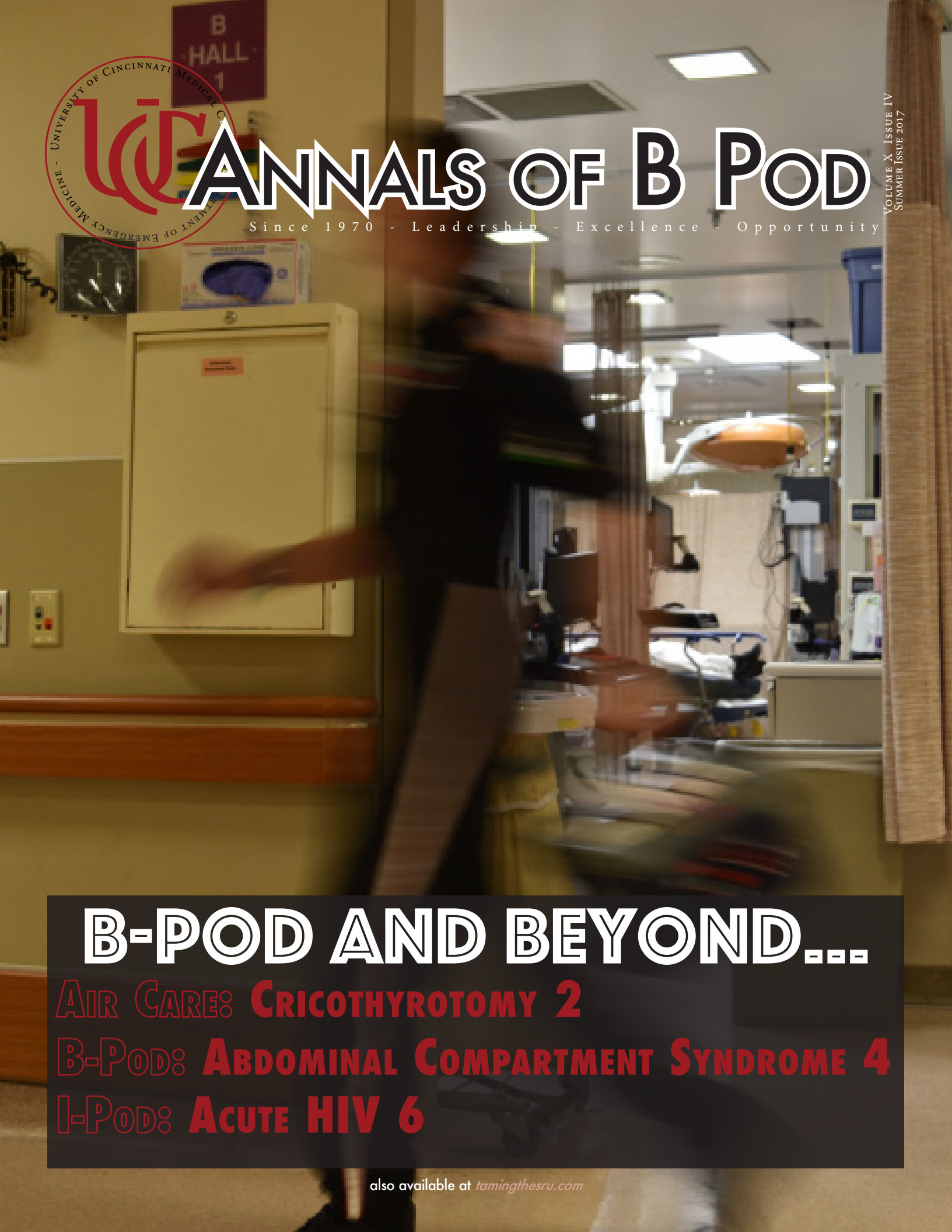

University of Cincinnati Medical Center - Department of Emergency Medicine

Annals of B Pod

Since 1970 - Leadership - Excellence - Opportunity

Volume X Issue IV
Summer Issue 2017

B-Pod and Beyond...

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UC Emergency Medicine
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From minor care to major injuries, this Summer 2017 edition of Annals of B Pod goes beyond B Pod. Dr. Murphy starts off with a discussion of the learning points from his prehospital cricothyrotomy in this month's Air Care column. From B pod, Dr. Harty discusses the management of abdominal compartment syndrome. Dr. Golden tells of a case of acute HIV presenting as thrombocytopenia. Dr. Baez's procedure piece describes how to measure bladder pressure for patients at risk of elevated intra-abdominal pressures. From I pod, Dr. Banning describes the diagnosis and treatment of Lisfranc injuries. In the monthly pharmacy article, we discuss reversal of neuromuscular blockade with sugammadex. Finally, Dr. Scanlon describes de Winter's morphology in the EKG corner.

aircare

Tim Murphy, MD
University of Cincinnati R2

To Cric or Not to Cric?

History of Present Illness

Air Care was dispatched to a scene for a male patient in his 20s who was involved in a motor vehicle collision. Prior to arrival, first responders report that the patient was entrapped in his vehicle with a metal pole entering adjacent to his mouth and protruding out the back of his neck. There was no further information on vitals or mental status.

Upon Air Care arrival, EMS had successfully extricated the patient from his vehicle by cutting the metal pole on both sides. The patient was initially combative, prompting EMS to administer 5 mg intramuscular midazolam to facilitate extrication. En route, the initial vital signs were reported to the Air Care crew as a blood pressure of 160s systolic, a heart rate of 130, and an oxygen saturation of 94% on room air. However, in the 10 minutes before Air Care's arrival, the patient became increasingly somnolent.

Physical Exam

Initial assessment revealed a young male laying in the right lateral decubitus position on a stretcher in the ambulance. There was a moderate amount of active bleeding coming from the oropharynx and pooling on the floor of the ambulance. The EMS providers were holding a non-rebreather mask over the patient's nose and mouth but were unable to secure it due to obstruction by the pole. His Glasgow Coma Scale (GCS) was 3, and vitals obtained by the crew were a blood pressure of 110/70, heart rate of 130, respiratory rate of 8, and an oxygen saturation of 92% on 15L. The patient had a 5 cm diameter pole entering the left side of his mouth, through his oropharynx and exiting the left posterior neck. He had ongoing intraoral bleeding but there were no other signs of head or facial trauma. Other obvious injuries included an open left forearm fracture with active bleeding. There were no external signs of trauma to his chest or abdomen. Breath sounds were diminished bilaterally, and the abdomen was soft. Peripheral pulses were present in all four extremities, and his pelvis was stable to compression.

Interventions on Scene

Given the active bleeding from his upper extremity, a tourniquet was immediately applied to the patient's left arm with cessation of hemorrhage. The next priority was managing the patient's airway. The patient had high predicted difficulty due to multiple factors included in the "LEMON" difficult airway mnemonic (table 1). On first look, the pole and active hemorrhage were distorting the anatomy of the face and oropharynx, making landmark identification challenging. A complete 3:3:2 evaluation and Mallampati classification of the airway were difficult to perform based on the foreign object, ongoing hemorrhage, and concern for unstable facial fractures. There was clearly a large obstruction to the airway, and neck mobility was limited due to the pole and concern for cervical spine injury. Additionally, rescue by bag-valve-mask

Difficult Airway Mnemonics

Laryngoscopy
Look Externally
Evaluate (3:3:2)
Mallampati Score
Obstruction/Obesity
Neck Mobility

Extraglottic Device
Restricted Mouth Opening
Obstruction
Disrupted/Distorted Airway
Stiff Lungs/Spine

Bag-Valve Mask
Mask Seal
Obstruction/Obesity
Age > 55
No Teeth
Stiff Lungs/Sleep Apnea

Cricothyrotomy
Surgery
Hematoma
Obesity
Radiation (or other distortion)
Tumor

Table 1: Helpful mnemonics for predicting airway difficulty.

(BVM) would be difficult as predicted by the "MOANS" assessment. The mask seal was prohibited by the patient's facial trauma and obstruction from the pole. Extraglottic device placement would also be problematic due to obstruction and likely distorted anatomy based on the "RODS" assessment. Finally, the patient did not have any true predictors of difficult cricothyrotomy based on the "SHORT" mnemonic.

As the patient was hemodynamically stable, this was not a crash airway. Consequently, the decision was made to give one attempt at an awake look using ketamine as analgosedation with immediate transition to cricothyrotomy if unsuccessful.

Prior to the intubation attempt, the plan was verbalized to the entire team including the flight nurse and EMS providers. Specific roles were assigned to each provider. Finally, the airway equipment, suction, bougie, and cricothyrotomy kit were positioned in the appropriate places to facilitate the initial intubation attempt and potential rescue cricothyrotomy.

The awake look was attempted after administration of 150 mg of ketamine. The base of the tongue was discernible; however, visualization of the cords was difficult due to the amount of active hemorrhage, distorted anatomy, and the pole protruding through the oropharynx. Given this, the team then immediately transitioned to cricothyrotomy. After palpation of landmarks, a 4 cm vertical incision was made through the skin. Blunt dissection was performed and the cricothyroid membrane was then re-palpated to confirm location due to blood obscuring any visual landmarks.

Image 1: Patient in the SRU after pre-hospital cricothyrotomy.

While firmly holding the trachea, a horizontal puncture incision was made through the cricothyroid membrane resulting in bubbling of air through the pooled blood. A finger was then passed into the trachea to enlarge the incision and stabilize the trachea. With the provider's finger still in the trachea, a bougie was passed alongside through the defect in the cricothyroid membrane. Finally, a 6.0 tube was passed over the bougie, and the cuff was inflated. This resulted in adequate end-tidal waveform and equal bilateral breath sounds. The patient was then loaded onto the helicopter and transported to the University of Cincinnati Medical Center. In route, oxygen saturations were in the mid 90s on 100% oxygen.

Hospital Course

Initial assessment in the resuscitation bay revealed a GCS of 3 with equal breath sounds bilaterally. Vital signs were remarkable for a systolic blood pressure in the low 100s and hypoxia to 85% on 100% oxygen. Chest x-ray showed a right mainstem intubation, so the tube was pulled back 6 cm. His oxygen saturation quickly improved to 100%. A right femoral trauma catheter was placed and two units of blood and plasma were given for borderline hypotension. CT scans were deferred due to concerns of artifact with the metal pole. The patient was taken emergently to the operating room (OR) with the trauma team, otolaryngology, and vascular surgery.

In the OR, a left neck exploration was performed which revealed intact carotid and jugular vessels. The metal pole was then removed with minimal bleeding. Additional injuries repaired in the OR included a left mandibular fracture, a right Le Fort I fracture, washout and closure of the posterior pharyngeal wound, and a complex facial laceration repair. The patient also received an open reduction and internal fixation of his left radius and ulnar forearm fractures. CT scans performed following the OR revealed numerous other injuries including multiple cervical spine transverse process fractures and an occluded left vertebral artery. A follow up MRA confirmed the vertebral artery occlusion and revealed a corresponding focal cerebral ischemic insult.

The remainder of the patient's hospital course was uneventful. He regained consciousness and had a GCS of 15 prior to discharge. His residual neurologic deficits at discharge included paralysis of

Continued on page 14

bpodcase

Abdominal COMPARTMENT Syndrome

Shaun Harty, MD
University of Cincinnati R1

History of Present Illness

The patient is a female in her 70s with a history of hypothyroidism and unspecified dementia who presented to the emergency department (ED) from her skilled nursing facility (SNF) due to altered mental status and abdominal distention. Per report, the patient had recently been diagnosed with a small bowel obstruction via a plain film obtained at her SNF. She may have been treated with an enema in the days prior to presentation. On the morning of her arrival, the patient had increasing abdominal distention with severe pain. She subsequently became altered, prompting transfer to the ED. Her blood sugar was within normal limits prior to transfer.

Past Medical History
Hypothyroidism
Dementia

Medications
Levothyroxine
Mirtazapine

Vitals

T 97.4 HR 103 BP 133/107 RR 33 SpO2 93% RA

Physical Exam

Exam reveals an ill-appearing elderly female that is not responsive to verbal or painful stimuli. Her eyes are open with a fixed rightward gaze deviation. Pupils are equal, round, and reactive to light bilaterally. Her upper extremities are contracted upwards. She is tachypneic but her lungs are clear to auscultation bilaterally. Cardiovascular exam reveals a tachycardic rate with a regular rhythm and no murmurs appreciated. Her abdomen is markedly distended and rigid. No significant lower extremity edema. No obvious signs of traumatic injury.

Labs and Imaging

BUN/Creatinine: 39/1.6
WBC: 2.7 H/H: 15/49
Lactate: 10.4 VBG: 7.09/63/21
Troponin: 0.05 BNP: 178
EKG: Sinus tachycardia

Abdominal X-Ray (Image 1): Sigmoid volvulus/pneumoperitoneum
CXR (Image 2): Pneumoperitoneum

Hospital Course

IV access was obtained and fluid resuscitation was initiated. Based on the patient's history and physical exam, there was concern for small bowel obstruction or other intra-abdominal pathology with resultant sepsis. Additionally, given the patient's neurological exam, there was concomitant concern for status epilepticus versus intracerebral hemorrhage or ischemic stroke. She was given 10 mg of intramuscular midazolam without improvement of her mental status and was felt to not be protecting her airway. As such, she was intubated and loaded with levetiracetam. Plain films of the chest and abdomen revealed pneumoperitoneum. She was started on piperacillin/tazobactam for her presumed sepsis. She became progressively hypotensive requiring push-dose vasopressors. Prior to being admitted to the acute care surgery service, a CT of the head was obtained which ruled-out intracranial hemorrhage. The patient was transferred to the surgical intensive care unit pending medical stabilization and a goals of care discussion with family.

The patient eventually underwent exploratory laparotomy, where she was found to have a sigmoid volvulus with infarction and perforation, cecal infarction with perforation, and abdominal compartment syndrome. She underwent a total colectomy. She was monitored with continuous electroencephalogram without evidence of seizures or non-convulsive status epilepticus. Her mental status did not improve. An MRI of the head and neck was obtained demonstrating bilateral cerebral and cerebellar infarcts. Her family ultimately pursued comfort care given her poor prognosis, and she was transferred to hospice.

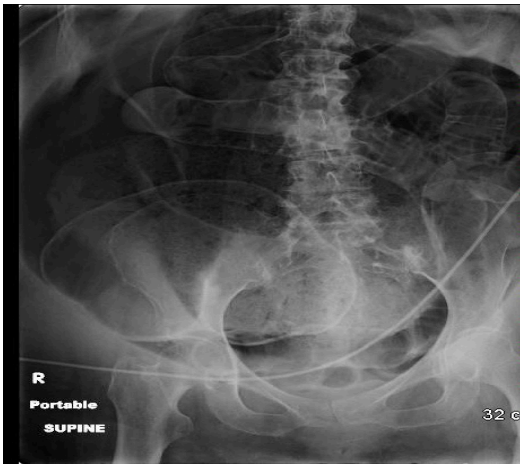

Image 1: Abdominal plain film revealing a sigmoid volvulus and pneumoperitoneum.

Discussion

Abdominal compartment syndrome is a rare but likely underrecognized clinical condition. Defined as intra-abdominal pressure greater than 20 mmHg with associated end-organ damage, abdominal compartment syndrome can be seen after recent surgery or due to any number of intra-abdominal pathologies.[1] Few studies exist that find agreement upon the true incidence and

Primary Etiologies

Hemoperitoneum
Abdominopelvic Trauma
Retroperitoneal Hemorrhage
Bowel Distension
Massive Ascites
Liver Transplantation
Pancreatitis

Secondary Etiologies

Aggressive Fluid Resuscitation (*e.g.,* sepsis, burn patients, shock states)

Figure 1: Etiologies of Abdominal Compartment Syndrome.

prevalence of the disease process, ranging from 1% to 14% depending on the exact etiology of the condition.[2,3] Although the precise incidence is unknown, it carries a high rate of morbidity and mortality, making early recognition by the ED physician paramount.

Patients presenting to the ED with abdominal compartment syndrome are typically critically ill, and therefore are not often able to provide a history, as seen with the above patient. If they are able to, they will often be experiencing abdominal pain and bloating, difficulty breathing, and lightheadedness. On physical examination, they will have a distended abdomen that is tender to palpation and possibly rigid or peritonitic if infarction and perforation has occurred. In critically ill patients, decreased urine output, increased ventilation requirements, hypotension, tachycardia, and a lactic acidosis in the setting of a distended abdomen should raise suspicion for abdominal compartment syndrome.

Abdominal compartment syndrome can be classified into primary or secondary causes. In general, a primary cause is from trauma or a direct medical or surgical etiology, whereas a secondary cause is typically iatrogenic from aggressive fluid resuscitation in sepsis and burn patients. Consider abdominal compartment syndrome if the patient has had any recent abdominal surgery or recent treatment of the pathologies listed in the table above (figure 1).

Intra-abdominal pressure may be measured by specialized pressure transducer instruments that can be passed into the stomach by a nasogastric route, into the colon from the rectum, or into the bladder using a Foley catheter. Measurement of intravesicular pressure by Foley catheter has become the standard method of screening.[4] Intra-abdominal hypertension exists when the pressure is sustained at greater 12 mmHg.[1] As stated above, abdominal compartment syndrome is recognized as an elevated abdominal pressure that is greater than 20 mmHg with evidence of end-organ damage.[1]

To better understand abdominal compartment syndrome, one must appreciate the physiology of the process that leads to it. Intra-abdominal pressure is related to a patient's body mass

APP = MAP - IAP

Figure 2: APP- Abdominal perfusion pressure; MAP- Mean arterial pressure; IAP- Intra-abdominal pressure

index and can be influenced by any recent abdominal surgeries or disease processes.[5] A prospective study looking to determine normal intra-abdominal pressure found a range of 0.2-16.2 mmHg with a mean pressure of 6.5 mmHg.[5] Different stages of intra-abdominal hypertension exist, ranging from normal for that individual towards a pathological increase towards abdominal compartment syndrome. The most obvious consequences of this pathology are the effects on the intra-abdominal organs.

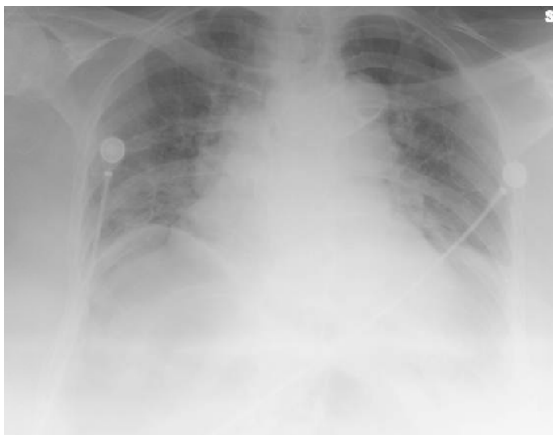

Image 2: Pneumoperitoneum in the right upper quadrant.

When intra-abdominal pressure rises, the abdominal perfusion pressure is reduced, leading to end-organ dysfunction (Figure 2). This is frequently exacerbated by the fact that these patients are often critically ill and have low MAPs to begin with, further reducing the abdominal perfusion pressure. This has multiple pathophysiologic effects, the majority of which have been studied in animal models.

In the abdominal compartment, increasing pressures leads to reduced perfusion of the abdominal viscera. This can result in progressive bowel ischemia, infarction, and perforation, as well as a significant lactic acidosis, all of which were seen in the presented patient.[6] The lactic acidosis is worsened by the fact that there is reduced hepatic perfusion and therefore inadequate lactate clearance. In addition, intestinal edema and necrosis can lead to subsequent bacterial translocation, sepsis, and multisystem organ dysfunction.[7]

Increased abdominal pressure also leads to compression of the renal veins, causing an increase in renal pressure and acute kidney injury.[8] As the intra-abdominal pressure increases, the patient will become progressively oliguric and may progress to anuria. In addition, the patient generally has reduced cardiac output with subsequent activation of the renin-angiotensin-aldosterone system (RAAS), leading to renal vasoconstriction and further kidney injury.[8]

Abdominal compartment syndrome also leads to cardiopulmonary and neurologic effects. These patients are typically hypovolemic and with the high abdominal pressure can have compression of the inferior vena cava with reduction in preload, all leading towards reduced cardiac output.[9] Activation of the RAAS system also causes increased systemic vascular resistance. Intra-thoracic pressure can become elevated with a reduction in compliance and difficulty with ventilation.[10] Cerebral venous outflow can be obstructed due to the increased intra-thoracic pressure, leading to a significant increase in intracranial pressure.[11]

Continued on page 15

bpodcase

Acute HIV

When rapid testing is not enough

Andrew Golden, MD
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History of Present Illness

This patient is a male in his late 20s with a past medical history of sexually transmitted infections, alcohol abuse, and intravenous drug use (IVDU) presenting to the emergency department (ED) with a rash. The patient states that five days prior to presentation, he noted progressive development of a pruritic rash on his trunk, arms, and legs. He reports having unprotected anal intercourse with other men and is concerned he was exposed to HIV three weeks ago. He presented to the ED at that time and was offered post-exposure prophylaxis but was unable to afford this medication. He also reports being treated for a skin infection with cephalexin and trimethoprim/sulfamethoxazole approximately two weeks prior to the development of his current rash. He denies fevers, genitourinary symptoms, nausea, and shortness of breath.

Past Medical History

Gonorrhea
Alcohol abuse
IVDU
Bipolar disorder

Medications

Clonidine
Diazepam
Trazadone

Vitals

T 36.9 HR 87 BP 126/75 RR 16 SpO2 97% in RA

Physical Exam

On physical exam, the patient is in no acute distress. He has palatal petechiae noted in his oropharynx (figure 1). His cardiovascular, pulmonary, abdominal, and neurologic exams are normal. His skin exam is notable for a blanching macular rash over his bilateral upper and lower extremities and chest. He also has petechiae over his upper and lower extremities (figure 2). His palms and soles are spared.

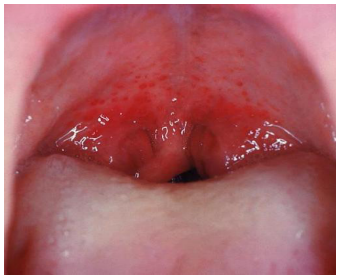


Figure 1: Representative image of palatal petechiae[1]

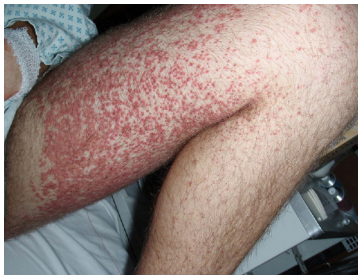


Figure 2: Representative image of lower extremity petechiae[1]

Labs & Diagnostics

WBC: 9.6 Hgb: 14.4
Platelets: 7
Creatinine: 0.97
HIV Screen: Negative
Hepatitis Panel: Nonreactive
Syphilis: Negative

Hospital Course

The patient was admitted to the hospital for severe thrombocytopenia. He received a platelet transfusion with a goal platelet count of greater than 10 given his lack of active bleeding. He was also started on steroids based on consultation with hematology in the ED. His initial HIV quantitative PCR was indeterminate. Two days later, his qualitative HIV RNA returned positive. However, his RNA quantitative levels were low. He followed up with infectious disease one week later and was found to have an HIV RNA count of greater than 10 million copies, confirming a diagnosis of acute HIV.

Discussion

Acute HIV Infection

According to the United Nations, as of the year 2012, an estimated 35.3 million people were living with HIV globally.[2] Additionally, there were 2.3 million new HIV infections.[2] While the number of new HIV infections globally continues to decrease, acute HIV infection continues to be a problem that can be diagnosed and managed in the ED. One of the difficulties of diagnosing acute HIV infection is the vague complaints with which patients may present. The most common complaints associated with acute HIV infection include fever (75%), fatigue (68%), myalgia (49%), skin rash (48%), and headache (45%).[3] These symptoms are slightly different between patients with sexual transmission versus transmission associated with IVDU. IV drug users more commonly present with fatigue and headache while patients with sexually transmitted infections more frequently experience fever, myalgia, and rash. Most commonly, these symptoms present between one and six weeks following exposure. Overall, the most common signs of acute infection are tachycardia and generalized lymphadenopathy.[4]

Due to these vague presenting symptoms, this diagnosis can be easily missed in the ED. Obtaining a thorough social history can help identify patients at risk for HIV, and testing should be considered in patients who endorse high risk behaviors such as multiple sexual partners, men who have sex with men (MSM), and IV drug users. This patient presented with thrombocytopenia as a manifestation of acute HIV. Approximately 10% of patients with acute HIV will present with thrombocytopenia, although it can occur at any point in the disease process. In addition, the degree of thrombocytopenia is associated with higher rates of viral replication and is often correlated with viral hepatitis co-infection. Therefore, in any patient presenting with new thrombocytopenia, HIV testing should be considered. Hepatitis serology may also be indicated.

The pathophysiology behind this finding is not fully elucidated. It

is thought to be due to increased peripheral platelet destruction and ineffective platelet production due to HIV infected megakaryocytes. Nonetheless, these immunocompromised patients are also at risk for opportunistic infections, many of which can lead to platelet destruction themselves. Involvement of an infectious disease specialist to help evaluate and treat concomitant infection is crucial for these patients. Ultimately, ED management of HIV-associated thrombocytopenia should focus on prevention of bleeding or achieving hemostasis in patients with active hemorrhage. Platelet transfusion thresholds and hospital admission indications vary based on each patient's clinical presentation but are generally similar to standard therapy. Platelet counts may improve with initiation of anti-retroviral therapy. Refractory thrombocytopenia in HIV-infected individuals may respond to steroids and IVIG, but a hematology consultation should be obtained in such cases.

The diagnosis of acute HIV infection in the ED is important for many reasons. Primarily, early initiation of antiretroviral therapy can have a significant impact on the morbidity associated with HIV. Furthermore, early detection can prevent transmission of the virus. The viral burden is highest in the acute phase of infection, and it is estimated that as the viral burden increases by a factor of 10, the risk of transmission increases by a factor of 2.5.[5] Approximately 50% of new HIV infections are thought to be acquired during acute HIV infection present in the source patient.[6] After a diagnosis of HIV is made, additional baseline labs should be obtained, including: CD4 count, viral load, genotypic testing (discussed below), glucose, lipids, complete blood count, renal panel, hepatic enzymes, urinalysis, and hepatitis serologies. Sending these studies from the ED once the diagnosis is made can expedite outpatient management for the patient.

The ED treatment of acute HIV infection largely includes the initiation of antiretroviral therapy if appropriate. While the guidelines regarding initiation of therapy in acute HIV infection vary in their recommendations, most seem to favor starting patients on antiretroviral therapy early in their diagnosis. This is supported by a 2015 study demonstrating that beginning antiretroviral therapy in HIV-positive patients decreased both AIDS- and non-AIDS-related complications and mortality.[7] Prior to initiating antiretroviral therapy, drug resistance testing should be obtained, as up to 20% of patients with acute HIV have at least one resistance mutation. This should not, however, hinder initiation of therapy.

CDC Recommendations for PEP

- Tenofovir disoproxil 300mg + emtricitabine 200mg (truvada) once daily

PLUS

- Raltegravir 400mg (isentress) twice daily OR dolutegravir 50mg (tivicay) once daily

Figure 3: Current CDC recommendations for post-exposure prophylaxis.

One commonly recommended regimen includes dolutegravir and tenofovir alafenamine-emtricitabine or tenofovir disoproxil-emtricitabine. Patients with any medical comorbidities or those taking long-term medications should be more comprehensively evaluated for the optimal medication regimen, as many contraindications do exist. The management of these patients is likely best handled in concert with an infectious disease consultant. Disposition for patients with newly diagnosed HIV is quite variable because the severity of presentation can be unpredictable. Evaluation and disposition should be tailored to the individual patient, keeping in mind the risk of opportunistic infections in untreated HIV patients. In general, most hemodynamically stable patients with newly diagnosed HIV can be discharged from the ED after initiation of antiretroviral therapy and establishment of follow-up.

Post-Exposure Prophylaxis

In the case presented, the patient sought medical attention after a possible HIV exposure, and post-exposure prophylaxis (PEP) was recommended. Over the past decade, the number of patients presenting to the ED after known exposure or possible non-occupational exposure to HIV has tripled.[8] The ED is an opportune setting for the initiation of PEP, as early institution of antiretroviral therapy can decrease the rate of HIV infection. There are a number of Centers for Disease Control and Prevention (CDC) recommendations regarding who should be offered PEP therapy for non-occupational exposures.[9] These include patients who have had unprotected sexual intercourse with an HIV-positive patient or exposure to blood from an HIV-positive individual. It may also be warranted if the source individual had an unknown HIV status but is part of a high-risk group, such as MSM. Importantly, PEP should only be definitively initiated when the exposure occurred within 72 hours of presentation. See figure three for current CDC recommendations on HIV PEP.

All patients who receive PEP should be tested for HIV infection status, preferably by rapid antigen/antibody testing.[10] However, PEP should not be delayed in a setting where this type of testing is unavailable. Other baseline blood tests to be drawn before initiating PEP include hepatitis studies, a renal panel, and a hepatic profile. Trending these labs is recommended, as renal toxicity is a possible side effect from first-line PEP regimens. Additionally, these medications are contraindicated when the patient's GFR is less than 60.

A 28-day course should be provided to all patients in whom PEP is initiated from the ED. It is important to evaluate the patient's current prescriptions and over-the-counter medications for potential interactions. Important interactions to this regimen may include antacids and anti-tuberculosis medications.[11] Follow up with a primary care provider should be established within two weeks for further testing of HIV status and repeat renal and hepatic studies.

Continued on page 13

Procedure Piece:

Measurement of Intra-Abdominal Pressure

Jessica Baez, MD
University of Cincinnati R2

In cases of suspected abdominal compartment syndrome, measurement of the intra-abdominal pressure is a crucial step in the diagnosis and management of these often critically ill patients. Early identification of elevated intra-abdominal pressures by the emergency medicine provider can be life and organ saving. Measurement of a patient's bladder pressure is the current standard of care for diagnosis of this rare disease process and should be considered in patients with severe abdominal distension and a tense or rigid abdomen.

There are many different ways to perform this procedure, and certain Foley catheters have the capability to obtain this measurement without additional supplies. Nonetheless, these are often difficult to find in the emergency department (ED), and locating them may lead to dangerous delays in patient care. Therefore, the following is a description of how to obtain this measurement with supplies that can quickly be found in the ED (image 1).

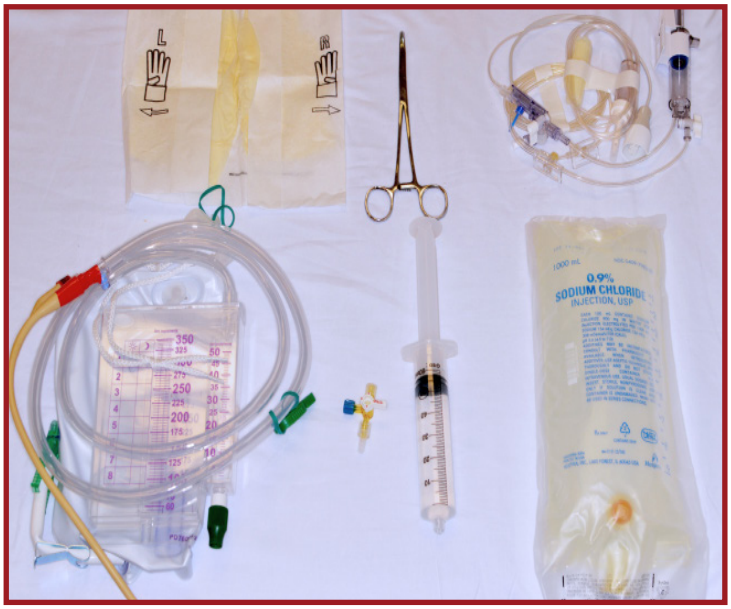


Image 1: Supplies needed for intra-vesicular pressure monitoring

Supplies

Foley catheter kit
Sterile gloves
Kelly forceps
60 mL syringe filled with sterile saline
Three-way stopcock
Arterial line pressure transducing tubing
1L normal saline
Pressure bag

The Procedure

1. Insert a Foley catheter and allow complete drainage of urine.

2. Prepare a standard arterial line set-up and prime it using the 1L bag of saline.

3. Connect the stopcock attached to a 60 mL syringe of saline to the end of the arterial line tubing.

4. Lie patient flat.

5. Clean Foley catheter access port with an alcohol swab.

6. Connect the three-way stopcock to the Foley catheter access port (image 2).

7. Zero the pressure transducer at the level of the iliac crest in the mid-axillary line.

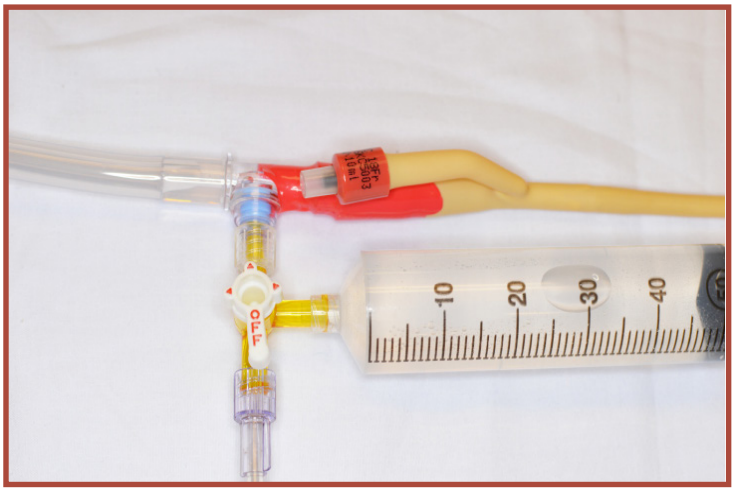


Image 2: The stopcock should be connected to the Foley catheter access port, a 60 mL syringe, and the arterial line tubing.

Continued on page 14

quickhit

Quick Hit: Lisfranc Injuries

Kathryn Banning, MD
University of Cincinnati R1

History of Present Illness

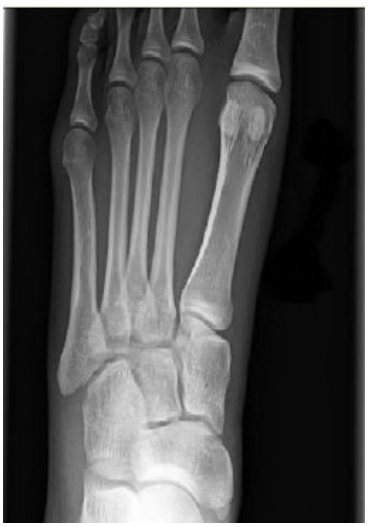
An otherwise healthy teenage female presents to the emergency department (ED) with left foot pain after being dared by her friends to jump out of a second story window. She reportedly landed directly on the plantar aspect of her left foot and was unable to bear weight on that foot following the incident. She reports no other injuries.

Physical Exam

The patient was well appearing and in no acute distress. Musculoskeletal exam was most significant for diffuse swelling and ecchymosis over the left anterior foot with tenderness over the base of the metatarsals. The foot was otherwise neurovascularly intact. Her cardiopulmonary, abdominal, and neurologic exams were within normal limits.

Imaging

The initial x-ray of the left foot was significant for soft tissue swelling without acute osseous findings. A weight bearing x-ray was then obtained that showed a linear lucency at the base of the first metatarsal with an osseous fragment between the base of the second metatarsal and medial cuneiform, with associated widening of the Lisfranc interval, concerning for a Lisfranc fracture. A CT of the foot confirmed the diagnosis of a Lisfranc fracture of the distal aspect of the medial cuneiform with multiple small ossific fracture fragments in the Lisfranc interval.



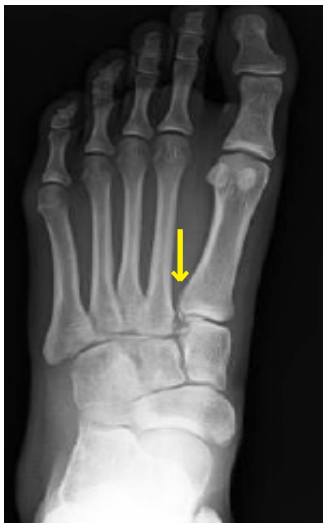


Figure 1: Unremarkable initial x-ray. Figure 2: Weight bearing film revealing widening of the Lisfranc interval and a small lucency on the medial aspect of the base of the first metatarsal (arrow).

Hospital Course

After CT results confirmed a Lisfranc fracture, the case was discussed with the orthopedist on call. The patient was placed in a posterior splint and was instructed to remain non-weight bearing until follow up. She followed up in orthopedic clinic four days later and was placed in a short boot brace and was scheduled for surgery within the next two weeks. She underwent successful open reduction and internal fixation of the left foot. In the operating room, the lateral displacement of the base of the second metatarsal was easily visualized. It was manually reduced and a screw was then placed from the base of the second metatarsal to the medial cuneiform. She was discharged with outpatient follow-up and is doing well.

Discussion

Lisfranc fractures and dislocations are rare and commonly overlooked in the ED setting. Because the standard workup is often unrevealing, approximately 20% of these injuries are missed on initial presentation.[1] Knowing the risk factors and clinical presentation of Lisfranc injuries can help providers identify when to suspect this diagnosis.

In order to assess for this injury, it is important to understand the anatomy that makes up the Lisfranc joint. This joint complex connects the midfoot to the forefoot, and is a critical area in stabilizing the arch of the foot to assist with ambulation. It is particularly crucial in ankle dorsiflexion and plantarflexion.[2] The Lisfranc joint complex consists of the five metatarsals of the forefoot articulating with the three cuneiforms and the cuboid of the midfoot.[3]

Of these bones, the second metatarsal in particular plays the largest role in stabilization of the arch and any disruption can often lead to displacement of the third through fifth metatarsals. There are multiple ligaments supporting the joint, with the strongest being the Lisfranc ligament connecting the medial border of the second metatarsal with the lateral aspect of the medial cuneiform. Additionally, there is also a transverse ligament that connects the second through fifth metatarsals. Disruption of this ligament in Lisfranc injuries characteristically results in displacement of the first metatarsal on imaging as discussed below.

The incidence of Lisfranc injuries has been reported at 1 in 55,000 per year, and it is two to four times more likely to occur in men than women. The average age of patients with this injury is in the fourth decade.[3] These injuries are often sustained by a high-energy mechanism

Continued on page 12

Sugammadex
Reversal of Neuromuscular Blockade

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Neuromuscular blocking agents (NMBAs) work by paralyzing skeletal muscles though varying mechanisms, leading to cessation of nerve impulses at the neuromuscular junction (NMJ). These agents are commonly utilized in the intensive care unit (ICU) or operating room (OR) to improve patient-ventilator synchrony, reduce muscle oxygen consumption, facilitate short procedures, or prevent unwanted movements in patients with elevated intracranial pressure. Available NMBAs are categorized as depolarizing or nondepolarizing agents. Succinylcholine, the only available depolarizing agent, binds tightly to acetylcholine receptors to cause sustained flaccid muscle paralysis. Nondepolarizing agents act as competitive antagonists of nicotinic receptors, blocking the action of acetylcholine. Nondepolarizing agents can be further divided into two structural groups, aminosteroids (pancuronium, vecuronium, and rocuronium) and benzylisoquinoliniums (atracurium, cisatracurium, doxacurium, and mivacurium).

Routine reversal of neuromuscular blockade (NMB) is relatively common after general anesthesia surgery in an attempt to reduce the risks of residual neuromuscular blockade, including pulmonary and respiratory complications. NMBAs may be reversed by either increasing the concentration of acetylcholine (Ach) in the synaptic junction or aiding in the elimination or metabolism of the drug. Traditional anticholinesterase reversal agents, including neostigmine, edrophonium, and pyridostigmine, compete with NMBA molecules at post-synaptic nicotinic receptors by increasing the concentration of acetylcholine through inactivation of acetylcholinesterase (AChE). Anticholinesterase reversal agents are only effective for reversal of nondepolarizing NMBAs, and are commonly administered with anti-muscarinic agents, glycopyrrolate or atropine, to offset unwanted side effects, such as nausea and vomiting, bradycardia, and increased salivary gland secretions. Additionally, it is recommended that antagonism of neuromuscular block with an anticholinesterase should not be attempted until partial muscle recovery is evident, as measured by two twitches of the train-of-four (TOF) twitch response.

Owing to the perceived limitations of anticholinesterases, there has been a quest for an ideal reversal agent. Sugammadex was developed to work independently of Ach and exhibits its effect by forming a 1:1 inclusion complex with steroidal non-depolarizing NMBAs (rocuronium, vecuronium, pancuronium), thereby removing these agents from the NMJ and facilitating the return of muscle function. Sugammadex has no effect on AChE, which obviates the need for co-administration of anticholinergic drugs. However, sugammadex is ineffective against depolarizing NMBAs (succinylcholine) and benzylisoquinoliums (atracurium and mivacurium) due to its inability to form the structural host–guest complex. Neuromuscular blockade of aminosteroids is rapidly terminated with phase III trials demonstrating the efficacy of sugammadex compared with neostigmine in antagonizing block produced by aminosteroidal NMBAs. In a randomized trial, 198 patients received sugammadex or neostigmine administered at the time when patients demonstrated two twitches on TOF monitors. Significantly faster recovery occurred after sugammadex. Median time to recovery was 1.4 (0.9–5.4) minutes for sugammadex as compared to 17.6 (3.7–106.9) minutes (P<0.0001) for neostigmine after rocuronium, and 2.1 (1.2–64.2) minutes versus 18.9 (2.9–76.2) minutes (P<0.0001), respectively, after vecuronium. Although clinically effective, concerns for routine sugammadex use include drug interactions, cost, and unknown incidence of adverse drug reactions. Currently, its use at the University of Cincinnati Medical Center (UCMC) is restricted to the indications above (figure 1).

Restricted indications for Sugammadex

- Emergent reversal of NMB in the event of loss of a difficult airway
- Reversal of NMB after conventional reversal
- Reversal of NMB for brief procedures where succinylcholine is contraindicated

Figure 1. Restricted indications for sugammedex at UCMC.

The detailed safety of sugammadex has not yet been established. Two previous systematic reviews did not include sufficient patients to be adequately powered to estimate the rates of significant adverse effects, including QTc prolongation, hypersensitivity, bronchospasm, and nausea and vomiting. Furthermore, due to its formation of ternary complexes, sugammadex may bind other drug structures within the plasma at therapeutic concentrations. For example, flucloxacillin doses greater than 500 mg should be avoided for 6 hours after sugammadex and missed dose advice should be followed in patients taking progesterone oral contraceptives who are exposed to sugammadex due to progesterone's steroid structure. Further identification of drug interactions is likely during post-market surveillance. Additional contribution to the hesitancy in sugammadex use is the estimated cost being five times that of routine neostigmine dosing as the cost is more than $100 for a single 200 mg sugammadex dose. Lastly, sugammadex does not have abundant literature supporting its use in special populations, such as the elderly and patients suffering from renal dysfunction.

Further data, including clinical outcomes and pharmacoeconomic analysis, is needed to routinely recommend sugammadex over traditional reversal agents, such as neostigmine, edrophonium, and pyridostigmine, all of which have activity against a broader class of NMBAs and are more readily available.

Agents for Reversal of Neuromuscular Blockade

	Method of Action	Indications	Dosing
Neostigmine	Acetylcholinesterase inhibitor	Reversal of nondepolarizing neuromuscular blockade after surgery	0.03mg/kg (rocuronium) to 0.07 mg/kg (vecuronium, pancuronium) Maximum total dose: 0.07 mg/kg or 5 mg (whichever is less)
Edrophonium	Acetylcholinesterase inhibitor	Reversal of nondepolarizing neuromuscular blockade after surgery	10 mg over 30 to 45 seconds; may repeat as necessary up to a maximum cumulative dose of 40 mg
Pyridostigmine	Acetylcholinesterase inhibitor	Reversal of nondepolarizing neuromuscular blockade after surgery	0.1 to 0.25 mg/kg/dose
Sugammadex	Modified gamma cyclodextrin, which forms a 1:1 complex with rocuronium or vecuronium in plasma	Routine reversal of rocuronium and vecuronium induced blockade	Moderate block (after appearance of T2): 2mg/kg IV as single dose Deep block (at least 1 to 2 post-tetanic counts but prior to appearance of T2): 4mg/kg IV as single dose

Table 1. Medications available for reversal of neuromuscular blockade, their indications, and appropriate dosing.

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Lisfranc Injuries
Continued from page 9

such as a motor vehicle collision, accounting for approximately two-thirds of all Lisfranc injuries. Certain low energy mechanisms can also result in Lisfranc injuries. A frequent reported mechanism is missing a step when coming down the stairs. Patients may also report direct trauma from a large external force that strikes the foot. As in the patient above, some patients experience an indirect force such as the foot hitting a stationary object and the weight of one's body becoming the additional force.

On physical exam, these patients usually have severe pain over the midfoot and often are unable to bear weight. One may see significant swelling throughout the midfoot or gross dorsal subluxation or lateral deviation of the forefoot. One highly specific exam finding suggestive of a Lisfranc injury is plantar ecchymosis. However, this finding is not always present in the setting of mild ligamentous strains or minor fractures.

Additionally, ecchymosis often does not appear until 24-48 hours after the injury. On rare occasions, compartment syndrome can occur due to significant swelling of the midfoot, and suspicion should be raised with pain out of proportion with passive extension of the toes. In subacute presentations, the Lisfranc joint may be dorsally subluxed with a provoked test by applying dorsal forces to the distal aspect of the midfoot while palpating the joint with the other hand.

When considering radiographic evaluation, if there is any concern for a Lisfranc injury it is important to order weight-bearing films to improve detection rates. Aggressive pain control should be pursued as obtaining true weight bearing films is often limited by patient discomfort. Anterior-posterior (AP), lateral, and oblique films should be obtained when evaluating for a Lisfranc injury.

On a normal foot x-ray, the medial cortex of the second metatarsal should form a continuous line with the medial cuneiform. Likewise, the intermetarsal joint space should be less than 2 mm. Dislocation or fracture of the Lisfranc joint complex should be suspected with a number of radiographic findings.

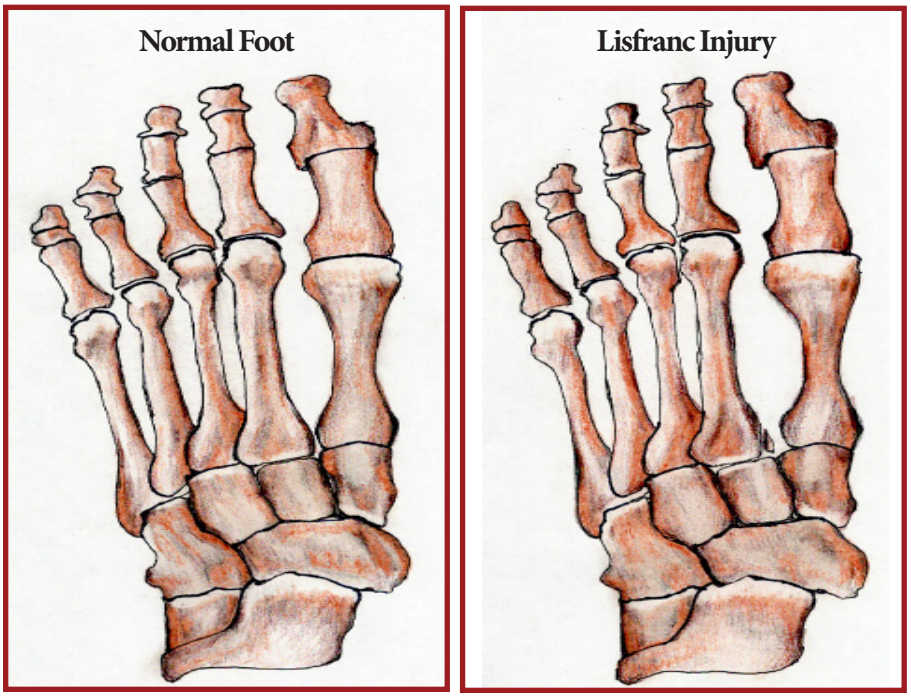

Figure 3: Note the loss of linearity of the second metatarsal and cuneiform, widening of the intertarsal space, and the "fleck sign" on the Lisfranc injured foot.

The AP film may show: 1) loss of linearity of the medial border of the second metatarsal with the medial border of the middle cuneiform; 2) increased intertarsal space greater than 2.7 mm; or 3) presence of a "fleck sign" at the base of the second metatarsal, which is pathognomonic for an avulsed or disrupted Lisfranc ligament.

On the oblique film, loss of normal alignment of the second through fourth tarsal-metatarsal joints will often be seen. Finally, dorsal displacement of the base of the second metatarsal may be evident on a lateral view of the injured foot.

If x-ray findings are not present, but clinical suspicion remains high, CT should be considered as even weight bearing radiographs have limited sensitivity and specificity for detecting Lisfranc injuries. One small observational study showed a miss rate of approximately 50% with weight bearing films. More recently CT has become the gold standard in imaging modalities as it demonstrates much higher detection rates. If the x-ray is diagnostic for Lisfranc injuries, a CT is not mandatory in the ED setting, although it can help with surgical planning.

Although this diagnosis does not often require emergent intervention, it is important that these patients receive timely follow up. Missed injuries can lead to osteoarthritis or debilitating deformities of the midfoot. Lisfranc injuries that would require urgent evaluation include open fractures, fracture patterns in which the soft tissue may be compromised and in danger of necrosis, or any concern for compartment syndrome.

Otherwise, it is recommended that all patients with evidence of a Lisfranc injury, whether minor or major, be evaluated by an orthopedist within one to two weeks to prevent these complications. All patients should be placed in a posterior slab splint, be made non-weight bearing, and instructed to ice and elevate the extremity.

Foot pain is a common ED complaint and Lisfranc injuries can be difficult to diagnose. Knowledge of the clinical presentation and management of these injuries can help prevent missed diagnoses and chronic complications.

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Title image obtained from https://pixabay.com/en/feet-foot-pies-man-person-160445/

Acute HIV
Continued from page 7

PEP is estimated to cost between $600 to $1000 for the full course. Understandably, this can lead to treatment delays in patients who are unable to afford these medications, such as in the presented case. Due to the Ryan White CARE Act which was first passed in 1990, government funding is allocated to providing healthcare for underinsured patients with HIV. Many programs exist that provide financial assistance for patients needing these medications. Therefore, providers are encouraged to discuss this with their patients and consider involving pharmacy and social work if financial needs are identified. Numerous provider resources regarding the initiation of PEP are available, including the national PEP hotline at UCSF.

Testing for HIV Infection

In order to understand options for HIV testing, it is essential to review the natural history and pathogenesis of the virus. HIV RNA can be detected in the serum early in the course of infection. One to three days later, p24 antigen and antibodies specific for HIV can be detected via enzyme-linked immunosorbent assay (ELISA). With the development of fourth-generation testing, the CDC released updated guidelines for HIV laboratory testing in 2014 (figure 4).[12] These recommendations include initial testing with HIV antigen/antibody combination immunoassay. This study identifies HIV-1 and HIV-2 antibodies, as well as p24 antigen to detect chronic and acute infections. These tests can distinguish acute infection as early as 10 days after HIV exposure. If positive, secondary testing should include antibody immunoassay to differentiate HIV-1 and HIV-2 antibodies. Specimens that are positive on the antigen/antibody combination immunoassay and negative or indeterminate on the antibody immunoassay should undergo HIV-1 nucleic acid testing. Following a diagnosis of HIV, more advanced testing, such as genotyping, will likely be required. This is used by infectious disease specialists to help tailor treatment regimens. For patients who participate in high-risk behaviors and test negative for HIV, follow up with primary care providers should be encouraged.

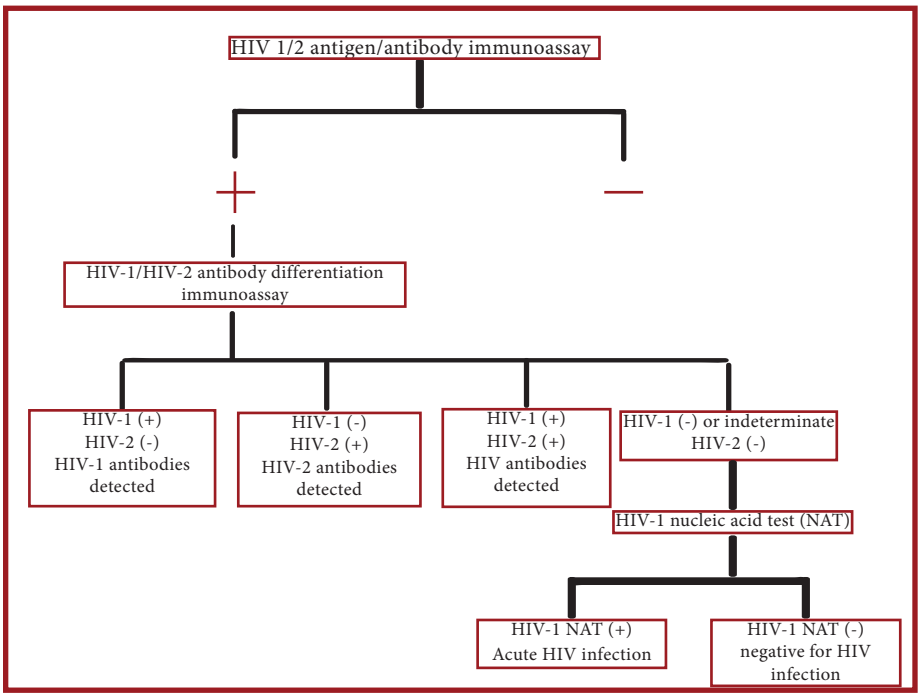

Figure 5: Flowsheet for HIV testing.

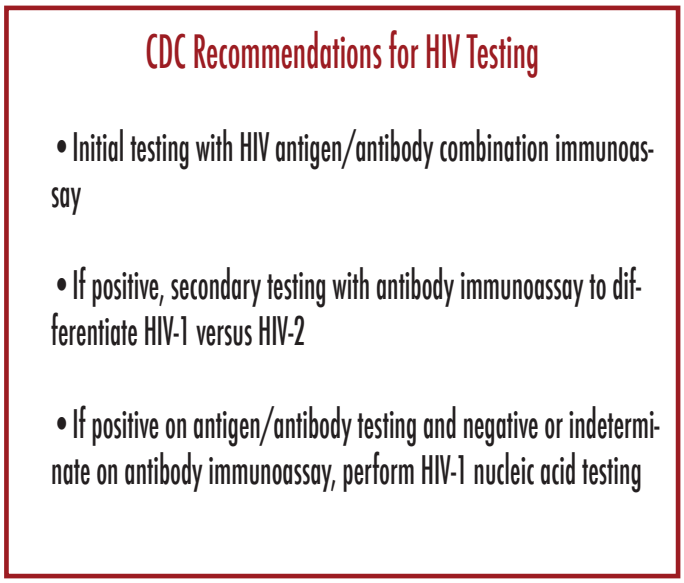

Figure 4: CDC recommendations for HIV testing

Ultimately, HIV, in both acute and chronic infection, is a medical problem that will continue to be seen in EDs across the country. Understanding the initial testing and management of patients with suspected acute HIV infection, as well as the importance of preventing infection following high-risk non-occupational exposures, is critical for emergency medicine physicians.

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To Cric or Not to Cric? Continued from page 3

his RUE secondary to the vertebral artery occlusion and ischemia. He was discharged to an inpatient rehab facility.

Discussion

Emergency providers are tasked with airway management on a daily basis, with the vast majority of intubations being successful on the initial attempt. Surgical cricothyrotomy is a rescue airway procedure that many hope to never have to perform, but can be life-saving when necessary. Indications for cricothyrotomy include the failure to oxygenate, failure to ventilate, or the inability to otherwise manage the airway. Success rates for surgical cricothyrotomy vary, but are generally high.[1] Details on the indications and technique of the procedure can be found in countless sources.[2] Because it is not commonly performed, providers often practice this procedure via simulations and with mental exercises. Nonetheless, these scenarios are often unrealistic when compared to the high stress situation of having to perform a cricothyrotomy on a live patient. Therefore, learning from those who have experience with this procedure can help providers prepare for the anxiety-provoking situation of the failed airway. The purpose of this discussion is to outline several practical considerations of the procedure based on the presented case.

Cricothyrotomy truly is a blind procedure.

The anterior neck has numerous vascular structures. Following the initial incision, blood often obstructs any further visualization of the surgical field. This makes it imperative that the provider can perform the procedure using tactile sensation only. Prior to incision, visual assessment can be performed and is helpful to locate landmarks. However, after the incision, landmarks must be palpated to identify the location of the cricothyroid membrane and allow for successful completion of the procedure.

The trachea is surprisingly mobile.

The trachea easily slides laterally during this procedure. Because of this, it is pivotal to have a firm grip on the trachea with the provider's non-dominant hand to stabilize it while making the incision to expose the cricothyroid membrane. It is easy to lose the landmarks if the trachea is not properly stabilized. Additionally, while making the horizontal puncture incision in the cricothyroid membrane, stability is needed to ensure that the incision is in the proper location.

Monitor the endotracheal tube depth.

The distance from the vocal cords to the carina is approximately 10 cm in an adult. The distance from the vocal cords to the cricothyroid membrane is approximately 1-2 cm. Therefore, the distance from the cricothyroid membrane to the carina is approximately 8 cm.[3] When inserting the endotracheal tube, stop as soon as the balloon disappears into the trachea. This will minimize the risk of a mainstem intubation. Be aware that the tube can easily migrate and needs to be reassessed frequently. In this case, there was difficulty oxygenating in the ED due to a mainstem intubation which was likely due to migration of the tube during transport.

Communicate with your team.

Difficult airways can be stressful for the provider, the team, and the patient. Vocalizing the airway plan for all involved is helpful to ensure that all team members know their roles and the plan going forward. Lack of knowledge that the backup plan involves a surgical airway can increase anxiety among the team members involved and lead to unnecessary errors or delays. In the case outlined above, each provider knew the initial plan and was aware of the alternative which helped to facilitate a seamless transition from the failed awake look attempt to the definitive surgical airway.

In summary, performing a surgical cricothyrotomy is one of the most stressful situations that emergency providers face. Realistic simulation for this procedure is difficult to replicate. Therefore, learning from the experience of others can help future providers save the life of the patient that cannot oxygenate, cannot ventilate, and cannot intubate.

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Measurement of Intra-abdominal Pressure Continued from page 8

8. Clamp the drainage bag of the Foley catheter just distal to the culture aspiration port using the Kelly forceps (figure 3, adjacent page).

9. Instill 60 mL of sterile saline into the bladder via the stopcock. Turn the stopcock off to the syringe and open to the patient upon completion of this.

10. Briefly unclamp the distal Foley to allow all air in the proximal collection tubing to pass into the distal tubing, then reclamp.

11. Allow 60 seconds for detrusor muscle relaxation prior to obtaining measurements.

12. The bladder pressure should be taken at end-exhalation with the patient completely flat in the supine position. Ensure a good waveform prior to recording the measurement.

13. Unclamp Foley when complete and remove pressure transducing tubing.

14. If strict measurement of urine output is necessary for the patient, subtract 60 mL from the total urine output.

Normal intra-abdominal pressure is less than or equal to 5 mmHg. Intra-abdominal hypertension occurs at pressures greater than 12 mmHg, while abdominal compartment syndrome is diagnosed at pressures greater than 20 mmHg in the setting of organ dysfunction. Ideally, bladder pressures should be obtained in paralyzed patients in order to ensure complete relaxation of the abdominal muscles. However, this is often unrealistic and not feasible. With appropriate instruction, accurate numbers can be obtained in alert patients. Complete supine positioning is also recommend-

ED treatment of abdominal compartment syndrome primarily involves recognition of the disease process and resuscitation of these often critically ill patients. Unfortunately, excessive fluid resuscitation can lead to increased intra-abdominal pressures due to third spacing. Some sources report that iatrogenic abdominal compartment syndrome can occur with only 5-7 liters of crystalloid in the first 24 hours of presentation. Therefore, judicious fluid administration is recommended. Recent studies suggest that targeting certain abdominal perfusion pressure goals may help guide resuscitation.[11] While not yet prospectively validated, maintaining an abdominal perfusion pressure of 50-60 mmHg has been shown to be associated with increased survival in these patients.

As vascular flow to the intestines is compromised due to increasing abdominal pressures, bacterial translocation can occur. Consequently, patients with abdominal compartment syndrome are at increased risk of intra-abdominal infections. ED providers are therefore encouraged to consider empiric antibiotic coverage for abdominal flora. While awaiting definitive intervention, gastric and colonic decompression can be performed by placement of nasogastric or rectal tubes. Likewise, if the patient has abdominal ascites, paracentesis can be used to help alleviate the pressure.[12]

Surgical management and decompression is completed by laparotomy. Practice patterns vary, with some surgeons waiting for intra-abdominal pressure to exceed 20 mmHg, whereas others will perform surgery if there is any end-organ damage present, or if the abdominal perfusion pressure is reduced. Temporary abdominal closure is common to prevent recurrence, with patients typically being brought back to the operating room within 48-72 hours to attempt primary closure.

Abdominal compartment syndrome should be suspected in patients presenting critically ill with a distended abdomen and any of the possible etiologies listed above. Diagnosis is made by measurement of the intra-abdominal pressure using an intra-vesicular pressure transducer, with a pressure greater than 20 mmHg and associated end-organ damage. Definitive management is by surgical decompression. Morbidity and mortality from abdominal compartment syndrome is high, with one prospective observational study finding a 36% mortality rate at one month, increasing to 55% at one year.[13] While abdominal compartment syndrome is rare in the ED, mortality increases with time, making the interventions by the ED provider crucial in improving patient outcomes.

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ed, as measurements will be artificially elevated if the head of bed is at all elevated. Ultimately, it can be helpful to obtain serial measurements using the same technique and positioning in order to assess for clinically significant changes in intra-abdominal pressures and to help guide response to treatment.

1. Delaet I, Vianne D, Vermeiren G, Schoonheydt K, et al. Effect of head of bed elevation on intra-abdominal pressure estimated via bladder and stomach: Preliminary results on the validation of the gastromanometer. Acta Clinica Belgica 2007; 62(Supplement 1); 259.
2. Intra-abdominal pressure monitoring. Department of Surgical Education, Orlando Regional Medical Center 2008. http://www.surgicalcriticalcare.net/Guidelines/intraabdominal_pressure_monitoring.pdf. Accessed 4/24/17.
3. Malbrain ML, Deeren DH. Effect of bladder volume on measured intravesical pressure: a prospective cohort study. Critical Care 2006; 10:R98.

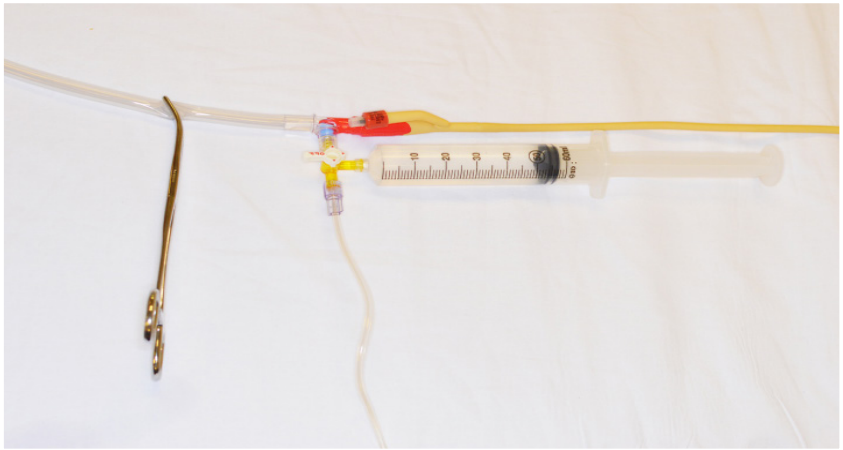

Image 3: Clamp the Foley just distal to the aspiration port

de Winter's

EKG focus

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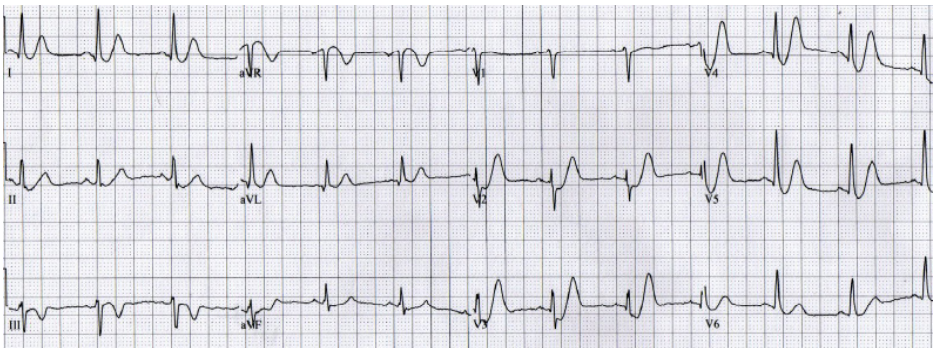


Figure 1. EKG showing classic de Winter morphology. Note the J-point depressions and symmetrical appearing T-waves.

History of Present Illness

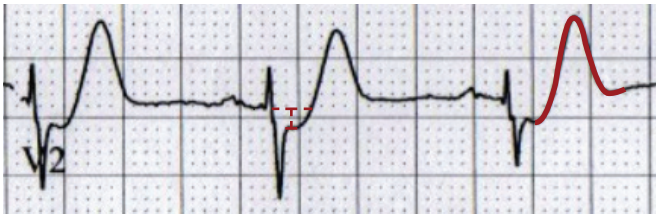
A middle-aged man with extensive family history of atherosclerotic disease presents to the emergency department with "crushing" precordial chest pain, diaphoresis, and shortness of breath. The patient's evaluation with electrocardiography was notable for J-point depressions in the precordial leads with large, sweeping T waves. His serum troponin was also notably elevated at 1.2 ng/mL. The patient was emergently taken to the cardiac cath lab, where coronary angiography demonstrated complete occlusion of the proximal left anterior descending artery.

Epidemiology of de Winter Morphology

de Winter morphology was identified in a 2008 case series by cardiology Dr. Robbert de Winter as a new EKG sign suggestive of LAD occlusion. de Winter's categorized his findings as 1-3 mm J-point depressions with large, peaked, symmetrical T waves found in the precordial leads (V1-V6).

A subsequent case series by Verouden (2009) reported similarly findings. Based on their observational study, de Winter morphology was found in roughly 2% of patients with acute proximal LAD occlusion. According to their epidemiological data, de Winter morphology was most common in young men with a history of hypercholesterolemia.

EKG Features of de Winter's



J-Point Depressions

1-3 mm J-point depressions are typically observed in the precordial leads (V_1-V_6). The J-point is defined as the point of transition from the QRS complex to the ST segment.

Symmetrical T-waves

Large, symmetrical T-waves are frequently observed concomitantly with J-point depression. The size of these T-waves is often quite striking and is one of the defining features of de Winter's morphology.

Management of de Winter's

Though formal recommendations have yet be to adopted, both studies recommended that de Winter morphology be treated as a STEMI-equivalent and that its identification prompt rapid cardiology consultation for definitive treatment with coronary angioplasty or thrombolysis.

EKG and Case referred by

Edward Otten, MD
University of Cincinnati
Professor Emeritus

1. Winter, R. J., Verouden, N. J., Wellens, H. J., & Wilde, A. A. (2008). A New ECG Sign of Proximal LAD Occlusion. New England Journal of Medicine, 359(19), 2071-2073. doi:10.1056/nejmc0804737
2. Verouden, N. J., Koch, K. T., Peters, R. J., Henriques, J. P., Baan, J., Schaaf, R. J., . . . Winter, R. J. (2009). Persistent precordial "hyperacute" T-waves signify proximal left anterior descending artery occlusion. Heart, 95(20), 1701-1706.

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

Please submit cases via EPIC In Basket message to Dr. David Habib. Make sure to include the R1/R4 involved in the case.

List of Submitted B Pod Cases

Case	Case Physicians
Tuberculous pericardial effusion	Jarrel/Kircher
Lung abscess resulting from dental abscess	Owens/Devries
Infected renal cyst	Spigner/Riddle
Ludwig's angina	Bernardoni
Penetrating atherosclerotic ulcer	Blanton/Kircher
Non-traumatic diaphragmatic hernia	Harty/McKean
High pressure finger injury	Woeste/LaFollette
Stokes-Adams syndrome	Owens/Golden/Mudd
New diagnosis of non-small cell lung cancer	Owens/Thomas