Twisted
Women’s Reproductive Health Issues in the Emergency Department
Can’t-Miss, Often-Missed Ovarian Torsion
Hemorrhagic Adenoma on Birth Control
Uterine Incarceration
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“This is bringing back some bad memories,” my patient said nervously, making the most severe understatement I’ve heard in residency.

About 10 years prior to our conversation, she had experienced a nightmare beyond words. In the third trimester of a very wanted pregnancy, her fetus died in her uterus. To make this devastating loss even worse, this went undetected for a few days as she became increasingly septic at home, and by the time she was transferred from a smaller ED to mine, she was in septic shock. She decompensated further, lost consciousness, and had a cardiac arrest requiring a resuscitative hysterotomy. The stillborn fetus was removed, and the patient was cannulated for ECMO, which she depended on for months afterwards. She spent the following year cycling through near-fatal complications including severe dilated cardiomyopathy necessitating a heart transplant and prolonged ventilator dependence requiring a tracheostomy, all while experiencing the emotional trauma of both her own hospitalization and the stillbirth of a desired third-trimester pregnancy.

Seeing her now, wearing her business casual work attire in the OB triage area, only a tracheostomy scar betrayed the long and arduous battle for her life she had fought years before. Despite her efforts to consistently use birth control with her husband, she was pregnant again. And she was scared.

These may not be the extreme circumstances of every person facing a pregnancy and considering termination, but many face other challenges that make pregnancy and childbirth life-threatening dilemmas. You have cared for patients with ectopic pregnancies, patients with precarious comorbidities, and patients who delivered a term fetus known in-utero to have severe unsurvivable malformations. For these people and so many more, legislation that forces them to continue a pregnancy can be extremely dangerous. For their obstetricians and emergency physicians, such laws make improving these people’s chance of survival by ending their pregnancy a crime for which doctors could potentially be prosecuted.

Even measures that do not legalize abortion but restrict it to life-threatening situations or cases of sexual assault and incest are fraught with inequity in how they are implemented. Attempts by lawmakers to prohibit some abortions before viability but not others often form logistical hurdles causing dangerous delays in care and worse medical outcomes for the pregnant person. These hurdles also disproportionately restrict treatment options for patients with limited resources. Those without transportation to far-away clinics, access to legal counsel, or money for abortion services are forced to proceed with a pregnancy that a person with more resources could terminate.

As emergency physicians, we care for patients of all backgrounds and means. We are often the ones to look into their eyes and break the news that they are pregnant. To put it bluntly, after residency we will also be some of the most wealthy and well-resourced people in some of our patients’ lives. Though I felt powerless when reading the leaked majority opinion of the Supreme Court that, if finalized, would overturn the decision of Roe v. Wade, this is only one battle in the war to protect our patients’ reproductive rights. If it is officially overruled, the stakes will be higher than ever for us to use what power we do have as physicians and voters at the state level.

Starting now, share your clinical experiences with legislators, friends, and family. Tell the de-identified stories of patients who have suffered from ectopic and high-risk pregnancies. Talk about your patient with endometritis or hemorrhage from an abortion done without medical supervision. Make the impact of this legislation as personal to lawmakers and voters as it is to the woman who has just learned she has a pregnancy that she may not survive. She was my patient. Now she’s yours, too. *
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Career Launchpad — Helping You Be the Best Doctor and Leader in EM

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I know what you’re after...a job!

The summer before your last year in residency is a great time to launch your job or fellowship search. When I began looking for jobs before starting my fellowship last summer, I felt lost. Myriad questions filled my mind: How do I decide whether to do a fellowship? What is the timeline for applications? Where do I find jobs? How do I get DEA and medical licenses? How much money do I need to have saved for moving and credentialing? Is it time to purchase own-occupation disability insurance? What kind of group business model do I want to practice in? Is this a fair job contract? The transition from residency to beyond can be daunting and overwhelming!

At the beginning of my term as your EMRA president, one of our priorities was for EMRA to be the leading source of career planning guidance as you work your way through residency, graduate, and tackle next steps. This month, I am proud to share that we are well on our way to making EMRA your go-to source for career planning.

EMRA Career Resources

Here’s a preview of what’s coming:

EMRA is building a landing page that will serve as a bridge during your transition and will answer all those important questions above, and more. Bookmark this page now! You can find it at https://www.emra.org/career-planning.

On EMRA’s career planning page, you can explore:

- The emCareers Job Match tool
- Guides to employment selection, group management types, and contract negotiation
- Resources to make your CV and cover letters shine
- Information on financial planning, loan refinancing, and disability insurance
- A moonlighting checklist for credentialing and preparedness

EMRA will be adding more resources to the site soon, making sure you have as much support as possible for the path ahead. We are in this with you, and we will adjust with the changing landscape of EM.

Need More? Just Ask!

Don’t see what you’re looking for? Just reach out to me at president@emra.org. We are happy to provide the information, resources, and connections you need to make your transition to independent practice as smooth as possible. 🌟
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Ovarian torsion, marked by pelvic and abdominal pain and considered a surgical emergency, can be difficult to diagnose but should not be overlooked, especially among pediatric patients.

A Cautionary Tale of Adnexal Torsion in an Adolescent Female

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Although a rare diagnosis, ovarian torsion is an emergent cause of pelvic and abdominal pain. As emergency department providers, we are primed to consider ovarian or adnexal torsion in our differential diagnosis in adult women presenting with pelvic pain, but are less likely to consider the diagnosis in pediatric populations. Although far less common in the pediatric population, occurring in approximately 4.9 per 100,000 women under the age of 20, ovarian torsion can occur in females of all ages, even fetuses and neonates, and remains a “can’t miss” diagnosis. Here, we present the case of an adolescent female who presented with fever and abdominal pain, and was initially misdiagnosed with spontaneous pelvic inflammatory disease, before the correct diagnosis of ovarian torsion was finally reached.

Case Presentation
An 11-year-old female with a history of obesity presented to the pediatric emergency department with left hip and left lower quadrant abdominal pain for 3 days. The patient reported that she fell on ice 4 days prior. She also endorsed nausea and vomiting. She had menarche 6 months prior and denied sexual activity. Vital signs included fever to 38.7°C (101.7°F), heart rate of 152, and blood pressure of 139/37. The physical exam revealed tenderness over the left lower quadrant without rebound or guarding, no bony tenderness to palpation over the greater trochanter or pelvis, and full range of motion of the hip. The patient ambulated with a steady gait. Labs were notable for white blood cell count of 25.5x10⁹/L, CRP of 178.9 mg/L, normal urinalysis, and negative rapid urine
gonorrhea and chlamydia.

Given the patient’s body habitus and her ill appearance, there was concern that an abdominal ultrasound would be insufficient to evaluate for intra-abdominal infection. A computed tomography of the abdomen and pelvis was performed, which showed a prominent cystic structure in the pelvis with a small to moderate amount of complex fluid. Subsequent formal transabdominal ultrasound demonstrated a cystic structure measuring 9.1 cm originating from the left ovary with low grade venous flow.

Gynecology was consulted and, given the patient’s fever and elevated inflammatory markers, admitted the patient with the presumed diagnosis of spontaneous pelvic inflammatory disease versus infected cyst. The patient continued to complain of left lower quadrant abdominal pain following admission despite initiation of broad spectrum antibiotics (cefotetan, doxycycline, and metronidazole). On hospital day 4, diagnostic laparoscopy revealed an enlarged and hemorrhagic appearing left ovary. The ovary and tube were torsed approximately 6-7 times. A paraovarian serous cyst was identified and drained. The ovary and fallopian tube were successfully de-torsed with improvement in color, and the patient was discharged with outpatient gynecology follow-up.

**Discussion**

Ovarian torsion is a surgical emergency and occurs when either the ovary or ovary and oviduct (termed adnexal torsion) twist on its ligamentous supports, resulting in congestion due to decreased venous return, decreased distal arterial blood flow, and ultimately ischemia and necrosis. The classic presentation of ovarian torsion is described as sudden onset, severe, colicky unilateral pain with guarding and adnexal enlargement on bimanual exam.\(^3\)\(^5\)\(^6\) However, even within adult populations, ovarian torsion can be difficult to diagnose as not all patients follow this pattern. In a 15-year retrospective review, only slightly over half of patients reported sudden onset pain, and 44% reported crampy or colicky pain.\(^2\) Moreover, one-third of patients had only mild tenderness on abdominal exam, and 29% had no tenderness of pelvic exam. As a result, clinicians failed to consider ovarian torsion in the initial differential diagnosis in more than half of cases.

Within the pediatric population, a high clinical suspicion is necessary as atypical presentations are even more common. Although the majority of patients will present with abdominal pain, nausea, and vomiting, this presentation can occur with a variety of clinical conditions, and the differential diagnoses include appendicitis, pelvic inflammatory disease, gastroenteritis, and renal colic.\(^3\)\(^4\) In one retrospective cohort study comparing presentation of ovarian torsion in premenarchal and postmenarchal patients, premenarchal patients were more likely to present with restlessness (14.6% versus 0%), fever (9.8% versus 1%), palpable pelvic mass (9.8% versus 0%), and diffuse abdominal pain (29.3% vs. 7.2%).\(^3\) Similarly, a retrospective chart review of 83 cases reported higher rates of tachycardia in younger patients (37% versus 8%) and found that 20% of cases presented with a leukocytosis.\(^6\)

Because pediatric patients are less likely to present with the typical symptoms associated with ovarian torsion, they are more likely to be initially misdiagnosed. In fact, compared to postmenarchal patients, a higher proportion of premenarchal patients were not evaluated by a gynecologist before surgery (12.2% compared with 0.5%\(^2\)). In another study, 38% of patients ultimately diagnosed with ovarian masses were originally diagnosed with appendicitis.\(^7\)

**Risk Factors**

The primary risk factor for ovarian torsion is an enlarged ovary or ovarian mass greater than 4 cm. Risk of torsion correlates with size of ovarian mass, and more than 80% of adult patients with ovarian torsion had ovarian masses greater than 5 cm.\(^8\) Underlying ovarian abnormalities are a primary risk factor for the development of ovarian torsion in pediatric patients as well.\(^3\)\(^4\) Due to hormone stimulation, there is a known increase in functional ovarian cysts around the fetal period, first year of life, and menarche. In fact, up to 20% of females have multicystic and enlarged ovaries at menarche.\(^3\) However, ovarian torsion can occur in patients with no underlying pathology and, compared with older women, pediatric and adolescent patients ultimately diagnosed with torsion are more likely to have normal ovaries. In a 22-year retrospective chart review of pediatric patients, only 42% of patients with torsion presented with an ovarian mass or calcification, the mean size of which was 5.5 x 6.5 cm.\(^4\) For example, in one retrospective review, slightly less than 50% (10/22) of girls experiencing torsion had normal ovaries.\(^9\) Of interest, in both adult and pediatric patients, malignancy is a rare cause of ovarian torsion.\(^3\)\(^4\) Additionally, in both adult and pediatric populations, ovarian torsion occurs more commonly on the right than on the left.\(^4\)\(^6\)\(^10\) This may occur because right utero-ovarian ligament is longer, because

**IMAGE 1. CT scan of abdomen and pelvis demonstrating a prominent cystic structure in the pelvis and a moderate amount of complex fluid in the pelvis concerning for a ruptured follicle or corpus luteum, ovarian torsion, or neoplastic process.**
of the presence of the sigmoid colon on the left, or because physicians are more likely to work up right-sided abdominal pain due to concern for appendicitis.

**Diagnostic Imaging**

In both adult and pediatric populations, the pelvic transvaginal ultrasound with doppler is the preferred diagnostic test. Findings on ultrasound can include an edematous, heterogeneous ovary, multiple small follicles (due to displacement by edema), and an enlarged ovarian or tubal cyst or mass. While it is common practice to obtain a transvaginal ultrasound in adult patients, transvaginal ultrasound is not appropriate for pediatric patients who are not sexually active. However, there is some data to suggest a transabdominal ultrasound may be sufficient. In Ashwal et. al., there were similar rates of ultrasound findings consistent with ovarian torsion in premenarchal patients, who exclusively had transabdominal ultrasounds performed, and postmenarchal patients, of whom 65% had a transvaginal ultrasound in addition to a transabdominal ultrasound. In pediatric patients, the most common finding is an enlarged ovary or an echogenic mass without visualization of the ovary.5,6

Regardless of modality, doppler can aid in the diagnosis of ovarian torsion if absent. However, the presence of normal arterial or venous doppler flow does not rule out ovarian torsion. The estimated sensitivity of doppler flow ranges from 43% to 92% and specificity of 100% to 97%, but data is inconsistent within pediatric populations.5,13 In one pediatric review, 62% of torsed ovaries had arterial or venous flow on doppler sonography.6 By comparison, in another review, 66% of patients had decreased or absent flow.2 And in Ashwal et. al., 53% of premenarchal and 63% of postmenarchal patients had either absent venous or arterial doppler flow.

The “whirlpool sign,” the ovarian vessels visualized wrapping around a central axis, is another ultrasound finding that is highly sensitive if present.6 In Ashwal et. al., 10% of patients had a positive whirlpool sign (two patients in the premenarchal group and 13 in the postmenarchal group).

Although not the gold standard, ultrasound is a helpful tool in the evaluation of ovarian torsion. If equivocal, MRI can be helpful in diagnosing ovarian torsion, as it can demonstrate components of a mass in more detail.8 Direct visualization is the only way to definitively make the diagnosis of ovarian torsion.8

**What is Spontaneous PID?**

In this case, the admitting diagnosis was spontaneous pelvic inflammatory disease (PID), a rare diagnosis. Following a literature review, we were only able to identify 14 case reports of spontaneous PID in the English language. Spontaneous PID in virginal patients is a polymicrobial infection, and is hypothesized to occur from bacterial seeding from bowel or ascension from the vagina. Hypothesized risk factors for spontaneous PID include obesity, constipation, chronic UTIs, poor hygiene, and cervical ectopy.13

One case describes an 11-year-old female with acute onset abdominal pain in the right lower quadrant associated with nausea and vomiting. She had a leukocytosis to $32.8 \times 10^9/L$, and an ultrasound showed a tubular structure in the right lower quadrant and moderate fluid in the pelvis. She was taken to the operating room with a presumptive diagnosis of appendicitis. However, on laparoscopy a large amount of purulent fluid in pelvis and posterior cul-de-sac were seen with an erythematous uterus and normal appendix. Pelvic fluid microscopy showed numerous polymorphonuclear leukocytes, but no growth of aerobic or anaerobic bacterial culture, and urine gonorrhea and chlamydia cultures were negative.

In another case report, a 13-year-old patient presented with abdominal pain, fever, and a cystic mass in the right lower quadrant. On laparoscopy she was diagnosed with a right pyosalpinx. After incision, purulent liquid was visualized, which ultimately grew *E. coli*.14

Despite these case reports, spontaneous PID is rare and poorly understood. However, it highlights that PID should be thought of as a polymicrobial disease, and that negative gonorrhea and chlamydia tests do not rule out PID.

**Case Resolution**

Two months following discharge, the patient was seen in a gynecology clinic for follow-up. The patient was asymptomatic at that time. Repeat ultrasound demonstrated a 4.8 cm complex cyst that was mostly cystic with solid components, likely representing residual clot from torsion. On a repeat visit four months after discharge, the patient remained asymptomatic. A repeat ultrasound was planned, and the patient has had no further follow-up visits in our system.
A 21-year-old female, G3P103, presented to the emergency department with a chief complaint of one month of right lower quadrant (RLQ) abdominal pain and one day of hematuria. The pain was constant, described as a “ball of pressure,” with occasional sharp shooting pain provoked by position change. She had no relief with ibuprofen. She also reported associated nausea, headaches, and lightheadedness, but denied fever, chills, dysuria, vaginal bleeding, or vaginal discharge.

One month prior, she was seen in the ED for the same pain but had no hematuria. An ultrasound showed that her intrauterine device (IUD) was in the myometrium of her uterus, and a large left adnexal cystic structure from her left ovary had crossed into the right adnexa, measuring approximately 8.2 x 4.2cm. She was told to follow up with OB/GYN but could not schedule an appointment. A urinary tract infection (UTI) was also diagnosed during that visit. It was successfully resolved with cephalaxin.

The patient’s past medical history included a cesarean section eight months prior and two complicated preterm vaginal deliveries within the last three years. The IUD was placed two months after the C-section.

Initial vital signs in the ED included HR 78, BP 108/74, RR 18, temp 98.6°F, SpO2 100%, and BMI 43.62. Upon physical exam, the patient was in noticeable pain and had mild abdominal tenderness to deep palpation in the right lower quadrant (RLQ) without rebound, guarding, or CVA tenderness. On pelvic exam, an IUD string was seen in the cervix but no swelling, erythema, discharge, or lesion. The remainder of the physical exam was within normal limits. The urinalysis was normal. The pregnancy test was negative. Apelvic transabdominal and transvaginal ultrasound redemonstrated a malpositioned IUD extending into the myometrial wall and a large left adnexal cystic structure. OB/GYN was consulted and attempted to remove the IUD in the ED, but was unsuccessful. Strings were visualized, grasped with ring forceps and gentle traction was applied but could not be removed. She was consequently admitted to have her IUD removed in the operating room (OR).

**Discussion**

Use of long-acting reversible contraceptive devices, such as IUDs, have increased dramatically in the past decade and therefore are seen more frequently in the ED. Serious complications are rare, but can include expulsion, pelvic inflammatory disease, contraception failure, ectopic pregnancy, and perforation.¹

Uterine perforations are uncommon and occur in approximately 1 in every 1,000 insertions. Perforation rates are greater when insertion occurs less than eight weeks postpartum.² It is possible that there is an association with perforations and scarred myometrium from cesarean deliveries and IUD placement while breastfeeding.³ A perforation can be complete or partial, fully entering the abdominal cavity or penetrating the uterine wall to varying degrees.⁴ Perforations can occur at the time of insertion or after the device has been successfully placed. Complete perforation will not protect against pregnancy and will frequently cause severe abdominal pain. The device is most commonly found in the Pouch of Douglas but can migrate in the abdominal cavity and attach to an organ, the bowel, the mesentery, and the omentum, resulting in further perforations and obstruction. Complete perforation, although unlikely, is very life threatening and must be addressed emergently.

Less severe and more frequently seen complications include malpositioned IUDs that are not perforating the myometrium, dysmenorrhea, amenorrhea, and normal pregnancy.⁵ Risk factors for malpositioning include obesity, prior uterine window or rupture, or copper IUD placement.⁶ Malpositioned IUDs are most frequently found in the lower uterus and can cause abnormal bleeding and pain, as well as increase the risk of pregnancy.⁷ Not all malpositioned IUDs require removal, but it is suggested due to possible lack of contraception. A transvaginal ultrasound or, less commonly, a CT scan can accurately diagnose malpositioning. If the IUD has partially perforated the myometrium and the strings are visible, removal by pulling the strings should be performed. If it is not easily removed, then removal under anesthesia is generally indicated. This patient had several risk factors that could have influenced the perforation of her IUD, including placement within eight weeks of cesarean, breastfeeding during placement, and prior vaginal deliveries.

**Case Conclusion**

While in the hospital, the patient underwent a successful hysteroscopy and removal of the IUD. The IUD had partially perforated her myometrium but remained in the uterus. There were no further complications postoperatively, and the patient was subsequently lost to follow up. Although rare, complications with IUDs do occur and can require emergent diagnosis and management. ED physicians should consider IUD complications in patients who present to the ED with abdominal pain, and note that patients may not voluntarily report an IUD as part of their medical history unless specifically asked.

References available online
One of the rare but feared complications of oral contraceptive pills (OCPs) containing estrogen is hemorrhage from an enlarging hepatocellular adenoma. The risk of developing hepatocellular adenomas in the absence of contraceptives is 1 in 1 million. However, the risk increases 30-40 fold with the use of OCPs, with the risk of significant bleeding ranging from 25-64%. Additionally, the larger the mass, the larger the risk of bleeding. In this case, we present a patient who suffered from the rupture of one of her hepatic adenomas while on Nexplanon® (etonogestrel), a progestin-only oral contraceptive that is implanted subdermally and lasts for up to three years.

Case

An 18-year-old female was referred to the ED from an urgent care facility with severe, constant, unrelenting back pain. On the urgent care provider’s initial assessment, the patient denied any abdominal pain. However, she had an episode of non-bloody, nonbilious emesis immediately upon palpation of her epigastrium.

At the urgent care facility, the patient’s laboratory studies revealed leukocytosis of 22,000 cells/mm³ and hypokalemia, with a potassium level of 2.8 mmol/L. Due to the concerning findings on initial assessment, the patient was transferred to the ED for further evaluation.

Upon the patient’s arrival to the ED, she reported severe abdominal pain that increased upon standing. The patient denied any relevant past medical or surgical history. When specifically asked, she denied any non-steroidal anti-inflammatory drug use, alcohol use, or family history of biliary colic. The patient’s only reported medication was a Nexplanon® implant.

The patient’s vital signs were within normal limits upon initial evaluation. Her exam was remarkable for severe epigastric and right upper quadrant abdominal tenderness with voluntary guarding. The patient’s repeat complete blood count (CBC) revealed leukocytosis of 25,000 cells/mm³ with an elevated absolute neutrophil count to 20. Her hemoglobin was found to be 9.2 gm/dL with no baseline for comparison. Comprehensive metabolic panel revealed mild transaminitis with AST and ALT values of 149 IU/L and 176 IU/L, respectively. Potassium was found to be 3.3 mmol/L. In addition, the patient had an elevated alkaline phosphatase of 131 IU/L. Bilirubin was unremarkable. Urine studies revealed red blood cells, and a pregnancy test was negative.

Point-of-care ultrasound of the patient’s right upper quadrant revealed a small amount of intra-abdominal free fluid in the right upper quadrant. Ultrasound did not identify any gallstones, pericholecystic fluid, or gallbladder wall thickening. However, there was an abnormal-appearing echogenicity to the liver (see figures). A stat CT scan of the abdomen and pelvis was ordered for further investigation.
While awaiting the results of the CT scan, the patient’s lactate was found to be elevated at 5 mmol/L. At that time, 2 L of normal saline was initiated.

CT scan results demonstrated a large hemorrhagic mass in the liver with contrast extravasation concerning for ruptured hepatic adenoma (see figures). Repeat CBC in the ED demonstrated a drop in hemoglobin from 9.2 gm/dL to 8.1 gm/dL.

Acute care surgery was consulted for further recommendations. The patient was then admitted to the hospital for serial hematocrit checks with plans to take her to the interventional radiology (IR) suite for embolization.

While admitted, the patient underwent Gelfoam® embolization of the right hepatic artery. The patient tolerated it well and transitioned to oral intake prior to discharge the following day, with instructions to follow up with surgical oncology. Chart review performed after the visit revealed that the patient had Nexplanon® implantation four months prior to this incident. No previous history of hepatic adenomas was documented in her previous office visit notes between 2016 and 2020.

Discussion

Ruptured hepatic adenoma is an uncommon diagnosis in the ED and thus not often considered in the initial differential diagnosis for abdominal pain. However, this patient did have one risk factor that should raise every emergency physician’s clinical level of suspicion for this disease process: the use of oral contraceptives.

Hepatic adenomas are known to be either caused or exacerbated by estrogen-containing OCPs. However, this case features a patient who developed liver adenomatosis, defined as more than 3 adenomas, after the implantation of Nexplanon®, a progestin-only contraceptive rod.5

Section 5.7 of the package insert of Nexplanon® includes the following: “It is not known whether a similar risk exists with progestin-only methods like Nexplanon®.”6

We found one other case report in which a patient developed liver adenomatosis with bleeding. However, that patient was previously on an estrogen-containing OCP prior to having Norplant (another progestin-releasing implant) inserted. In this case, though, the patient was not on any contraceptives previously and had Nexplanon® inserted approximately four months prior to her ED visit. The case presentation is in contrast with the common teaching that patients on progestin-only contraception have little to no risk of enlarging hepatic adenomas. *
Acute Urinary Retention

A Rare Case of Incarcerated Uterus in the Gravid ED Patient

When a pregnant woman, especially at 14 to 16 weeks gestation, shows signs of acute urinary retention, consider retroverted incarcerated uterus as a potential cause.

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Background
Uterine retroversion is considered a normal variant in an estimated 15% of first-term pregnancies. In most cases, retroversion self-corrects around the 10th to 14th week of pregnancy as the gravid uterus ascends from the pelvis into the abdomen. In some cases, the uterus becomes impacted between the sacral promontory and symphysis pubis, and the patient commonly presents with urinary complaints such as acute urinary retention or urinary frequency. Prompt diagnosis in the ED is paramount to reducing obstetric and/or urogynecologic complications. We present a case of a retroverted, incarcerated uterus presenting as acute worsening of urinary retention in a gravid ED patient.

Case Report
The patient is a 32-year-old G2P1 woman with an intrauterine pregnancy at 13 weeks gestation by dates. She presented to the ED complaining of urinary retention. Her symptoms had started about a week earlier and had...
worsened over the past 12-24 hours. Her symptoms were initially relieved by positional changes (i.e., lying on her side, standing up, sitting on a yoga ball) but then worsened, prompting her visit to the ED. She denied fevers, chills, vaginal bleeding, or other acute systemic symptoms. Her past medical history was relatively non-contributory but included depression, anxiety, insomnia, and undifferentiated connective tissue disease.

The patient’s vital signs were: temperature 36.8 C; blood pressure 120/69; heart rate 89; respiratory rate 16; and oxygen saturation 100% on room air. A physical examination revealed mild abdominal fullness of the lower abdomen as well as a gravid, non-tender uterus. An initial pelvic exam was deferred until a chaperone could be present.

Soon after arrival, a point-of-care ultrasound revealed a single, live intrauterine pregnancy (IUP) with a low-lying uterus, and a large bladder without evidence of hydronephrosis. Additional point-of-care ultrasound imaging was performed for further evaluation of IUP that was grossly normal and consistent with dates. Urine samples were taken to evaluate for asymptomatic bacteriuria of pregnancy.

Given the urinary retention, a Foley catheter was placed with resolution of symptoms. External genitalia examination with a chaperone present revealed a cervix visible at the level of the introitus, raising concern for uterine prolapse causing urethral hypermobility and resultant urinary retention. The diagnosis of retroverted incarcerated uterus was also considered upon review of the literature. Obstetrics was not immediately available for consultation, but the patient was instructed to follow up with family medicine and her midwife, and therefore was felt to be safe for discharge and outpatient follow-up.

The patient followed up with her obstetrician, and while the radiology ultrasound was unrevealing, a physical examination confirmed the diagnosis. The uterus was unable to be manually manipulated in the outpatient setting, though a subsequent trans-rectal reduction under anesthesia was successful.

With the availability of point-of-care ultrasound (POCUS) in many emergency departments, it is important to note that if there is not high clinical suspicion for incarcerated uterus, imaging studies can be easily misinterpreted, leading to a missed diagnosis.

**Discussion**

Urinary retention is not an uncommon presenting symptom in ED patients. However, urinary retention in a gravid patient is cause for concern. The differential diagnosis in a gravid patient typically includes: infectious causes such as cystitis or urethritis; neurologic causes like multiple sclerosis; medication effect; and uterine prolapse with urethral hypermobility. While it is considered an obstetric emergency, gravid uterine incarceration is rarely included as it is relatively uncommon, occurring in 1 in 3000 pregnancies.

Uterine incarceration occurs when the uterus becomes stuck between the sacral promontory and the symphysis pubis. This typically occurs between the 14th and 16th week of gestation, and the patient commonly presents with urinary symptoms, such as this patient described. The exact mechanism of this process is thought to be from progressive elongation and anterior displacement of the cervix as the gravid uterus expands into the pelvic cavity. This causes obstruction of the bladder and urethra, leading to common presenting symptoms such as urinary retention, dysuria, urgency, and paradoxical incontinence. Other common, non-specific presenting symptoms include: abdominal, pelvic, perineal, or back pain; vaginal bleeding; tenesmus; and constipation. Identified risk factors for this condition include history of prior incarceration, pelvic inflammation, endometriosis, adhesive disease from abdominal surgeries, and uterine anomalies.

Due to the highly non-specific nature of presenting symptoms, gravid uterine incarceration can be an easily missed diagnosis. While historically diagnosed clinically, several case reports have noted the ability of imaging studies such as ultrasound and MRI to augment diagnostic potential.

With the availability of point-of-care ultrasound (POCUS) in many emergency departments, it is important to note that if there is not high clinical suspicion for incarcerated uterus, imaging studies can be easily misinterpreted, leading to a missed diagnosis.

Treatment differs depending on gestational age, though prompt recognition and obstetric consultation are vital to mitigate complications. Given the non-specific presenting symptoms, incarceration of a gravid uterus is rarely diagnosed in the ED setting. However, it is a condition with potentially fatal consequences, and the ED clinician should consider it in the differential for the gravid patient presenting with urinary retention.

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**TEACHING POINTS**

- Consider retroverted incarcerated uterus as a cause for acute urinary retention in gravid patients, especially between 14 and 16 weeks gestation.
- MRI is preferred to ultrasound for diagnosis, and transabdominal is preferred to transvaginal ultrasound, though an incarcerated uterus is best diagnosed through history and physical exam.
- Consult OB if there is concern for this diagnosis, as the patient may require urgent reduction, close follow-up, and monitoring for the remainder of the pregnancy.
Introduction

Although historically thought to be a rare cause of Acute Coronary Syndrome (ACS) and Myocardial Infarction (MI), recent evidence suggests that Spontaneous Coronary Artery Dissection (SCAD) might be the culprit in 1-4% of all ACS cases, in 35% of MIs in women under 50 years old, and in 43% of MIs in the peripartum period. This case reports a rare case of postpartum cardiac arrest secondary to SCAD in a woman with Methylene tetrahydrofolate Reductase (MTHFR) Gene Mutation and Plasminogen Activator Inhibitor type 1 (PAI-1) deficiency.

Case Report

A 37-year-old G5P4A2 female, three days postpartum from spontaneous vaginal delivery of twins at 38 weeks gestation, presented to the ED in cardiac arrest. She was found by her husband with seizure-like activity before becoming pulseless. Her husband started CPR as directed by EMS dispatch and, on arrival, EMS found her pulseless in ventricular fibrillation (VF). Prehospital ACLS interventions included chest compressions, intubation, naloxone, magnesium, multiple rounds of defibrillation, and epinephrine, resulting in brief return of spontaneous circulation (ROSC) with re-degeneration to pulseless electric activity on route.

Return of spontaneous circulation (ROSC) was attained during the initial pulse check in the ED, 42 minutes after being found by her husband. Post-ROSC ECG showed anterolateral ST- elevation MI (STEMI) (figure 1), and bedside cardiac echocardiography (ECHO) demonstrated dilated akinetic left ventricular apex, hypokinetic anterior wall, and no signs of right ventricular...
strain. Arterial blood gas showed pH 6.804, pCO\(_2\) 58.3, pO\(_2\) 83.6, HCO\(_3\) 8.9, base deficit 25.3.

She was given aspirin and heparin while P2Y12 anti-platelet was withheld per interventional cardiology recommendation. Computed tomography angiogram (CTA) did not reveal pulmonary emboli or aortic dissection. Emergent left heart catheterization revealed multi-vessel spontaneous coronary artery dissection involving entire left anterior descending artery (LAD) and its diagonal branch, and the proximal segment of the left circumflex artery (LCX) (figure 2), requiring emergent triple coronary artery bypass grafts (CABG).

Hospital course was complicated by refractory hypoxemia requiring veno-venous extracorporeal membrane oxygenation, multi-vessel deep vein thrombosis, heart failure with reduced ejection fraction (HFrEF) secondary to ischemic cardiomyopathy (left ventricular ejection fraction [LVEF] of 31%), anemia, and consumptive coagulopathy necessitating several blood transfusions. Nonetheless, this patient recovered astonishingly well and was discharged without any neurologic sequelae with a preventative life vest, warfarin, and guideline-directed medical therapy for HFrEF on hospital day 15. Two months later, an echocardiogram demonstrated interval improvement (LVEF 40%), and full-body CTA did not reveal fibromuscular dysplasia. The patient completed cardiac rehab and was happy to be home with her four children.

### Discussion

The mechanism of SCAD is thought to be partially related to inflammatory changes and, in peripartum women, a consequence of hemodynamic changes and hormonal effects on weakening the coronary arterial walls. Unlike atherosclerotic arterial dissections in which expansion is limited by vessel scarring and stiffening, SCAD can rapidly expand given vessel fragility. The spontaneous tear on the intimal layer leads to bleeding of the vasa vasorum creating a false lumen. Eventually the blood forms an intramural hematoma leading to vessel occlusion, and subsequently ischemia.1,2

Predisposing factors for SCAD include female sex, fibromuscular dysplasia (FMD), physical or emotional stress, postpartum status, connective tissue disorders, and multiparity.2 While a recent study of 168 patients with SCAD found FMD in 72% of cases,6 most peripartum cases have been observed in women over 33 years old, underlining the likely link with advanced maternal age.1

In addition to her sex, age, multiparity, and postpartum status, our patient had MTHFR gene mutation and PAI-1 deficiency. MTHFR is the rate-limiting enzyme in the methionine metabolism that breaks down homocysteine. Hyperhomocysteinemia is a known risk factor for cardiovascular disease through oxidative stress and the stimulation of thrombosis, vascular smooth muscle, and collagen.3

Although there have been a few case reports of SCAD associated with hyperhomocysteinemia in literature,4,5 spontaneous cervical artery dissection and aortic dissection have been linked with this gene mutation.2,4 On the other hand, PAI-1, which is known to be associated with ACS, suppresses fibrinolysis via inhibition of tissue plasminogen activator, leading to decreased plasmin production, and consequently thrombosis, which in theory should have been a protective factor for this patient.6

SCAD patients presenting with an acute MI or hemodynamic instability are often treated with revascularization by percutaneous coronary intervention; yet the procedure is exceedingly complex and risky given the instability of the vessel walls. SCADs involving the LAD are often treated with CABG just as in atherosclerotic ischemic MI.

While most patients with SCAD present with non-ST-elevation MI (NSTEMI), 25-50% present with STEMI, with the LAD being most frequently affected, followed by the left main coronary artery (LM).1,3 While postpartum SCAD is a predictor for recurrence, VT/VF at presentation is associated with worse outcomes, including unplanned revascularization, heart failure, need for implantable defibrillator, and in-hospital death.7

Acid-base status is another important outcome predictor in cardiac arrest. A large study reported mean survivor blood pH of 7.22, mean base deficit 8.8 mmol/l, no survivors with pH < 6.72, base deficit > 25 or HCO\(_3\) < 8.6 mmol/l, and only 7.3% of patients with base deficit > 13.2 mmol/l surviving without serious neurological deficits,8,9 making our patient an extreme outlier.

**Take-home Points**

- SCAD is a separation of the coronary arterial walls that often leads to ACS in the absence of atherosclerosis or trauma.
- While seldom diagnosed, it is associated with cardiac arrest in the young, particularly females in the peripartum period.
- Fortunately, most patients will have an elevated troponin with the ACS symptoms, leading to the same management.
- In suspected SCAD, P2Y12 anti-platelets should be avoided in the ED as it can preclude a patient’s eligibility for CABG.
Introduction

In the United States, more than 350,000 out-of-hospital cardiac arrests occur each year. One-third present to ambulance services with ventricular fibrillation (VF) or pulseless ventricular tachycardia. VF is often the result of significant left coronary artery disease (>70% stenosis), and there is a dismal survival rate of 8-15% when refractory to treatment. Cardiac catheterization can provide definitive treatment, however, patients often die before reaching the cardiac catheterization lab. Extracorporeal cardiopulmonary resuscitation (ECPR) involves the application of percutaneous veno-arterial extracorporeal membrane oxygenation (VA-ECMO) for management of cardiac arrest in the emergency department. ECPR functions as a bridge therapy, enabling temporary systemic organ perfusion until the cause of cardiac failure can be definitively treated. We present a case of ECPR-enabled cardiac stenting and post-arrest care, leading to a full neurological recovery in a patient who otherwise may not have survived refractory VF.

Case Report

A 54-year-old man walked into the emergency department waiting room complaining of chest pain. Suddenly, he collapsed into the arms of a triage nurse and turned ashen gray and unresponsive as he was hoisted onto a stretcher and wheeled back to the resuscitation bay. Cardiopulmonary resuscitation (CPR) began promptly when the team was unable to palpate a pulse. Per brief history from the patient’s wife, he had developed chest pain early in the day and drove himself to the emergency department. He had a history of hypertension and no prior cardiac disease. Cardiac monitoring revealed ventricular fibrillation and the patient was defibrillated with 200 joules while resuscitation efforts began. During the first several minutes of resuscitation, the patient drifted in and out of consciousness, pushing away CPR providers as he did. This is a phenomenon described in medical literature as cardiopulmonary resuscitation-induced consciousness (CPRIC), an indicator of good cerebral perfusion and CPR. With each sign of CPRIC, compressions were immediately stopped and pulse checks were performed. However, there was no palpable pulse and the patient lost consciousness shortly after cessation of CPR. He received multiple defibrillations, epinephrine, and amiodarone. He was intubated, and an EKG (Figure 1) confirmed ventricular fibrillation. A second set of defibrillation pads were placed in the anterior-posterior position for dual synchronous defibrillation. This patient was in refractory ventricular fibrillation and a call was made to evaluate the patient for ECPR. The cardiothoracic surgical team began cannulation 52 minutes after cardiac arrest, and full flow VA-ECMO was achieved 58 minutes post-arrest. Due to a high suspicion for ischemia-mediated dysrhythmia, the patient was emergently transported to the cardiac catheterization laboratory where coronary angiography confirmed a 99% occlusion of the left anterior descending artery. This “widowmaker” lesion was successfully treated with one drug-eluting stent, and the left ventricle was decompressed with placement of an Impella (Abiomed, Danvers, MA, USA). Following revascularization, defibrillation was performed with successful and durable conversion to normal sinus rhythm. The patient was admitted to the cardiothoracic intensive care unit for post-arrest care. The VA-ECMO circuit enabled targeted temperature management for the first 24 hours.
Echocardiography revealed severe left ventricular dysfunction with an ejection fraction of 10-15%. The Impella enabled ventricular decompression, allowing the myocardium to rest and recover. On day three, repeat echocardiography showed a recovered ejection fraction of 45% and the patient was successfully decannulated. The patient’s care continued in the medical intensive care unit where he returned to his pre-arrest neurological baseline and was discharged home.

**Discussion**

**What are the indications for ECPR?**

ECPR is a bridge therapy for patients with cardiac arrest refractory to ACLS. ECMO has been employed in a range of cardiac and respiratory failure patients with reversible etiologies; e.g. obstructive coronary artery disease, massive pulmonary embolism, COVID-19, acute respiratory distress syndrome, hypothermia, electrolyte derangements, cardiotoxicity due to drug overdose, and submersion injuries. VA-ECMO and more specifically, ECPR, has been used and studied predominantly in VF, caused by left heart disease in 87% of cases. When VF is refractory to traditional advanced cardiac life support, the survival rate is extraordinarily low (8-15%). While clinical gestalt, experience and team readiness continues to play a large role in present day application, ECPR indications include: young age, witnessed arrest, bystander CPR, initial shockable rhythm, correctable causes such as a cardiac etiology, and no return of spontaneous circulation within three defibrillations. Patients in refractory ventricular fibrillation often have reversible etiologies and represent a large population who benefit from well-developed ECPR programs.

**Which patients are ideal candidates for ECPR?**

ECPR should be considered for patients in cardio-pulmonary arrest with both a reversible etiology and a high likelihood of return to a comparable neurological baseline. Ideal candidates are younger in age and have fewer comorbidities, witnessed cardiac arrest, shockable initial rhythm on cardiac monitoring, brief down-time, and brief time to cannulation. These variables factor into the SAVE score, a tool developed and externally validated to predict survival for patients receiving ECMO for refractory cardiogenic shock. Studies in the last decade have shown up to 54% survival rates with full neurological recoveries in out-of-hospital cardiac arrest patients in refractory VF. Most recently, The University of Minnesota completed the ARREST trial, a small RCT to evaluate ECPR versus standard ACLS. The trial was stopped early when clear benefit was observed in the ECPR arm versus standard ACLS. While small, this is the first RCT to evaluate the efficacy of ECPR over standard of care. Contraindications in the literature, while not absolute, include patients with poor physical activity levels, permanent neurological injury, noncardiac cause of arrest, prolonged CPR without return of spontaneous circulation, and aortic injury such as dissection.

**What times matter in ECPR resuscitation?**

Favorable neurological outcomes with ECPR are achieved by minimizing downtime, known as no-flow, and time to cannulation, known as low-flow. Out-of-hospital arrests that are witnessed and have prompt initiation of bystander CPR minimize no-flow time and have better outcomes. The literature highlights optimal outcomes when no-flow is less than five minutes, and low-flow is less than 60 minutes. Few institutions have even developed mobile ECPR centers with a goal of under 30 minutes from arrest to cannulation.

**How does the heart recover?**

VA-ECMO helps stabilize patients with cardiogenic shock, however, there are significant physiological changes that occur due to the retrograde flow towards the aortic valve. This physiologic alteration elevates afterload pressures that the injured ventricle must overcome. Ventricular over-distension can cause pulmonary edema, stasis, arrhythmias, and increased wall stress and myocardial oxygen demands.

Subendocardial ischemia worsens LV function and recovery. To prevent ventricular over-distension, clinicians recommend left ventricular venting using medical therapy, a surgical vent, or a percutaneous ventricular assist device. These devices, such as the intra-aortic balloon pump, Impella, or TandemHeart, optimize flow and enable myocardial recovery. In this patient, myocardial recovery occurred within the first three days and the patient was able to be safely decannulated with an ejection fraction of 45%. Per analysis of the Extracorporeal Life Support Organization registry, there is an increasing chance of survival up to four days, then a decreasing chance to day twelve. Thus, patients that survive the early period are more likely to survive if weaned shortly thereafter. Three quarters of patients receive treatment for less than one week.

**Conclusion**

ECPR can be a life-saving therapy in refractory ventricular dysrhythmias. This case report provides an example of successful implementation of ECPR from the prehospital arena to the intensive care unit. We believe this report can serve as an introduction for providers who wish to better understand ECPR, and for emergency departments developing multi-disciplinary ECPR pathways.
We present an uncommon case of a total knee arthroplasty (TKA) dislocation. Knee dislocations are rare, representing a mere 0.02-0.2% of orthopedic injuries in the general population. In the even smaller subset of patients with TKAs, the risk of knee dislocation is 0.15–0.5%. As uncommon as these injuries are, they constitute a true emergency; what may appear at first glance to be a simple joint dislocation could be hiding more serious neurovascular damage.

**Case**

A 72-year-old male with a history of autoimmune hepatitis status post-transplant and, nine years earlier, a left TKA, was brought in by EMS to the emergency department shortly after a mechanical trip and fall. The patient stated that while taking out the garbage, he fell and landed on his left leg. He experienced no pain but was then unable to stand. On exam, the patient was hemodynamically stable. His left knee was immobilized in a splint from EMS and demonstrated gross deformity with a positive sag sign (Image 1). There were no breaks in the skin. Distal pulses and sensation were intact, and the patient was able to actively plantar and dorsiflex.

Differential diagnosis included...
After reduction, the neurovasculally intact knee should be immobilized in full extension until outpatient follow-up. Even with non-operative management, a return to full activity takes months of physical therapy and rehabilitation.
Recognizing and Managing Epidural Abscess

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Unlike in the adult population, back pain is an uncommon chief complaint in the pediatric population, representing 0.4% of all emergency department encounters in one study.\(^1\) It is estimated that up to 77% of back pain presentations in the pediatric population are due to benign causes.\(^2\) These benign causes include muscle strains, back contusions, and sports-related injuries. However, there are more serious, but less common, causes to be considered in adolescents with back pain, especially when patients present with fever and labs point to a possible infection.

Epidural abscess is a rare but serious condition that should be considered in patients with fever and localized back pain. Failure to appropriately diagnose and treat epidural abscess in a timely manner can cause significant irreversible neurological deficits.\(^3\)

Case Report

A previously healthy 13-year-old male presented to the emergency department with right flank pain. The pain had started the night prior, initially located between his shoulder blades, and had migrated to the right flank by the time of presentation. The pain was constant but worsened by inspiration. He also had a home temperature of 100.2°F. He had taken 200 mg of ibuprofen overnight without relief. He denied trauma or previous similar episodes of flank pain. He also denied nausea, vomiting, abdominal pain, dysuria, or hematuria.

His triage vitals were significant for an oral temperature of 100.2°F, heart rate of 121, and a respiratory rate of 22. Blood pressure was normal at 123/61, and the patient had a peripheral oxygen saturation of 100% on room air. His initial exam was significant for right costovertebral angle tenderness and mild right lower quadrant abdominal tenderness.

His initial workup included a CBC showing mild leukocytosis at 13,500/mm\(^3\) with a neutrophilic predominance, an unremarkable complete metabolic panel, and a urinalysis that only showed trace protein and no hematuria. COVID and influenza PCR tests were negative, although a Covid-19 spike antibody test was positive, indicating prior infection. An initial abdominal X-ray and abdominal ultrasound did not show any abnormalities. At this point, the patient was admitted to the hospital for further diagnostic workup. A CT scan of the abdomen and pelvis with contrast showed only mild bilateral atelectasis and possible infiltrates at lung bases. It was negative for nephrolithiasis or appendicitis.

At this point, a presumed diagnosis of community acquired pneumonia was made, and his back pain was ascribed to general myalgias in the setting of his pneumonia. The plan was to discharge the patient on amoxicillin and azithromycin with outpatient follow-up. However, the patient’s pain worsened in his back and right flank. He also remained tachycardic and spiked a fever to 104.8°F so he remained in the hospital for observation. He later developed nausea and had 4 episodes of non-bloody emesis. On day 2 of hospitalization, the overnight team noted moderate tenderness to palpation of the spine from the upper thoracic to the sacral area. At this point, blood cultures and an MRI of the spine were ordered.

The MRI findings were significant for a moderate-sized epidural abscess extending from the T3 to T10 level, along with a paraspinal soft tissue abscess near the spinal canal at the T10 level. The patient then underwent an aspiration procedure with interventional radiology where a 10 Fr drain was placed under ultrasound guidance at the soft tissue abscess site. Neurosurgery was consulted, and they did not recommend surgical intervention in the absence of neurological deficits.

Upon recognition of the paraspinal and epidural abscess, he was started on ceftriaxone and azithromycin. Blood cultures came back positive for MSSA bacteremia, which was also consistent with abscess drain cultures. The patient was switched to IV nafcillin after day 4 of hospitalization.

Infectious disease was consulted and discovered that the patient had a distant history of a submental abscess 5 months ago, which was treated with antibiotics.
Rare, but possible, diagnosis of epidural abscess must be considered when a pediatric patient presents with back pain. When diagnosed, carefully evaluate the risks and benefits of medical versus surgical management.

**Discussion**

Pediatric spinal epidural abscess is an extremely rare infection of the central nervous system that can have significant neurologic sequelae if not diagnosed and treated in a timely manner. Its prevalence is estimated to be 0.6-1.5 per 10,000 admissions. This condition is characterized by pus in the space between the dura and the periosteum of the vertebrae. The presenting symptoms are commonly nonspecific, which makes spinal epidural abscess a challenging diagnosis that requires a high index of suspicion. The classic triad of symptoms is fever, localized back pain, and neurological deficits. Although highly specific, patients rarely present with all three symptoms at the initial encounter, and this triad is found only in about 13% of patients with a spinal epidural abscess. Neurological deficits appear later in the course of the disease if not treated appropriately. It is important to have a high clinical suspicion for epidural abscess in certain patients, even in the absence of neurological symptoms.

Some of the risk factors associated with spinal epidural abscess are diabetes mellitus, alcoholism, immunodeficiency syndrome, IV drug use, and malignancy. Since these risk factors are more predominant in the adult population, most cases of epidural abscesses occur in adults. Spinal epidural abscess occurrence in the pediatric population is not only extremely rare, but also the clinical presentation can be very nonspecific. In our patient, he initially complained of right flank pain in the emergency department. Initial concerns included pyelonephritis, urinary tract infection, or nephrolithiasis, although his unremarkable urinalysis made these diagnoses less likely. Because he had leukocytosis and a fever, an infectious etiology was suspected, and the patient was admitted for further evaluation.

Our patient did not have any predisposing risk factors or neurological complaints. However, he did have a history of submental abscess formation 5 months prior. There are laboratory studies that can increase the clinical suspicion for epidural abscess, such as erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP). In a meta-analysis, it was found that ESR was elevated in 94% of patients diagnosed with epidural abscess. Although we did not have an ESR available for our patient, his CRP was elevated at 181.2mg/L.

The most common causative agent in epidural abscess is *Staphylococcus Aureus*. The blood cultures and abscess cultures of our patient were both positive for Methicillin Sensitive *Staphylococcus Aureus* (MSSA). The goal of management is early decompression of the site of maximal compression to prevent neurologic deficits as well as isolating the causative agent. The decision for medical versus surgical management is somewhat controversial. One study found that more than 40% of patients who were treated medically later required surgical management due to failure of treatment. Factors associated with medical treatment failure include: diabetes, CRP greater than 115 mg/L, white blood count (WBC) greater than 12,500/mm, and bacteremia. Our patient had an elevated CRP, WBC, as well as bacteremia, which all made medical management unlikely to be successful. Consequently, he underwent drainage of his abscess by interventional radiology, and was started on 4-6 weeks of antibiotic treatment. On his follow-up visits with the infectious disease clinic, he was found to be neurologically intact and clinically improving.

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Missed Ruptured Abdominal Aortic Aneurysm in a Paraplegic Patient

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Introduction

Aneurysms develop as a result of degenerative breakdown of the arterial wall, brought on by a loss in collagen and elastin fibers. AAAs classically present with abdominal or flank pain, hypotension or shock, and a pulsatile mass. However, more than half of cases lack one or more of these characteristics, and the first presentation may be the aneurysm rupture itself. The relatively nonspecific clinical presentation has resulted in AAAs commonly going undetected or being mistaken for other disease processes, leading to an often lethal outcome.

Without surgical intervention, which is generally recommended if the aneurysm is greater than 5.5 cm, AAAs can reach almost 100% mortality.

The rising prevalence of bedside ultrasound and implementation of the Rapid Ultrasound for Shock and Hypotension (RUSH) exam in most emergency departments has, fortunately, led to better detection of AAA quickly at bedside. Here, we describe a case of a missed ruptured AAA (rAAA), measuring 7.5 cm, in a paraplegic patient not fitting the classic demographic presenting in shock, where the RUSH protocol likely would have reduced delays in care and improved the outcome.

Case

A 54-year-old male with a history of hypertension and T2 paraplegia secondary to a gunshot wound many years ago presented to the ED via ambulance for evaluation of fatigue with associated nausea and decreased appetite. Per patient and paramedics, the fatigue has been ongoing for the past five days. He normally could transfer himself from the bed to the wheelchair, but was unable to do so for the past two days. He also performed intermittent straight catheterizations as a result of chronic urinary retention. Otherwise, the patient denied any fevers, chills, cough, dyspnea, syncope, new focal weakness or numbness, vomiting, diarrhea, or blood in stool. Regarding his T2 paraplegia, he endorsed no sensation or motor function below T2. He denied any surgical or pertinent family history.

He admitted to daily chronic alcohol and tobacco abuse, but no illicit drug use. On ED presentation, the patient was afebrile, mildly tachycardic at 105 beats per minute, hypotensive with a blood pressure of 80/44 mmHg, and saturating well on room air. Physical examination revealed an alert patient, answering questions and following commands appropriately, with baseline neurologic deficits of lower extremity paralysis and lack of sensation below T2. His abdomen was soft and nondistended without peritoneal signs or palpable masses. The patient’s lower extremity compartments were soft to palpation with 2+ dorsalis pedis pulses.

Given his initial vital signs, the patient was activated for sepsis with our rapid response team. Bedside ultrasound revealed a hyperdynamic left ventricle with complete collapse of the inferior vena cava upon inspiration and absent B-lines on all lung fields. As a result, he was administered 30 mL/kg of intravenous fluids and started on empiric Zosyn and Vancomycin. Status post-administration of intravenous fluids, his blood pressure normalized to 122/70 mmHg, but the patient became bradycardic with a heart rate dropping into the 40s. Given his lack of appetite over several days, there was concern for metabolic/electrolyte derangements which were confirmed by a point-of-care
potassium level of 9.2 mmol/L and peaked T waves on electrocardiogram. He was placed on transcutaneous pacer pads and administered medications to temporize his hyperkalemia (calcium gluconate, sodium bicarbonate, albuterol, furosemide, and insulin with dextrose). The patient’s bradycardia was noted to have resolved after these medications as well.

On laboratory analysis, a complete blood count was significant for leukocytosis with a white blood cell count of 27.8 (normal range: 4–10 x 10^9 per L) and anemia with hemoglobin levels of 8.7 g/dL (normal range: 13–16.5 g/dL). Comprehensive metabolic panel was significant for hyperkalemia of 9 mmol/L (normal range: 3.6–5 mmol/L) as well as acute renal failure with an estimated glomerular filtration rate of 6.23 mL/min/1.73m² (normal range: ≥ 60 mL/min/1.73m²), creatinine of 9.4 mg/dL (normal range: 0.70–1.3 mg/dL) and BUN of 84 mg/dL (normal range: 7–18 mg/dL).

Due to the patient’s bladder scan having revealed no urine output and a repeat potassium at 8.3 mmol/L, the decision was made to consult nephrology for emergent hemodialysis and admit the patient to the medical intensive care unit (MICU) for further management. While boarding in the ED, hemodialysis was initiated overnight.

Once hemodialysis was completed, he was deemed stable to obtain a computed tomography (CT) scan of his abdomen/pelvis to investigate the acute renal failure. After returning from the CT scanner, the patient began to become more tachycardic and borderline hypotensive; nurses also noted faint or absent dorsalis pedis pulses with cool extremities. Radiologists immediately notified our providers that the CT scan revealed a 7.5 cm fusiform abdominal aortic aneurysm (Figures 1, 2, and 3), with surrounding retroperitoneal hemorrhage concerning for abdominal aortic aneurysm rupture. It was suspected that the dialysis worsened the ongoing rupture given his hemodynamic decompensation. The MICU team immediately consulted vascular surgery, and the patient was subsequently taken to the operating room for abdominal endovascular aneurysm repair (EVAR). Although the EVAR was performed successfully, the patient’s right lower extremity was unfortunately unsalvageable secondary to acute limb ischemia and underwent cryoamputation. The patient was hospitalized for the next two months and ultimately discharged to a long-term care facility.

**Discussion**

An aortic aneurysm is defined as a weakened aortic wall that has dilated by over half of its original diameter, most commonly occurring between the renal and inferior mesenteric arteries. The single most important risk factor for developing AAA is a history of smoking, with age greater than 65, being of the male sex, and having family history of AAA following close behind. Additional risk factors include uncontrolled hypertension, coronary artery disease or atherosclerosis, and previous MI.

While the overall incidence of AAA has been decreasing, attributed in part to improvements in tobacco use and dietary changes, mortality rates secondary to rAAA have remained high at almost 100% without immediate surgical intervention. As aneurysm diameter increases, so does risk of rupture, with 7 cm aneurysms exhibiting a 50% chance of rupture.

**Physical Exam**

Although AAA has been documented with a classical presentation of severe abdominal or back pain, hypotension, and a pulsatile abdominal mass, this triad is rarely clinically seen in its entirety. Many patients present to

![FIGURE 1](image1.png)

![FIGURE 2](image2.png)
the ED only after the aneurysm has ruptured, yet still demonstrate vague and nonspecific symptoms leading to a missed diagnosis of AAA. On physical exam, emergency physicians should assess for pulsatile abdominal masses, increased tenderness over the aneurysm, and signs of lower extremity hypoperfusion such as weakened distal pulses, loss of hair, or non-healing foot wounds. A comprehensive physical examination should also include inspection for signs of associated aneurysms including iliac artery aneurysms, which are the most common, and peripheral aneurysms.

Ultrasound Screening

With sufficient screening and early detection, AAA mortality can be decreased to about 1-2%. According to the U.S. Preventive Services Task Force, all men between the ages of 65 and 75 who have ever smoked should undergo a one-time AAA ultrasound screening. Conversely, screening is not recommended for those who have not smoked or for women at all.

Thus, performing a dedicated ultrasound exam in the ED can offer a noninvasive yet sensitive screening modality for detecting a dilated abdominal aorta in at-risk patients.

Physical evaluation for AAA in the case of our paraplegic patient was insufficient due to the lack of sensation below the T2 vertebral level. Furthermore, our patient initially presented without any pulsatile mass and had strong distal pulses in the lower extremities. In this instance, the RUSH exam would have saved valuable time in identifying the patient’s AAA prior to obtaining a diagnostic CT. A study by Moore et al reaffirms this by indicating that bedside ultrasound screening by an experienced emergency physician takes no more than 3 minutes of scan time, and measured aneurysm diameter by ultrasound was in line with diagnostic imaging results.

RUSH Exam

The RUSH exam consists of evaluating the heart, intravascular status, and large arteries in the setting of shock. More specific to this particular case, however, is how the RUSH exam dictates visualization of the abdominal aorta. Following RUSH protocol, emergency physicians can utilize a phased-array or curvilinear transducer in the transverse orientation to visualize the abdominal aorta as a circular vessel anterior to the vertebral column. Moving the transducer from the xiphoid process inferiorly to slightly below the umbilicus, while applying constant pressure to displace bowel gas, will allow for proper visualization of the abdominal aorta. It is imperative to pay special attention to the infrarenal aorta as this is where most AAA are located. While ultrasound can provide valuable insight on the dilation of the aorta, it is limited in its ability to discern whether the aneurysm has ruptured due to the aorta being a retroperitoneal organ.

In a hemodynamically stable patient, CT and angiography is appropriate to evaluate for leakage of blood. However, hypotensive patients should be assumed to have acute rupture and sent for emergency repair. A study by Kuhn et al revealed that even emergency physicians with limited training and experience in ultrasound techniques can successfully identify the presence — or lack thereof — of AAA, further emphasizing the utility of the RUSH protocol.

AAA in Setting of Paralysis

Literature review revolving around AAA in patients with previous spinal cord injuries is rather limited. A retrospective study conducted by Jacobs et al through the U.S. Department of Veterans Affairs found that out of 31 patients identified, none presented with symptoms associated with AAA and that the finding of AAA was largely incidental. Jacobs et al noted that while delayed diagnosis of an AAA could be due to the sensory deficits and the lack of pain perception associated with an aneurysm, patients with sensory-motor deficits are also predisposed to develop complications that would warrant imaging on routine visits. The increased number of abdomen/pelvis imaging and work-up for complications such as nephrolithiasis, urinary tract infections, or pressure sores could play a role in an increased incidental diagnosis of AAA when patients present for unrelated complaints. This retrospective study concluded that AAA repair in patients with longstanding spinal cord injuries can be performed by minimal added mortality. Namely, hemodynamic instability was deemed to not be problematic as these patients present with a compensated degree of lower extremity vasodilatation. Furthermore, the lack of pain perception in patients such as those identified by Jacobs et al, and in our own case, serves to reinforce the utility of the RUSH exam as a valuable tool in risk-stratifying AAA.

TAKE-HOME POINTS

1. AAAs are generally asymptomatic before rupture and often lethal due to delays in diagnosis and care, as most are missed for alternative diagnoses before hemodynamic compromise occurs.
2. Traditional physical exam findings can be unreliable in diagnosing AAA ruptures; this was the case with our paraplegic patient who had no sensation below the T2 level.
3. The RUSH exam is a rapid ultrasound protocol that can be easily performed by emergency physicians for evaluation of undifferentiated shock.
4. CT scans are the imaging modality of choice, but ED ultrasound is readily available and has high sensitivity and specificity in diagnosing AAA. This may reduce delays in care, especially in the setting of hemodynamic instability.
5. Most AAA cases rupture into the retroperitoneal space, making ultrasound less useful in identifying AAA after rupture.
There’s an Eagle in my Throat!

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A 30-year-old male with a history of hypertension presented to the ED with a foreign body sensation in his throat. He denied eating anything recently and did not recall something that may have gotten stuck. He was swallowing well and tolerating secretions. He stated that he tried carbonated drinks and water, but nothing alleviated his symptoms. He also reported an occasional burning sensation. His vitals on arrival were: HR 65; RR 16; temp 36.7°C; BP 197/136; and O2 100%.

Physical examination revealed a patient whose airway was patent with no pooling of secretions or stridor. Oropharynx was non-erythematous without signs of uvular deviation or peritonsillar abscess. No masses or swelling palpated in the submandibular space or around the neck.

Soft tissue neck radiograph was ordered to evaluate for potential foreign body and demonstrated a calcified stylohyoid ligament. This is associated with a foreign body sensation called Eagle’s Syndrome.

**Discussion**

Eagle’s Syndrome was described by otolaryngologist Dr. Watt W. Eagle in 1937. It is characterized by calcified stylohyoid ligament or an elongated styloid process. The average length of the styloid process varies between 20mm and 30mm in adult Caucasians, and between 15.4mm and 18.8mm in the Asian population. Some estimates suggest that approximately 4-7% of the general population have elongated styloid processes, with about 4-10% of them being symptomatic. This process can occur bilaterally or unilaterally. Females have an increased likelihood of presenting with Eagle’s Syndrome, and it is more common among patients older than 50 years. It can also be seen in patients after throat trauma or tonsillectomy.

The causes of elongation are unclear, but include: 1) congenital elongation, 2) calcification of stylohyoid ligament due to an unknown process or associated with conditions increasing heterotopic calcification, such as in disorders of calcium or phosphorous metabolism or patients with chronic renal failure, or 3) growth of osseous tissue at the insertion point of the stylohyoid ligament.

Eagle initially described two types of syndromes: 1) classic styloid and 2) carotid artery. The former is associated with cranial nerve impingements related to compression of CN V, VII, IX, X. This was more commonly described after tonsillectomy as a result of fibrous tissue distorting nerve fibers in the tonsillar fossa. The latter is associated with vascular or ischemic pain as well as sympathetic plexus irritation, and was not associated with tonsillectomy.

As mentioned above, presentation of Eagle’s Syndrome can range in symptomatology. Some described symptoms include difficulty swallowing, foreign body sensation, craniofacial pain from neurovascular irritation, pain on neck rotation, hypersalivation, voice changes, otalgia, tinnitus, and headache or dizziness if the carotid artery is compressed. Furthermore, compression of perivascular sympathetic fibers may lead to a presentation of Horner Syndrome. As these symptoms are nonspecific and may mimic other clinical presentations, it is important to rule out life-threatening etiologies and consider other causes and assess for airway compromise.

CT or radiographs can both be helpful in diagnosis. CT — especially a 3D-CT scan — is considered superior, given its ability to provide accurate evaluation of the styloid process, its anatomic relationship to surrounding neck structures, and for surgical planning. CT will demonstrate a calcified styloid process. Radiographs may include panoramic or lateral views. Radiographs will demonstrate a calcified structure extending from the base of the skull toward the hyoid bone. On physical exam, the styloid process may be palpated in the tonsillar fossa, as styloid processes that are normal in length are usually not easily palpable. Palpation of styloid process may elicit pain or exacerbation of symptoms.

Definitive treatment is surgical, requiring styloidectomy to shorten the styloid process via an intraoral or extraoral approach. The intraoral approach has been widely criticized given its increased risk of deep neck space infections and poor visualization of surgical field as it is performed through the mouth. The extraoral approach is preferred, although it may lead to external scarring or facial nerve injuries. Surgical styloidectomy has a cure rate of about 80%. More conservative, medical management can be guided by symptomatology. Medication includes pain control with NSAIDs, anticonvulsants such as carbamazepine or gabapentin, and SSRI. Transphyaryngeal injection with steroids and anesthetics have also been shown to help with pain. In our case, we also suspected our patient may have a gastroesophageal reflux component, and therefore treated the patient for such.

**Case Conclusion**

Findings were discussed with the patient, and he was discharged with famotidine and ENT follow up.
Patient Boarding During COVID-19

A Literature Review

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“Boarding” is a term used to describe the phenomenon of patients remaining in the emergency department for an extended period after the ED care team has decided to admit or transfer them. While boarding has been shown to have negative impacts on patient care, those with mental health disorders are particularly vulnerable to the effects of boarding, as most EDs are not equipped to handle the unique challenges of caring for the mentally ill patient for a prolonged period of time in a chaotic environment with limited resources.

In December 2015, The Joint Commission released a QuickSafety article stating that psychiatric boarding in the ED was leading to multiple problems when it came to treating both psychiatric and medical patients. Some of the problems included: increases in psychological stress on patients who may already be in depressed or psychotic states; worsening ED crowding; and delays for other ED patients, some of whom had life-threatening conditions.

Since the beginning of the COVID-19 pandemic, emergency departments across the country have seen a well-documented increase in patient volume and boarding. Though the study of psychiatric boarding since the pandemic began is lacking, available data indicates likely challenges ahead.

Review of Literature

1) Mass. Physicians Call On State to Address ER ‘Boarding’ of Patients Awaiting Admission (Jolicoeur & Mullins³)

In February 2021, WBUR News released an interview with Dr. Jesse Rideout, president of the Massachusetts chapter of the American College of Emergency Physicians (MACEP). The data provided by MACEP showed that psychiatric boarding time in the ED had increased by more than 25 hours between July 2019 and January 2021 (Figure 2). For pediatric behavioral health patients alone, the average wait was even longer at 59 hours. And the total number of hours that psychiatric patients had boarded in emergency departments had increased from 4,555 hours in January 2019 to 10,527 in January 2021 (Figure 1), marking the highest total that MACEP had ever recorded, according to Rideout. The MACEP data also showed that 25% of ED beds were occupied by boarding psychiatric patients awaiting an inpatient bed at any given time.

2) ED Visits for Suspected Suicide Attempts Among Persons Aged 12–25 Years Before and During the COVID-19 Pandemic — United States, January 2019–May 2021 (Yard et al⁴)

The CDC reported during 2020 there was a 31% increase in the proportion of mental health–related ED visits among adolescents aged 12–17 years. The CDC also noted in May 2020 there was an increase in ED visits for suspected suicide attempts among the same age range. In 2021, the month between February 21 and March 20 saw a 50.6% increase in suspected suicide attempts among girls ages 12-17 years, compared to the same time period in February and March 2019.

3) Trends in U.S. Emergency Department Visits for Mental Health, Overdose, and Violence Outcomes Before and During the COVID-19 Pandemic (Holland et al⁵)

This national cross-sectional study was made using data from The National Syndromic Surveillance Program, which captures approximately 70% of U.S. ED visits from more than 3,500 EDs that cover 48 states and Washington, DC. The study looked into the ED visit changes for mental health conditions (MHCs), suicide attempts (SAs), overdose (OD), and violence outcomes from Dec. 30,
Acute Inflammatory Demyelinating Polyneuropathy After COVID-19

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A 49-year-old male with no significant past medical history presented to the emergency department reporting generalized weakness, with lower extremities weaker than upper extremities and bilateral facial numbness. The patient had multiple falls without loss of consciousness or head trauma and described lower extremity weakness that had progressed to his upper extremities. He denied recent fever, chills, chest pain, shortness of breath, nausea, vomiting, diarrhea, or abdominal pain. He also denied recent travel. His symptoms began after having COVID-19 in November 2020, two months prior to his ED visit.

The patient had unremarkable CT head, CTA head and neck, CBC, and CMP, but had an elevated ESR, CRP, and CK. Due to concern for post-COVID myositis or Guillain-Barré syndrome (GBS), the patient was admitted to neurology for further workup. The patient’s EMG/NCS was supportive of a subtype of GBS known as acute inflammatory demyelinating polyneuropathy (AIDP), then confirmed by lumbar puncture. Further workup included multiple sclerosis panel with elevated IgG index and positive oligoclonal bands, unremarkable flow cytometry, and negative ganglioside antibody panel. The patient started plasmapheresis and intravenous immunoglobulin. Overall improvement was seen in the patient’s global weakness after IVIG and plasmapheresis. The patient was later discharged with outpatient physical therapy.

Discussion
GBS is an acute immune-mediated polyneuropathy and is one of the most common causes of acquired weakness often provoked by an antecedent infection. Campylobacter jejuni gastroenteritis and upper respiratory infection are the most common triggers of GBS. Since the COVID-19 outbreak in 2020, COVID-19 has been known to cause a myriad of post-infection complications such as stroke and encephalopathy, and multiple cases of GBS/AIDP have been reported in the literature. Although post COVID-19 fatigue and weakness are common and often benign complaints, high suspicion for GBS should be upheld when evaluating these patients.
Favorable Neurologic Outcomes Possible with Prolonged CPR

Abstract
A previously healthy 6-year-old male presented to the emergency department with a submersion injury. Upon arrival, the patient was in cardiac arrest, had a GCS of 3, severe metabolic and respiratory acidosis, and required CPR for a total of 57 minutes prior to return of spontaneous circulation. Despite these findings, the patient had an excellent neurocognitive outcome. A review of the literature of pediatric submersion injuries was performed and showed that our case is unusual because survival after submersion injury complicated by cardiac arrest requiring more than 30 minutes of CPR is extremely rare.

Case
A 6-year-old male was found by his friends submerged in sea water. Family reported he had been submerged for about 10-15 minutes. Lifeguards brought him to shore and noted he was spontaneously breathing but unresponsive. Bag valve mask ventilation was initiated prior to EMS arrival to the scene. When EMS arrived, the patient had a pulse and was breathing spontaneously. Patient was placed on a non-rebreather mask and taken to the emergency department. In route, the patient became pulseless and apneic; initial rhythm was pulseless electrical activity. EMS began CPR, intubated the patient, and arrived at the ED after 15 minutes of CPR.
On arrival, the initial rhythm was PEA. Pediatric Advanced Life Support resuscitation was continued. Vascular access was achieved via IO. Endotracheal tube placement was confirmed. CPR was continued with a total of four rounds of epinephrine 0.01mg/kg. Finally, return of spontaneous circulation was achieved after 42 minutes in the ED. Initial vitals were BP 62/42, temperature 34.3°C, HR 50, O₂ saturation 95%, and GCS 3.

A point-of-care ABG revealed pH less than 6.85, PaCO₂ 72 mmHg, base excess −25, PaO₂ 158 mmHg, and HCO₃ 10 mmol/L (Table 1). Chest X-ray showed diffuse bilateral pulmonary interstitial and airspace infiltrates with predominantly perihilar distribution. Oxygenation to fractional oxygen ratio (PaO₂/FiO₂) was 158, indicating moderate acute respiratory distress syndrome (ARDS).

Ventilator settings were set and titrated according to lung protective ventilation strategies. Initial ventilator settings were tidal volume of 6 ml/kg, RR 25, PEEP 10; and FiO₂ of 100%. Settings were then titrated to a goal oxygen saturation of 88% to 95%.

Bicarbonate pushes at 1mEq/kg were administered to correct the acidosis noted on the initial arterial blood gas. Two lactated ringer boluses at 20cc/kg were given due to hypotension, which improved blood pressure. Shortly after, the patient became hypotensive again and an epinephrine and dopamine drip were started, resolving the hypotension.

Repeat CXR showed worsening of infiltrates consistent with ARDS. Second ABG demonstrated persistent metabolic acidosis with pH of less than 6.85, but improved PaCO₂ from 72 to 56. PEEP was then increased to 18 (Table 1).

The third arterial blood gas showed improved pH of 7.01, and the PaO₂ decreased to 45mmHg, with the SpO₂ range between 50%-80% (Table 1). On exam, an air leak was appreciated and the patient required reintubation. After successful reintubation, the air leak resolved and the SpO₂ improved to 75%-85%.

The patient was clinically improving and after 3 hours in the ED was transferred to a nearby tertiary care center. The receiving hospital continued lung protective ventilation strategies and the repeat blood gas showed an improved pH of 7.20. Repeat chest X-ray showed improvement of ARDS. The patient was weaned to BiPAP on hospital day 7, high-flow nasal cannula on hospital day 10, and on hospital day 14 the patient was stable on room air and no longer required any oxygen support. At a 2-year follow-up, the parents reported that the patient made a complete neurologic recovery and continued to be a straight-A student.

**Discussion**

Drowning is defined as respiratory impairment secondary to submersion or immersion in liquid. Drowning accounts for more than 500,000 deaths per year and is one of the leading causes of death in the pediatric population. The most common risk factors for drowning are male sex, poverty, concomitant drug or alcohol use, history of epilepsy and dysrhythmias, and age under 14 years. For every 1 drowning death, there are 4 non-fatal drowning victims who seek care at emergency departments in the United States. Survival is influenced by a myriad of factors such as the initial rhythm and duration of CPR, but specific differences that predict mortality and neurocognitive outcomes are difficult to characterize. Unfortunately, there are currently no definitive guidelines or robust clinical evidence on which to base the duration of CPR in pediatric drowning cases.

The causes of death in drowning are often multifactorial. In toddlers and younger children, drowning can occur quickly with minimal flailing or struggle. The initial mechanism is usually due to water aspiration leading to cough, possible laryngospasm, and more fluid accumulation in the lungs. This can lead to surfactant washout, atelectasis, and ventilation/perfusion mismatch, causing ARDS. It is estimated that as little as 1-2.2 mL/kg can cause significant alteration in oxygenation. There is no proven difference between saltwater or fresh water drowning in relation to pulmonary injury. Cardiac arrhythmias are usually caused by hypoxia, hypothermia, electrolyte disturbances, and acidosis. Most drowning patients

### Table 1. Arterial Blood Gas

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Initial ABG</th>
<th>ABG at Max O₂</th>
<th>ABG (Air Leak)</th>
<th>ABG Post Reintubation</th>
<th>Reference Range &amp; Units</th>
</tr>
</thead>
<tbody>
<tr>
<td>pH</td>
<td>&lt;6.85</td>
<td>&lt;6.85</td>
<td>7.01</td>
<td>6.96</td>
<td>(7.35-7.45)</td>
</tr>
<tr>
<td>PaCO₂</td>
<td>72</td>
<td>56</td>
<td>63</td>
<td>77</td>
<td>(35-45 mmHg)</td>
</tr>
<tr>
<td>PaO₂</td>
<td>158</td>
<td>252</td>
<td>45</td>
<td>41</td>
<td>(80-90 mmHg)</td>
</tr>
<tr>
<td>HCO₃</td>
<td>10</td>
<td>8</td>
<td>16</td>
<td>17</td>
<td>(20-29 mmol/L)</td>
</tr>
<tr>
<td>BE</td>
<td>-25</td>
<td>-25</td>
<td>-15</td>
<td>-15</td>
<td>(-2.2 mmol/L)</td>
</tr>
<tr>
<td>TCO₂</td>
<td>12</td>
<td>10</td>
<td>18</td>
<td>19</td>
<td>(23-27 mmol/L)</td>
</tr>
<tr>
<td>SaO₂</td>
<td>96</td>
<td>99</td>
<td>58</td>
<td>48</td>
<td>(95-100 %)</td>
</tr>
<tr>
<td>FIO₂A</td>
<td>21</td>
<td>21</td>
<td></td>
<td>%</td>
<td></td>
</tr>
<tr>
<td>Lactate</td>
<td>15.9</td>
<td>11.17</td>
<td>7.63</td>
<td>(0.50-2.00 mmol/L)</td>
<td></td>
</tr>
</tbody>
</table>

### Table 2. Szpilman Clinical Score System

<table>
<thead>
<tr>
<th>Grade</th>
<th>Mortality</th>
<th>Signs and symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>Grade 1</td>
<td>0%</td>
<td>Cough (no foam in oropharynx)</td>
</tr>
<tr>
<td>Grade 2</td>
<td>0.6 – 1.2%</td>
<td>Rales in some fields</td>
</tr>
<tr>
<td>Grade 3</td>
<td>3.6 – 5.2%</td>
<td>Acute pulmonary edema</td>
</tr>
<tr>
<td>Grade 4</td>
<td>19.4 – 22%</td>
<td>Acute pulmonary edema with signs of shock</td>
</tr>
<tr>
<td>Grade 5</td>
<td>31 – 44%</td>
<td>Isolated respiratory arrest</td>
</tr>
<tr>
<td>Grade 6</td>
<td>88 – 93%</td>
<td>Cardiopulmonary arrest</td>
</tr>
<tr>
<td>Dead body</td>
<td>100%</td>
<td>Rigor mortis, lividity, putrefaction (submersion time &lt;1 hour)</td>
</tr>
</tbody>
</table>
present in asystole and PEA arrest, but ventricular dysrhythmias have been reported.\textsuperscript{4,5} Sepsis, cerebral edema, and disseminated intravascular coagulation can occur within the first 72 hours from the initial insult.\textsuperscript{7}

Our case report demonstrates the effectiveness of PALS-guided resuscitation and the benefits of lung-protective ventilator strategies in the initial management of the severe drowning patient. Other reports have led to guidelines that limit resuscitation efforts based on duration of CPR, submersion times, or water temperature.\textsuperscript{8} The likelihood of intact neurological function after a submersion injury is multifactorial. A combination of variables, including the circumstances surrounding the submersion injury and the treatment course, affects the likelihood of intact neurological function after drowning.

The Szpilman clinical score system has been used to prognosticate the clinical outcome and course of pediatric drowning.\textsuperscript{1} As per the Szpilman classification, drownings can be classified from grades 1 to 6 (Table 2). Our patient belonged to grade 6—a drowning complicated by cardiopulmonary arrest, which is the most severe type of drowning.

In a South Korean pediatric drowning study, it was shown that poor prognosis after submersion was associated with lower consciousness levels, higher Szpilman scores, greater need for intubation and mechanical ventilator support, and longer duration of oxygen therapy. Poor prognosis was also associated with lower bicarbonate levels as well as higher sodium, AST, and ALT levels.\textsuperscript{9}

Our patient had all of the above risk factors yet had an excellent outcome. The CPR duration in out-of-hospital pediatric cardiac arrest is often limited to 30 minutes.\textsuperscript{10} However, our patient had a CPR duration of 57 minutes. The appropriate duration of CPR for pediatric out-of-hospital cardiac arrests (OHCA) remains unclear.

In a national retrospective study from Japan, it was found that prehospital EMS-initiated CPR duration for pediatric OHCAs was independently and inversely associated with 30-day favorable outcomes. The duration of prehospital EMS-initiated CPR, beyond which the chance for 30-day favorable outcomes diminished to <1%, was 42 minutes, which is much shorter than the duration of CPR administered in this case.\textsuperscript{11}

In a 2015 Dutch study, the authors published data on 160 pediatric drowning cases that required CPR for more than 30 minutes and followed the long-term outcomes. One of the indicators reported to measure the neurological disability was Pediatric Cerebral Performance Category (PCPC). PCPC is a score from 1-6 where higher scores indicate poor neurologic function. In 98 (61%) of these children, resuscitation was performed for more than 30 minutes; 87 (89%) of those children died, and the 11 who survived all had a PCPC score more than 4, which indicates moderate to severe neurological disability. In the 62 (39%) of children who did not require prolonged resuscitation, 17 (27%) survived, with a PCPC score less than 3 after one year; 10 (6%) had a good neurological outcome (score 1); 5 (3%) had mild neurological disability (score 2); and 2 (1%) had moderate neurological disability (score 3).\textsuperscript{10}

**Conclusion**

Pediatric drowning complicated by cardiac arrest must be treated on a case-by-case basis, and in select patients, a duration of CPR longer than 30 minutes is clinically indicated. The patient in this case was successfully treated and had meaningful neurological recovery after prolonged CPR and judicious use of lung-protective mechanical ventilation strategies. This demonstrates that even with prolonged CPR and severe metabolic derangements, favorable neurologic outcomes are possible. \*
No Need to Fear Fournier’s with POCUS

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A 44-year-old male with a history of diabetes and hypertension presented to the emergency department with gradually worsening pain and swelling of his testicles over the prior few days. The patient denied fever, dysuria, urinary incontinence, nausea, vomiting, and diarrhea. Initial vital signs showed a temperature of 37°C, heart rate of 117, blood pressure of 126/68, and respiratory rate of 18. A physical exam revealed induration and crepitus of the perineum, scrotal edema, and mild tenderness to palpation of bilateral testicles. Laboratory testing was significant for a leukocytosis to 21 x10^9/μL, sodium of 128 mmol/L, glucose of 476 mg/dL, lactate of 2.9 mmol/L, and creatinine of 1.9 mg/dL.

Point-of-care ultrasound (POCUS) was performed to evaluate for Fournier’s Gangrene (FG).

Ultrasound Findings/Technique

POCUS was performed using a high-frequency linear transducer in the affected area and demonstrated several definitive features of FG, including a thickened perineal and scrotal wall with streaks of linear hypoechoic fluid and punctate, hyperechoic foci with posterior acoustic shadowing and reverberation artifact. These echogenic foci lacked clear margins (“dirty shadowing”) and represented the pathognomonic intrascrotal gas found in FG.

The testes and epididymides are usually normal in appearance and echotexture due to separate blood supply from the scrotum. Although there is limited literature for the use of POCUS to diagnose FG, ultrasound has been shown to reliably diagnose necrotizing fasciitis in limbs with a reported sensitivity and specificity of 88.2% and 93.3%, respectively.

Discussion

FG is a rare, life-threatening necrotizing fasciitis involving the perineal, perianal, and genital region predominantly in men with mortality rates ranging from 15% to 50%. The infection is commonly caused by the synergistic activity of aerobes and anaerobic organisms that produce exotoxins and enzymes leading to rapid tissue destruction, gas formation, and spread of infection. Diabetes mellitus is the major predisposing factor in addition to other risk factors of alcoholism, hypertension, and immunosuppression. Early recognition and treatment with broad-spectrum antibiotics, surgical debridement, and hemodynamic support is crucial to management. Empiric antibiotic therapy such as piperacillin-tazobactam and vancomycin includes coverage for gram-positive, gram-negative, and anaerobic organisms as well as methicillin-resistant Staphylococcus aureus (MRSA). Clindamycin is also a commonly included antibiotic due to its suppression of toxin production from streptococci bacteria. Delay in treatment leads to extremely poor prognosis due to the rapid progression of the disease — in some cases, the rate of fascial necrosis can spread as fast as 2-3 cm per hour.

Although FG is most commonly diagnosed clinically, it can be challenging to identify an early presentation. The Laboratory Risk Indicator for Necrotizing Fasciitis (LRINEC) score, which stratifies likelihood of necrotizing fasciitis based on white blood cell count, hemoglobin, sodium, glucose, creatinine, and C-reactive protein, can be used to distinguish soft tissue infections from necrotizing fasciitis. Computed tomography (CT) is the primary imaging modality and has greater sensitivity and specificity (88.5% and 93.3%) at detecting fascial gas than with plain radiography (48.9% and 94%), but is time-consuming and can delay treatment. In contrast, POCUS can be performed at bedside quickly and noninvasively, though sensitivity and specificity have not been well-established. Furthermore, ultrasound can detect subcutaneous gas even prior to development of physical exam findings of crepitus in the early stages of FG. This case highlights the value of POCUS in diagnosing this relatively early presentation of FG and ultimately expediting management of this patient’s potentially lethal infection.

Case Resolution

General surgery and urology were promptly consulted, and the patient was concurrently started on IV fluids and broad-spectrum antibiotics. CT of the pelvis was performed to evaluate for underlying pathology and surgical planning, and revealed extensive soft tissue gas within the scrotum tracking into the left inguinal canal consistent with Fournier’s Gangrene. The patient was taken to the operating room emergently for surgical debridement. Wound cultures grew polymicrobial organisms including Peptostreptococcus anaerobius, Staphylococcus epidermidis, Proteus mirabilis, Enterococcus faecalis, and Klebsiella pneumoniae. The patient returned to the operating room five times over three weeks for extensive debridement of the scrotum, perineum, penis, testes, and skin graft placement. The patient was eventually downgraded from the surgical intensive care unit and discharged from the floor.

References available online
We have been witnessing a humanitarian emergency of epic proportions in the days since Feb. 24, when Russia launched a large-scale invasion of Ukraine. The conflict has displaced millions of people and has thrust Ukraine into a global spotlight. The invasion has included documented attacks on Ukraine’s healthcare facilities, including the reported bombing of a hospital’s maternity ward and a children’s clinic in Mariupol. Sadly, even medical personnel, who under international humanitarian law are to be protected from hostile action during times of war, have been targeted.

This military activity is taking place in the eastern region of the country, where infrastructure had already been devastated by prior attacks in the conflict between Russia and Ukraine. Local and foreign healthcare workers wishing to provide aid now face numerous challenges including limited medical facilities, lack of medical supplies, and obstruction of humanitarian aid organizations trying to deliver essential supplies and medications.

Expertise in emergency care is a critical need as this conflict continues and evolves. As emergency physicians, we want to help. With some sensitive considerations, we are well-positioned to contribute triage, clinical, and systems management skills during all phases of humanitarian catastrophes. But first, we need to do our homework.

Emergency Medicine in Ukraine: History and Context

The practice of emergency medicine in Ukraine is markedly different from EM in North America. The Ukrainian model of emergency care was heavily influenced by health policies of the former Soviet Union. Key historical differences between US and USSR physician training can be observed in all stages of medical education, beginning with prerequisites for medical school.

To illustrate, in the 1910s, state licensing boards began requiring U.S. medical schools to raise the bar on admission standards and implement stricter curriculum requirements, increasing the exclusivity of the field. In contrast, the Soviet Union issued a resolution of “free and open access to medical education for all” in 1919. Furthermore, American physicians had to undergo extensive postgraduate medical training starting in the 1920s, while Soviet physicians experienced the addition of residency training for the first time in the 1970s.5

Ukrainian medical training was influenced and shaped by these types of events. Our emergency physician colleagues in Ukraine attend 6 years of medical school, followed by 18 months of pre-hospital physician training at an emergency medical service (EMS) base station. Roughly 1,000 residency-trained EMS physicians practiced in Ukraine leading up to the war, with approximately 150 residents currently in training in 12 accredited residency programs.7 In Ukraine, EMS physicians operate predominantly in a pre-hospital setting. Ukraine follows a Franco-German model, in which ambulances are usually equipped with basic diagnostic and treatment modalities, one physician, occasionally a paramedic or physician assistant, and one or two nurses. When a person requires immediate medical attention, they call an ambulance instead of going to the emergency department. When the ambulance arrives, a physician evaluates the patient, establishes a preliminary diagnosis, and starts treatment if
How Can Emergency Physicians Help?

The War in Ukraine

Without government financial support, digital angiography suites and donations. Without government financial support, digital angiography suites and essential medicines, and medical supplies are limited, leaving emergency department. Instead, patients call ambulances for emergency care. EMS physicians are trained primarily in procedures that can be completed pre-hospital, with limited exposure to cardiac pacing, central venous access, bedside ultrasound, pericardiocentesis, procedural sedation, and lumbar punctures.

Even prior to the current crisis, funding has been the largest challenge for the development of emergency medicine in Ukraine, given the current model of medical financing by the central government — a relic of the Soviet era. Budgets for ambulances, EMS physicians, essential medicines, and medical supplies are limited, leaving emergency clinicians dependent on private corporate donations. Without government financial support, digital angiography suites and cardiac ambulance services are only accessible if local businesses provide financial assistance.

Do No Harm

Historically, well-meaning medical providers have responded to crises with the intention of providing direct care. The presumption is that their training and experience can be readily implemented and produce a beneficial response. The reality is more complex. A medical degree and residency training are not enough to work effectively in a humanitarian crisis. To be effective, providers need the following:

- Specialized training or experience working in resource-limited or conflict environments
- The support of an established humanitarian organization that has been granted permission by the host country to provide care and has an established logistics package
- The physical and emotional capacity to work in a crisis environment

This applies to attempting to volunteer directly in Ukraine or bordering nations. The reality is that medical personnel who lack experience in resource-limited environments and knowledge of the local culture and healthcare system may generate more work than they accomplish. At worst, they generate the potential for injury or harm to themselves or others around them.

Another common, well-intentioned response is to gather and send medical supplies. The World Health Organization has very clear guidance regarding the medical donation process. Pharmaceutical and non-pharmaceutical donations generate a substantial logistical burden to transport, store, and safely use. This is expensive and requires its own infrastructure. Governments and the humanitarian sector have established systems and networks to expedite medical aid at the request of host nations. Donating funds to established aid organizations is often more impactful. All wars end, and this one will end too. In the years to follow, Ukraine will be rebuilt. The recovery phase of disaster is a valuable time for modernization. American emergency physicians can help strengthen EM education and practice in Ukrainian healthcare. North America has specialists in EM education, simulation, administration, and healthcare policy.

We can use these valuable skills to aid Ukraine in developing more robust emergency care. It is only by building a robust emergency care system that we can ensure improved disaster response and ensure access to acute care for everyday emergencies.

HOW TO HELP

Right now, in this time of war, Ukrainian physicians are serving their patients around the clock. There are no shifts or time off. They are always at work, and they are always needed. In times like this, emergency medical knowledge is especially valuable. But even more than that, these brave medical professionals need supplies and financial support.

There are direct and concrete ways we can help Ukrainian physicians on individual and collective levels. Here are a few:

- **Donate funds to recognized aid agencies.** Many American residency programs organized fundraising events in support of Ukraine over the past weeks. (Scan the QR code for reliable nonprofit organizations.)

- **Donate medications and medical supplies.** Send medications and medical supplies directly to Ukrainian hospitals by reaching out to your supply manager. (Scan the QR code for a list of most-needed supplies.)

- **Share your knowledge.** Physicians experienced in tactical emergency medicine and in low-resource settings can create and share open access educational content. Content should be evidence-based and easy to access from the bedside. To provide the maximum benefit for physicians in war zones, it is essential to first listen to them, and allow them to identify their needs and their available resources. (Scan the QR code for a list of Ukrainian hospitals’ emails.)

- **Advocate.** Use your authoritative voice as a medical professional to advocate for an end to human rights abuses. For guidance on advocating as a physician, refer to EMRA’s Emergency Medicine Advocacy Handbook, Physicians for Human Rights’ advocacy toolkits and letters, and ACEP’s advocacy guides.

References available online
International EM has faced challenges in recent years due to the pandemic and other factors, but with the right roadmap, it can still be done. EM physicians are finding new ways to get involved, secure fellowships, and serve remotely.

I was optimistic at the start of residency that I could pursue projects in global health, but reality played out differently. Global health has seemed elusive over the past two years for a variety of reasons: the coronavirus pandemic, a shift from in-person to remote work, and the changing landscape of job security within emergency medicine. These challenges were in addition to pre-existing barriers: demanding clinical rotation schedules, restrictive elective time (usually one month for the entirety of residency), and limited funding and resources.

So what now? Recent events have limited us to domestic work, but many global health projects still require attention. If anything, the past two years have demonstrated that global health events have a profound effect on local health as well. Though global health may conventionally seem less accessible, your involvement may actually be more feasible during residency through a more creative approach.

A Resident’s Current Guide to Navigating Global Health

What Now?

Anisa Mughal, MD
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Where do I begin?
Identify the area of global health in which you are most interested. General categories include medical education, residency development, systems and capacity building, and research. Which specific topics within these larger categories appeal to you? An alternative is to identify a location of interest and develop an idea or attach to an existing project. Ask friends and colleagues for any contacts within global emergency medicine.

How do I find a project?
There are two ways to approach this: find a mentor you enjoy working with and assist with an existing project, or identify a project or location of interest and subsequently find a mentor who has experience in those areas. Do not be afraid to “cold email” faculty at institutions with robust global health departments, especially if your home residency does not offer formal global health training or electives. Pursuing an opportunity outside your program demonstrates your dedication to your interests. Communicating with another institution regarding global health is in no way binding, but you should close the loop with mentors you contact regarding your final decision. Good places to find programs and contacts are the EMRA Fellowship Guide chapter on International Emergency Medicine, the Global Emergency Medicine Fellowship Consortium website, and the EMRA Match database for fellowships. Many faculty members at these programs are involved in a multitude of projects and would welcome resident interest.

What ethical questions should I consider?
It is important to consider what impact your involvement will have on projects. It is almost always better to consider a community’s needs and join projects that have an established relationship with a site. If you have to withdraw from a project, what impact will that have on the outcome and on your team? Does this project serve an existing need in the community, and is the community amenable to your involvement? What political or social dynamics regarding hospital leadership, site administration, or project history need to be taken into account?

In what capacity can I be involved?
In the current travel climate, remote involvement in projects is the most feasible. Despite the drawbacks of remote work in such situations, consider this: Remote involvement allows for a longitudinal relationship with a site, scheduled check-ins, and team meetings. Site visits can be time consuming, expensive, and risky. As residents, we are certainly short on time. We often are not granted additional funding, and financing travel is not always feasible. Consider remote involvement as a new avenue to global health involvement, with the ability to establish online resources for community health workers, a digitized trauma registry, or virtual lectures for conferences. As our digital infrastructure grows, so will our capacity to provide healthcare remotely.

How will this impact my clinical care at my home institution?
Involvement in global health has educational and clinical benefits, including improving your clinical care for underserved populations during residency, becoming cost and resource conscious, and understanding cultural impacts on health care. There are many similarities between working in global health and working with underserved populations at our own home institutions.

What are other tips for success?
Clarifying time allotment, specifying deliverables, and discussing outcomes with your mentor are good ways to lay the foundation for successful teamwork.

TAKE-HOME POINTS
- Identify a mentor and project in your location of interest.
- Leverage remote involvement as a longitudinal project that can be scheduled around shifts.
- Consider ethical questions regarding your involvement.
- Set a timeline, anticipate delays, and develop contingency plans.

EMRA Resources
For those interested in pursuing opportunities in global health, EMRA offers a few valuable resources.

One place residents can turn to is our book, "The Nuts & Bolts of Global Emergency Medicine." Readers will learn about terminology within the realm of global EM, ethics and cultural awareness, and travel-related safety considerations, among other topics. Plus, there's a whole section dedicated to educational opportunities, which includes chapters on mentorship, research, and funding. The book is accessible online and available for free download.

Additionally, the Global Emergency Medicine Student Leadership Program (GEMS LP), formerly known as the International Ambassador Mentorship Program (AMP), was established in 2018 as a joint effort of EMRA and the ACEP International Ambassador Program. It provides guidance to the growing number of EM-bound medical students seeking GEM mentorship.

Goals of the mentor-mentee initiative are:
- To connect EMRA medical student members interested in global emergency medicine with ACEP International Ambassador Mentors.
- To familiarize students with the current state of emergency medicine as a specialty in different countries around the world.
- To educate students about professionalism, leadership, and teamwork through participation in ACEP International Ambassador projects.
- To provide an overview of global emergency medicine work including research, education, development, humanitarian work, and ethical implications.
- To provide mentees with a network of contacts in global emergency medicine that they can utilize to further their professional interests.

This program aims to expose medical students to the field of GEM through participation in global health projects, engagement in a thought-provoking journal club, mentorship from ACEP Ambassadors participating in EM programs worldwide, and networking with like-minded peers. In addition to fostering interest in GEM, the program seeks to expose students to some of the complex issues in global health and the importance of having an ethical, equitable, and sustainable approach to global health involvement.
Be specific, and be smart, about documenting, charting, and coding patient encounters. Underbilling results in lost opportunities for physicians and their institutions.

In modern medicine, there has been an increasing trend of analyzing metrics to evaluate patient outcomes, patient satisfaction, and provider productivity. Metrics-driven performance measures have been implemented in various industries, although only recently making a frontier in medicine. Across different specialties, productivity is measured. For a hospitalist, it might be the number of admissions; for a surgeon, the number of cases; for operating room staff, the turnover time between surgical cases; or for a researcher, the number of papers published. For an emergency medicine clinician, productivity is frequently measured by patients per hour treated and the number of relative value units (RVUs) generated per hour. In every setting, academic or community, and by every employer, including hospitals, independent groups, or contract management groups, EM physicians’ productivity is constantly measured and compared.

For EM clinicians, there can be significant variability in productivity,
depending on multiple factors such as the site of practice, staffing model, patient volume, and patient acuity. There is not only variation between sites, but also between providers within a site. Through all the factors that a clinician cannot control, the one tool that a clinician can best leverage for better productivity is appropriate charting. The metric of RVUs generated is dependent on billing and coding, which is reliant on a physician’s ability to document fully the patient encounter. In seeing the same number of patients, a clinician has the potential to generate higher RVUs if documentation accurately captures all the care that was delivered. Documentation at its core serves three purposes:

1) relaying important information to others,
2) a medical record to protect against liability, and
3) a tool for billing. Although during medical training residents receive ample education on the clinical and medicolegal purposes of charting, the nuances of billing are frequently mystified despite its impacts on personal compensation and financial durability of an ED.

The two main billing components that generate RVUs are the level of the patient encounter as per charting requirements and the procedures billed. A general teaching is that a high acuity ED visit should be billed at a level 4-5 (with 5 being the highest level), so long as the minimum criteria are met as described by the Centers for Medicare and Medicaid Services (CMS). Level 1-3 charts are more befitting of low acuity visits, frequently seen in urgent care settings or low acuity dedicated sections of the ED. Attention must be paid to documentation requirements as a high acuity encounter billed as a level 3 would produce approximately half of the revenue compared to a level 5 chart. For example, if a chest pain encounter is documented appropriately, a physician will generate more revenue from one patient encounter compared to three patient encounters with similar complaints that are charted poorly. With smarter documentation, physicians can leverage the billing and coding of their charts to maximize RVUs generated, bill accurately for services provided, and help ensure department financial stability. Without department financial stability, we risk loss of access to emergency care for many members of our community.

Common causes of underbilling in the emergency medicine encounter are inadequate documentation of procedures and “critical care” time. Two frequent errors in procedure documentation that negatively impact physician metrics, and ultimately lead to loss of ED revenue, are under-documentation of the complexity of a procedure, and failure to explicitly document a billable procedure. The loss of revenue related to critical care billing often stems from misunderstanding about the CMS billing definition of critical care time and lack of documentation of critical care time in the chart.

**Complex Procedures Are Not so Complex**

Complexity of a procedure is frequently thought by clinicians as a subjective measure of conducting the procedure, but there are CMS definitions of complexity for specific procedures. Looking at the example of a complex abscess incision and drainage (I&O) versus a simple abscess I&D, CMS describes an I&D as complex if the abscess was probed or loculations were broken up during the procedure, if there were multiple abscesses, or if it was packed. Probing and breaking up loculations are fundamentals of an emergency department I&D, making nearly all abscess I&Ds completed in the ED complex per the CMS definition. The implication of documenting simple I&Ds, which generate 2.77 RVUs, instead of complex I&Ds that bill 5.12 RVUs, is the loss of tens of thousands of dollars in revenue for a department annually. Similarly, laceration repairs are coded by the length of the laceration. Lacerations less than 2.5 cm generate 1.27 RVUs, while those greater than 2.5 cm generate 1.67 RVUs or more. Although it might not be general practice to measure exact wound size with a ruler, an easy way to accurately estimate wound length is by the number of sutures used. Traditionally, sutures are placed 0.5 cm apart, so one can estimate that the laceration size must be greater than 2.5 cm when more than four sutures are placed in one layer. It is also worth noting that when performing laceration repairs, using a skin-adhesive or steri-strips in place of sutures still counts as a laceration repair and should be documented as such. It is important for ED physicians to learn about these details in procedural documentation, so that there is accurate compensation for their work.

**Missed Opportunities in Documenting Billable Procedures**

Another frequent cause of under-billing is not separately documenting all billable procedures. Although we are usually immaculate in placing procedure notes for things such as endotracheal intubations and central lines, many physicians fail to recognize that procedures such as cardiopulmonary resuscitation (CPR) and cardioversions can be billed separately. Thus, along with critical care time charted (although this cannot include time spent on separately billed procedures), we can bill for CPR, which significantly increases RVUs generated from the patient encounter, leads to increased departmental revenue, and more accurately represents the care delivered. One way to make procedural documentation easier is by leveraging the Electronic Medical Record (EMR) to make dot-phrases or saved dictation texts. These tools are shortcuts to longer documentation and can ensure that any procedure can be documented efficiently. When documenting a patient encounter, we merely have to say a phrase like “insert CPR” or type “.cpr” to prefill a procedure note and adjust accordingly for a specific patient encounter.

**Understanding Critical Care Time**

Underbilling related to critical care time documentation often stems from the difference in how EM physicians think about critical care compared to the actual CMS billing criteria for “critical care.” EM physicians frequently use subjective measures of hemodynamic instability or obvious immediate life-threatening illness to determine when to bill for critical care time, which can lead to severe underbilling of care provided.
CMS defines critical care time as care for a patient with a “high probability of imminent or life-threatening deterioration” or illness that “impairs one or more vital organ systems,” requiring “frequent personal assessment and/or manipulation.” This definition and coding system is used for all providers through the healthcare system and is not specific to emergency medicine. Many diagnoses and clinical conditions that EM physicians manage and treat on a regular basis do not meet our intrinsic threshold to be considered “critical,” but they often are based on the definition by CMS. For example, any use of non-invasive ventilation (BiPAP or CPAP) infers a patient is in a critical state, and charting critical care time should be considered. Other common missed opportunities to document critical care time include hyperkalemia management that requires a corrective measure such as calcium. Hypoglycemia that needs dextrose administration with three finger stick glucose checks throughout the patient’s ED course can also be considered for critical care time. There are numerous such examples of underbilled critical care time as per the CMS definition, leading to loss of revenue for emergency departments. Becoming familiar with diagnoses and intervention requirements that consist of critical care time can be immensely worthwhile to ensure that the RVUs generated from a patient encounter accurately reflect the care we provide.

**Conclusion**

By knowing the minimum criteria for charting accurately, EM physicians can more reliably get credit and compensation for the care they deliver. Using the tips of documentation described above, residents can ensure that after graduating they are capturing high RVUs to maximize their measured productivity. In many employee contracts, compensation is linked to metrics recorded for an individual clinician, so RVUs generated can significantly impact a clinician’s salary. For example, salary variability could range between $250,000 to $400,000 for a provider, with the ultimate decision dependent on metrics such as RVUs. By knowing how to bill appropriately, physicians can ensure that the metrics reflect the care provided AND increase revenue generated for the ED. Most importantly, increased revenue for the department allows us to be financially viable and better prepared to increase care capacity. Additional resources enable departments to hire more providers, fund more projects, and acquire the latest technology, which ultimately improves the care we provide and increases access to emergency care for members of our communities.

**TAKE-HOME POINTS**

1. “Complex” billed procedures aren’t so complex. “Critical care” time billed isn’t always what we think of as critical.
2. Remember to recognize and document all billable procedures.
3. Maximizing RVUs generated allows us to continue delivering emergency care to patients who need it most.

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**Annals of Emergency Medicine**

Resident Editorial Board Fellowship Appointment

The Resident Fellow appointment to the Editorial Board of *Annals of Emergency Medicine* is designed to introduce the Fellow to the peer review, editing, and publishing of medical research manuscripts. Its purpose is not only to give the Fellow experience that will enhance his/her career in academic emergency medicine and in scientific publication, but to develop skills that could lead to later participation as a peer reviewer or editor at a scientific journal. It also provides a strong resident voice at Annals to reflect the concerns of the next generation of emergency physicians.

**Selection Process**

The Resident Fellow is chosen in a competitive selection process in July. Members of the selection committee, including the Editor in Chief, the deputy editors, Annals’ Editorial Director, and the current Resident Fellow, review all curriculum vitae and select the top three candidates. These candidates are interviewed by the Editor in Chief, who then makes the final selection. Preference will be given to senior-level residents; applicants currently in training as EM fellows are not eligible.

**Application Process Requirements**

**Required materials:**

- Completed application form
- Cover letter
- 1-page personal statement
- Current curriculum vitae
- 2 letters of recommendation
- 2-3 proposed topics for future “Residents’ Perspective” columns

**Deadline: July 25, 2022 • Download the application packet at www.annemergmed.com**

**Questions?**

Please contact Martha Villagomez, Editorial Associate, *Annals of Emergency Medicine*

mvillagomez@acep.org or 800-803-1403, ext. 3223

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References available online
Scholarship Winner Helps Implement Early HCC Screening in Tanzanian EDs

EMF/EMRA research grant recipient and EMRA scholarship award winner James Ford spent four weeks in Tanzania on a pilot project that employs rapid Hepatitis B testing and ultrasound in the ED to screen for hepatocellular carcinoma.

In April, I had the wonderful opportunity to participate in and complete a 4-week international ED rotation in Arusha, Tanzania, at Arusha Lutheran Medical Center. The primary purpose of my elective was to conduct a prospective observational study titled, “Use of Point-of-Care (POC) Hepatitis B Virus (HBV) Testing and Ultrasound to Screen for Hepatocellular Carcinoma (HCC) in a Tanzanian Emergency Department (ED).” This project was funded by the Emergency Medicine Foundation (EMF)/Emergency Medicine Residents’ Association (EMRA) Resident Research Grant. My travel was graciously funded by the EMRA International EM Rotation Scholarship Award.

The purpose of this rotation/study was to pilot a novel approach to community infectious diseases and liver cancer screening by employing rapid HBV POC testing and POC ultrasonography in the ED. HBV is the most common risk factor underlying the development of HCC, which is the second leading cause of cancer-related death in the world. The largest epidemiologic burden of HBV and HCC occurs in low- and middle-income countries, and sub-Saharan Africa is one of the most disproportionately affected regions. In Tanzania, the primary care infrastructure is underdeveloped. Thus, many patients do not seek medical attention unless they are ill and do not have access to routine infectious disease screening.

To bridge this gap in healthcare access, this program will capitalize on a patient’s presentation to the ED and offer him or her important medical screening. Initiatives aimed at increasing HBV and HCC awareness and screening in Tanzania and other parts of Africa have the potential to save millions of lives. This pilot study, which places the ED at the center of the public health system, could serve as a model for the delivery of public health services in the region and will help extend the potential impact and growth of emergency medicine in Africa.

During my short time there, we were able to screen more than 700 patients for HBV. Patients who were positive had POCUS performed to assess for HCC and were referred to the clinic for treatment. The study will continue to enroll patients, under the guidance of local co-investigators, and we hope to screen more than 2,000 patients over the next two months.

One goal of this rotation was to train local co-investigators in how to perform research so that they might independently conduct their own methodologically robust studies. I also had the opportunity to work with emergency medicine and intensive care providers and provide bedside POCUS teaching. During my time shadowing local ED providers, I learned an immense amount about what it is like to practice EM in a resource-limited setting. I am humbled by the resiliency and resourcefulness of my Tanzanian colleagues.

In summary, I believe my experience will be transformative in helping me become a more well-rounded clinician and researcher. My cultural immersion and exposure to the Swahili language gave me a glimpse into a new way of life. I will forever cherish the memories made with my new Tanzanian friends and colleagues.

Hakuna Matata.
James Ford, MD
Illegal and unregulated marijuana markets in parts of our country have given rise to dangerous and easily obtainable synthetic options. The ill effects of this predictable phenomenon are being felt in EDs. To combat this trend and cut down on risky black-market alternatives to marijuana, the author asserts we must abolish our prohibitionist approach to drug policy.

Synthetic Cannabinoids Highlight the Need to Abandon the War on Drugs

Timothy Kelly, MD
Emergency Medicine PGY2
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As an emergency medicine resident in New York City, I am regularly confronted with a chief complaint that I had not seen during medical school on the West Coast — K2 intoxication. While prevalence numbers are hard to come by, poison control calls in 2021 due to K2, otherwise known as synthetic marijuana, were significantly higher in New York state compared to Washington state even after accounting for difference in size of population. While the population of New York is roughly three times that of Washington, the number of K2-related calls to poison control that originated from New York was approximately 12 times the number of calls from Washington.1

At the risk of simplifying a complex issue, the divergence in the two states’ political approaches to marijuana policy can explain much of this disparity in prevalence. That is to say, it is my view that our country’s ongoing embrace of prohibition is directly responsible for the scourge of K2.

Marijuana is classified as a Schedule I drug — the government...
possible medical benefits were rarely and yet research proposals investigating have no established medical benefits — the government has created a Catch-22. due to regulatory and financial barriers, therapeutic potential of marijuana. On Drug Abuse (NIDA) investigated the year 2015, only 16.5% of cannabis-related grants funded by the National Institute whose aims were to investigate the only federally available at the University of Mississippi. Until recently, the only federally available source of marijuana for research was a single strain produced in an outdoor farm. As alcohol makes abundantly clear, a substance need not have a medicinal benefit to have a valuable role to play in society. And as alcohol also demonstrates, the potential to cause harm does not necessitate prohibition. Being Schedule I means it is exceedingly difficult to obtain permission to perform any research on marijuana. Until recently, the only federally available source of marijuana for research was a single strain produced in an outdoor farm at the University of Mississippi. Not only was this supply of marijuana deemed inadequate by many scientists and other experts, but up until recently, the vast majority of marijuana research proposals that were granted federal funding were those whose aims were to investigate the potential harms of the plant. In fiscal year 2015, only 16.5% of cannabis-related grants funded by the National Institute on Drug Abuse (NIDA) investigated the therapeutic potential of marijuana. Thus, due to regulatory and financial barriers, the government has created a Catch-22. The substance is Schedule I — deemed to have no established medical benefits — and yet research proposals investigating possible medical benefits were rarely federally approved and funded, thereby ensuring that marijuana would maintain its Schedule I status.

A Novel Workaround to Research Restrictions

In part as a response to these federal restrictions on research, organic chemist Dr. John Huffman circumvented this conundrum by creating a novel compound based closely upon delta-9 tetrahydrocannabinol (THC), the primary active ingredient in marijuana. By creating a substance with a distinct molecular structure, yet with effects that mimicked THC, he was able to sidestep this Catch-22. In doing so, he was able to research endocannabinoids — the body’s naturally produced neurotransmitters similar in structure to THC and the other psychoactive compounds found in marijuana — without going through the arduous, near-impossible federal approval structure for marijuana research.

This first compound was initially synthesized in 1995, and the first studies investigating binding affinities of endocannabinoid receptors in humans were published in 1998. However, as exemplified by the history of lysergic acid (LSD) and methylenedioxymethamphetamine (MDMA), when a novel compound with pleasant psychoactive effects is created in a lab for scientific evaluation, it tends to find its way out of the lab and into the bodies of eager consumers. And so it was with synthetic cannabinoids. In 2008, Dr. Huffman received an email alerting him that one of his novel synthetic cannabinoids, JWH-018, had been identified in herbal supplements being sold in Germany.

Here was a substance whose effects were virtually indiscernible from whole marijuana yet not federally illegal. In the early 2010s, synthetic cannabinoids became ubiquitous. The chemical was sprayed on inert vegetative matter; sold in attractive, psychedelic-inspired packaging; and legally marketed as incense and “not for human consumption.” These products were easily obtained — online, in gas stations, and at smoke shops — with a number of companies attempting to carve out their corner of the market with intriguing product names, including Spice and K2.

Inevitable Harms From Our Prohibitionist Drug Policies

In response, the Drug Enforcement Agency (DEA) did what it often does and moved to outlaw this new substance with little input from the scientific and medical community and without comprehensive data about actual harms posed by these substances. But by this point, it was too late. An appetite had been built for synthetic cannabinoids, and entrepreneurial black-market chemists realized there was profit to be made. Instead of being deterred by the new illegality of their product, these chemists were instead inspired to outmaneuver the DEA. They made small changes to the molecule, hoping to preserve its familiar psychoactive effects while being dissimilar enough in structure to the original synthetic cannabinoids as to no longer be covered by the DEA’s emergency scheduling.

This set off a predictable game of whack-a-mole between the DEA and their chemist adversaries. Each time a new version of synthetic cannabinoids would be released to the market, the DEA would make it illegal, prompting yet another round of revisions to chemical structure to produce yet another novel and legal psychoactive substance.

The DEA emergently scheduled synthetic cannabinoids for the first time in 2011, and as of 2021, there were 40 synthetic cannabinoids listed under Schedule 1. The DEA identified 84 novel cannabinoid clinical studies investigating binding affinities of endocannabinoid receptors in humans were published in 1998. However, as exemplified by the history of lysergic acid (LSD) and methylenedioxymethamphetamine (MDMA), when a novel compound with pleasant psychoactive effects is created in a lab for scientific evaluation, it tends to find its way out of the lab and into the bodies of eager consumers. And so it was with synthetic cannabinoids. In 2008, Dr. Huffman received an email alerting him that one of his novel synthetic cannabinoids, JWH-018, had been identified in herbal supplements being sold in Germany. In response, the Drug Enforcement Agency (DEA) did what it often does and moved to outlaw this new substance with little input from the scientific and medical community and without comprehensive data about actual harms posed by these substances. But by this point, it was too late. An appetite had been built for synthetic cannabinoids, and entrepreneurial black-market chemists realized there was profit to be made. Instead of being deterred by the new illegality of their product, these chemists were instead inspired to outmaneuver the DEA. They made small changes to the molecule, hoping to preserve its familiar psychoactive effects while being dissimilar enough in structure to the original synthetic cannabinoids as to no longer be covered by the DEA’s emergency scheduling.

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history of human use, are regularly being synthesized and placed in the quasi-legal market and into bodies of willing consumers. The problem, however, is that you can only make so many changes to a chemical structure before it starts to have less predictable effects and really begins to belong to a class of drugs all its own.

Some may be surprised by the fact that intoxication with the first incarnation of K2 was virtually indiscernible from natural marijuana. Many of us, for one reason or another, are familiar with the effects of cannabis. We may even have cared for a patient or two who presented to the emergency department with a chief complaint of marijuana intoxication — like the mother-daughter pair I helped treat after they accidentally consumed marijuana edibles in their quest to find a natural sleep aid. These experiences of marijuana intoxication are an entirely distinct clinical phenomenon from our patients inebriated by K2. That’s because what started off as a predictable mimicker of THC now has a profound array of disturbing possible effects that include catatonic states, violent behavior, paranoia, kidney injury, stroke, seizures, and heart attacks.9

I believe that our federal drug policies are directly responsible for this Frankenstein-esque drug, K2. Seemingly unwilling to accept the reality that nearly every society has documented use of psychoactive substances, most of our elected leaders remain hell-bent on achieving Ronald Reagan’s quixotic drug-free world. While an impartial and evidence-based evaluator would almost certainly determine that the potential harms of marijuana use are far outweighed by the harms of an unregulated and illegal marijuana market, most of our laws are more inspired by the cult-classic “Reefer Madness” than by science.

This prohibitionist approach, only recently rejected in New York state with the cannabis legalization bill signed into law in March 2021, plays a large role in why I frequently see K2 intoxication as a chief complaint in my ED. In the absence of a regulated marijuana market, many people turn to the quasi-legal, bodega-available, cheap K2. Instead of a mild case of anxiety and tachycardia that can be seen with marijuana intoxication, those who consume K2 are often ending up chemically sedated while being monitored for the rare but concerning aforementioned side effects.

**A Necessary Alternative**

Nearly as striking as treating a K2 patient in my New York City ED is the relative absence of K2 in my hometown of Seattle. That is, in part, because marijuana enjoys widespread acceptance in Seattle. Washington state’s first step toward legal recognition was a medical marijuana law in 1998, followed by a Seattle law instructing cops to make marijuana enforcement their “lowest enforcement priority,” and then outright legalization of recreational marijuana in 2012.8,10 What this means is that people who want marijuana as a way to recreate, connect, or even self-medicate have relatively easy access to a safe and regulated supply without risk of legal consequences. It makes sense, then, that there was not, and is not, much of a market for K2 among most Washingtonians. However, there are populations in the state within which K2 is more prevalent: the military, individuals who are incarcerated, and employees who are subjected to drug tests. Case in point: These are populations that are barred access to the state’s otherwise legal supply of cannabis.8

And lest one think this phenomenon is unique to marijuana, one only has to look at synthetic opioids to see that this troubling trend of increasingly dangerous novel drugs will continue. Heroin is expensive, bulky, and requires growing large fields of poppies — making it an unnecessarily risky enterprise in a world of prohibition. Synthetic opiates are the opposite — extremely potent, far less bulky, and cheaply made in a lab. As we have seen, this has led to fentanyl and its more dangerous derivatives flooding markets and all but replacing the heroin that consumers are often seeking, leading to the overdose crisis we are all too familiar with. But in an attempt to evade the illegality of heroin and fentanyl, other opioid derivatives — such as nitazenes — are making their way, legally, onto the market.11 And as was the case with synthetic cannabinoids, I believe it’s only a matter of time before these derivatives have had one too many changes to the original opioid chemical structure and they stop acting like a predictable opiate. Or even more worrisome, they stop responding to naloxone.

We must use our experience on the frontline of the unintended, yet inevitable, consequences of the war on drugs to lobby our elected officials for policies that reduce, not perpetuate, harm — policies that acknowledge that people will always seek to alter their consciousness, that this doesn’t make them inherently criminal, and that doing so does not have to be dangerous to their health. By embracing harm reduction and evidence-based drug policies, we can reduce the prevalence of relatively dangerous synthetic cannabinoids while also decreasing the chances that our EDs will be flooded with other harmful derivatives of commonly used psychoactive substances. Indeed, this act of courage — of being willing to challenge our unjust but widely accepted prohibitionist drug policies — is not only the right thing to do, but required of us by our field’s own code of ethics.12-13
The New Reality of EM and the Rise of Peri-Primary Care

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As recently as the 1950s, the "emergency room" often consisted of nothing more than just that: a room in the hospital reserved for emergencies. Staffed by rotating hospital physicians, residents, and, in some cases, medical students, the early emergency departments were conceived of as places only for the interim management of severe injuries and acute illnesses. Over the last century, the role of the ED has shifted rapidly from that of an isolated adjunctive to inpatient care to its current status as a lynchpin of hospital functioning. The ED now serves as the source of admission for more than half of hospitalized patients over age 65. During this time, the number of patients visiting EDs annually has skyrocketed, far outpacing the growth in US population. Just as the number of overall ED visits has increased, so too has the number of ED encounters for "non-urgent" issues. Increasingly, EM physicians are tending to patients who have come in not for life-threatening injuries or illnesses, but for management of more chronic medical conditions. Elevated at-home blood pressures, mild asthma exacerbations, chronic headaches — all issues broadly considered to be the realm of primary care — are increasingly common causes of a trip to the ED.

The root of this trend is not fully understood, but is likely due to the combination of a variety of factors. For many without insurance, outpatient primary care is not easily accessible or is prohibitively expensive. At understaffed, underfunded free clinics, patients often have to wait hours to be seen for a 15-minute visit. Patients working multiple jobs, or those who cannot get time off without risking their employment, may not be able to make it to most clinics during normal office hours. Some simply feel that the ED "doc in a box" format better serves them when they are experiencing a minor aggravation but cannot find an available outpatient appointment for another three weeks.

Whatever the reasons, the general dialogue regarding this trend often maintains that these problems are outside the purview of an EM physician’s practice. While it may be true that many of these non-urgent issues would be better addressed by regular outpatient follow-up, EM physicians are already well-versed in adapting care to the reality of the patient in front of them. In the same way that EM physicians may alter prescriptions for different patients — IM haloperidol for a patient having trouble with oral psychiatric meds, or generic drug prescriptions when the brand name is not covered — so too may they adapt care according to the primary care status of their patients. If a patient has regular, easy access to ambulatory care, the management of their hypertensive meds may well be deferred to their PCP. But for a patient coming in for the third time with a BP of 180/120 and no established PCP, the EM physician who accepts his/her responsibility as a provider of peri-primary care is the one who is really going to save this patient’s life. Of course this ED peri-primary care will never, and should never, serve as a full substitute for regular access to primary care, but in the absence of any primary care, it is a critical alternative.

While it diverges from the more traditional understanding of EM, we should try to understand this ED primary care, or "peri-primary care," not as an inconvenience but as the vital next phase in the ever-adapting field of EM. In the past 70 years, emergency medicine has grown from a limited non-specialty staffed by unequipped medical students and residents to a well-established, integral part of hospital functioning staffed by highly trained physicians. As the field continues to grow, so too will its purview.

We can reframe the responsibility of EM physicians so as to include not only acute, life-threatening emergencies, but also chronic conditions that can be equally as life-threatening. In the end, a patient with uncontrolled diabetes and no regular primary care faces equally as dangerous complications as the patient with acute frostbite. The time course may be different, but the outcome is still the same. Emergency medicine as a specialty was developed to handle acute emergencies, but as the field grows and ED utilization rates continue to rise, we would do well to expand our understanding of the purview of EM to include chronic emergencies as well.

References available online
EMRA Case-Con is a poster presentation contest featuring fascinating emergency medicine cases. The event includes a 5-minute presentation followed by 2 minutes of group discussion. Presentations will be judged by a panel of EM residents and faculty.

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CASE.
A 36-year-old female with a PMH of alcohol use disorder and type 1 DM presents with substernal chest tightness, nausea, vomiting, and diaphoresis.

What is your interpretation of her ECG?

See the ANSWER on page 52
ECG Challenge

HYPERKALEMIA

This ECG shows a regular wide complex rhythm with a ventricular rate of 100 bpm, no discernable P-waves, right axis deviation, and a prolonged QRS complex duration of 160 msec with an intraventricular conduction delay. There are large T-waves in leads I, II, aVL, aVR, and V4-V6, STE in leads III, aVF, aVR, and V1-V2, and STD in leads I, aVL and V4-V6.

This EKG could easily be interpreted as a STEMI, especially given the patient’s symptoms, but the constellation of findings suggests hyperkalemia which can cause STE, especially in leads V1-V2, that mimic a STEMI. This patient was aggressively treated with IV calcium gluconate (8 amps in the first 20 minutes), insulin with dextrose, and IVF. Nebulized albuterol was deferred due to COVID and a lack of available negative pressure rooms. Figures 1-3 demonstrate the dynamic ECG changes seen over the first hour of treatment.

**HYPERKALEMIA LEARNING POINTS**

- ECG is specific but not sensitive for hyperkalemia (ie, hyperkalemia can present with minimal to no ECG changes)²
- ECG changes are not always sequential/progressive and include:
  - Tall, narrow, peaked T-waves (best seen in the precordial leads)
  - P-wave flattening and PR interval prolongation
  - Prolonged QRS complex duration, ranging from minimal to maximal
  - Conduction abnormalities (AV blocks, fascicular and bundle branch blocks)
  - Bradycardia
  - Sino-ventricular rhythm (loss of P-waves, extremely widened QRS) with normal or slow rate
  - Ventricular dysrhythmias
- Hyperkalemia can mimic a broad range of pathologies
  - Can cause STE (common in leads V1-V2 and aVR) that mimics a STEMI or Brugada pattern
  - Can also occur simultaneously with hyperacute T-waves and obscure early changes seen in an anterior or anteroseptal AMI

**CASE CONCLUSION**

This patient’s initial labs showed multiple abnormalities consistent with severe DKA, as well as a potassium level of 7.8 mEq/L. She was admitted to the ICU for further treatment.

Regarding the dose of calcium used in this case, there are no universally accepted guidelines for the dosing of calcium in the treatment of hyperkalemia. In cases of severe hyperkalemia, it is reasonable to treat aggressively with calcium as the risk of inadequately treated hyperkalemia outweighs the risk of iatrogenic hypercalcemia.★
1. Which statement applies to the innervation and sensation of pain in the abdomen?
   A. Distention of abdominal organs results in somatic pain and is associated with autonomic symptoms
   B. Extra-abdominal disorders such as pneumonia can result in abdominal pain
   C. Irritation of the colon causes visceral pain that manifests as upper abdominal pain
   D. Irritation of the peritoneum causes dull, poorly localized visceral pain

2. A mother brings in her 2-day-old son for evaluation of feeding difficulties. She says he has been coughing and gagging with all feeds since birth and that "he drools way more" than her other children did. He was born at home with assistance from a doula at 37 weeks' gestation, and there were no delivery complications. The mother received normal prenatal care and had no complications during the pregnancy. What is the most likely diagnosis?
   A. Esophageal web
   B. Gastroesophageal reflux
   C. Meckel diverticulum
   D. Tracheoesophageal fistula

3. Which patient should be evaluated for a significant immunocompromising condition?
   A. 17-year-old who has itchy, red, raised plaque on the inner thigh extending to the groin
   B. 22-year-old who has fever, cough, and night sweats following a recent trip to Arizona
   C. 32-year-old who has white plaques in the posterior pharynx with odynophagia and redness
   D. 38-year-old who has multiple well-demarcated spots of lighter skin on the chest and back

4. A 37-year-old woman presents with intermittent, severe, left lower quadrant abdominal pain over the past 24 to 48 hours. She denies fever, light-headedness, nausea, and vomiting. A physical examination reveals a nondistended abdomen with rebound tenderness in the left lower quadrant. A urine pregnancy test is negative. An ultrasound examination demonstrates an enlarged left ovary with a 7-cm cyst and normal Doppler flow noted. Which diagnosis might this patient have that requires urgent obstetrics consultation?
   A. Acute ovarian torsion
   B. Endometrioma
   C. Hemorrhagic corpus luteum
   D. Ovarian cyst rupture

5. In which scenario is cardioversion most indicated?
   A. 23-year-old man presenting with palpitations for the past 4 days with an ECG demonstrating atrial fibrillation with rapid ventricular response
   B. 57-year-old man with a history of hypertension presenting with chest pain and orthopnea with an ECG demonstrating third-degree AV block
   C. 63-year-old woman with a history of hypertension and diabetes presenting with chest pain and diaphoresis with an ECG demonstrating a wide-complex tachycardia
   D. 75-year-old woman with a history of heart failure and COPD presenting with acute shortness of breath with an ECG demonstrating multifocal atrial tachycardia

ANSWERS
1) B, 2) D, 3) C, 4) A, 5) C
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