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From the President

A Celebration of a Leader

Matt Keadey, MD, MHA, FACEP

Leadership comes in many forms and it may mean something different to each of us. Leaders may be charismatic with the power to move people through their words. Leaders may move us through by their actions setting an example of what they feel we should be and do. Leaders may support us through being a servant and helping us achieve our goals. Leaders may tell us what to do or maybe ask our opinion and lead by the will of the group. Leaders may use a combination of the above styles or maybe none of the above. Leadership is not easy and often it means putting your personal goals aside for the greater good of the group. Often leaders are easy to pick out amongst a group as others often follow their lead and gravitate to them in difficult times. Leadership will never be easy and for those select few who elect to receive the mantle of leadership, you are a special breed.

John Rogers was one of those who elected to receive the mantle of leadership. John entered medicine at a time when emergency medicine was still a relatively novel specialty. Board certified emergency medicine specialists were still relatively rare and most emergency departments where staffed by a hodge podge of providers. John trained in general surgery but found his love in emergency departments. Due to only bad timing, John found himself without the ability to become board certified without completing another residency. Since that time, John has toiled in rural emergency departments for the betterment of all our patients and become a legacy emergency physician.

I want to be very clear that this editorial is not meant to be a treatise on board certification. I believe that residency training should be the only means to board certification. I also believe that board certified emergency medicine physicians provide the best care to emergency patients. However, I am also a realist and understand the past. We stand on the shoulders of giants, most of whom were never certified in emergency medicine but represented out specialty well and lead us to where we are today. In addition, it takes a village to care for a community. We currently do not have enough emergency physicians to staff all emergency departments. In many cases rural emergency departments are staffed by non-boarded emergency medicine providers out of necessity.

John is one of the last giants (although he would not tell you this himself). He has become more than just a rural "ED doc" just trying to make a difference. He has worked tirelessly for emergency medicine and our specialty giving much of himself when others would not. If you saw John's speech to the council last year, clearly the council selected the right person to lead our specialty for the coming year. He had vision, was inclusive, and lived the principles of ACEP. The council had spoken and selected John as the next president of our national society. He was the best choice even though he was not board certified similar to many of our leaders in the past. The council saw what I have had the chance to see over the last 10 years, John Rogers is a leader and he was the right man to represent emergency medicine. I am proud to know John and glad to have him in the house of emergency medicine especially in Georgia.

However, I am also embarrassed for our specialty by the events that have unfolded over the last month. Although John resigned his position as president elect of ACEP of his own accord, the events surrounding his resignation give me pause and concerns for our future. In fact, my resolve in support of John is even that much stronger through his resignation and reaffirms my belief that he would have been the right leader for our organization. We are at a tipping point and divisive rhetoric will only make us weaker as a specialty. We have a number of vital issues to our specialty on the horizon and need a strong leader to see us through. Now more than ever, we need to be inclusive and any rhetoric that excludes one

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group or another must be squelched. I have no doubt that our interim president elect is a great leader, and will no doubt represent us well. However, I am saddened by the loss of John and what would have been a great year for ACEP. If you are concerned like I, get involved and make a difference. That is what John did and that is what you should too!

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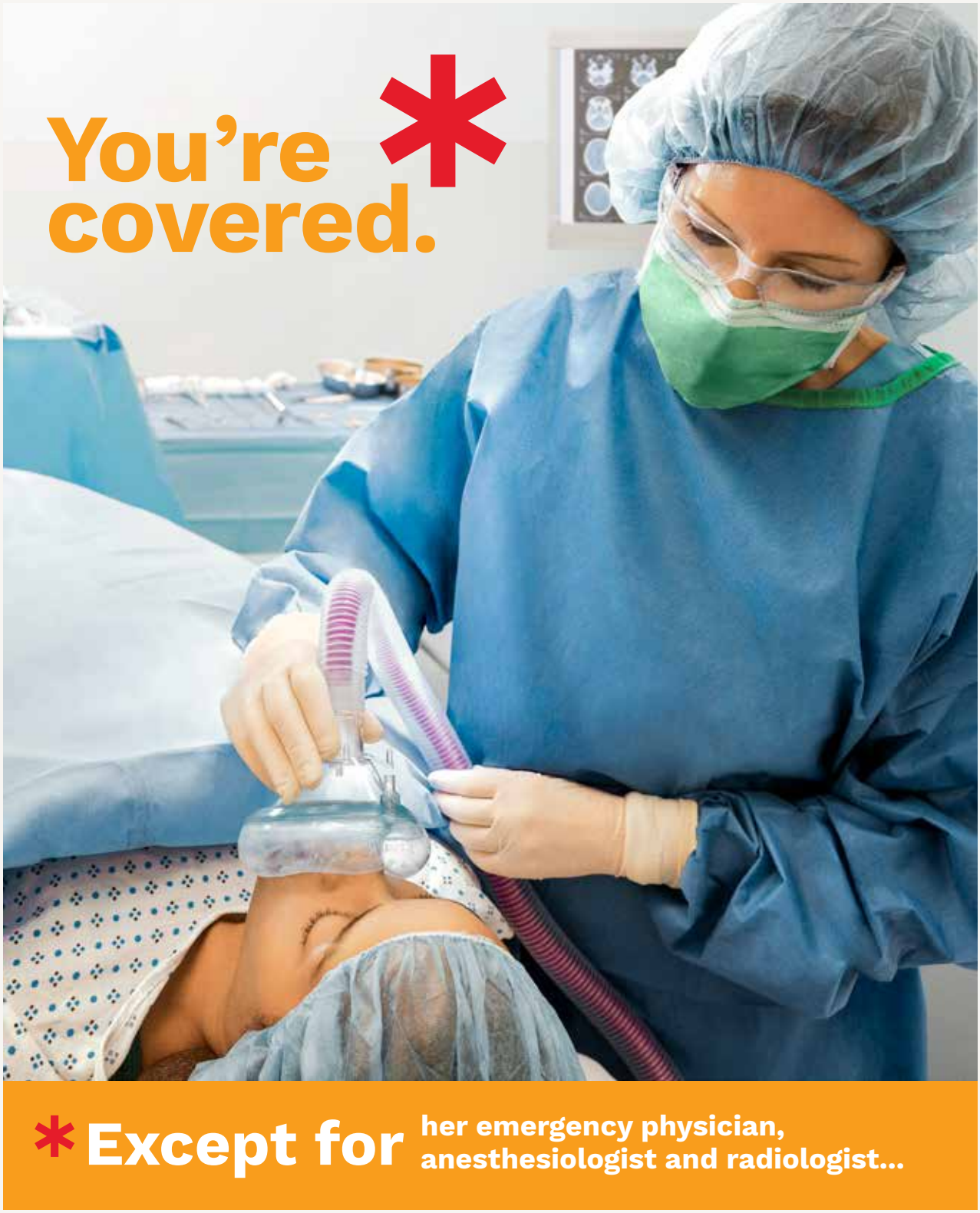

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Pearls in PEM: Cases in Myocarditis

Carmen Sulton, MD and Colleen K. Gutman, MD

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Case 1: A 16-month-old boy presents to the ED with difficulty breathing. He has had congestion and cough for three days. On arrival to the emergency department, he has a temperature of 39.4C, heart rate 180, respiratory rate 52, BP 85/60, and SpO2 95% on RA. He appears tired and is clinging to his father, with copious clear rhinorrhea. He has mild subcostal retractions and clear breath sounds. He is tachycardic without a gallop or murmur. He has strong central pulses and cap refill is 3-4 seconds. His abdomen is soft and he has no hepatomegaly. He is given acetaminophen, and on re-evaluation his temperature is 37.4, heart rate 160, respiratory rate 48, BP 85/60, and SpO2 95% on RA. A chest x-ray shows mild cardiomegaly. Cardiology is consulted and obtains an echocardiogram, which shows moderate left ventricular dysfunction. He is admitted to the general pediatrics floor and receives intravenous immunoglobulin (IVIG) for treatment of myocarditis.

Case 2: A 9-year-old girl presents to the emergency department with two days of fever, fatigue, nausea, and vomiting. While in the waiting room, she experiences a syncopal episode. Her parents report that she had a similar episode a day prior. She regains consciousness and is found to have a heart rate of 37. She is afebrile with a normal respiratory rate and her blood pressure is 110/74. She appears fatigued but is arousable. She has strong central pulses and her distal capillary refill is slightly prolonged. An EKG shows third degree heart block (Fig 1). Cardiology is consulted and recommends initiation of isoproterenol. An echocardiogram shows a mildly hypertrophied left ventricle with mildly depressed left ventricular function and normal right ventricular size and function. She is admitted to the pediatric cardiac intensive care unit and requires trans-venous pacing while receiving IVIG for treatment of myocarditis. At the time of discharge, her heart block has resolved and her echocardiogram has normalized.

Pediatric myocarditis is a rare disease with high mortality. Most often caused by viral infection, pediatric myocarditis can have a gradual onset with an initially subclinical presentation or can rapidly progress to fulminant myocarditis and cardiogenic shock. Although rare, mortality rates can range from 25 – 75%.[1] Additionally, myocarditis is likely responsible for a significant portion of pediatric sudden death.[2] Recognition of pediatric myocarditis is essential in the emergency department, as appropriate stabilization and management is key. Although data to support its use is limited, intravenous immunoglobulin (IVIG) is often used as part of the management for pediatric myocarditis.

Children with myocarditis often present with widely varied signs and symptoms, and the index of suspicion for evaluation and diagnosis must be high. In 2007, Freedman et al. published a retrospective review of all patients diagnosed at a single academic children's hospital over a six-year period.[1] In this cohort, the incidence of myocarditis was 5/100,000 emergency department visits, with 31 cases of myocarditis diagnosed over the six-year period. Children under age 10 were most likely to present with respiratory symptoms and children over age 10 were most likely to present with cardiac symptoms, such as chest pain or palpitations (Table 1). In addition, nearly a quarter of all children presented with signs of hypoperfusion, including lethargy, dizziness, syncope, or seizure. A small number of children also presented with gastrointestinal symptoms. Tachypnea and/or respiratory distress were found in most children. Nearly two-thirds of children were tachycardic on presentation. Only one-third of children had abnormal heart sounds and a similar number had hepatomegaly. With these widely varied presentations, a quarter of the children ultimately diagnosed with myocarditis were not suspected to have this diagnosis in the ED.

Although signs and symptoms of pediatric myocarditis can be widely variable, an EKG and echocardiogram can be more revealing (Table 1). In the study by Freedman et al, nearly all patients had an abnormal EKG; the majority of abnormalities were changes in the ST/T segments, but also included axis deviation, ventricular hypertrophy, and heart block. The EKG was abnormal in 93% of patients, and the echocardiogram was abnormal in 87% of patients. Chest X-ray abnormalities (including cardiomegaly, venous congestion, and effu-

sion) were seen in half of patients. Cardiac biomarkers are not well studied in children and can lack specificity. However, in 2012 Eisenberg et al. published a retrospective cohort study of all previously healthy children who had cardiac Troponin T (cTnT) sent as part of the evaluation for myocarditis and found that a cutoff of 0.01 ng/mL had a sensitivity of 100% and a specificity of 85%3. None of the children in their cohort with cTnT < 0.01 ng/mL had myocarditis. Although most of the children with positive results did not have myocarditis, many were later diagnosed with other serious illnesses (i.e. shock, rhabdomyolysis, pulmonary embolism).

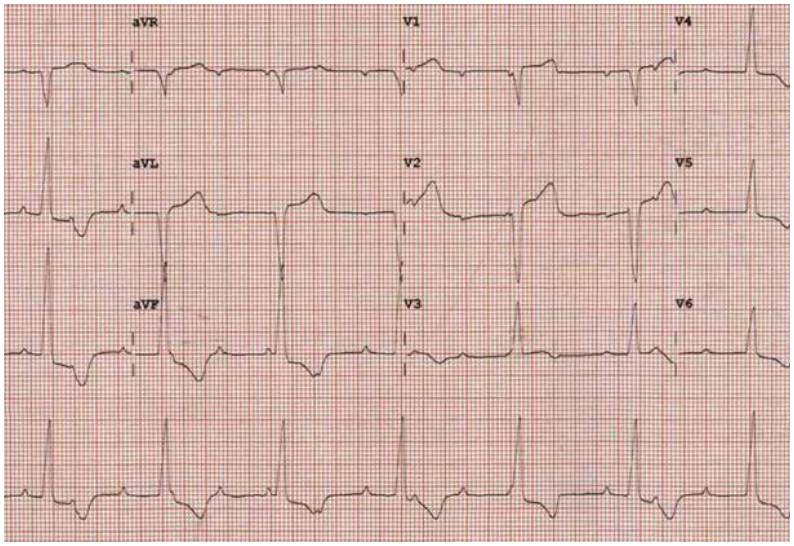

Figure 1 Case 2—EKG showing third-degree heart block

Take Away Points

Pediatric myocarditis, although uncommon, has high associated morbidity and mortality and the presentation can be subtle and nonspecific. Although many older children present similar to adults, with typical cardiac complaints, nearly half present for other concerns, such as cough or syncope. Younger children are unlikely to present with a cardiac-related chief complaint and are more likely to present with symptoms that may be confused for pneumonia, asthma, acute gastroenteritis, or dehydration. Tachypnea and respiratory distress, followed by tachycardia, are the most commonly abnormal physical exam findings, but these are not present in all children. Clinical signs of heart failure, such as abnormal heart sounds and hepatomegaly, are not common. The initial evaluation for myocarditis should include an EKG and CXR, although the latter may be normal. Consider obtaining cardiac biomarkers as well. Early consultation with a pediatric cardiologist is recommended.

Table 1. Presentation and findings in children with myocarditis[1,3]

Presenting Signs and Symptoms by Age	
Less than 10 years	
Respiratory illness (rhinorrhea, cough, dyspnea)	47%
Hypoperfusion (lethargy, dizziness, syncope, seizure)	20%
Kawasaki-associated	20%
Gastrointestinal illness (nausea, vomiting, diarrhea, abdominal pain)	13%
Greater than 10 years	
Cardiac symptoms (chest pain, palpitations)	56%
Hypoperfusion (lethargy, dizziness, syncope, seizure)	25%
Respiratory illness (rhinorrhea, cough, dyspnea)	19%
Findings on Exam and Work-Up	
Abnormal respiratory exam (distress, tachypnea)	68%
Tachycardia	58%
Hepatomegaly	36%
Abnormal heart sounds	32%
Fever	30%
Hypotension	23%
Abnormal CXR (cardiomegaly, pulmonary venous congestion, pleural effusion)	55%
Abnormal EKG (ST/T wave changes, axis deviation, ventricular hypertrophy,heart block, decreased voltage, atrial enlargement)	83-93%
Abnormal echocardiogram	78-87%
Elevated cardiac troponin-T	
0.01-0.09 ng/ml	10%
0.1-0.99 ng/ml	50%
> 1.0 ng/ml	30%

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Pediatric Update: Emory Univ. School of Med.

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The Emory Department of Pediatric Emergency Medicine celebrated the graduation of three fellows. Ning Chan, MD will be staying on as faculty here at Emory University in Atlanta, GA. Her academic interests include medical student education. Nicholas Jones, MD accepted a faculty position at Vanderbilt University in Nashville, TN. His academic interests include procedural sedation and resident education. Mahati Reddy, MD will join the faculty at New York University Children's Hospital in New York City. Her research interests include improving emergency department care and pain management of sickle cell patients. All of our graduating fellows have presented research at national meetings, served Children's Healthcare of Atlanta on numerous committees and educated medical students and residents within the school of medicine. We wish them well!

Our Division will soon see a change in leadership. Dr. Naghma Khan, who has served Children's Healthcare of Atlanta and Emory University for over 21 years will be stepping down as Division Chief. After 21 years, Dr. Khan has served as longest Division director in CHOA history. She was also the recipient of the GCEP Medical Director of the Year Award. She has grown the Pediatric Emergency Medicine Division to over 45 physicians, fellows, and mid-levels. She has invested time into our ultrasound, teaching, sedation and quality endeavors as well as increasing physician productivity. She has secured millions of dollars in federal funding, developed the largest PEM Fellowship program in the southeast, and laid a groundwork of success for future leaders in one of the highest volume pediatric emergency departments in the country. Under her leadership, the majority of graduates from the PEM fellowship program have remained within the state of Georgia and have gone on to develop teaching programs on the local, regional, national and international levels.

Dr. Naghma Khan

Dr. Srikant Iyer

In keeping with her visionary track record, she and her search committee have successfully recruited another well-known national leader to take the helm for the division. Dr. Srikant Iyer will assume the position of Director of Emergency Services for CHOA and director for the Emory Division of Pediatric Emergency Medicine in August 2018. The entire CHOA and Emory communities are thankful for decades of leadership under Dr. Khan and are excited to embark on the journey with Dr. Iyer.

Emory PEM Class of 2018 (l-r): Ning Chan, MD; Mahati Reddy, MD; and Nicholas Jones, MD

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CLINICAL

A Rare Case of Pyelopnephritis Associated Macroscopic Ureteral WBC Casts

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Introduction

Urinary tract infections and pyelonephritis are fairly common maladies treated in the emergency department (ED). A population-based study of acute pyelonephritis in the United States found overall annual rates of 15-17 cases per 10,000 females and 3-4 cases per 10,000 males. Most cases are treated as outpatients, but 3-4/10,000 women and 1-2/10,000 men required admission.[1] While it is common to find microscopic white blood cell casts in the urine of patients diagnosed with pyelonephritis, a systematic literature review did not yield any descriptions of macroscopic ureteral white blood cell casts.[2] We present a rare case of a patient presenting with a macroscopic white blood cell cast formed in the ureter as a result of a pyelonephritis infection.

Case report

A 33-year-old previously healthy man presented to the ED with a one-week history of painful urination, body aches, fatigue, nausea, vomiting and chills. On the morning of presentation, he reported passing worm-like "tissue" from his urethra during urination. The patient was afebrile and his vital signs were within normal limits. Physical examination was unremarkable other than left lower quadrant abdominal and left flank tenderness. Examination of the specimen revealed two long thin fragments of tan soft to mushy material. The larger fragment measured 4.3 x up to 0.5 x up to 0.3 cm and the smaller fragment measured 3.0 x up to 0.5 x up to 0.3 cm. (Figure 1). Laboratory testing was remarkable for a white blood cell (WBC) count of 20,600 cells per millimeter cubed (mm3), moderate urine leukocyte esterase, and too numerous to count white blood cells were observed on microscopic examination of the urine. An abdominal CT scan showed severe left pyelonephritis with early intrarenal abscess formation, a possible ureteral cast protruding into the bladder and marked inflammatory changes of the left ureter with bladder wall edema at the left ureterovesicular junction. Urine culture grew Gram negative lactose-fermenting bacilli. One of two blood cultures was positive for Klebsiella pneumoniae. A portion of the "tissue" was sent for culture and grew Klebsiella pneumoniae. The remainder of the specimen was sent to pathology, which reported that the material most likely represented a tubular cast formed in the patient's ureter by abundant purulent material secondary to his recently discovered pyelonephritis.

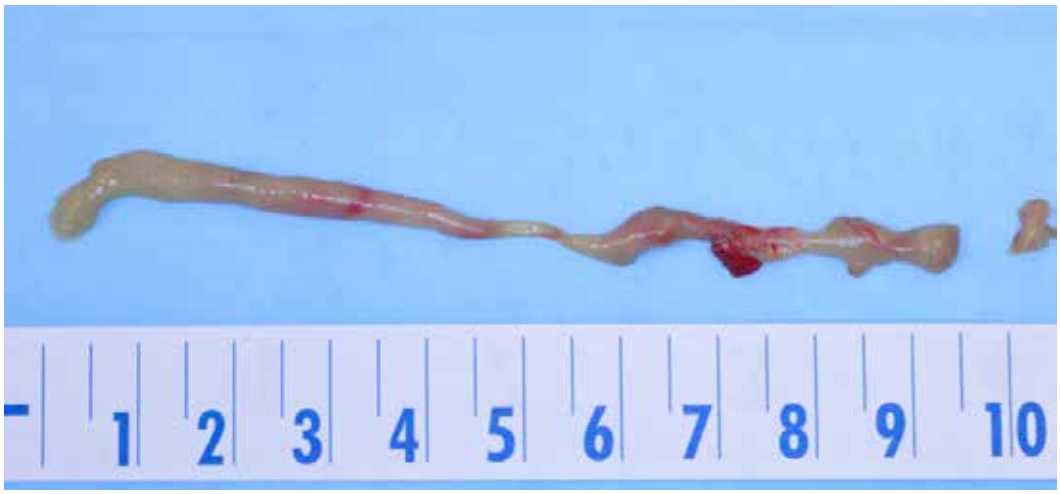

Figure 1: Macroscopic white blood cell cast associated with pyelonephritis.

The patient was evaluated by urology resident physicians in the emergency department who determined there was no need for and urologic interventions; and the patient was admitted to the internal medicine service for treatment of severe pyelonephritis. During the four days of hospitalization the patient received intravenous piperacillin and tazobactam. At discharge he was treated with oral ciprofloxacin for an additional ten days.

Discussion

This case describes a macroscopic white blood cell cast that likely formed in the ureter of a patient with pyelonephritis. While microscopic WBC casts are commonly found in the urine of patients with pyelonephritis, to our knowledge this is the first report of a macroscopic WBC cast.

Urinary casts are microscopic cylindrical structures formed in the renal tubules by protein, cells, and debris. White blood cells (WBC) in urine are suggestive of infection or inflammation. Because WBC casts are formed in the tubules of the kidney, their presence suggests kidney disease rather than a source in the lower urinary tract. The presence of a hyaline matrix allows differentiation between a WBC cast and a clump of WBCs. WBC casts can be found in pyelonephritis, glomerulonephritis, acute interstitial nephritis, lupus nephritis, and acute papillary necrosis.[2]

Pyelonephritis is caused by bacterial invasion of the renal pelvis or parenchyma. Bacteria usually reach the kidney by ascending from the lower urinary tract, often resulting from untreated or undertreated cystitis. Bacteria can also reach the kidney through the bloodstream. Pyelonephritis caused by gram negative organisms accounts for 70-95% of cases and is almost always the result of ascending infection.[3] Infection caused by Gram positive organisms is usually the result of hematogenous spread. Uncomplicated pyelonephritis is rarely fatal and can be treated on an outpatient basis with oral fluoroquinolones, cephalosporins, aminoglycosides, or trimethoprim/sulfamethoxazole, either alone or in combination.[4] The development of complications significantly increases the morbidity and mortality. A urinary source is the cause of 20-30% of sepsis cases, with sepsis carrying a mortality rate of 20-40%.[5,6] In rare cases acute renal failure can result from pyelonephritis and recovery is slower than when renal failure is caused by other factors.[7] Emphysematous pyelonephritis is rare, mainly affecting immunocompromised patients, and is associated with significant morbidity and mortality.[8]

The patient in this case is an otherwise healthy 33-year old male with no history of urinary tract infections. He presented to the ED after a week of symptoms and the disease process was already fairly advanced. The patient did not require any procedural interventions and was treated with antibiotics. His condition improved with treatment, he was able to be discharged home after four days of hospitalization and made a complete recovery.

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CLINICAL

Emergency Department Management of Penetrating Neck Trauma: A Literature Review

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Introduction

Penetrating neck trauma is not only relatively common, but associated with approximately 50% incidence of serious vascular, aerodigestive, or neurologic injuries, as well as significant risk of morbidity and mortality.[1] These injuries present several concomitant challenges for emergency physicians, as they require elements of airway management, hemorrhage control, hemodynamic resuscitation, performance of the trauma survey and other components of advanced trauma life support, knowledge about potential injuries and other complications of penetrating neck trauma, and the ability to perform rapid evaluation and initial management of penetrating neck trauma.[2,3]

Stable patients who do not require immediate intervention for vascular or airway compromise should undergo early assessment of the wound. Local anesthetic may be required in order to facilitate an exam which is thorough enough to determine whether or not the platysma has been breached. By definition, penetrating neck injuries must violate the platysma; those which do not are superficial neck injuries.[4] Several important structures may be injured as a result of penetrating neck trauma.[1,4] One quarter of these patients will have arterial injury, with 80% of those involving the carotid arteries.[5] One quarter will have aerodigestive injuries,[2] which are plagued by a lack of initial physical findings and markedly increased morbidity and mortality associated with delayed diagnosis.[1]

Trauma Association Guidelines and Recommendations

According to the 2013 guidelines of the Western Trauma Association (WTA) regarding the management of penetrating neck trauma, management of these injuries should include: following Advanced Trauma Life Support protocols for identification of life-threatening injuries and initiation of immediate life-saving interventions; temporizing hemorrhage control of active bleeding via manual pressure or balloon catheter tamponade; establishment of secure airway, including surgical airway if necessary due to failure of orotracheal techniques; and emergent surgical intervention for patients exhibiting hemodynamic instability or hard signs.[2] Hard signs include airway compromise (indicated by hoarseness, stridor), massive subcutaneous emphysema or gurgling wound with air bubbling through it, shock, active bleeding, expanding or pulsatile hematoma, other signs of vascular injury such as bruit or pulse deficit, dysphagia, hematemesis, and neurologic deficit.[1,2] Any symptomatic zone II injuries, which WAT defines as those above the line of the clavicles, should also proceed to the operating room (OR), for further evaluation in a controlled setting where escalation of care is readily available.[2]

For stable patients with a penetrating wound and suspicion of resultant injuries but no hemodynamic instability or hard signs, non-operative evaluation via advanced imaging is appropriate for most zone I and III injuries, as well as asymptomatic zone II injuries.[2] As compared to mandatory surgical exploration, selective operative management of zone II penetrating neck trauma has shown equivalent patient safety diagnostic accuracy, with reduced incidence and associated costs of unnecessary operations.[1,2,6] Eastern Association for the Surgery of Trauma (EAST) recommendations agree with these findings and recommend selective surgical management as well1. For these patients, computed tomography angiography (CTA) is recommended, as it is highly sensitive for identifying significant injuries which need further operative management, while decreasing the incidence of unnecessary operations.[1,3,7,8] Esophageal injuries are best evaluated by contrast esophagography or esopha-

goscopy, if suspicion warrants further evaluation after negative CTA.[1,3] Venous injuries are not uncommon, but rarely require surgical intervention.[1,2,4]

For stable patients without suspicion of serious injuries, observation and serial examination versus radiographic evaluation is appropriate.[2,6] If the former option is chosen, it should be done cautiously, keeping in mind that while physical examination is quite reliable for excluding arterial injuries, it is less dependable for excluding aerodigestive injuries.[7] For transcervical gunshot wounds, the observation strategy is controversial, as these wounds have a higher incidence of significant injuries and are more likely to involve multiple zones.[1,2] For this reason, EAST recommends that physicians have a low threshold for utilizing advanced imaging.[1] Patients who are candidates for observation should undergo serial examinations every 6 to 8 hours for a total of 24 to 36 hours.[3]

To Zone or Not to Zone

Anatomic zones are much more relevant to the surgical approach than they are to the initial evaluation and management.[2] While the line between zone I and zone II has been changed from the clavicles to the cricoid cartilage, this has been without great significant clinical relevance,[9] and many still prefer the former definition, which was based on surgical approach and exposure rather than on presumed underlying injuries.[2,3] WTA guidelines have opted to utilize the initial boundaries, where zone I lies below the level of the clavicles.[2]

For initial evaluation and management of penetrating neck trauma outside of the OR, recent evidence suggests that anatomic zones should no longer be a major consideration. The apparent anatomic zone of external wounds does not appear to reliably correlate with underlying internal injuries,[10] and the use of anatomic zones in management algorithms does not appear to improve patient outcomes.[11] As a suggested alternative, all patients with hemodynamic instability and/or hard signs should go to the OR emergently regardless of zone, and all stable patients with concern for serious underlying injury should be evaluated with CTA,[4,12] which is now readily available and reliable at detecting and excluding injuries regardless of zone.[1,12] The exception to the zoneless approach would be for injuries beneath the clavicles, for which management should be based on the potential for thoracic and axillary injuries.[3,4]

Airway

Airway management can be more difficult in penetrating neck trauma than in many other situations, due to the possibility of disruption of normal anatomic airway structures, or occlusion by hemorrhage or hematoma.[3,4] Such cases require the physician to be prepared ahead of time to work through a difficult airway algorithm.[4,13]

Many of the hard signs of penetrating neck injury are important indications of potential airway injury—including hoarseness, stridor, dyspnea, subcutaneous emphysema, or air bubbling through the wound.[3,4,14] In the absence of obvious structural deformity of the airway anatomy, rapid sequence orotracheal intubation should be attempted and has a satisfactory success rate.[13,14] In the presence of anatomic deformities, alternative techniques are preferred. For compression by a large hematoma, for example, fiberoptic intubation should be attempted if available,[3,13] both because it allows the physician better visualization to avoid intubating a false tracheal lumen created by the injury plane, and because it allows the patient to remain spontaneously breathing as long as possible.[4]

When there is massive airway or midface deformity, inability to visualize or identify the airway during direct or fiberoptic laryngoscopy attempts, or any otherwise failed intubation with inability to obtain adequate oxygenation or ventilation; surgical airway is immediately indicated. Cricothyrotomy is preferred for its safety and the quickness with which it can usually be performed; unless there are contraindications such as transection of the trachea below the level of the cricothyroid membrane,[3,4,15] in which case exposure and intubation through the tracheal defect,[3,13] or tracheostomy will be necessary.[3,4,15] Airway injuries inferior to this point may be very difficult to identify and manage, and confer higher mortality. When tracheal intubation is unable to ventilate these patients due to lower airway injuries, extra corporeal membrane oxygenation may be considered.[13]

Hemorrhage

Hemorrhage is a significant cause of mortality in patients with penetrating neck injuries; therefore hemorrhage control is a crucial aspect of management. Hemorrhage control may be attempted with direct external compression,[3,4,16] but if this is not successful, internal digital compression, gauze packing,[3] or a foley catheter balloon may be used to tamponade the bleeding from within. When the balloon catheter is used, the wound may be closed tightly around the balloon catheter insertion site to facilitate tamponade. The balloon should be left in place until surgical repair is performed, or if angiography is performed and excludes arterial injury, for approximately 72 hours to allow hemostasis.[3,4,6,16-18]

Cervical Spine

While penetrating neck injuries of other mechanisms have not been associated with clinically significant cervical spine injuries in the literature,[19,20] a low proportion (0.4 – 1.4%) of gunshot wounds to the neck do result in unstable cervical spine fractures or cervical spinal cord

injuries.[20,21] These injuries are almost always associated with neurologic deficit on initial assessment, and their incidence is much lower than that of other injuries such as vascular injuries.[19,20] In such cases, cervical spine immobilization (CSI) should be considered if there significant and specific concern for such an injury.[4,21]

In most other cases, cervical spine immobilization (CSI) should not be routinely performed for penetrating neck trauma, because it is low yield and takes time and focus off of the assessment and management of other injuries.[4,19] CSI makes pre-hospital transport significantly more time-consuming,[19] and will inevitably impair physicians' ability to safely and efficiently assess and manage the airway and vascular injuries.[4,19]

Conclusion

Penetrating neck trauma presents a complex set of challenges for emergency physicians,[1,2,4,5] who are in a position to reduce morbidity and mortality by preparing to evaluate and manage these injuries in a timely and evidence-based manner.[2,3,16] In summary, unstable patients or those with hard signs should be taken to the OR emergently after a definitive airway is secured and hemorrhage is controlled.[2-4] Those who are stable without hard signs should be evaluated with CTA,[1,3,7,8] and additionally, contrast esophogram or esophagoscopy if there is lingering concern for esophageal injury.[1,3] Observation management should be selected with caution, as aerodigestive injuries lack early signs and are associated with much higher morbidity and mortality if diagnosed late.[1,2,7] Airway management is more difficult in these patients, due to airway injuries and compression or obstruction by blood or adjacent injuries.[3,4,13] Physicians should be prepared to work through difficult airway algorithm,[3,4,13-15] including techniques such as performing a cricothyrotomy,[3,4,15] or intubating through an exposed tracheal injury.[3,13] Hemorrhage control is also extremely important, and can usually be accomplished with techniques such as direct external compression, digital internal compression, gauze packing, or foley catheter balloon tamponade.[3,4,16] Cervical spine immobilization should not be routinely used, as it delays evaluation and management of more likely life-threatening injuries;[4,19] with the exception of gunshot wounds or patients with neurologic deficits, in which case it should be considered.[20-21]

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Diagnosing Panic Disorder in the ED—Beware the Clinical Inconsistency

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Anxiety is a fairly common associated symptom experienced by patients in the ED. After all, fear of illness combined with unfamiliar surroundings, lack of access to their primary care doctor and the sights, sounds, smells of the chaotic environment of the ED makes for a situation in which it qualify as almost abnormal not to be apprehensive. That said, incidental situational anxiety differs greatly from the panic attack, an emergent condition that can produce incapacitating symptoms of such severity that they may be the precipitant of the visit to the ED itself.

The capricious nature of these symptoms along with their overlap with those of potentially dangerous conditions can make caring for these patients a diagnostic and therapeutic challenge. Deciphering the serious organic from the purely functional anxious condition is our prime directive when evaluating these patients. Symptoms such as chest pain, shortness of breath, numbness, nausea and diaphoresis are all commonplace in panic disorder but alternately can present as a harbinger of other more potentially lethal conditions that run the gamut from ACS, Pulmonary embolus, severe anemia, acidosis and a variety of other causes.

To safely manage these patients and minimize the risk of misdiagnosis it behooves us to always retain a posture of wariness and skepticism when confronted with perplexing symptomatology and diligently work through a prudent differential diagnosis prior to assigning a patient an isolated diagnosis of anxiety.

Many of us tend to rely on our gut in these matters, a "feeling" that may be derived from a combination of patient demeanor on presentation, a history of recent circumstantial stress, and what strikes us as a set of disparate symptoms that do not seem to coalesce into a coherent unifying diagnosis. Though intuition is a critical tool that aids us every day in medical decision-making, it can be quite perilous to depend on it in isolation without consistent supportive clinical findings. We must likewise be especially cautious and mindful to avoid the trap of confirmation bias in clinging to the initial gut diagnosis in the face of contradictory findings.

Panic should always be a diagnosis of exclusion and history is critical. One should ascertain from the get go if the patient has a past history of panic attacks presenting with similar symptomatology (reassuring) and/or of relevant organic disease (should give us pause). One should be particularly cautious when diagnosing new onset panic disorder without careful workup in those of more advanced age with coexistent past history of cardiac, pulmonary or neurologic disease. New onset of panic disorder in the later years of life is not unheard of but is fairly uncommon.

When making the diagnosis one must make sure that all components of clinical evaluation combine to support your clinical impression. Clinical outliers must be identified and explained in the record. Predictors of potential for misdiagnosis are several and should be recognized as such, worked up completely and dispositioned safely.

Vital signs are a sensitive indicator for organic pathology. Certainly presence of fever or hypotension is inconsistent with panic disorder and should immediately guide one toward consideration of other etiologies for presenting symptoms. Similarly, significant tachycardia, though common and consistent with panic attack, is nonspecific and may be indicative of other more sinister pathologies such as pulmonary embolism, sepsis, cardiac issues and metabolic abnormalities to name just a few. When present it is reasonable to initially treat with anti-anxiety agents and fluids. If the pulse rate normalizes and the patient feels better, dis-

position home is reasonable. Should tachycardia persist however, then organic causes become more likely and more in-depth workup and admission for observation should be considered. Though sensory phenomena such as paresthesias are also quite common in panic disorder, any unusual distribution such as unilaterality involving more than one limb and/or the unilateral face with forehead sparing should generate concern for neurologic pathology such as stroke or TIA. Of course, presence of any new onset focal motor deficits should put the diagnosis of anxiety in serious doubt and admission for observation with neurology consultation is advised. Even if these sensorimotor deficits were described subjectively and have resolved prior to arrival in the ED, one must consider TIA as a potential etiology and recognize it as a threat for recurrence and evolution into future stroke.

In conclusion, as with all of our patients, thin slicing is a well-developed and important skill for ED clinicians to aid in managing flow through a busy ED. Nevertheless, to minimize risk and provide optimal care to our patients we must always maintain that wide differential diagnosis and double back after initial evaluation to perform further drill-down on history, exam and testing to verify our initial impressions regarding diagnosis and disposition in these sometimes challenging cases.

Thank you to all who contributed to our legislative advocacy fund this year. Without this help we would not have had the resources to fight very bad legislation in the state legislature. We need your continued support the "Stop the Insurance Gap" campaign.

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5 Ways to Lower the Cost of Disability Income Insurance

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While you may think that your investment portfolio or your home is your biggest asset, for most emergency medicine physicians your ability to produce income (your future income stream) usually dwarfs the current assets you own — especially in the first half of your career. Because of this, protecting your future income stream by purchasing disability income (DI) insurance is a must, but I've encountered many physicians who are reluctant to buy DI insurance because of the relatively high premiums. [In full, disclosure I do not sell insurance.]

However there are several strategies to make disability coverage more affordable:

1. Lengthen the elimination period

The elimination period is the period of time after which the insurance company will start paying you benefits. The longer the elimination period, the lower your premium. This means that you've got to have enough reserves to support you and your family for a period of time when you have no income. The most common elimination periods are 90 days and 180 days but can be longer than that if you choose. If you have an adequate emergency fund, other sources of income (spouse works for example), or your expenses are modest then you should consider a longer elimination period. You may also want to skip buying short term disability insurance (usually offered through group coverage) altogether.

2. Shorten the benefit period

If you're just starting out in your EM career, you're better off choosing a policy which pays you benefits until your retirement age — usually age 65. If you've done a good job of saving and building wealth and reducing debt over your career, you can lower your premium by shortening the benefit period which is the number of months or years that the DI policy pays you. In that case a benefit period of say 5 years might make sense — premiums could be 20-30 percent lower.

3. Lower your coverage

When you go through the underwriting process to buy DI insurance, usually the insurance company will quote you the maximum DI insurance you're eligible for. They won't replace all of your pre-disability income. Instead it's usually capped at 60-70% of your pre-disability income.

But what if your monthly expenses are much lower than the amount of DI insurance benefit quoted to you? In this case you can opt to lower the DI insurance benefits instead of accepting the maximum amount offered to you. CAUTION: This is risky because it's possible that your expenses could go up when you are disabled. Again, you'll need a large investment portfolio or other source to cover the gap in this situation.

4. Eliminate riders

There are a number of riders you can add to the base DI insurance policy such as catastrophic disability coverage, cost of living adjustments, own occupation coverage, residual disability coverage and others. In my opinion several of these riders are quite valuable — such as own occupation coverage which pays you disability benefits even if you work in another occupation.

But if your goal is to reduce your premium so that you're more likely to get at least some DI insurance coverage rather than having none, then skipping the riders could save you a good chunk of money.

5. Buy group disability insurance

Individual DI insurance is usually more expensive than group insurance. If you're an employee of a hospital system or other EM group, take a look at the benefits guide from your employer and see if they offer group disability insurance. Sometimes the employer pays the premiums for you for a basic long term DI insurance policy. However you may have the option to purchase more for a modest deduction from your paycheck. Realize that group DI insurance policies have limitations such as a less favorable definition of disability and the inability to take the policy with you if you leave the group. Also realize that the premiums tend to go up as you age. If this is the only type of disability coverage you can afford, or if you already have health problems or can't otherwise get coverage, having group disability insurance is a lot better than having none.

Realize that with insurance — like anything you buy — you can't just look at the cost. You've got to consider the potential benefit if the policy pays out and the consequences of not having adequate coverage. You'll have to decide whether it's worth risking the quality of coverage to save some money, but if the pain of the premium is too high for you, then the strategies outlined might soothe some of that.

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