University of Cincinnati Medical Center
Department of Emergency Medicine

Annals of B Pod

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Take a Look...
#beneaththesurface

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EMERGENCY MEDICINE
ANNALS OF B POD
SUMMER 2016

#beneaththesurface

It's that time: the academic year has come to a close. Interns are spending their last few shifts in B Pod, and the R2s are testing their taming skills in the SRU. The R3s are stepping up to the educator role, and the R4s are looking onwards to their future careers. We spend these last few weeks balancing the excitement of our transitions and the purpose of our job: somewhere between the hustle and noise of B Pod, there is a critical illness waiting to be found, #beneaththesurface.

In this issue, we look at the difference between how something appears on the surface and its true nature. A simple DVT hides something more. A chronic wound takes a patient to the ICU. Our patients challenge us every day to keep our skills fresh and our index of suspicion high, because #beneaththesurface, your patient may be hiding something more.

Mycotic aneurysm:
A rare cause of lower extremity DVT

Robert Whitford, MD
University of Cincinnati R1

History of Present Illness

The patient is a middle-aged male with a past medical history of a recent right popliteal deep vein thrombosis who presents with worsening pain and swelling of his right lower extremity.

The patient was seen three days ago at an outside hospital for acute onset right lower extremity pain and swelling. A venous doppler was performed and showed a right popliteal deep vein thrombosis. He was started on rivaroxaban and discharged. He presented to our ED for worsening pain and swelling. He reports taking his rivaroxaban as prescribed as well as oxycodone, but states that despite this the pain is worsening and preventing him from sleeping. He denies chest pain, shortness of breath, numbness, weakness, fevers, or cough. He has no history of prior deep vein thrombosis or pulmonary embolisms. He denies a personal history of cancer or recent unintentional weight loss. He smokes one pack per day, reports moderate alcohol use, and admits to injecting IV drugs, although denies use in the last three years.

Past Medical History	Past Surgical History	Medications
None	None	Rivaroxaban

Physical Exam

T100.2 HR 105 RR 16 BP 153/95 O2 Sat 97%

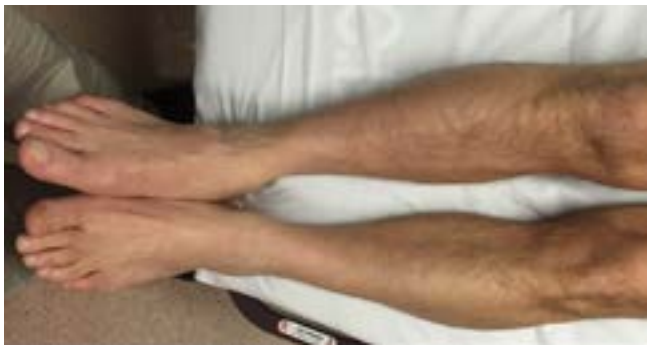

Image 1: Venous distention in the right lower extremity of a patient previously diagnosed with a DVT.

The patient is an alert, well-developed male in no acute distress. He is tachycardic but has a normal S1/S2 with no murmurs, rubs or gallops. He has clear and unlabored breath sounds bilaterally. His neurologic exam is normal. His skin exam reveals no rashes or lesions. He has 2+ pulses in all four extremities. His right lower extremity is notable for 1+ non-pitting edema from the shin to the mid-thigh with visibly dilated superficial veins without erythema when compared to the left lower extremity (Image 1).

Labs

WBC: 13.9 H/H: 11.6/35.7 INR 1.8
BMP: 131/4.0/97/27/17/0.8/129

Hospital Course

Given the patient's worsening symptoms despite being on appropriate medical therapy for his known deep vein thrombosis, the decision was made to obtain a repeat right lower extremity venous ultrasound to evaluate for proximal spread of the clot. The ultrasound was concerning for a mid to distal thigh pseudo-aneurysm, as well as an acute occluding deep vein thromboses of the right popliteal, gastrocnemius, soleal, posterior tibial, and peroneal veins.

To better characterize this vascular abnormality, a CT angiogram of the lower extremities was ordered. It showed a large pseudo-aneurysm or contained rupture of the distal right superficial femoral artery measuring up to 2.8 cm (Image 2). Vascular surgery was consulted and they admitted the patient with plans for operative repair.

Shortly after admission, the patient developed a fever to 102.2 F. Blood cultures were drawn and he was started on vancomycin and ampicillin/sulbactam. The patient was taken to the operating room on hospital day 1 and was found to have a 3 cm ruptured mycotic right popliteal artery aneurysm. This was repaired and the patient underwent a femoral to below knee popliteal bypass graft. Intraoperative culture swabs of the aneurysm were negative for bacteria, fungus, anaerobes, and mycoplasma. Ultimately, the aneurysm was believed to be secondary from injecting intravenous drugs.

During his hospital course he had a negative workup for endocarditis including a normal trans-throracic echocardiogram and negative serial blood cultures. He was discharged on hospital day 10 with a PICC line for six weeks of antibiotics.

Discussion

When diagnosing a patient with a deep vein thrombosis, the provider should attempt to determine if the clot was provoked or unprovoked. A provoked deep vein thrombosis should have an inciting factor such as prolonged immobility, recent surgery, trauma, vascular anomalies, or active malignancy. A deep vein thrombosis without a clear inciting factor is classified as unprovoked, and may merit further evaluation for a primary clotting disorder such as protein C or S deficiency, anti-thrombin deficiency, or factor V Leiden deficiency. Select patients with unprovoked deep vein thrombosis may merit further work up for an occult malignancy.

In our patient, the cause of his deep vein thrombosis was likely compression of the popliteal vein by his mycotic right popliteal artery aneurysm. Causes of arterial aneurysms include atherosclerosis, hypertension, trauma, congenital anomalies, infection, and syphilis.

The term mycotic aneurysm was coined by William Osler in 1885 to describe the mushroom shaped aneurysms associated with bacterial endocarditis. Today, the term is used more generally to refer to all aneurysms of infectious etiology. Popliteal vein thrombosis secondary to popliteal artery aneurysm is most often seen in patients with aneurysms due to atherosclerosis.[2] Although less frequent, they have also been seen secondary to mycotic aneurysms. Mycotic aneurysms are an uncommon but known complication of IV drug abuse with a rate amongst IV drug users of 0.14%.[4]

Mycotic aneurysms are most commonly caused by septic emboli from endocarditis. However, they have also been observed in IV drug users without endocarditis and are thought to be due to direct needle trauma and inoculation of the vessels. They have been described in the popliteal, femoral, radial, brachial, and subclavian arteries, as well as intracranially.[5] The predominant causative organism is methicillin resistant Staph aureus, but other organisms have been identified, including Strep viridians, Staph epidermis, Campylobacter, E. Coli, and Mycobacterium tuberculosis.[6, 7] Diagnosis of popliteal arterial aneurysms can be difficult. Findings may include a pulsatile popliteal fossa mass or swelling, fever, leukocytosis, limb ischemia, DVTs, elevated ESR or CRP, and foot drop.[8] Complications include aneurysm rupture, thrombosis, embolization, compartment syndrome, limb ischemia, need for amputation, and death from exsanguination.[9]

In summary, when evaluating a patient with a deep vein thrombosis, careful attention should be paid to eliciting a provoking factor, as venous obstruction from an unrecognized arterial aneurysm is associated with high morbidity and mortality. IV drug use may lead to the development of vascular abnormalities that provoke deep vein thrombosis. In light of the ongoing national opioid epidemic, providers should be aware of the risk of mycotic aneurysms in IV drug users and their potential to provoke deep vein thrombosis. Vascular surgery consultation is integral to the management of these patients.

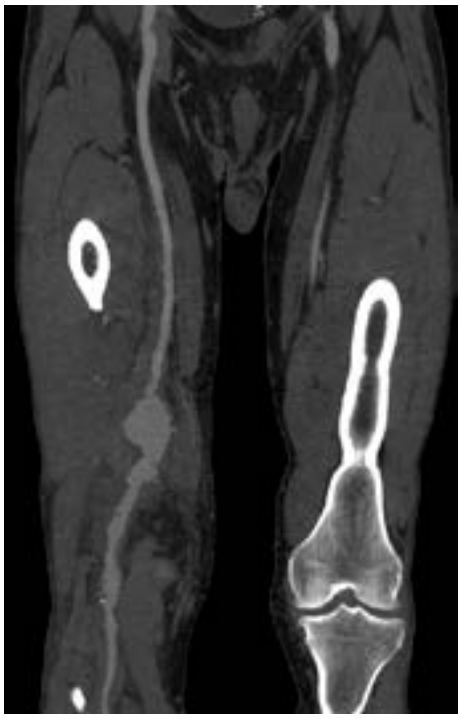

Image 2: Mycotic aneurysm of the right distal superficial femoral artery.

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#bpodcase

Necrotizing Fasciitis

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History of Present Illness

The patient is a female in her 50s with history of hypertension, insulin-dependent type 2 diabetes mellitus, and hyperlipidemia who presented with bilateral lower extremity swelling and right foot pain. The patient states that approximately two weeks ago she began noticing swelling in her feet. Since then, she has noted worsening edema and redness of the right foot that now extends up her leg. The patient also remarks that she noticed some bleeding from her right heel with worsening pain in the right foot and lower leg. She has still been able to ambulate despite these symptoms, but does note that she is generally very sedentary. The patient does not know how long she has had the wound on her foot.

She endorses some numbness in her bilateral feet, but states that this has been a chronic problem for her that has not worsened during this current illness. She denies fever, chills, chest pain, shortness of breath, cough, abdominal pain, nausea, vomiting, diarrhea, or dysuria. The patient admits that she has stopped taking all of her medications for at least one month, including her insulin.

Past Medical History

Hypertension, Type 2 Diabetes mellitus, Hyperlipidemia

Past Surgical History

Hysterectomy

Social History

Occasional alcohol use

Allergies

None

Medications

None

Physical Exam

The patient is an alert, obese, unkempt female in no apparent distress. Her pupils are equal, round, and reactive. Her mucous membranes are moist. She has clear breath sounds with good air entry bilaterally and normal respiratory effort. Her cardiovascular exam reveals a normal rate and regular rhythm with no murmurs, rubs, or gallops. She has 2+ dorsalis pedis and posterior tibial pulses bilaterally. Examination of her lower extremities reveals bilateral non-pitting edema to the level of the mid-shin, greater in the right leg than the left. She has tenderness over the right heel, dorsal right foot, and right shin. There is warmth and erythema of the right foot extending up to the level of the mid-calf and shin. There is a large ulceration encompassing the entire right heel with a considerable amount of eschar. There is no palpable crepitus or purulent drainage. She has 5/5 strength in the bilateral lower extremities.

Lab Work-up

Glucose 657, Na 125, BUN 53, Cr 2.98, Anion Gap 19, Lactate 2.8.
pH 7.34, pCO2 35, HCO3 19
WBC 24.2, Neutrophils 92.3%
CRP 373.5, ESR 74. CK 262

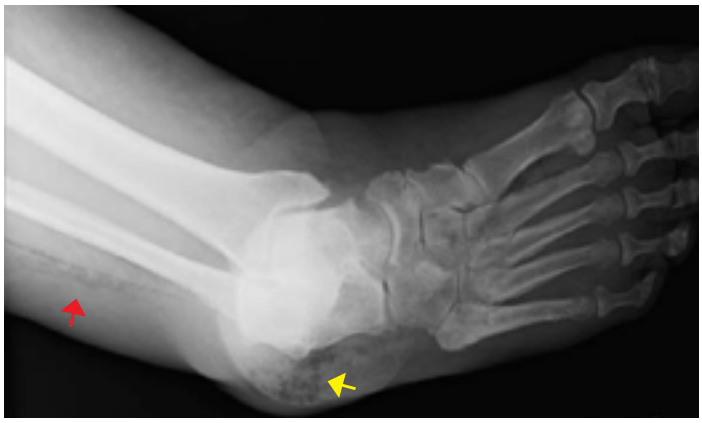

Image 1. Right ankle xray showing multiple foci of subcutaneous gas within the ankle (yellow arrow) and subcutaneous gas tracking up the leg (red arrow).

Hospital Course

The patient's history, exam and imaging studies (Image 1) were most consistent with a diagnosis of necrotizing fasciitis. In addition, her laboratory studies indicated that she was in DKA with an AKI. She was resuscitated with normal saline and started on broad-spectrum IV antibiotics (piperacillin-tazobactam, vancomycin, and clindamycin). The patient was admitted to the SICU where a central line was placed and she was started on an insulin drip for her DKA. She then underwent a right below knee amputation, which was extended to an above knee amputation two weeks later. Wound culture and blood cultures grew MSSA. She completed a 10-day course of doxycycline and ceftriaxone. Her DKA resolved, but her renal function did not recover. It was believed patient likely had underlying chronic kidney disease and she was ultimately started on intermittent hemodialysis that was continued after discharge. After a 3 week hospitalization, she was discharged to a SNF where she continued her antibiotics to complete a 4 week course.

Discussion

Necrotizing fasciitis is a deadly diagnosis. Fortunately, it is an extremely rare condition, with an estimated annual incidence of only 1000 cases in the United States.[1] It is a rapidly progressing infection of the subcutaneous soft tissues with a significant mortality rate of 20% or higher. Untreated, its mortality rate approaches 100%. As such, it is crucial that EM physicians recognize the early signs of necrotizing fasciitis and initiate appropriate treatment.

Although necrotizing fasciitis can occur anywhere on the body, the most common location is the lower leg. Patients at increased risk for this clinical presentation include those with diabetes, vascular insufficiency, and immunosuppression. In 80% of cases, necrotizing fasciitis is the result of direct extension from a skin lesion. The type of skin le-

sion can be quite variable and includes penetrating trauma, surgical incisions, diabetic eschars, pressure ulcers, IV drug injection sites, burn injuries, varicella infection, and injuries from childbirth.[2] When infection occurs in the perineal, genital, or perianal regions, it is referred to as Fournier gangrene. This is more commonly seen in males, diabetics, the immunosuppressed, and chronic abusers of alcohol. [3]

Once bacteria have breached the skin, infection sets in involving the superficial fascia. The bacteria produce and release proteins, enzymes, and exotoxins that trigger an inflammatory response and cause breakdown and necrosis of the fascial layers. Exotoxins also inhibit neutrophils which can facilitate bacterial growth. As the bacteria multiply, they spread along the fascial planes, which is why initially there may not be significant overlying skin changes. The bacterial enzymes and toxins can result in thrombosis of the arteries and veins, causing tissue ischemia and further necrosis.[4]

There are three main types of necrotizing fasciitis. Type I is polymicrobial and is usually seen in the elderly, diabetics, or those with chronic medical illnesses. This typically affects the trunk and perineum. Often there is no history of trauma, but rather breakdown in the skin secondary to pre-existing abscesses, perforations or bacterial translocation from the GI or GU tracts. Type II is caused by Group A strep or staphylococci and is commonly referred to as "flesh-eating disease." Type III is due to Clostridia perfringens and is commonly called "gas gangrene." Some reports also describe a Type IV that is caused by fungal infections (candida and zygomecetes), but this is by far the least common. There is no significant difference in the clinical course, morbidity, or mortality between these different types. For EM physicians, timely recognition and treatment is more important than identifying the type of infection or the causative agent.

Early in the disease course, symptoms may be vague with pain in the affected area and possibly some associated flu-like symptoms (e.g., fever, nausea, general malaise). Within several hours to days, the site will start to swell and may develop a purplish/reddish rash. Distinguishing between necrotizing fasciitis and simple cellulitis can be challenging. However, there are some findings that are suggestive of more serious infection, including systemic toxicity, severe pain, pain out of proportion to exam findings, and crepitus. Alternatively, there may be the absence of pain once necrosis destroys the peripheral nerves. Unfortunately, these findings can be nonspecific and have low sensitivity. Although subcutaneous air and crepitus are traditionally taught to be classic findings, they are rarely seen. Quite possibly the most important information a physician can obtain on initial history and physical exam is the chronicity. Unlike simple cellulitis, necrotizing fasciitis usually advances at a much more rapid pace. Significant progression will be noted over the order of several hours rather than several days.

There have been studies to look at more objective findings for predicting the diagnosis of necrotizing fasciitis. In two studies by Wall et al., they demonstrated that a white blood cell count <15,000 cells/mm^3 and a sodium level >135 mmol/L had a 99% negative predictive value and 90% sensitivity for ruling out necrotizing fasciitis.[5, 6]

CRP (mg/L) ≥150: • 4 points
WBC count (×10^3/mm^3): • <15: 0 points • 15–25: 1 point • >25: 2 points
Hemoglobin (g/dL): • >13.5: 0 points • 11–13.5: 1 point • <11: 2 points
Sodium (mmol/L) <135: • 2 points
Creatinine (mg/dL) >1.6: • 2 points
Glucose (mg/dL) >180: • 1 point

Table 1: Laboratory Risk Indicator for Necrotizing Fasciitis (LRINEC) Score Parameters. Wong et al, 2004.

A few years later, Wong et al. proposed a scoring tool for predicting whether a patient was likely to have necrotizing fasciitis. Known as the Laboratory Risk Indicator for Necrotizing Fasciitis (LRINEC), it factors in CRP, WBC count, hemoglobin, serum sodium, creatinine, and glucose, assigning point values as shown in Table 1. Ninety two percent of patients with a LRINEC score of six or more were found to have true necrotizing fasciitis. Furthermore, of all the patients with a total score of less than six, only 4% had necrotizing fasciitis.[7] The disadvantage of this tool that clinicians must bear in mind is that it is derived from a single study and has never been validated. It is plausible that other conditions could produce the same abnormal lab values, and this makes it especially difficult to apply in conditions where other inflammatory states are present. As such, while this can be a useful tool to take into consideration in a diagnostic dilemma, it should be interpreted with caution within the context of the entire clinical picture and should not be the sole determinant in clinical decision-making. Ultimately, a high index of clinical suspicion is necessary to initiate a broader workup for patients at risk for necrotizing fasciitis.

Once the clinician has determined that there is significant concern for possible necrotizing fasciitis, the most important immediate actions are to consult surgery and start empiric broad-spectrum antibiotic coverage. Imaging studies may be obtained but there are two disadvantages to doing so. First, if obtaining the imaging creates any delay to antibiotic administration or surgical management, they are more likely to be of more harm than utility. Second, there are no well-designed or adequately powered studies to compare what is the best imaging modality to aid in this diagnosis. Plain films are the most commonly obtained to assess for subcutaneous gas; however, despite this being a specific finding, it is not particularly sensitive and cannot be used to definitely rule out necrotizing infection.

The initial choice for antibiotics should include coverage of gram-positive, gram-negative, and anaerobic organisms. A typical regimen includes vancomycin and clindamycin, along with a broad-spectrum β-lactam (*i.e.*, piperacillin-tazobactam or ampicillin-sulbactam). Intravenous immunoglobulin (IVIG) has been proposed as an adjunctive therapy, with the theory that it binds exotoxins and super antigens produced by staphylococcal and streptococcal bacteria, thereby limiting the systemic inflammatory response.[8] The evidence that exists supporting the use of IVIG comes mostly from underpowered and non-randomized studies and there have been no randomized controlled trials that have confirmed its benefit.[9] There does not appear to be significant harm associated with its use, however, and may be worth consideration in a critically ill patient who continues to deteriorate despite appropriate antibiotics and large surgical debridement.

Ultimately, the most important interven-

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#bpodcase

Erythroderma

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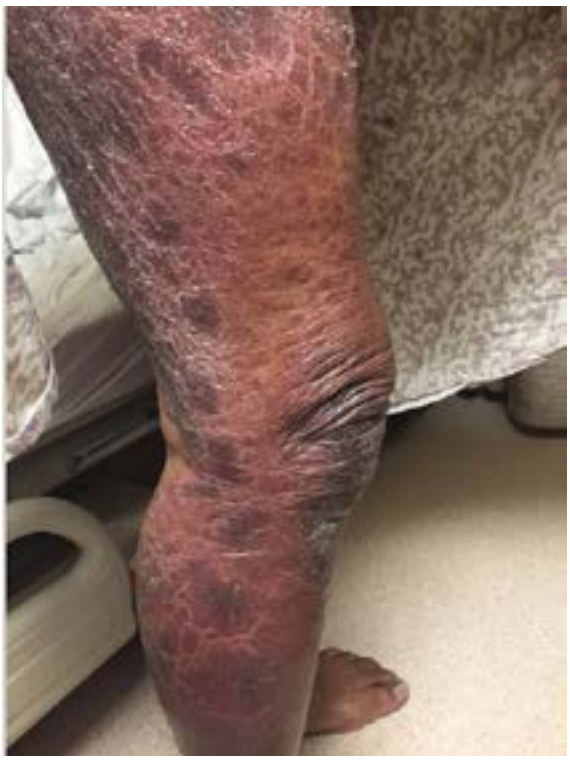

Image 1: The classic lesions of erythrodermic psoriasis.

History of Present Illness

The patient is an African-American male in his 70s with a history of psoriasis presenting with spreading redness and scaling. The patient states that approximately five months ago he began to have diffuse scaling that has now spread throughout the majority of his body, including his hairline and ears. He reports malaise, chills, and increased thirst for one week. He also reports some pain in his right elbow with movement, which he describes as a prickling sensation of the skin. He was prescribed triamcinolone 0.1% ointment initially with no improvement. Antipruritics and T-gel shampoo were added shortly after this with minimal relief, as well as a trial of methotrexate (MTX). He denies fever, peripheral edema, cough, dysuria, hematuria, melena, or joint swelling.

Past Medical History

Diabetes mellitus, Hypertension, Hypercholesterolemia, Gout, Psoriasis, Chronic kidney disease

Allergies

Aspirin

Social History

Denies tobacco, alcohol or drug use

Medications

Allopurinol, colchicine, hydrochlorothiazide, lisinopril, metformin, methotrexate, nifedipine, saxagliptin, simvastatin

Physical Exam

T 98.3 HR 105 BP 179/104 RR 18 SpO2 99%

Exam reveals a well-nourished male, appearing his stated age and in no apparent distress. He is borderline tachycardic, normal S1/S2 with 2+ pulses in all extremities. Lung sounds are clear without focality. MSK exam reveals no peripheral edema, focal tenderness or joint swelling. He is alert and oriented with intact cranial nerves and grossly normal strength and sensation in all extremities. Skin exam is remarkable for numerous, erythematous, thick scaling plaques with desquamation involving the scalp, ears, neck, chest, abdomen, back and all extremities. There is involvement of >80% of the total body surface. There is one small crack in the skin in the right posterior flank approximately 2 cm in length.

Hospital Course

The patient received 1L NS in the ED for a mildly elevated lactate of 3.5 and new acute kidney injury with a creatinine of 2.8. His tachycardia responded appropriately to the fluids. He was started on 125cc/hr maintenance IV fluids for the next 48 hours, which brought his creatinine back to his baseline of 1.3. The patient then transitioned to oral fluids only and maintained his baseline creatinine.

Dermatology was consulted from the ED. In addition to analgesia and hydration, they recommended admission to medicine for erythrodermic psoriasis. The patient underwent a daily regimen of a warm shower, treatment with triamcinolone 0.1% ointment to all affected skin areas and Kerlix wrap over the ointment. He was instructed to continue the full body treatments three times daily until he followed up with his primary dermatologist several days after discharge. He received hydroxyzine for pruritus. Systemic therapy was held until he was screened for Hepatitis B, C and tuberculosis, all of which returned negative. As an outpatient, he began systemic therapy with ustekinumab, a monoclonal antibody indicated for severe plaque psoriasis. He reported significant improvement on his most recent outpatient visit.

Discussion

Psoriasis is an immune-mediated chronic skin disorder most commonly characterized by well-demarcated erythematous plaques with silver scale. Erythroderma is a rare dermatologic condition with an incidence of 1/100,000 annually. A potentially life-threatening condition,

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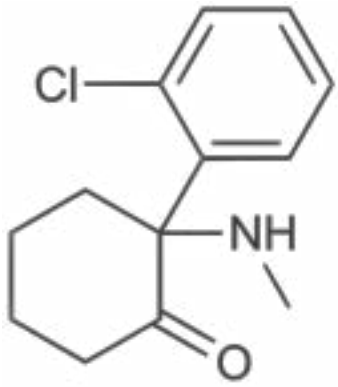
#pharmconsult

Continuous Ketamine



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Edited by: Madeline Foertsch, PharmD, BCPS and Nicole Harger, PharmD, BCPS

Ketamine hydrochloride is a dissociative agent and a rapidly acting general anesthetic with both sedative and analgesic properties. Primarily acting as an N-Methyl-D-aspartate (NMDA) receptor antagonist, ketamine effectively disconnects the central nervous system from external stimuli.[1-3] Typically, IV doses > 0.5 mg/kg will cause this dissociative state. Ketamine also acts as a potent analgesic that is used for post-surgical analgesia, palliative care, acute pain, and chronic pain management.[2,3] Ketamine's analgesic mechanism is multifactorial, but is in large part due to its effects on mu and kappa opioid receptors.

Ketamine produces concurrent sedation and analgesia while preserving respiratory effort, cardiovascular stability, and airway reflexes in most cases.[2,3] These qualities—in addition to low cost, availability, high versatility, and a large therapeutic window—have made ketamine a popular agent for use in procedural or dissociative sedation in the emergency department.

Reservations for liberal use of ketamine are related to possible emergence reactions, overstimulation of the central sympathomimetic system, and possible elevation in intracranial pressure (ICP). Emergence reactions occur in up to 12% of patients, manifesting as vivid dreams, hallucinations, irrational behavior, and frank delirium, and are due to its activity on NMDA receptors in the cortex and limbic systems.[3,4] As a result, it is recommended to avoid ketamine in patients with a psychiatric history, and ketamine is contraindicated in patients with a history of schizophrenia.[2,3,5] Pretreatment with benzodiazepines has been shown to decrease the incidence of emergence reactions.[1,3]

Additionally, close monitoring of mean arterial pressures, systolic blood pressure, and heart rate is necessary while utilizing ketamine due to its augmented effects on catecholamine release. It is recommended to avoid ketamine in patients with decompensated heart failure or cardiogenic shock due to concern of negative ionotropic effects leading to cardiovascular instability.[3-5] Increases in heart rate and blood pressure are more common with bolus dose administration and increased doses of ketamine.[2-3] Studies show that ketamine administration in tachycardic and hypotensive patients may lead to improvements in heart rate and systolic blood pressure while preserving cardiac output.[3]

To address the final concern with ketamine administration, the theory of increased ICP has been historically argued within the neurosurgical community. However, a 2014 meta-analysis demonstrated that ketamine does not increase ICP in severe traumatic brain injury patients who are sedated and ventilated.[6] Of note, in patients who are chemically paralyzed, ketamine infusions should be used with caution and preferably not as the sole sedative agent due to variability in achievement of retrograde amnesia and the deeper level of sedation necessitated in chemically paralyzed patients.

Current studies investigating ketamine as a continuous infusion for sedation focus primarily on monitoring for respiratory, cardiovascular, and psychiatric adverse effects. Ketamine's optimal dosing, duration, and adverse effect profile for maintenance sedation via continuous infusion have not been well defined in the literature. There have only been small studies and case series examining the use of ketamine continuous infusions (for summary, see table 1).

One of the larger studies was a single-center retrospective study consisting of 30 mechanically ventilated adult patients undergoing sedation with continuous infusion ketamine for greater than twenty-four hours.[2] This study assessed the incidence of tachyarrhythmias and agitation requiring the addition of another sedating agent or the discontinuation of ketamine. All patients received continuous sedation utilizing a ketamine infusion initiated at 0.5 mg/kg/hr with no bolus. The drip was titrated to achieve appropriate sedation, defined as a goal Motor Activity Assessment Score (MAAS) of 2.

Fifteen patients (50%) had fentanyl drips concurrently running with the ketamine infusion and none of the patients received propofol, dexmedetomidine, or benzodiazepines during ketamine therapy. The median dose of ketamine administered was 2 mg/kg/hr (IQR, 1.1-2.5; equivalent to 33 mcg/kg/min) with a mean duration of 59.6 hours. Two patients (6.7%, 95% CI, 2%-21%) experienced new onset atrial fibrillation. However, both patients were on one vasopressor, which were started prior to the development of atrial fibrillation.

An additional two patients (6.7%) (95% CI, 2-21%) had agitation attributed to ketamine. Overall, the adverse event rate was 13% (95% CI, 5%-30%) with a median ketamine dose of 2.25 mg/kg/hr (IQR, 2-2.9; equivalent to 37.5 mcg/kg/min) in those patients experiencing adverse effects. The average MAAS sedation score in the overall population was 1.9. In this study, the use of ketamine appeared to have a frequency of adverse effects similar or lower to that of common sedatives and achieved goal sedation.

Continuous infusion ketamine has also been proposed for use as an adjunctive sedative agent. A small retrospec-

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The Pediatric Abdomen

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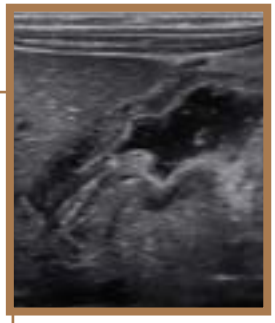
Acute abdominal pain in the pediatric population presents a significant diagnostic challenge. When compared to adults, the differential of abdominal pain in children is dynamic and varies markedly with the age of the patient. One of the most challenging aspects of managing children with abdominal pain is remembering what pathologies should be considered in a given age group. Below is a timeline to help you hone your differential based on the patient's age.

PYLORIC STENOSIS

An abnormal hypertrophy of the pyloric muscle leads to gastric outlet obstruction presenting as post-prandial projectile non-bloody non-bilious emesis and poor weight gain. Patients are often described as "hungry vomiters" as they desire to be fed immediately after emesis. This condition is seen commonly in first born males with a 5:1 male-to-female predominance.[1] The diagnosis is made by an ultrasound demonstrating hypertrophy (attached image). The treatment is surgical incision of the hypertrophied pyloric muscle.



HIRSCHSPRUNG'S

Typically diagnosed within 48 hours of birth, Hirschsprung's disease is caused by the congenital absence of autonomic plexus in the intestinal wall. The overall incidence is 1 in 5000 live births.[1] Denervation of the distal colon prevents peristalsis, and infants typically present with findings of distal bowel obstruction including obstipation, abdominal distension, and emesis. The diagnosis is often suspected when the child fails to pass meconium. The diagnosis is suggested by an abnormal Barium enema and confirmed with rectal biopsy. Treatment is surgical resection of the affected colon.

INTUSSUSCEPTION

Caused by the telescoping of one segment of intestine into another, 65% of cases of intussusception present prior to 1 year and 80-90% prior to 2 years of age. The classic presentation is colicky abdominal pain, non-bloody non-bilious emesis, "currant-jelly stool", and a "sausage-like-mass" in the right lower quadrant. The diagnosis is made by ultrasound and the treatment is reduction with air-enema or, if unsuccessful, surgery.

HSP

HSP is an IgA-mediated small vessel vasculitis. HSP is characterized by the triad of lower extremity palpable purpura, arthralgias, and abdominal pain. The diagnosis is primarily clinical. The most serious complication is the development of chronic kidney disease. Treatment is primarily supportive and the use of steroids is controversial. In severe cases, steroids may be considered as some studies have shown improvement in pain and kidney injury.

NECROTIZING NTEROCOLITIS (NEC)

otizing enterocolitis (NEC) is a condition ncertain etiology that leads to intestinal osis. It occurs primarily in preterm infants or neonates. Patients will present with feeding culty, bloody or bilious emesis, abdominal nsion, and bloody stool. If not recognized , NEC can progress to intestinal perforation, onitis, sepsis, and death. X-rays, as e, may show intraluminal gas known as matosis intestinalis. Treatment is supportive IV fluids, NG suctioning, antibiotics, and nteral nutrition. Surgery is required in % of infants.

MECKEL'S

Meckel's diverticulum is a congenital sacculation of the distal ilium caused by a failure of the vitelline duct to atrophy. Heterotopic gastric tissue that secretes HCl may cause adjacent ulcers in the terminal ilium. Roughly 2% of Meckel's diverticula develop abdominal pain or painless bloody stool. Forty-five percent of cases are diagnosed around 2 years of age. The diagnosis is confirmed by an abnormal radionuclide scan. Symptomatic Meckel's diverticula should be referred to a surgeon for resection.

COLIC

Colic is characterized by periods of excessive crying during which the child draws their knees against their abdominal and appears to be in severe pain. To make the diagnosis, the child must demonstrate such behavior > 3 hours per day for >3 days per week over at least a 3-week period. Treatment options include increasing soothing activities (*e.g.*, rocking, swinging, swaddling), more frequent feedings, use of pacifier, and dietary changes such as removal of cow's milk.

OVARIAN TORSION

Ovarian torsion has been reported in children as young as 2 years of age. Risk factors include known ovarian cyst, adnexal mass, or pregnancy. Classically, ovarian torsion presents with sudden onset of severe, unilateral lower abdominal pain associated with nausea and vomiting. Fever, if present, is usually a late finding and indicates necrosis of the affected ovary. The diagnosis is made by ultrasound with color Doppler analysis. The treatment is emergent surgical intervention.

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APPENDICITIS

HENOCH-SCHONLEIN PURPURA (HSP)

ALSO CONSIDER
MESENTERIC LYMPHADENITIS
URINARY TRACT INFECTION
CONSTIPATION
ACUTE GASTROENTERITIS

OVARIAN TORSION

4 years 5 years 6 years 7 years 8 years 9 years 10 years 11 years

Mastering Minor Care

Benjamin Ostro, MD
University of Cincinnati R4

FINGERTIP INJURIES

While injuries to the fingertip may appear small, the high concentration of sensory nerve endings make real estate in this area very expensive. Sensation at the fingertip as well as an intact support structure (nail apparatus, volar pad, and distal phalanx) are vital to a fully functioning hand. As such, injuries to this area can result in significant morbidity if not managed properly. Despite the importance of treating these injuries appropriately, management strategies in this field of wound care vary considerably. This is in part due to the fact that there are few controlled studies looking at fingertip and fingernail injuries. Joining us again for this installment of Mastering Minor Care is wound management guru, Dr. Alexander T. Trott, to address the management of some common fingertip injuries.

Subungual Hematomas and Nail Bed Lacerations

The management of subungual hematomas has simplified in recent years. It was once taught that if the size of the hematoma was greater than 50% of the nail surface, it was likely that a nail bed laceration was present and would need repair. Today, regardless of the size of the hematoma, if the nail itself is intact and not loose, simple trephination alone is carried out. This can be done with an electrocautery device or with a simple 18 gauge needle, as described below. Any underlying nail bed laceration will heal well without the need of sutures.

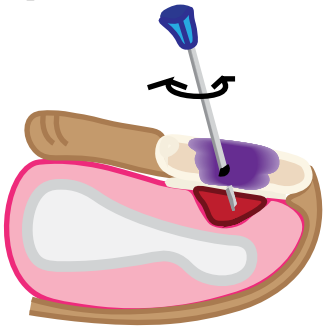
However, if the nail is loose, it should be taken off and the nail bed explored. Any laceration should be repaired to provide stability of the bed. This is typically done with 6.0 or 7.0 absorbable sutures, such as chromic. However, tissue glue such as Dermabond has recently been proposed as an alternative to standard suture repair of nail bed lacerations. A recent study showed no significant difference between the two repair techniques in terms of functional or cosmetic outcomes, and procedure time was significantly shorter for those physicians using tissue glue.[1] Any adjacent soft tissue injury should be repaired in the standard fashion.

If the nail is removed and the bed repaired, is has been routine to temporarily replace the nail to prevent the nail fold from adhering to the nailbed and impeding nail regeneration. Although some recent studies have called this practice into question, it is still standard of care to use the proximal nail to stent open the nail fold and protect the germinal matrix. If the original nail is significantly damaged or missing, non-adherent dressing (*e.g.*, Telfa) or the sterile metal packaging from the suture packet can be used instead. The nail or nail substitute should then be sutured or glued into place.

In healthy patients, prophylactic antibiotics are not indicated for simple nail bed lacerations. Accompanying distal tuft fractures have been classically considered open fractures requiring antibiotics. This practice is also controversial, and local practice may vary. These injuries should be wrapped with a non-adherent, bulky dressing, stabilized with a malleable finger splint, and given hand surgery follow up.

How to Drain a Subungual Hematoma

A subungual hematoma can be drained using an 18 gauge needle. Holding the hub of the needle between your thumb and index finger, position the tip of the needle over the center of the hematoma.



Rotate the needle back and forth to slowly bore through the nail plate. When performing this, it is important to not bore through to the sensitive nail bed.

Avulsion-Tissue Loss Injuries

These injuries can vary from a small oval avulsion from dicing a tomato to a large, mutilating injury from a machine tool accident. The decision to manage the wound by the emergency physician or involve a consultant depends to some degree on the skill set of the practitioner. Exposed bone, complex fractures, tissue distortion, and necrosis are indicators for consultation, as is probable need for an amputation-revision procedure.

The standard of care for superficial fingertip tissue loss that measures 1 cm squared or less can be managed by spontaneous healing (*i.e.*, secondary intention). This assumes that no bone is exposed and the nail apparatus is intact. Some studies show that larger avulsions of up to 1.5 cm by 2 cm in size can be managed similarly. Even if the tip of the distal phalanx can be seen or a small portion of the nail bed is involved, spontaneous healing will provide excellent coverage,

Continued on page 13

#quickhit

Seymour Fracture

Lucia Derks, MD
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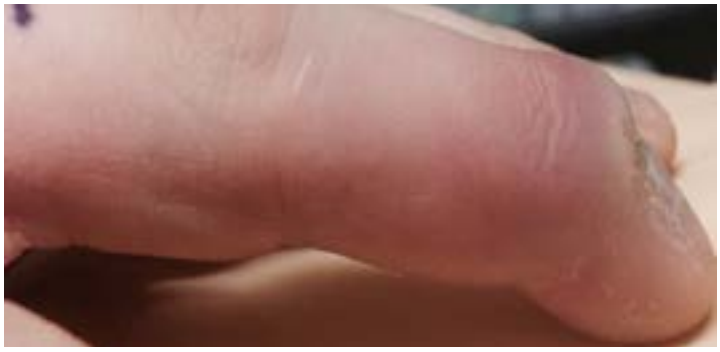

Image 1: The patient' s right fourth digit held in mild flexion at the DIP

The Case

The patient is a teenage, right hand dominant male with no past medical history who presents with three days of worsening right ring finger pain and swelling. He reports that he injured his finger ten days ago while playing football. He was seen by an athletic trainer who told him to buddy tape his fingers. He took acetaminophen for pain which initially seemed to help. However, approximately three days ago after swimming in a river, he developed increased pain and difficulty flexing or extending his finger at the distal joint. He denies fevers and the remainder of his ROS was negative.

Physical Exam

The patient is well-appearing with normal vital signs. The patient's right fourth digit can be seen in Image 1. The digit is held in mild flexion at the DIP joint. There is erythema present just proximal to and surrounding the eponychial fold with underlying hypopigmentation concerning for purulence. There is tenderness to palpation over the dorsum of the distal fourth digit, and notable resistance to active and passive extension at the DIP joint. There is intact range of motion of the PIP and MCP joints.

Discussion

X-rays of the patient's finger demonstrated a Salter Harris II fracture of the right fourth distal phalanx (Image 2) with an overlying nail bed laceration, consistent with a Seymour fracture. Seymour fractures were first described in 1966 by Dr. Seymour of Sheffield, England.[1] A Seymour fracture is a displaced fracture of the distal phalanx that involves the physis with an underlying nailbed laceration, typically thought of as an open fracture. These are primarily seen in children and adolescents due to the incomplete closure of growth plates of the distal phalanx.[2]

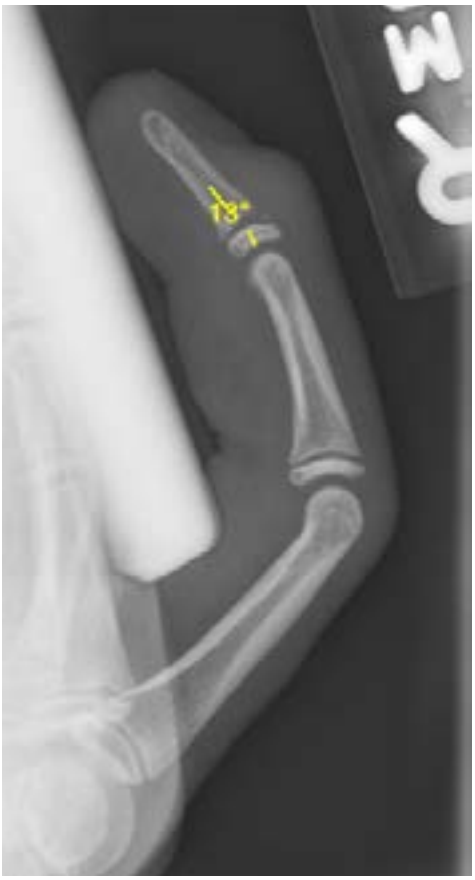

Image 2: Xray showing a Salter Harris II fracture with 13 degrees of angulation.

In addition to pain, redness, and swelling, physical examination of these patients will reveal an inability to fully extend at the DIP joint. This is due to a disruption of the extensor tendon insertion site at the injured epiphysis of the distal phalanx. The injury is limited to the bone and the extensor tendon itself is not injured. In contrast, the flexor tendon inserts at the intact metaphysis and its function is spared. The unopposed flexion is responsible for the classic mallet finger deformity seen with Seymour fractures. Given its similar appearance to a simple nail bed infection, Seymour fractures are often misdiagnosed as a paronychia. Range of motion deficits and the classic mallet finger deformity can help distinguish an isolated infectious process from a more complicated injury such as this.[3] Seymour fractures require special attention due to the involvement of both the nail bed and growth plate. Without proper recognition and treatment, this injury can result in nail deformity, physeal arrest, and chronic osteomyelitis.

A retrospective analysis by Krushe-Mandl et al evaluated operative versus conservative management in a cohort of patients with Seymour fractures.[4] Their primary end-points were range of motion, nail growth disturbance, pain, infection rate, and patient satisfaction. Based on the study results, the authors recommended operative management for all Seymour fractures with an open injury and for those closed injuries that fail closed reduction. Closed injuries that undergo successful reduction can be splinted and managed conservatively. Given the complexities of this injury, risk of serious morbidity, and frequent need for operative intervention, a hand specialist should be consulted to assist with management.

In this case, plastic surgery was consulted. Because of his overlying nail bed laceration, the wound was treated as an open fracture. He was started on IV antibiotics and taken to the OR for debridement, open reduction, and nail bed repair, which was successful. He did well postoperatively and was discharged on oral antibiotics.

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3. Ugular, M, Saka, G, Saglam, N, Milcan, A, Kurtulmus, T, Akpinar, F. Distal phalanx fracture in adults: Seymour-type fractures. Journal of Hand Surg.
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#quickhit

Patella ALTA

Patellar Tendon Rupture

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The Case

The patient is a 35-year-old male with no significant past medical history who presents with left knee pain and inability to ambulate after an assault. The patient works as a bouncer. Shortly prior to arrival in the ED he fell backwards while restraining a rowdy bar patron. The patron fell directly on his left knee while his left foot was planted and his knee was flexed. On exam, the inferior border of the left patella can be palpated 5 cm above the tibial tubercle. With his knee held in extension, he cannot lift his heel off the bed. He has no other knee instability. X rays were obtained and are shown in Image 1.

Discussion

X-ray of his left knee reveals a high-riding patella, also termed 'patella alta', confirming a complete patellar tendon rupture (Image 1).

Patellar tendon rupture, as its name implies, is disruption of the tendon that connects the inferior patella to the tibial tubercle. Trauma is the most common mechanism of rupture, occurring most frequently when a flexed knee resists a powerful contraction of the quadriceps muscle. Patients with systemic inflammatory disease, diabetes, chronic kidney disease, or chronic patellar tendinitis are at increased risk of atraumatic patellar tendon rupture.[1]

Physical exam in patients with patellar tendon rupture reveals a high-riding patella, diffuse swelling, and inability to ambulate on the affected leg. Additionally, patients have an impaired extensor mechanism such that they are unable to lift their heel with an extended knee. Our patient demonstrated all of these cardinal examination findings. Significant force is needed to rupture the patellar tendon, so care should be taken to evaluate patients for other ligamentous injury, as ACL and meniscal tears have been known to occur simultaneously with patellar tendon rupture.[2]

X-ray is the imaging modality of choice to diagnose patellar tendon rupture. Patella alta will confirm the diagnosis, and calcification of the patellar tendon inferior to the patella may indicate chronic patellar tendinitis. However, if the diagnosis cannot be established with x-ray and physical exam, MRI is indicated.[3] Ultrasound has also shown promise as an accurate imaging modality to diagnose patellar tendinopathy.[4] A patellar tendon rupture will be apparent by increased tendon thickness in the sagittal plane due to tendon retraction, as

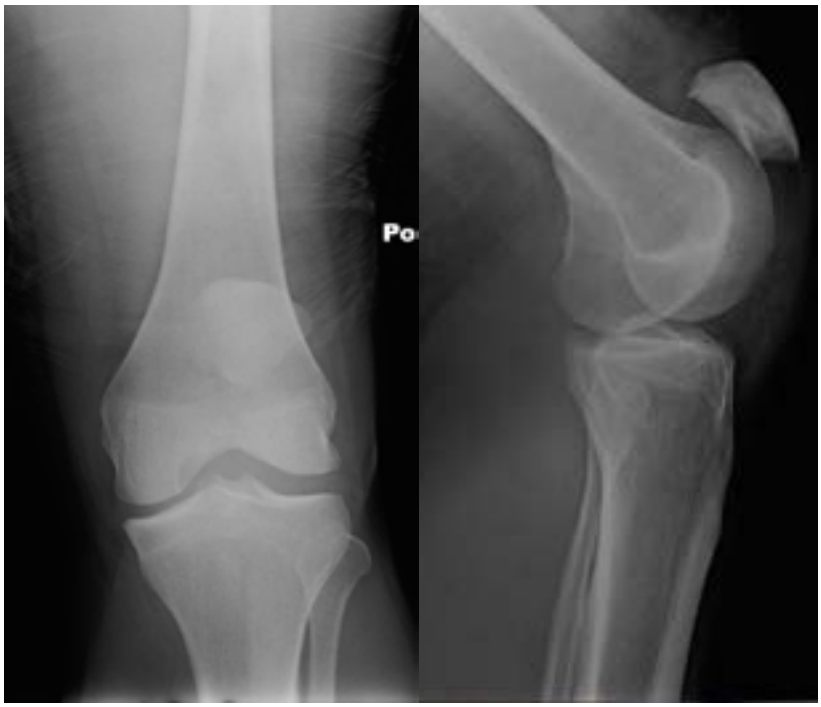

Image 1: Xray of the left knee xray showing a high-riding patella.

Treatment of patellar tendon rupture is early primary operative repair, as delay can cause tendon contracture, scar tissue formation, and quadriceps muscle atrophy.[5] Therefore, urgent orthopedics follow-up must be established for patients. To prevent tendon contracture, the affected knee should be placed in a knee immobilizer and the patient should be non-weightbearing until they can be seen by an orthopedic surgeon.

Our patient was placed in a knee immobilizer and was discharged with urgent orthopedic surgery follow-up. He underwent successful open repair 2 weeks later. He has since followed up with orthopedic surgery and is healing well. He is currently able to ambulate without assistance.

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Necrotizing Fasciitis
Continued from page 5

tion is surgery. The goal of the EM physician should be to minimize the delay in getting the patient to the OR. There have been multiple studies over the years that have demonstrated that an increase in the amount of time from admission to surgical intervention is associated with increased mortality.[10, 11] The same authors who developed the LRINEC score reported that there was a nine-fold increase in the mortality risk if surgical treatment was delayed by >24 hours from the time of diagnosis. In a more recent study, it was found that a delay of >12 hours to initial surgical debridement was associated with an increase in the number of surgical procedures required, as well as a higher incidence of septic shock and acute renal failure.[12]

Necrotizing fasciitis is a rare condition with high morbidity and mortality that relies on rapid recognition and prompt intervention. Emergency physicians must be able to identify the findings by history and on physical exam that point toward a higher risk for necrotizing infection, including rapid progression, pain out of proportion to exam, peripheral anesthesia, and possibly skin findings such as erythema (Image 2), bullae, ecchymosis, and crepitus. They may also apply the LRINEC score if the clinical diagnosis is more ambiguous.

While laboratory and imaging studies may provide further support of the diagnosis, administering appropriate antibiotics and getting surgery involved should never be postponed if there is high clinical suspicion based on history and physical alone. Definitive diagnosis and treatment can only be achieved in the operating room. Given the severe consequences of delaying treatment, it is always better to err on the side of caution if this deadly diagnosis is on the differential.

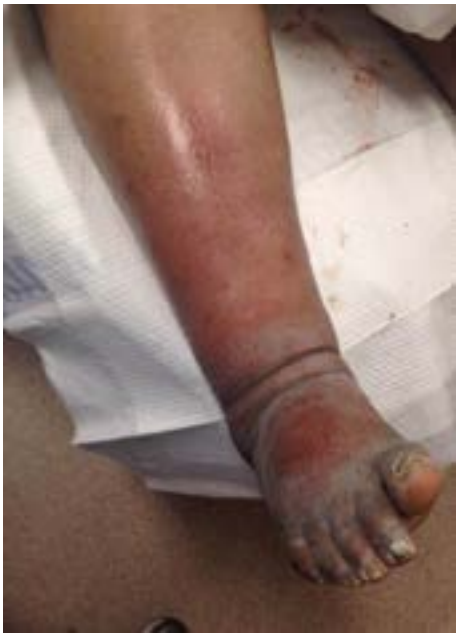

Image 2: Skin changes of the lower extremity seen in a patient with necrotizing fasciitis.

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Fingertip Injuries
Continued from page 10

with sensation restored. The complication rate is virtually nil, whereas it is 20-25% with grafting or flap repair. Larger or more complex wounds require surgical consultation (see summary below).

It takes 3-4 weeks for an avulsion injury to heal. The patient needs to keep the wound clean with soap and water. One or two band-aids, changed every 2-3 days after the initial non-adherent dressing is removed, provides adequate protection.

1. Strauss et al. A prospective, randomized, controlled trial of 2-octylcyanoacrylate versus suture repair for nail bed injuries. J Hand Surg Am. 2008; 33(2):250.

Guidelines for Management of Avulsion Injuries

If NO BONE is exposed...

- If the defect is <1 cm in diameter, spontaneous healing is the treatment of choice.
- If the defect is >1 cm, but with an intact nail apparatus, the provider can consider conservative management as an alternative to grafting, especially in children. Local practice may dictate management.

If BONE is exposed...

- For injuries with exposed bone, consultation is recommended.

If the NAIL APPARATUS is involved...

- For significant nail apparatus involvement, repair or revision of the matrix is necessary. Consultation may be required.
- With minor nail apparatus involvement, conservative management is reasonable.

Dressings and Antibiotics

- Proper dressings for fingertip avulsions include a non-adherent base, such as Xeroform or Adaptic, with a sponge covering and gauze wrapping. Dressings should be changed every 2-3 days. Antibiotics are suggested for injuries with exposed bone.

Erythroderma
Continued from page 6

it presents with diffuse erythema and scaling involving a significant portion of the skin. It is the clinical presentation of a wide range of cutaneous and systemic diseases including psoriasis (~38% of cases), atopic dermatitis, hematologic and systemic malignancies, hypersensitivity drug reactions, connective tissue disorders and skin infections.[1] Approximately 30% of cases are idiopathic. In patients with psoriasis, triggers of erythroderma include the abrupt discontinuation of systemic corticosteroids or other immunosuppressant therapy, systemic illnesses, emotional stress, medications, or HIV infection.[2] The pathogenesis is poorly understood but is thought to be a complex interaction of cytokines, including interleukins, tumor necrosis factor and intercellular adhesion molecules. These signalling molecules recruit massive amounts of inflammatory cells to the skin, causing elevated epidermal turnover.

Erythroderma may be acute or chronic. Patients will present with generalized erythema and scaling, as well as systemic symptoms including chills, malaise, and fatigue. Due to the loss of adequate barrier protection, these patients are at high risk for sepsis. Additional complications include electrolyte and protein abnormalities due to fluid losses through the exposed dermis, as well as hypothermia, high output heart failure, and ARDS.[2] Laboratory studies are typically nonspecific and show elevated inflammatory markers.

The initial emergency department treatment of erythrodermic psoriasis is management of the ABCs. In severe cases or in patients with underlying cardiopulmonary comorbidities, the severe systemic inflammation and leaky pulmonary capillaries seen with erythroderma can lead to ARDS, requiring advanced airway management. IV crystalloid is typically sufficient to restore volume loss, but if complicated by sepsis or other underlying illnesses, vasopressors may be necessary for blood pressure support.

Any electrolyte abnormalities should be addressed and quickly replaced. The patient's temperature should be monitored closely, and the patient should be kept in a warm and humid environment to prevent hypothermia. Low to moderate potency topical steroids (*e.g.*, triamcinolone acetonide 0.1% ointment) are recommended initially due to of risk for systemic absorption of high potency forms secondary to the extensive body surface area and enhanced cutaneous permeability.[2] Systemic steroids should be avoided if psoriasis is the underlying etiology of the erythroderma based on anecdotal evidence that their use or sudden withdrawal poses a risk to the development of flaring the disease.[4] Oral antihistamines may be added for symptomatic relief of pruritus. Systemic antibiotics are only required for patients with secondary infection. [5, 6]

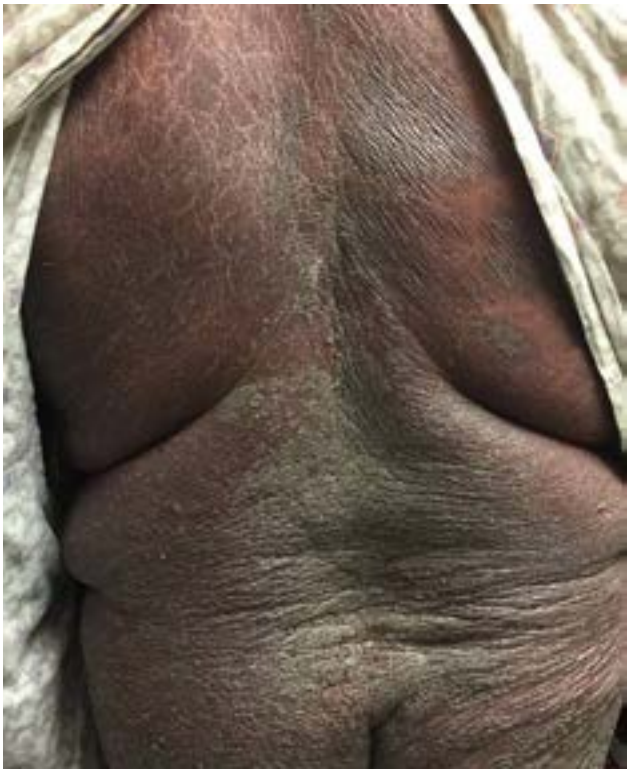

Image 2: The back of a patient exhibiting erythrodermic psoriasis.

Our patient responded well to IV rehydration, maintenance of warm ambient temperature, and topical steroids. Kerlix was used to mimic the barrier protection that his skin would usually provide. Immunosuppressive treatment provided definitive control of his symptoms.

Erythroderma is a rare clinical manifestation of numerous underlying systemic or cutaneous diseases. Exacerbation of a preexisting inflammatory dermatosis such as psoriasis is the most common cause of erythroderma. Emergency Medicine providers must be mindful of the acute complications of erythroderma including kidney injury, hypovolemia, respiratory compromise, sepsis and electrolyte abnormalities. Initial therapies include IV fluids, electrolyte replacement, oral antipruritics and topical low-to-moderate potency steroids. Early dermatologic consultation in the ED can assist in the appropriate treatment of this serious skin disorder.

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Continuous Ketamine
Continued from page 7

tive study of 43 patients described the efficacy and safety of continuous infusion ketamine for adjunct sedation in mechanically ventilated critically ill patients.[7] Sedation was assessed using the Riker Sedation Agitation Score (SAS). On average, ketamine was initiated at 0.16 mg/kg/hr (range 0.05–1 mg/kg/hr) and continued for 3.6 days with a mean dose of 0.44 mg/kg/hr (range 0.05–2 mg/kg/hr). A goal SAS of 3 was achieved in 69% of patients. Twenty-six patients (60%) had at least one alternate sedative decreased or discontinued after the initiation of ketamine. Mean arterial blood pressure increased from 77 to 81 mmHg (p=0.15) compared to baseline in the initial 24 hours of the ketamine infusions without significant change in heart rate (p=0.97). Overall, ketamine helped to achieve goal sedation while minimizing the use of other sedative agents.

There are still many questions that remain regarding the use of continuous infusion ketamine, including optimal patient population, dosing, and duration. The literature that does exist is limited by small sample sizes, lack of consistent dosing, varying definitions of ideal sedation, and the use of different sedation scales and scoring systems. There are also some UCMC specific limitations to consider when initiating a ketamine infusion. Per the Ohio State Board of Nursing, nurses are unable to titrate ketamine infusions and may only be titrated by a physician order. Therefore, each dose adjustment must be ordered by a physician prior to the adjustment taking place. Ketamine may be used as a continuous maintenance sedative agent; however, a large prospective clinical trial is necessary to further clarify the utility of ketamine for this specific indication.

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Study	Design	Sample size	Patient population	Dosing Strategy	Outcomes
Arnold, et al[8]	Case series	12	MV MICU	Median: 0.2-0.8 mg/kg/h Max: 2.8 mg/kg/h	• Discontinued in 2 patients d/t increased HR and HTN • Patients who received concomitant BZD had no hemodynamic changes
Bourgoin, et al[9]	P, R, DB	25 (12 received ketamine)	MV severe TBI	Initial dose: 50 mcg/kg/min Mean dose at day 4: 94 mcg/kg/min	• Ketamine/midazolam vs. sufentanil/midazolam • Increased HR in ketamine on day 3 and 4, otherwise no significant differences
Groetzinger, et al[7]	Retrospective	43	MV	Initial mean dose: 0.16 mg/kg/hr Mean overall dose: 0.44 mg/kg/min	• Goal sedation achieved in 69% of observations • 26 (60%) had at least one alternate sedative decreased or discontinued • MAP increased compared to baseline in the initial 24 hours of ketamine infusion with no significant change in HR
Hijazi, et al[10]	Case series	12	Neurotrauma ICU	2mg/kg bolus then 2mg/kg/h x 2 hours	• Primarily evaluated pharmacokinetics in critically ill patients • No significant changes in hemodynamics
Kolenda, et al[11]	P, R, DB	24 (12 received ketamine)	Moderate to severe TBI	Initial dose: 2.7 mg/kg/h Median overall dose: 4.3 mg/kg/h	• Ketamine vs fentanyl • MAP was significantly higher in the ketamine group and decreased use of vasopressors
Umunna, et al[2]	Retrospective	30	MV ICU	Initial dose: 0.5 mg/kg/hr Median overall dose: 2.0 mg/kg/hr (IQR, 1.1-2.5).	• Ketamine was switched to another sedative agent due to ADE in 4 patients (13%)
Zakine, et al[12]	P, R, DB, Controlled	50 (27 received ketamine)	abdominal surgery	0.5mg/kg bolus then 2 mcg/kg/min x 48 hours post op	• Lower morphine consumption and nausea/vomiting in the ketamine group • No difference in sedation score or psychiatric disorders

Table 1: Evidence for continuous ketamine infusions
P = prospective; R = randomized; DB = double blind, MV = mechanical ventilated

Brugada Syndrome

EKG focus

Grace Lagasse, MD
University of Cincinnati R2

History of Present Illness

A 25-year-old male presented after an episode of presyncope after working out. The patient denies any chest pain associated with this event. The patient had an EKG that was consistent with a type 3 Brugada EKG pattern. Electrophysiology recommended that the patient undergo a procainamide challenge in the EP lab to see if a type 1 Brugada EKG pattern could be elicited with sodium channel blockage.

Brugada Syndrome

Brugada Syndrome (BrS) is a cardiac conduction abnormality due to a sodium channelopathy that is often found in structurally normal hearts. It can result in sudden cardiac death, and definitive treatment is ICD placement.

Diagnosis

Type 1 EKG: spontaneously occurring or induced

AND

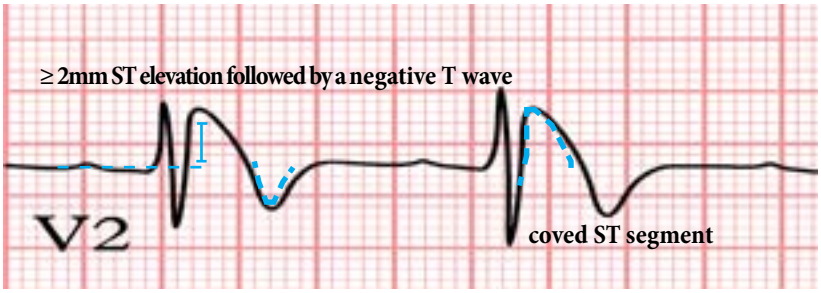
Any one clinical characteristic:
1. Documented VF or VT
2. Family history of sudden cardiac death
3. Family history of Type 1 EKG
4. Patient history of syncope or nocturnal agonal respirations
5. Electrophysiology induced VF or VT

AND/OR

Known genetic mutation, SCN5A is the most common

1. Antzelevitch C, Brugada P, Borggrefe M, Brugada J, Brugada R, Corrado D, Gussak I, LeMarec H, Nademanee K, Perez Riera AR, Shimizu W, Schulze-Bahr E, Tan H, Wilde A. Brugada syndrome: report of the second consensus conference: endorsed by the Heart Rhythm Society and the European Heart Rhythm Association. Circulation. 2005 Feb 8;111(5):659-70. Epub 2005 Jan 17. Review
2. Berne P, Brugada J. Brugada syndrome 2012. Circ J. 2012;76(7):1563-71. Epub 2012 Jun 13. Review.
3. Brugada R, Campuzano O, Brugada P, Brugada J, Hong K. Brugada Syndrome. 2005 Mar 31 [updated 2014 Apr 10]. In: Pagon RA, Adam MP, Ardinger HH, Wallace SE, Amemiya A, Bean LJH, Bird TD, Fong CT, Mefford HC, Smith RJH, Stephens K, editors. GeneReviews® [Internet]. Seattle (WA): University of Washington, Seattle; 1993-2016.
4. Priori SG, Napolitano C, Gasparini M, Pappone C, Della Bella P, Giordano U, Bloise R, Giustetto C, De Nardis R, Grillo M, RonchettiE, Faggiano G, Nastoli J. Natural history of Brugada syndrome: insights for risk stratification and management. Circulation. 2002 Mar 19;105(11):1342-7.

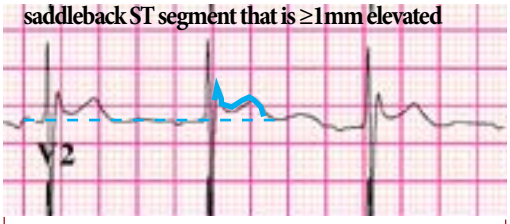
Type 1 Brugada Pattern



Type 1 EKG: ≥ 2mm elevation of ST elevation followed by a negative T wave OR coved type ST segment configuration that gradually declines in at least one of the right precordial leads (V1-V3)

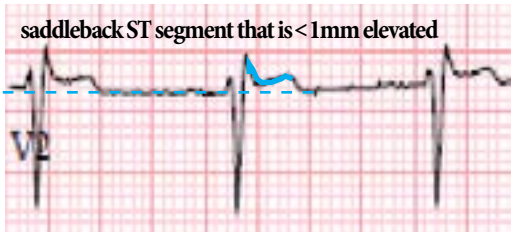
Types 2 and 3 Brugada EKG patterns are only *suggestive* of Brugada Syndrome.

Type 2 Brugada Pattern



Type 2 EKG: ≥ 2mm elevation of J wave, positive or biphasic T wave, and saddleback ST segment configuration that is ≥ 1mm elevated in at least one of the right precordial leads (V1-V3)

Type 3 Brugada Pattern



Type 3 EKG: ≥ 2mm elevation of J wave, positive T wave, and saddleback ST segment configuration that is < 1mm elevated in at least one of the right precordial leads (V1-V3)

EKG and Case referred by

Jordan Bonomo, MD
University of Cincinnati

Annals of B Pod is looking for YOU to submit your interesting cases of B Pod - There is a composition book at the R4 desk - please make sure to include the R1/R4 involved in the case, a brief synopsis and a patient sticker
annalseditors@gmail.com

List of Submitted B Pod Cases

Case	Case Physicians
Patellar tendon Rupture	Ostro/Shaw
Submassive PE with catheter-directed TPA	Doerning/Fritch
Mycotic aneurysm and DVT	LaFollette/Whitford
Dislocated lens	Mann/Sabedra
Type A aortic dissection	Mann/Sabedra
Necrotizing fasciitis	Curry/Sabedra
Malignant otitis externa	LaFollette/Fananapazir
Nephrotic syndrome	Selvam/Continenza
Myasthenic crisis	Mann/Merriam