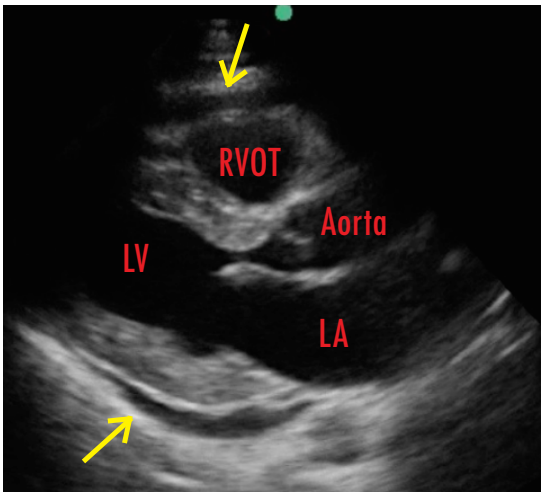

Figure 1: Representative echo image showing a pericardial effusion in the parasternal long axis. The yellow arrows highlight the pericardial effusion. Also note the left ventricle (LV), left atrium (LA), right ventricular outflow tract (RVOT) and aorta labeled in red.

Hospital Course

This patient presented with coffee-ground emesis, melenic stools, and a low hemoglobin concerning for a significant upper gastrointestinal hemorrhage. His diagnostic evaluation also showed acute on chronic renal failure with significant uremia, as well as a new pericardial effusion. Given the severity of his GI bleed, the patient was transfused with four units of packed red blood cells in the Emergency Department prior to transfer to the Medical Intensive Care Unit for further management.

Upon admission, the patient's family was informed that the patient would likely need hemodialysis given the severity of his renal failure. Though noted to have a pericardial effusion, the patient remained hemodynamically stable with no evidence of tamponade. As such, pericardiocentesis was not deemed a priority given the patient's more critical comorbidities. The admitting team also recommended that the patient undergo esophagogastroduodenoscopy (EGD) to evaluate for the source of his hemorrhage.

In discussing the potential complications of EGD, the likely need for intubation, and the patient's likely need for lifelong hemodialysis, his family ultimately requested that patient be discharged home on hospice. The patient passed away at home roughly two weeks after discharge from the hospital.

Discussion

A pericardial effusion is an abnormal collection of fluid within the pericardial sac. The pericardial sac is a potential space formed between the visceral and parietal layers of the fibrous covering of the heart. This potential space normally contains a small volume (~50 mL) of serous fluid similar in composition to plasma ultrafiltrate that serves as a lubricant, allowing the two layers to slide over one another during the movements of the heart.[1] The volume of this pericardial fluid is regulated by various lymphatic systems that drain the pericardium.

The onset of a pericardial effusion can be categorized as acute (≤1 week), subacute (>1 week and ≤3 months), and chronic (>3 months).[2] Similarly, the size of an effusion may be categorized semiquantitatively using ultrasonography by measuring the diameter between the visceral and parietal pericardial tissue in diastole (smallest size during cardiac cycle while in M Mode).

Various perturbations in normal physiology or exogenous insults (e.g., infections) may lead to inflammation of the pericardial tissue (pericarditis) or a fluid collection in this potential space that exceeds the lymphatic drainage. Rapid accumulation of fluid may lead to tamponade physiology, wherein the collection of material within the inflexible pericardium may lead to a build up of pressure that exceeds the resilience of the ventricular walls. This can limit diastolic filling and thus decrease stroke volume and cardiac output, with consequential hemodynamic instability. Fluid that accumulates more insidiously, however, may lead to a gradual dilation of the pericardial sac. Volumes exceeding 2L of fluid may accumulate via this gradual dilatation prior to the onset of tamponade physiology. [2,3]

Detailing the chronicity of pericardial effusions is frequently complicated by their often subtle presentations. Perhaps more meaningful, then, is to determine evidence of hemodynamic instability or compromise secondary to an effusion. Common physical exam findings in patients with significant effusion or tamponade physiology include dyspnea, tachycardia, and hypotension. Patients may present with jugular venous distention or pulsus paradoxus (a drop in systolic blood pressure >10 mmHg during inspiration). The presence of elevated jugular pressure, hypotension and muffled heart sounds is known as Beck's triad and is pathognomonic for cardiac tamponade.

Electrocardiography is often a useful adjunct in the diagnosis of pericardial effusion. Widespread, upward-concave ST segment elevations may be indicative of significant peridcardial inflammation.[4] Diffuse low-voltage lead tracings may be suggestive of a large pericardial effusion, whereas electrical alternans is often cited as pathognomonic for a large pericardial effusion or tamponade. Literature indicates that the sensitivity of these findings, however, is quite poor.[5]

Echocardiography may also be used in the diagnosis of tamponade physiology. Findings suggestive of tamponade physiology include collapse of any of the cardiac chambers (most often the right atrium and ventricle, due to their comparatively thinner, less muscular walls) and inferior vena cava plethora (>20mm).[2,6] The most specific marker for tamponade is diastolic collaspe of the right atrium.[2]

Diagnosing Cardiac Tamponade

- Collapse of right atrium and ventricle on echocardiogram
- Inferior vena cava plethora (>20 mm) on echocardiogram

- Pulsus paradoxus
- Jugular vein distention (JVD)
- Muffled heart sounds
- Hypotension

Table 1: Key physical exam or imaging findings suggestive of cardiac tamponade from a pericardial effusion.

As mentioned previously, pericardial effusions may stem from a number of etiologies including numerous infectious agents (tubercular, bacterial, viral, fungal, parasitic), autoimmune processes, postinfarction inflammation, malignant effusions, traumatic (pneumo- or hemopericardium), and metabolic derangements (including uremia).[2] The incidence of these diseases varies dramatically on the country and demographics examined.

Uremic pericarditis and pericardial effusion, as seen in this patient, are well-known complications of azotemia, though the exact pathophysiologic mechanism is poorly understood. Uremia refers to an abnormally high level of urea (BUN >60 mg/dL) and other nitrogenous waste products retained in the blood. The kidneys play an essential role in the elimination of nitrogenous waste from the blood and, as a result, uremia invariably stems from renal dysfunction.

There are numerous nephropathies that may lead to progressive renal dysfunction and uremia. Interestingly, the incidence of pericarditis and pericardial effusion correlates with the severity of a patient's uremia/azotemia and not the etiology of patient's kidney disease.[7] It has been speculated that platelet dysfunction and consequential coagulopathy in uremic patients may in part contribute to pericardial effusions, though the pathogenesis is again poorly understood.[8]

Pericardial effusion is a clinical diagnosis, though confirmation studies with echocardiography have become the standard of care. Patients presenting with symptoms concerning for pericardial effusion may be evaluated with a complete blood count, basic metabolic panel, coagulation studies, thyroid function, electrocardiography, and plain films of the chest.[4] Erythrocyte sedimentation rates and C-reactive protein may be elevated in the event of inflammatory processes.

Serum troponin levels may be elevated in patients with significant pericardial inflammation, large effusions, traumatic injuries or tamponade physiology leading to myocardial injury.[9] Furthermore, rheumatologic testing may be indicated in young female patients due to the higher incidence of autoimmune disease. Fluid samples collected from the pericardium may be sent for Gram staining, bacterial/fungal cultures, acid-fast staining and/or mycobacteria-specific cultures, tailored to patient's clinic presentation and regionally-endemic pathogens.

Emergent pericardiocentesis performed

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Quick Hit: Achilles Tendon Rupture

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History of Present Illness

The patient is a 30-year-old female with no past medical history who presents to the Emergency Department shortly after stepping through a picture frame that was propped up on her floor at home. The glass shattered, lacerating the posterior aspect of her left lower leg. She is complaining of difficulty walking, but denies loss of sensation distal to the injury. Her tetanus is up to date.

Physical Exam

The patient is an alert, well-developed female who is hemodynamically stable and in no acute distress. Her musculoskeletal exam is significant for a linear transverse laceration superior to the left calcaneus. She has intact dorsalis pedis and posterior tibialis pulses, and her sensation is intact through the sural, saphenous, tibial, and deep and superficial peroneal distributions. She has decreased plantar flexion of the left foot with a positive Thompson test.

Imaging

Plain film radiographs of the left heel showing a large soft tissue defect without evidence of osseous abnormalities or foreign bodies.

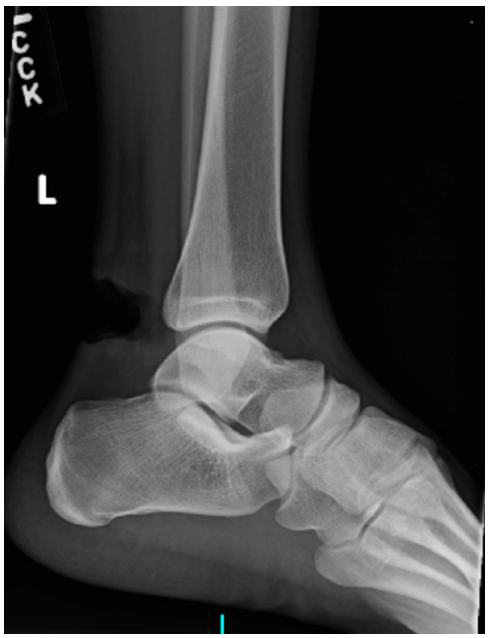

Figure 1: X-ray imaging showing a large soft tissue defect just superior to the left heel.

Hospital Course

The patient presented with an Achilles tendon laceration. She was started on prophylactic antibiotics with IV cefazolin. Her pain was managed with hydromorphone and the orthopedic surgery team was consulted. After bedside washout, skin closure, and application of a plantar flexion splint, she was admitted to the orthopedic service for operative repair.

In the operating suite, the wound was explored and found to have spared her medial neurovascular bundle as well as the peroneal tendon sheath. The site was irrigated with sterile saline and bacitracin and the ends of the tendon were reapproximated and sutured together. The wound was then closed, and a splint was placed to hold the foot in maximal plantar flexion (equinus splint).

The patient had an uneventful postoperative hospital course. She was discharged home the following day on non-weight-bearing status with the equinus splint still in place. At her one month follow up visit she was transitioned to weight-bearing status with a boot and is reportedly doing well with physical therapy to this day.

Discussion

Achilles tendon rupture occurs with a reported yearly incidence of 18 per 100,000,[1] and is most common in men aged 30-40. Specific risk factors include episodic physical exertion (*e.g.*, "weekend warriors"), steroid injections, and fluoroquinolone antibiotics. With regards to antibiotics, the FDA recently updated its black-box warning for fluoroquinolones to clarify that they carry a small but known risk for tendinitis and tendon rupture, and should only be used in patients who have no alternative treatment options for acute bronchitis, sinusitis, and cystitis.[2]

The Achilles tendon attaches to the muscles in the posterior leg responsible for plantar flexion of the foot at the ankle (*i.e.*, gastrocnemius, soleus, and plantaris). Unlike in this patient, rupture usually occurs from blunt trauma involving forced or sudden dorsiflexion of a plantarflexed foot, often during sports. Open injuries are more rare than closed ruptures, but have been reportedly more common in cultures where floor-level toilets pose a unique risk factor.[3]

The Thompson test is a critical physical exam maneuver that should be performed whenever there is suspicion for Achilles tendon injury. To perform the test, the practitioner should

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Hypertensive Emergency

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Edited by: Madeline Foertsch, PharmD, BCPS and Jessie Winter, PharmD, BCPS

Hypertension is one of the most common disease states in the United States, with more than 75 million Americans afflicted. Of these patients, approximately 1% will develop an acute hypertensive emergency each year.[1] Hypertensive emergency is defined as severely elevated blood pressure (typically above 180/120 mmHg) with associated end-organ damage.[2,3] Symptoms of end organ damage include neurologic symptoms (*e.g.*, agitation, confusion, seizures, coma), nausea or vomiting, chest pain, or shortness of breath. Diagnostic tests concerning for end-organ damage in the setting of severe hypertension include EKG changes, elevated creatinine, proteinuria, elevated cardiac enzymes, pulmonary edema on chest x-ray, and evidence of neurologic compromise on CT or MRI. Initial management of patients with hypertensive emergency in the Emergency Department includes lowering blood pressure in a controlled manner with intravenous agents. Table 1 (page 13) shows the characteristics of recommended intravenous anti-hypertensive agents for treatment of hypertensive emergency. However, blood pressure goals and agents of choice depend significantly on the patient's presentation and underlying comorbidities. Below are four different clinical scenarios that discuss hypertensive emergencies and their specific management.

Hypertensive Emergency in Baseline Hypertension

According to Joint National Commission 7 (JNC 7) guidelines, mean arterial blood pressure should be reduced by no more than 25% within the first hour. If the patient remains stable, practitioners should target a goal BP of 160/100 mmHg within the next 2-6 hours.[4] Instances when blood pressure should not be lowered to normal ranges in hypertensive emergency include patients with baseline long-standing hypertension.

Patients with long-standing hypertension have adapted to chronically high blood pressure and developed compensatory mechanisms to perfuse vital organs at a higher baseline blood pressure. Therefore, end-organ damage may not develop until blood pressure is above 200/150 mmHg, or even higher. Lowering these patients' blood pressure to normal ranges can cause relative ischemia. It is also important to note that most patients in hypertensive emergency are volume depleted. Therefore, repletion with crystalloids can help with organ perfusion and prevent relative hypotension and underperfusion that can occur with antihypertensive treatment.

Hypertensive Emergency in Ischemic Stroke

In patients with ischemic strokes, perfusion to the brain is disrupted. It is thought that due to impaired cerebral autoperfusion blood flow to the brain is dependent upon mean arterial blood pressure. Although many patients presenting with acute ischemic strokes have poorly controlled hypertension at baseline, they are allowed to be more hypertensive in order to perfuse the ischemic penumbra. Studies have shown an increase in long-term morbidity and mortality in patients with ischemic strokes who have had aggressive blood pressure control. As such, guidelines recommend targeting a blood pressure of 180/110 mmHg in patients who are treated with intravenous thrombolytics, and 220/120 mmHg in patients who are not.

Hypertensive Emergency in Aortic Dissection

One of the most concerning and potentially fatal complications of a hypertensive emergency is acute aortic dissection, with approximately 2,000 cases per year in the United States. If left untreated, two-week survival is as low as 25%. However, if properly treated, 5-year survival is up to 75%, depending on the location of the defect.[2,6]

When treating hypertensive emergency with a likely aortic dissection, goals of therapy focus on lowering blood pressure as well as reducing heart rate, which can decrease the shear forces on the aorta. For these patients, the 2014 European Society of Cardiology guidelines recommend a goal systolic blood pressure of 100-110 mmHg and a goal heart rate of 60 beats per minute. These blood pressure and heart rate parameters should be obtained as long as the patient maintains end organ perfusion.[7] Selective beta-1 blockers, such as esmolol, primarily decrease heart rate and have little effect on blood pressure; if utilized for the treatment of aortic dissection, additional anti-hypertensive agents, such as nicardipine, may need to be added in order to achieve blood pressure goals.

Hypertensive Emergency in Acute Hemorrhagic Stroke

Patients with hemorrhagic strokes often have increased blood pressure as a result of pain, stress, and increased intracranial pressure. However, unlike in acute ischemic stroke, studies have consistently found that decreasing blood pressure in these patients is associated with better outcomes. Higher systemic pressures have been associated with hematoma expansion and increased risk of death and neurologic disability. However, there is no consensus as to the optimal blood pressure goals for these patients.

The 2015 AHA guidelines recommend lowering systolic pressures to 140 mmHg.[11] More recently, the ATACH-2 trial found that intensive blood pressure

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bpodcase

Hyperleukocytosis

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History of Present Illness

The patient is a male in his mid 20s presenting with several weeks of progressively worsening bilateral lower extremity edema with new shortness of breath for the past 10 days. The swelling began spontaneously and has continued to increase in girth and climb higher up the leg, now to the level of his groin. He also notes painless abdominal distention that preceded the swelling in his legs. His shortness of breath started as mild dyspnea on exertion without chest pain, but has worsened in past few days such that he is now short of breath at rest.

He denies any associated or preceding chest pain, nausea or vomiting, fevers, weakness, or paraesthesias in the lower extremities. He denies cough, sputum production, hemoptysis, and pulmonary embolism or tuberculosis risk factors. He has no history of abdominal surgery and has no other pertinent medical history. He continues to pass stool and flatus. He denies any history of intravenous drug use or frequent alcohol use. On review of systems, he does endorse approximately fifteen pounds of unintentional weight loss over a similar time course as his other symptoms.

Past Medical History

None

Medications

None

Vitals

T 36.1 HR 117 BP 140/84 RR 20 SpO2 95% on 2L NC

Physical Exam

The patient is a thin male in mild respiratory distress. He has moist mucous membranes and anicteric sclera. No lymphadenopathy is appreciated on the neck. There is jugular venous distention to the angle of the mandible. Auscultation of the heart and lungs is unremarkable. His abdomen is firm and non-tender with no fluid wave. His spleen is palpable approximately five finger widths below the costal margin. There is bilateral +2 pitting edema in the lower extremities to the level of the inguinal crease. His distal pulses are intact and there are no rashes observed on his skin.

Labs and Imaging

Na 129 K 6.0 Cr 0.78 Ca 8.3 Mg 2.3 Phos 2.9
Alk Phos 314 Albumin 3.1
Uric Acid 8.8
WBC 433.1 Hemoglobin 7.6 Platelets 329

Chest x-ray: Volume overload consistent with pulmonary edema

Hospital Course

The patient presented in mild respiratory distress with a new oxygen requirement, and was found to have both pericardial and pleural effusions. His lab work was most notable for hyperleukocytosis with a white blood count (WBC) of 433K, concerning for a new diagnosis of leukemia. In the ED, he was given 3L of IV fluid. He was admitted to the oncology service and started on hydroxyurea for hyperleukocytosis and allopurinol for tumor lysis syndrome, which were continued for the course of his admission. The patient received one round of leukapheresis, which decreased his WBC and resolved his respiratory distress. Flow cytometry revealed chronic myelogenous leukemia (CML) with a positive Bcr-Abl mutation. On hospital day seven, he was started on dasatinib, an oral Bcr-Abl tyrosine kinase inhibitor.

At the time of discharge, his WBC had decreased to 66K and his respiratory status and edema were much improved. He was continued on dasatinib and has been followed up in outpatient oncology clinic with ongoing improvement. His most recent WBC was 10K.

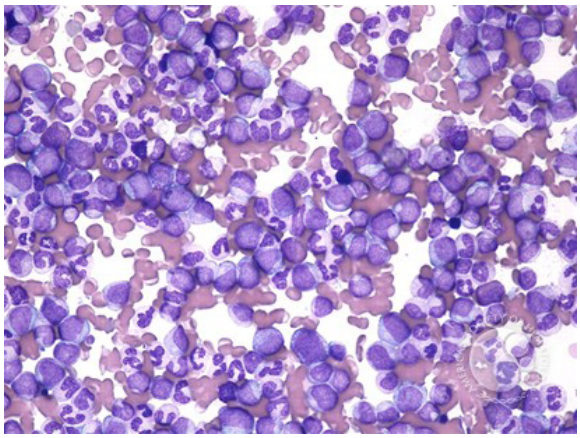

Image 1: Representative histology slide showing hyperleukocytosis in a patient with CML. Image from hematology.org.

Discussion

This patient presented with respiratory distress and hyperleukocytosis, concerning for new diagnosis of leukemia. Hyperleukocytosis is defined as a WBC count greater than 100,000/mm^2. While there are reported cases of hyperleukocytosis from leukemoid reactions with infectious etiologies, hyperleukocytosis is almost always a signal of hematological malignancy. [1,2] The Emergency Physician should be aware of the complications and management of hyperleukocytosis, including leukostasis, tumor lysis syndrome, and disseminated intervascular coagulation.

Leukostasis

One of the most serious emergent presentations associated with hyperleukocytosis is leukostasis. At critically elevated levels, WBCs can impede blood flow and cause local tissue damage that presents with variable symptoms depending on the affected tissue. The cause of leukostasis is likely multifactorial, but is hypothesized to involve physical obstruction of microvasculature by increased blood viscosity and an abundance of larger, less deformable blast cells. Leukostasis is most commonly seen in acute myeloid leukemia (AML) and CML blast crises, as the myelogenous blast cells are larger and more rigid than their mature counterparts, and thus more likely to cause obstruction.[9]

In addition to physical obstruction, leukemic cells have increased oxygen metabolism and elevated cytokine production, which may lead to local ischemia, endovascular damage, hemorrhage and infiltration of leukemic cells into local tissues.[3,4] Occlusion of pulmonary vasculature can lead to dyspnea, hypoxemia, respiratory distress or failure and acute respiratory distress syndrome (ARDS).[5]

This patient presented with mild respiratory distress and plain film imaging suggestive of volume overload. While leukostasis is known to present with respiratory symptoms, leukemic patients are also immunocompromised by their disease and at increased risk of pulmonary infections. Differentiating infection from leukostasis in the ED may be difficult, as plain film imaging and CT can appear similar to volume overload and local infiltration.[4,5,13] The early use of broad-spectrum antibiotics may benefit patients with infection, and can be tapered or removed once infection is ruled out. Other diagnostic considerations include pulmonary embolism, transfusion-related acute lung injury and chemotherapy toxicity.

For patients with more severe respiratory distress or ARDS, intubation and mechanical ventilation may be required. Management of leukostatic respiratory distress may differ slightly from normal supportive protocols in that diuretic use is typically discouraged, as it will further increase blood viscosity and may paradoxically worsen respiratory symptoms.[9] If blood gases are being used to manage therapy, it is important to note that leukocytes continue to metabolize oxygen in the blood after a sample is drawn, an effect which is exaggerated in hyperleukocytosis. This can lead to falsely low measures of oxygen concentration, particularly if samples are not analyzed in an expedient manor.[17,23]

In the central nervous system (CNS), leukostasis frequently presents with headache, vision changes, altered mental status, and stroke-like symptoms. Patients with leukostasis may also have an ischemic pattern on imaging. Case reports of acute stroke symptoms in the setting of hyperleukocytosis are rare, and no clear consensus on proper treatment is available.[20] Patients with hyperleukocytosis frequently present with severe thrombocytopenia, which is an absolute contraindication for thrombolytic therapy.[21] Presumed microvascular hemorrhage from leukostasis and high rates of DIC, as discussed below, likely further increase the risk for complications. There is currently no safety data available for the use of thrombolytics in patients with hyperleukocytosis. However, case reports have shown neurological improvement in patients undergoing cytoreductive therapy.[22] While CNS and respiratory symptoms are most common, leukostasis can also present with cardiac symptoms, including acute myocardial infarction, abdominal pain, gastrointestinal ischemia or hemorrhage, and rarely with priapism, renal vein thrombosis and limb ischemia.[6,7]

System-based Effects of Leukostasis

Pulmonary
- Dyspnea
- Hypoxemia
- Respiratory Distress
- ARDS

Central Nervous System
- Headache
- Vision Changes
- Altered Mental Status
- Stroke-like Symptoms

Other
- Acute myocardial infarction
- Immunocompromised state
- Abdominal Pain
- Gastrointestinal Ischemia
- Gastrointestinal Hemorrhage
- Priapism
- Limb Ischemia
- Renal vein thrombosis

Table 1: System-based effects of leukostasis

In general, management of leukostasis revolves around decreasing the viscosity of circulating blood. Most immediately, aggressive intravenous fluids (IVF) are used to dilute the viscosity due to elevated leukocrit. Fluids are also an essential part of treating and preventing another major complication, tumor lysis syndrome, as discussed below. There are no published guidelines for management of hyperleukocytosis, and aside from early induction of chemotherapy, most treatment modalities are controversial. Cranial radiation has been used for CNS symptoms, but was not shown to improve outcomes and is no longer recommended.[10,11] Dexamethasone has been hypothesized to decrease the adhesive properties of leukemic blasts, but evidence to support this is lacking.[10-13] Direct reduction of leukocytes with either leukapheresis or exchange transfusion is frequently practiced.

Our patient experienced resolution of his dyspnea immediately following leukapheresis. Similar short-term clinical improvement from leukapheresis has been documented in many case reports.[14-16] Larger retrospective studies have also shown decreases in early death for patients receiving leukapheresis; however, this has not been shown to translate into improved long-term survival, and has even been loosely correlated with higher long-term mortality.[13,18-19]

Induction chemotherapy is the definitive therapy for hyperleukocytosis and leukostasis. Hydroxyurea, which was started on admission for this patient, is frequently used in conjunction with or in advance of definitive chemotherapy, particularly if any delays in chemotherapy are anticipated, as it directly decreases the WBC count. All patients will require admission and consultation with oncology specialists for definitive treatment.

Tumor Lysis Syndrome

Tumor lysis syndrome (TLS) is a life-threatening complication in which the intra-cellular contents of dying neoplastic cells are released into the system faster than they can be metabolized. Patients most at risk are those with fast growing cancers (which would include all blood malignancies), a large tumor burden, and with cancers that are particularly

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Procedure Piece: Pericardiocentesis

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Introduction

As fluid accumulates in the pericardial space, it exerts pressure on the heart, ultimately resulting in equalization of diastolic filling pressures as it continues to progress. This can impair ventricular filling and may eventually result in decreased cardiac output leading to pulseless electrical activity and death. Pericardiocentesis can be a life-saving procedure in patients with such presentations.

In the emergency department setting, pericardiocentesis is indicated in hemodynamically unstable patients with evidence of tamponade[1]. This diagnosis should be suspected in those with an effusion on bedside ultrasound or clinically with Beck's triad (hypotension, distended neck veins, and distant heart sounds) or pulsus paradoxus (inspiratory decrease in systolic blood pressure). Classically, this procedure is relatively contraindicated in traumatic pericardial effusions, myocardial rupture, serious bleeding disorders, and aortic dissection due to concerns for rapid reaccumulation of the effusion. However, in hemodynamically unstable patients, there are no absolute contraindications to performing this procedure, as it may be the only available treatment for life-threatening pericardial effusion. Nonetheless, it should be considered a temporizing measure, and definitive treatment for the underlying etiology of the effusion should be pursued.

Supplies

- Sterile gloves
- Betadine (or other similar cleaning solution)
- 18 gauge spinal needle
- 20 cc syringe
- 3-way stop-cock
- Ultrasound (if available)

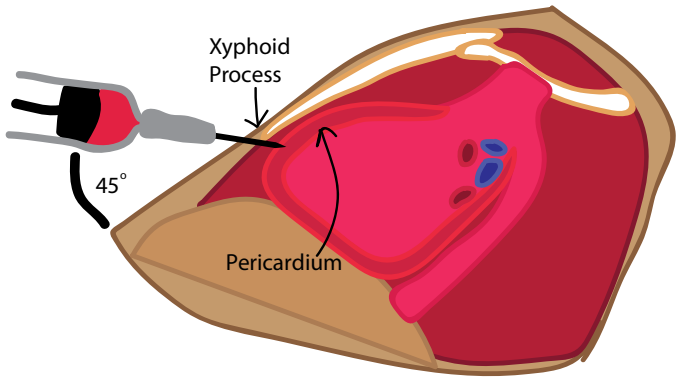

Figure 1: A representative illustration of a sagittal cut through the chest of a patient with a pericardial effusion, with a spinal needle angled at 45 degrees inserted under the xiphoid process.

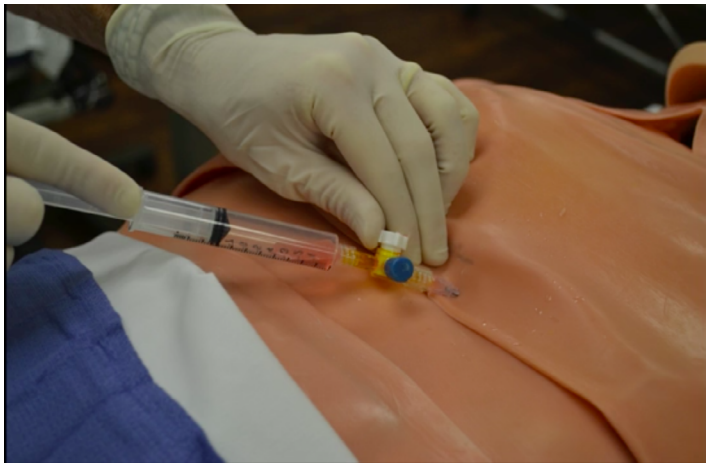

Figure 2: A video of the setup of this procedure and a step-by-step walk through of performance of the procedure can be found at www.tamingthesru.com/blog/acmc/pericardiocentesis or by accessing the online version of this article at www.tamingthesru.com/annals-of-b-pod/

Procedure

- Cleanse a wide area of skin just above the xiphoid process using Betadine or other cleansing solutions.[2]
- Palpate the xiphoid process to determine landmarks. Visualize the effusion if using ultrasound.
- Insert the spinal needle with the stylet in place just inferior to the xiphoid process. Aim superiorly and angled toward the left shoulder.
- Once the skin has been punctured, remove the stylet and attach the stop-cock and 20 cc syringe to the spinal needle.
- Advance the needle at a 45 degree angle while aspirating-toward the largest fluid collection if using ultrasound.
- If available, IV tubing may be attached to the third port on the 3-way stop-cock in order to allow quick access for additional fluid drainage if necessary.
- Withdraw fluid until hemodynamic stability is obtained, and then turn the stop-cock toward the needle to stop the flow of fluid.
- At this point, the syringe may be removed. Keep the needle and stop-cock assembly in place so that additional fluid may be drained if the patient again becomes hemodynamically unstable.
- After the procedure, obtain an ultrasound to evaluate for improvement in cardiac function in addition to a chest x-ray to evaluate for pneumothorax.

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Pericardiocentesis
Continued from page 10

While pericardiocentesis can be a life-saving procedure, it is not without potentially dangerous complications, which occur in up to 10% of all cases of pericardiocentesis.[3] There is a 20% rate of life-threatening complications if this procedure is performed without ultrasound guidance, but that number is reduced to 3% with the use of ultrasound, making its use the standard of care. Cardiac puncture occurs in 1% of cases and may be subtle, resulting in a persistent effusion and potential redevelopment of tamponade. Management frequently necessitates involvement of cardiac surgery for repair.

Misdirection of the needle through the pleural space can result in pneumothorax or pneumopericardium, both of which may be detected on the post-procedure chest x-ray. Chest tube placement may be indicated in the case of pneumothorax, while close observation is often sufficient for pneumopericardium because it often spontaneously resolves.[3]

Pericardial decompression syndrome is a term used to describe the development of pulmonary edema after pericardiocentesis, a phenomenon which is seen in patients with both acute and chronic pericardial tamponade. It is thought to be due to a greater increase in the right ventricular end-diastolic volume when compared to the left ventricular end-diastolic volume after the procedure.[3] One study showed that after pericardiocentesis, right ventricular stoke volume increased an average of 76%, while left ventricular stroke volume increased by only 64%. This is thought to result in a disproportionate increase in pulmonary blood flow resulting in pulmonary edema. The management of this syndrome is supportive.

Finally, iatrogenic injury to surrounding structures such as the liver, diaphragm, and stomach are not uncommon, highlighting the importance of ultrasound guidance. Thus, Emergency Department pericardiocentesis is reserved for hemodynamically unstable patients for whom surgical treatment is not readily available. Even so, it can provide life-saving time to transport such patients to definitive care, making it an important skill for the emergency physician.

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Pericardial Effusion
Continued from page 5

in the Emergency Department under ultrasound guidance is indicated in patients with evidence of severe hemodynamic instability (cardiac tamponade).[6] Approaches to pericardiocentesis are varied; the most common approaches include parasternal or subxiphoid needle insertion with transthoracic echocardiographic guidance. Ultrasonography should be used in all emergent cases, as blind pericardiocenteses are associated with a high rate of complications (>20%), including pneumo- or hemothorax, laceration of the coronary arteries, puncture or rupture of the ventricular walls, and injury to the thoracic vasculature (e.g., internal mammary and intercostal arteries).[6,10] Large but hemodynamically stable effusions may be drained nonemergently under fluoroscopic guidance or by cardiothoracic surgery. In both instances, fluid analysis may be performed to determine the etiology of the effusion.

Small or moderate sized effusions without tamponade physiology may be managed pharmacologically. Good evidence exists for the use of anti-inflammatory agents in both treating and preventing recurrence of effusion.[2] Various drug classes, including NSAIDs (specifically ibuprofen and aspirin), corticosteroids (prednisone), and atypical agents (such as colchicine), have relatively robust evidence for their use. Generally, first line treatment involves the scheduled use of nonsteroidal anti-inflammatory drugs with subsequent assessment to verify remission or resolution of the effusion.[2] In addition, patients should receive appropriate treatment for the underlying pathophysiologic process, e.g., active infection, malignancy, or rheumatologic disease.

Many of the above interventions — specifically NSAIDs and corticosteroids — have demonstrated somewhat mixed results in patients with uremic pericardial disease.[11,12] Current evidence supports the use of dialysis (to remove nitrogenous waste) and colchicine in the management of pericardial effusions in patients with renal disease.[13,14] Unfortunately, recurrence is common in patients with pericardial effusion, particularly in patients with uremia. Recurrent disease may require more aggressive management, including chronic immunomodulation via prolonged steroid or colchicine use, creation of a pericardial window, and pericardiectomy.[14]

Pericardial effusions are multifactorial in etiology and can have varying clinical presentations. Emergency Physicians should have a low threshold to evaluate for recurrent disease in patients with a history of pericardial effusion presenting with concerning symptoms, or in any patient with risk factors and concerning symptoms or pertinent physical exam findings.

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Intra-aortic Balloon Pump
Continued from page 3

should be performed as usual in these patients without fear of damaging the balloon. Secondly, when set to "auto-mode," the newer machines will automatically switch between ECG and pressure trigger mode depending on the signal quality. This prevents the machine from sensing R-waves from artifact and impeding systemic and cardiac perfusion when compressions are being done. The pressure mode will sense the increased pressure in the aorta from external compressions and time the inflation cycle to match and improve coronary perfusion.

Additionally, defibrillation may be used if the patient is in a shockable rhythm. The machine is grounded, but everyone should stand clear of the machine to prevent being shocked if it is not plugged into a wall outlet. Finally, the pump should not be placed in "standby mode" for greater than 30 minutes due to the risk of thrombosis on the balloon itself, as these patients are not typically systemically anticoagulated while the IABP is in place.

For the prehospital or ED provider, it is important to understand the basic workings of balloon pumps and their complications. Ultimately, care of the patient with an IABP should proceed as usual when treating the underlying condition necessitating the pump.

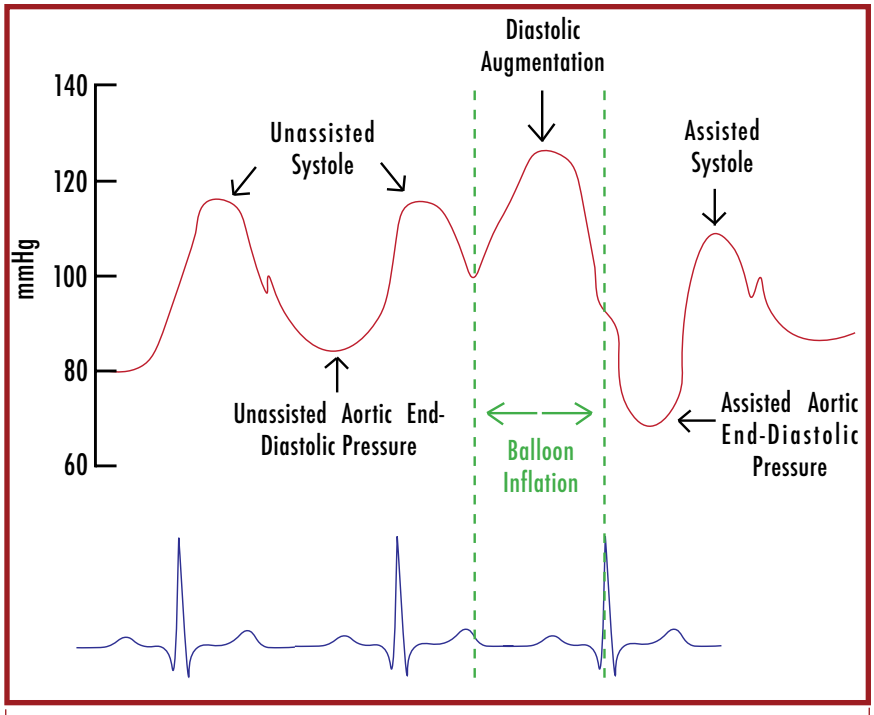

Figure 2. This cartoon illustrates how IABP inflation, starting at the beginning of diastole, augments diastolic blood pressure. By augmenting and increasing the diastolic pressure, coronary perfusion is improved. IABP balloon deflation, starting at the end of diastole, decreases the aortic end-diastolic pressure, decreasing afterload.

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Achilles Rupture
Continued from page 6

have the patient lie prone on the exam table with their feet extending past the edge. Squeezing the calf in this position should elicit plantar flexion of the foot. If it does not, the practitioner should strongly suspect injury to the Achilles tendon.[4] Howver, sensitivity of the Thompson test has ranged from 96% to only 78% in other studies, so it is important to keep a high clinical suspicion in the right clinical context.[8]

Plain films should be obtained in closed ruptures to evaluate for avulsion fractures or other associated osseous injuries. Kager's triangle is a space bounded by the Achilles tendon, calcaneus, and the tibia, which may be filled with blood and appear as a dark space on x-ray in such cases.[5] In open injuries and lacerations, plain films should be obtained to check for foreign bodies.

Several studies have shown the utility of ultrasonography in helping diagnose Achilles tendon rupture, especially in differentiating partial from full-thickness tears. One study reported that ruptures diagnosed in this way were confirmed by direct intraoperative findings with a high degree of accuracy,[6] and another smaller study reported 100% sensitivity and 83% specificity for distinguishing full from partial thickness tears.[7] Ultrasound is also useful to our orthopedic colleagues in following the process of tendon healing.

In the ED, the focus should be on supportive care and early orthopedic consultation. In the case of an open injury such as this one, proceed as with any open orthopedic injury with pain control, intravenous antibiotics (usually cefazolin), and updating tetanus as necessary. Complete ruptures require operative management. For closed partial ruptures, there is some debate in the literature regarding operative versus nonoperative management, and these decisions should be performed in consult with our orthopedic colleagues.

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Hypertensive Emergency
Continued from page 7

lowering to a systolic blood pressure of 110-139 mmHg was not statistically different for the primary outcome of death or disability compared to patients with a systolic pressure goal of 140 mmHg.[12]

Hypertensive emergency has a high rate of associated morbidity and mortality if not treated rapidly and appropriately. Understanding the characteristics of intravenous anti-hypertensive agents can aid in the quick selection of appropriate treatment of these patients in the Emergency Department.

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Drug	Mechanism	Physiologic Effects	Time to Onset	Duration of Activity	Dosing	Drug of Choice	Cost/ Formulary Status
Esmolol*	Selective B-1 blocker (heart)	• Decreases HR • Minimal BP effect	< 60 seconds	10-20 min	Bolus dose: 0.5-1 mg/kg Continuous infusion: 50-300 mcg/kg/min	Aortic dissection[6]	$8.80 for 100mg/ 10mL Formulary
Fenoldopam	Dopamine-1 agonist (vasculature, kidneys)	• Increases HR (dose-related) • Decreases BP • Increases renal perfusion, UOP, and Na excretion	5 min (max effect 30-60 min)	1 hour	0.1-0.3 mcg/ kg/min	----------	$452.33 for 10mg/1mL Non-formulary
Labetalol*	Alpha-1 (vasculature), B-1 (heart), and B-2 blocker (lungs)	• Decreases HR • Decreases BP • No change in CO, cerebal perfusion, or coronary perfusion	2-5 min (max effect 5-15 min)	2-4 hours	20mg loading dose, then: 20-80mg q10min OR 0.6-6mg/min continuous infusion	Severe HTN in pregnancy (or oral nifedipine or hydralazine)[10], aortic dissection[6]	$3.36 for 100mg/20 mL Formulary
Nicardipine	DHP-calcium channel blocker (primarily vasculature, heart)	• Decreases HR • Decreases BP • Causes cerebral and coronary vasodilation	5-15 min	4-6 hours	5 mg/hr, increase by 2.5mg/hr q5min to 15mg/hr Max: 30mg/hr	Acute ischemic stroke[8]	$200.61 for 20mg/ 200mL Formulary
Nitroprusside**	Direct vasodilator via nitric oxide (NO) release	• Increases HR • Decreases BP • Decreases afterload and preload • Decreases CPP • Increases ICP	Seconds	1-2 min	Do not use with hepatic or renal dysfunction 0.3-0.5 mcg/kg/min Max: 2 mcg/kg/min to decrease toxicity	----------	$1057.06 for 50mg/2mL Formulary

Table 1: Characteristics of recommended intravenous anti-hypertensive agents for treatment of hypertensive emergency.
HR = Heart Rate, BP = Blood Pressure, DHP = Dihydropyridine, CPP = Cerebral Perfusion Pressure, ICP = Intracranial Pressure
*Consider avoiding beta-blockade in a cocaine-induced hypertensive emergency; conflicting evidence currently exists relating to unopposed adrenergic predominance in this population
**Caution with nitroprusside – associated with significant toxicity (cyanide accumulation in hepatic and renal failure, unexplained cardiac arrest, coma, encephalopathy, convulsions, irreversible focal neurologic abnormalities, metabolic acidosis); has increased mortality if used within 12 hours post-myocardial infarction

Hyperleukocytosis
Continued from page 9

sensitive to chemotherapies. The characteristic electrolyte pattern includes hyperkalemia, hyperuricemia, hyperphosphatemia and hypocalcemia. Dying cancer cells release potassium, which can cause fatal cardiac arrhythmias. Uric acid precipitates in the collecting duct and causes kidney injury. Phosphate, which is highly concentrated in immature blasts, binds to ionized calcium, causing hypocalcaemia with symptoms such as peri-oral numbness, muscle tetany, and seizure. Calcium phosphate precipitation also contributes to kidney injury.[9,24] The most widely accepted diagnostic definition is the Cairo-Bishop (Table 2), which defines TLS as two or more laboratory changes within three days before or seven days after cytotoxic therapy.

Management of TLS involves aggressive IV hydration, correction of significant electrolyte abnormalities, reduction of uric acid, and frequent monitoring of laboratory studies. IVF and electrolyte corrections should be initiated immediately in the ED. Fluid goals can be aggressive, and reach up to 6-9 liters/day for an average adult.[2,4] Quarter normal saline with 5% dextrose is recommended for both TLS24 and leukostasis.[6]

Cairo-Bishop Laboratory Criteria for Tumor Lysis Syndrome
(-3 to +7 days from cytotoxic therapy)

two or more of the following:
- uric acid ≥ 8 mg/dL
- potassium ≥ 6 mg/dL
- phosphorus ≥ 4.5 mg/dL
- calcium ≤ 7 mg/dL [or > 25% change from baseline]

Table 2: Key laboratory findings consistent with tumor lysis syndrome

Hyperkalemia should be managed in a standard fashion including insulin/dextrose, sodium bicarb and albuterol as needed. Calcium replacement with calcium gluconate should be given for dysrhythmia or seizures. However, calcium should not be routinely replaced in asymptomatic patients, as additional calcium phosphate will precipitate, possibly leading to obstructive uropathy. Phosphate binders are recommended for moderate hyperphosphatemia (>2.1 mg/dL). In severe cases, hemodialysis can be used to correct all electrolyte abnormalities associated with TLS.[9,24]

The indications to prevent and treat hyperuricemia depend on the initial serum uric acid level and the type of malignancy, if known. American Society of Clinical Oncology guidelines rate malignant subtypes as low, intermediate and high risk for TLS, and direct initial prophylactic strategies accordingly.[24] Allopurinol is frequently utilized as a first line agent. Allopurinol prevents uric acid formation by inhibiting xanthine oxidase, the enzyme that degrades purine bases into uric acid, but will not actively decrease elevated serum levels. Alternatively, rasburicase, a recombinant form of urate oxidase, will actively cleave uric acid and is recommended as initial therapy for significantly elevated uric acid levels (>7.5mg/dL), as well as the highest risk malignancy subtypes.[24] Of note, rasburicase is known to cause hemolytic anemia and is contraindicated in patients with G6PD deficiency. Alkalinization of the urine with sodium bicarbonate was previously recommended, but evidence for its efficacy is equivocal and current guidelines no longer recommend this practice.[24] Initiation of uric acid prophylaxis will likely be performed in consultation with an oncologist, and should typically precede induction chemotherapy for hematologic malignancies. Patients with laboratory or clinical TLS should usually be admitted to an intensive care unit for frequent lab draws and cardiac monitoring.[9]

Disseminated Intravascular Coagulation

A frequent complication of hyperleukocytosis and of myelogenous leukemia in general is disseminated intravascular coagulation (DIC). Some leukemia subtypes trigger DIC by producing procoagulant tissue factor. Up to 40% of patients with hyperleukocytosis will present with DIC.[12] DIC is a consumptive coagulopathy that can present clinically with symptoms of hemorrhage or thrombosis. Serum studies will frequently show elevated PT/INR, decreased fibrinogen, elevated D-dimer and decreased platelets. Treatment in the emergency setting involves platelet transfusion for thrombocytopenia and cryoprecipitate or fresh frozen plasma for hypofibrinogenemia (>100).

The definitive treatment of DIC is to remove the underlying cause, and supportive measures should be quickly followed by chemotherapy when possible. Increased WBC production in leukemia depresses the production of other cell lines, and patients frequently present with anemia and thrombocytopenia. DIC may further contribute to this trend.

For patients with hyperleukocytosis, it is important to note that packed red blood cell (PRBC) transfusion may increase the blood viscosity, worsening leukostasis. PRBC transfusion for anemic patients with symptoms of leukostasis is a significant risk/benefit calculation.[9] Platelets, however, do not increase viscosity and should be transfused to maintain levels at least above 10K, and perhaps above 20K for patients with hyperleukocytosis to decrease the risk of cerebral hemorrhage.[9]

Hyperleukocytosis is an emergent condition and almost always indicates a hematologic malignancy. Leukostasis, tumor lysis syndrome and DIC are life-threatening complications seen in these patients. Aggressive IV hydration, electrolyte replacement and emergency stabilization can be initiated in the Emergency Department until definitive treatment of the underlying malignancy is achieved.

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CARA: A New Law to Help Opiate Addiction

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We're all seeing the devastating effects of opioid abuse in the Emergency Department, as Ohio leads the nation in suffering from the opioid epidemic. This month, the Surgeon General released a statement regarding the magnitude of the addiction epidemic in the United States. While the ED stands on the front lines of the fight, are policy-makers doing anything to address this issue? Thanks in large part to US senator Rob Portman of Ohio, the answer is yes.

Sponsored by Portman, the Comprehensive Addiction & Recovery Act (CARA), was passed this year and was lauded as a solid first step in the fight to stem the tide of opioid abuse blanketing the south and midwest. It passed by a majority of 92-2 in the Senate, and 407-5 in the House, galvanizing rare bipartisan support. It was signed into law by President Obama in July of this year. Action was desperately needed, as the Centers for Disease Control reports continually rising rates of opioid-related deaths, with over 15,000 people dying of overdoses each year in the United States (see Figure 1).

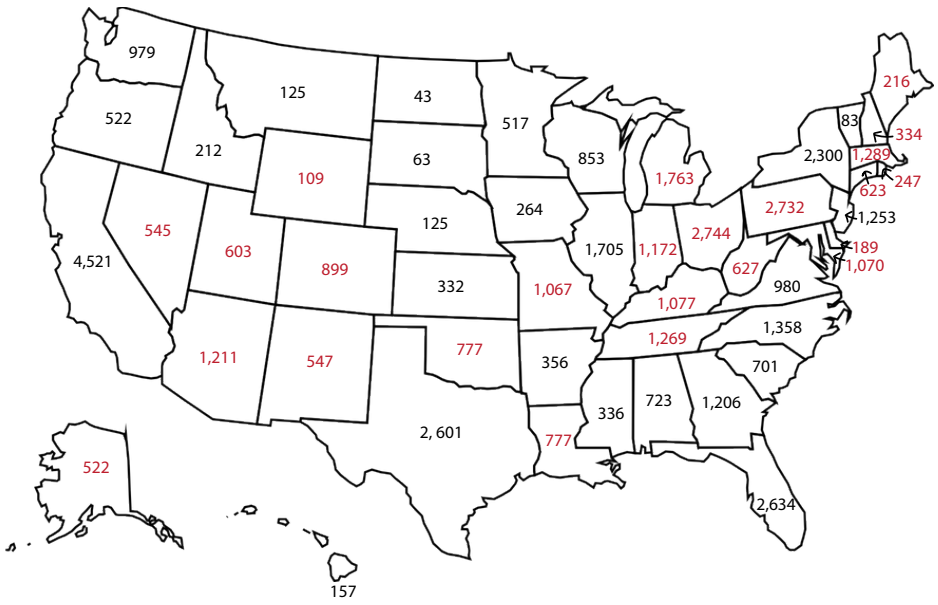

Figure 1: Map of the number of opioid related deaths in 2014[4]. Those states in red had an age-adjusted death rate from opioids of >16 in 2014.[4]

CARA authorizes $180 million for a comprehensive response to heroin and other opioids. Its goal was to create a balanced strategy to expand education and prevention while simultaneously focusing on treatment and recovery. Specifically, it focuses on treating addiction with individualized treatment plans, develops community-based prevention services and creates recovery programs. It also expands disposal sites for unwanted prescription pills.

Additionally, it expands the availability of naloxone (narcan) to first responders, as well as training law enforcement personnel in overdose reversal techniques. On the legal side of things, it expands the treatment for individuals in the criminal justice system and provides diversion programs for addicted individuals into treatment instead of jail.

Opioid abuse is a problem all Emergency Physicians will see. CARA is one way the US is fighting back.

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HYPERLEUKOCYTOSIS

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Sgarbossa Criteria

EKG focus

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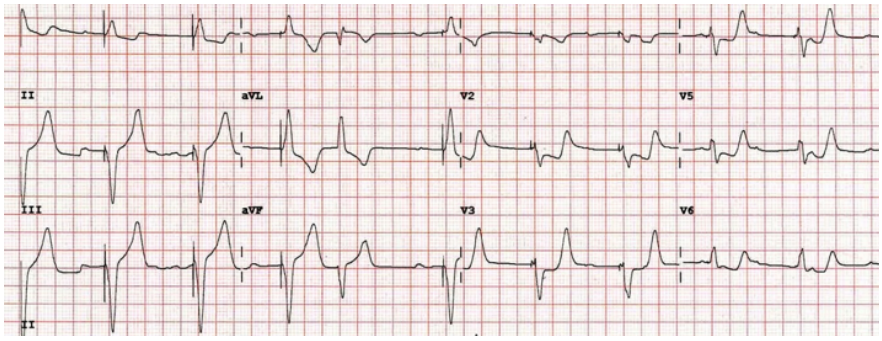


Figure 1. A representative EKG showing a ventricularly paced rhythm. In leads V2 and V3, there is ≥ 1 mm of concordant ST segment depression, meeting the second Sgarbossa criteria.

History of Present Illness

A male in his 70s presents with two hours of crushing substernal chest pain that is associated with shortness of breath and diaphoresis. The patient states that he has a history of an abnormal heart rhythm that required him to have a pacemaker implanted.

Sgarbossa Criteria

The Sgarbossa criteria, first published in 1996, was developed to help diagnose acute myocardial infarction (AMI) in the setting of a left bundle branch block (LBBB) or ventricular pacing.

In LBBBs, discordance is normal: the ST segment and/or T wave are in the opposite direction to the QRS complex. In AMIs, there is inappropriate concordant elevation or depression (Table 1), or excessive discordant elevation. In the original weighted criteria, a minimum of three points was required for the diagnosis of AMI.

Since the publication of the Sgarbossa criteria, attempts at validation have found to it have good specificity but poor sensitivity. This led to the development of the modified Sgarbossa criteria, published in 2012 (Figure 2).

≥ 1 mm of concordant ST segment elevation	5 points
≥ 1 mm of concordant ST segment depression in lead V1, V2, or V3	3 points
≥ 5 mm of discordant ST segment elevation	2 points

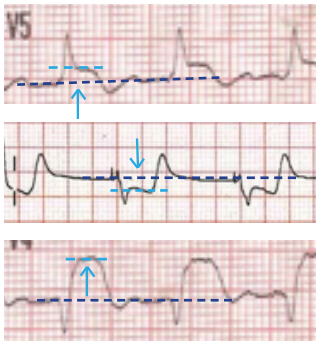
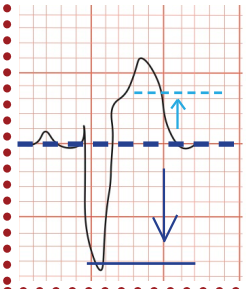


Table 1. Sgarbossa criteria with EKG examples highlighting each criteria. The navy dashed line indicates the baseline as defined by the TP segment. The dashed light blue line indicates the ST segment. The light blue arrow highlights the concordance or discordance of the ST segment. The points listed next to each criteria shows the values given to each criteria as defined in Dr. Sgarbossa's initial paper.

Modified Sgarbossa Criteria



The modified criteria replace the weighted score with a 3 point score, any of which, if positive, are concerning for STEMI. Discordant ST segment elevation out of proportion (≥25%) of the depth of the preceding S wave is concerning for AMI. The modified criteria has shown improved sensitivity compared to the original Sgarbossa criteria.

Figure 2. A cartoon illustration highlighting examples of the modified Sgarbossa criteria. The thick navy dashed line indicates the baseline, defined by the TP segment. The S wave depth is indicated by the solid navy line. The dashed light blue line indicates the discordant ST segment. The arrows represent ST segment and S-wave deviation from the TP segment. In both examples the ST segment is > 25% of the depth or height of the S wave.

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EKG and Case referred by

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Annals of B Pod is always looking for interesting cases to publish!

Please submit cases in the composition book located in B Pod. Make sure to include the R1/R4 involved in the case.

List of Submitted B Pod Cases

Case	Case Physicians
Achilles tendon rupture	Boyer/Ham
GI bleed/pericardial effusion	Winders/Hani
Anoxic brain injury after overdose	Scupp/Banning
Symptomatic hyponatremia	Winders/Bensman
Lupus/rash	Mckean/Spigner
Cecal volvulus	Scupp/Kenny
Hyperleukocytosis	Lagasse
Inferior ramus fracture	Winders/Banning
Torsades	Winders/Banning