University of Cincinnati Medical Center
Department of Emergency Medicine

Annals of B Pod

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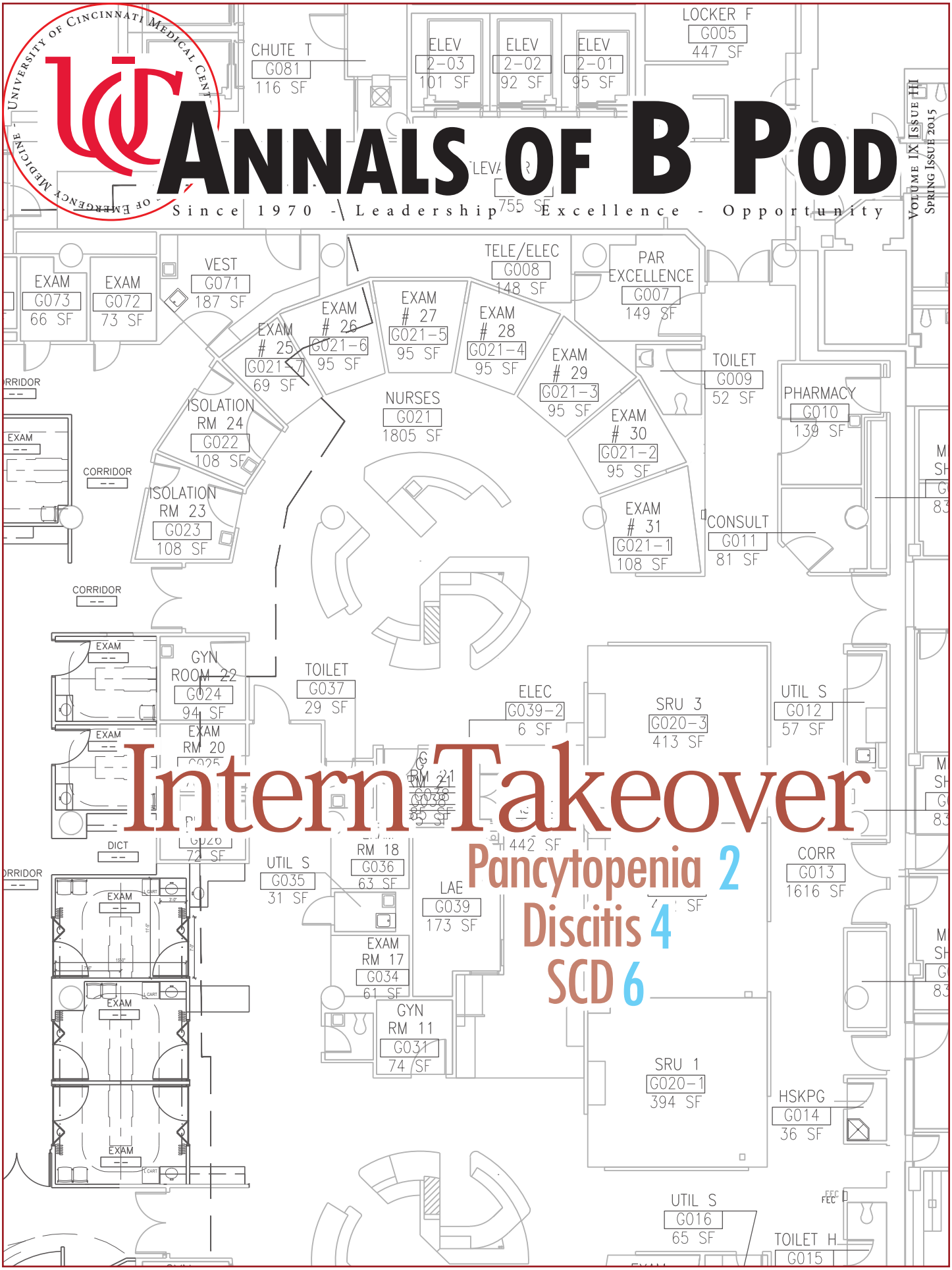

Intern Takeover

Pancytopenia 2
Discitis 4
SCD 6

UC Emergency Medicine
Annals of B Pod
Spring 2015

A Time for Turnover

It's a special time of year, step-up shifts abound and everyone is looking forward to what comes next. The R1s are zipping on their flight suits and stepping into C-pod, the R2s are applying two years of clinic knowledge in the SRU while learning how to balance endless phone calls and consultants, and the R3s are making the ultimate transition back to B-pod, into the teaching role that accompaines the R4 desk. Staying true to the step-up theme, this issue is dedicated to our R1s, who "stepped up" and authored all of the B-pod cases presented in this issue themselves

Doc, I'm tired

and I done fell out

Walker Plash, MD
University of Cincinnati R1

case originally seen by Walker Plash, R1 and Megan Redmond, R4

History of Present Illness

The patient is a 61 year old female with a past medical history of iron deficiency anemia, hypertension, and poorly controlled type II diabetes who presented to the emergency department after an episode of syncope. The patient reported she noticed a gas leak in her apartment the night of presentation. Prior to being exposed to any noxious fumes, she left her apartment. While standing outside, the patient had an episode of syncope, which convinced her to come into the emergency department to be evaluated. In the several months leading up to presentation, the patient reported worsening light headedness and generalized weakness. She also reported epigastric abdominal pain, polyuria, nausea, and nonbloody, nonbilious vomiting over the week prior to presentation. The patient denied shortness of breath, chest pain, palpitations, bleeding, melena, head trauma, loss of control of bowel or bladder, or post-ictal state upon regaining consciousness.

Past Medical History

Iron deficiency anemia, Hypertension, Diabetes, Abnormal pap smear

Past Surgical History

None

Medications

Clobetasol ointment, Clotrimazolebetamethasone, Gabapentin, Mycostatin, Simvastatin

Allergies

None

Social History

Unremarkable

Temp
96.5
Heart Rate
109
Resp Rate
11
BP
117/85
O2 Sat
100%

Labs

WBC: 2.0	B12: <159 (nl 313-618)
HgB: 7.6	Folate: >20 (nl 2.76-17)
Plt: 62	Haptoglobin: <5.83 (nl 36-195)
MCV: 104.6	LDH: 12,342 (nl 313-618)

Physical Exam

General: Thin female lying in bed, appears in no acute distress
HEENT: Head atraumatic, pupils equal round and reactive to light, extraocular movements intact, sclera clear, mucus membranes dry, oropharynx nonerythematous
Neck: Supple, no lymphadenopathy
Pulmonary: Clear to auscultation bilaterally, no wheezes, rhonchi, or rales
Cardiac: Regular rate and rhythm, normal S1S2, no murmurs, rubs, or gallops
Abdomen: Soft, mildly tender in the epigastrium, nondistended, no rebound and no guarding
Musculoskeletal: No obvious deformities, no tenderness to palpation
Vascular: 2+ radial pulses bilaterally
Skin: Warm, dry, well perfused, no rashes
Neuro: A&Ox4. CN 2-12 intact. Unsteady gait. Sensation intact. Strength grossly equal and symmetric. No deficits with finger-nose-finger.

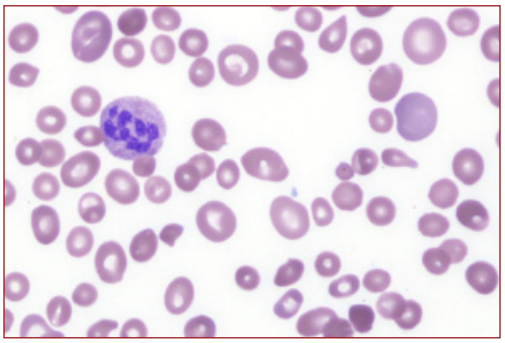

Figure 1: Representative peripheral blood smear of a patient with Vit B12 deficiency. Note the hypersegmented neutrophils, which are pathognomonic for megoblastic anemia. (http://www.bloodjournal.org/content/124/11/1844)

ED & Hospital Course

The patient presented with syncope and generalized weakness. On initial laboratory testing, the patient was found to be pancytopenic, with significantly decreased WBC, RBC, and platelets. Given her symptomatic anemia, the patient was admitted to the general medicine service. Based on the patient's elevated MCV, there was a concern for Vitamin B12 deficiency. These levels were checked, and were below the detectable range. The patient's intrinsic factor antibody was positive, and she was diagnosed with Pernicious Anemia. The patient did require transfusion of 2 units of blood as repeat hemoglobin fell to 6.7. Post transfusion, her hemoglobin responded appropriately and remained stable. She was started on daily subcutaneous B12 shots, with a significant increase in her reticulocyte count, suggestive of positive response to the B12 injections. She was discharged home on weekly B12 injections and followed up with hematology/oncology. One month post admission, her hemoglobin had improved to 13.3.

Discussion

The patient presented with pancytopenia of unclear origin. Generally, pancytopenia can be broken into three categories: reduced production by the bone marrow, increased destruction in the periphery, or a combination of the two mechanisms (Table 1)[1]. In newly diagnosed pancytopenia, these patients typically require admission for further workup and management of their pancytopenia.[2] After easily reversible causes are ruled out (such as B12 deficiency), these patients will undergo a bone marrow biopsy to establish a diagnosis.[1]

Pernicious Anemia

In this case, the patient was found to have a pancytopenia secondary to pernicious anemia. Pernicious anemia is mediated by an autoantibody to intrinsic factor, the factor responsible for absorption of vitamin B12 in the ileum. This leads to the destruction of gastric parietal cells, atrophic gastritis, and further decreased secretion of intrinsic factor, causing a B12 deficiency. There is also some thought that helicobacter pylori plays a role, though this is not completely understood. Patients deficient in vitamin B12 present with a variety of insidious onset symptoms, including megaloblastic anemia and pancytopenia with resultant fatigue and other symptoms of anemia. Patients also have symptoms of gastritis, including reflux and diarrhea, as well as glossitis. Finally, these patient's also have progressive neurologic symptoms secondary to demyelination, including ataxia, paresthesias, and mild cognitive impairment. Apart from gastric symptoms, pernicious anemia cannot be easily distinguished from other forms of B12 deficiency on history and physical.[3]

Historically, pernicious anemia was diagnosed with the Schilling test. In this test, patients were initially given a dose of intramuscular B12 to saturate receptors in the body, and thus cause any absorbed B12 from the GI tract to be excreted into the urine. The patient was then given two separate doses of radiolabeled B12, with and without intrinsic factor, and excretion in the urine is compared between the two as a marker of GI absorption. This test is rarely used any longer since tests specific for the antibody now exist.[3]

Treatment for pernicious anemia is repletion of vitamin B12. This can be done with intramuscular B12 or extremely large doses of oral B12, leading to absorption even without intrinsic factor. Once B12 is replaced, the anemia is usually reversed. Neurologic symptoms usually persist, but do not progress further.[3] Reticulocyte response, indicating response to B12 replacement, is generally seen within 4 days.[2] Prior to response to therapy, indications for transfusion are the same as other forms of anemia, including hemoglobin <7 as in this patient, hypoxia, cardiac ischemia, end organ damage, or hemodynamic instability. [2]

If a decision to transfuse packed red blood cells is made, regardless of pure anemia or pancytopenia, it is helpful to send labs prior to transfusion, including a lactate dehydrogenase (LDH), iron, and transferrin saturation. When transfusions are given, LDH is artificially increased, creating a false picture of intravascular hemolysis.[4] Iron and transferrin saturation can either be artificially increased or decreased, which can cloud the origin of anemia.[5,6] However, ferritin levels are not affected by transfusion.[7] Other labs that are not affected by transfusion, but can be generally sent to aid the admitting team include haptoglobin, total iron binding capacity, folic acid, and B12.[7,8]

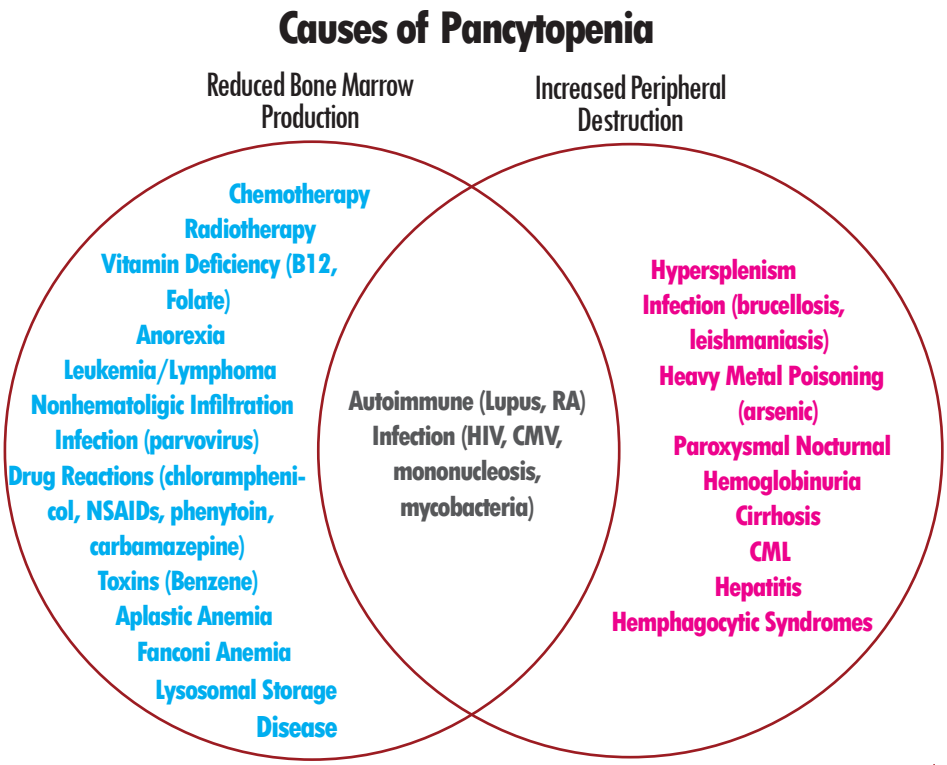

Table 1: Three main categories of Pancyotpenia: Reduced Bone Marrow Production, Increased Peripheral Destruction, or a combination of those processes

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Diffuse pain in a 59 year old

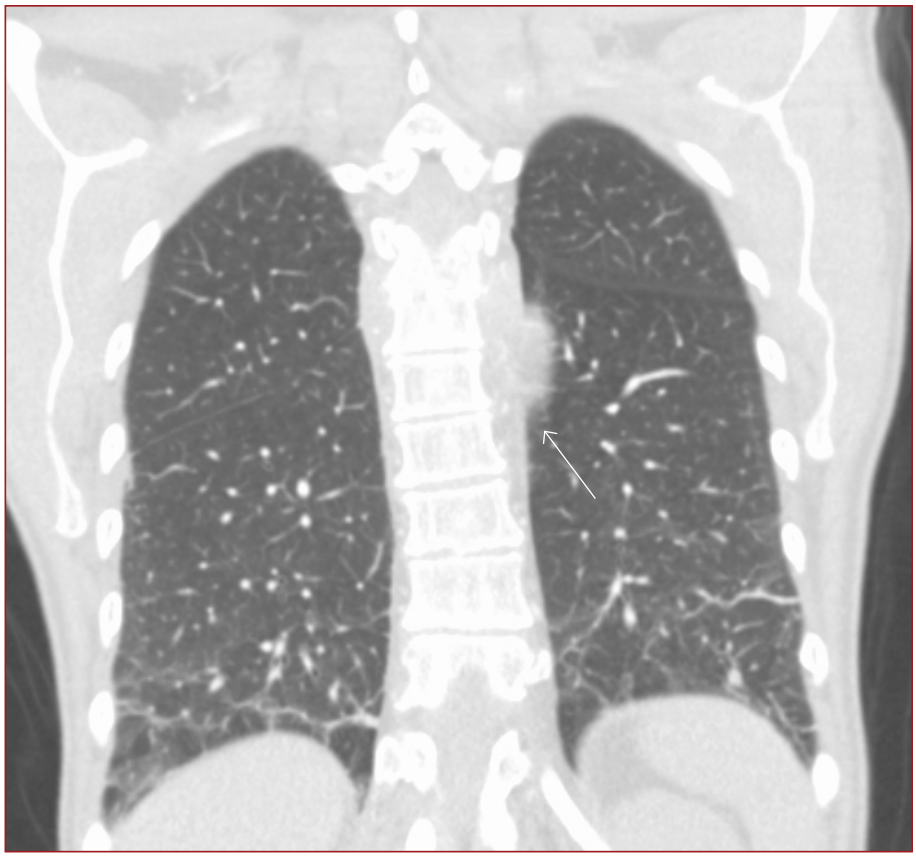
A Case of Discitis

Maika Dang MD
University of Cincinnati R1
case originally seen by Maika Dang R1 and Phil Moschella R4

History of Present Illness

59 year-old female with a history of hypertension, thyroid nodule, and reported history of intravenous drug use presented to the emergency department with a complaint of chest and back pain for the past month. She was recently diagnosed and treated for community acquired pneumonia two weeks prior with azithromycin. The chest and back pain had an insidious onset, described as a deep, sharp pain that comes in waves. It was exacerbated by movement of her trunk and upper extremities, 10 out of 10 in severity that started in her mid-back and radiated throughout her chest and bilateral flanks. The pain improved with antibiotic treatment, although it had progressively worsened over the the week prior to presentation.

The patient reported chills and subjective fevers, non-productive cough, shortness of breath, dysuria, and increased urinary frequency during the previous month. She had several episodes of non-bloody non-bilious emesis over the previous two days. She reported a history of intravenous heroin use, last injecting in bilateral upper extremities 7 days prior. Of note, she denied any trauma, history of back surgery, and symptoms of urinary retention, bowel incontinence, saddle anesthesia, or weakness or numbness in her lower extremities.



Coronal chest CT shows acute spondylodiscitis with paraspinal phlegmon at the T5-T6 level. No evidence of pulmonary embolism

Past Medical History

Hypertension, thyroid nodule

Past Surgical History

Partial thyroidectomy at age 15

Medications

None

Allergies

No known

Social History

Unemployed. Recently incarcerated 3 months ago. Current smoker, 20 pack/year tobacco history. Former alcoholic, stopped 10 years ago. Intravenous heroin drug use, active user. Monogamous relationship for 8 years, uses protection.

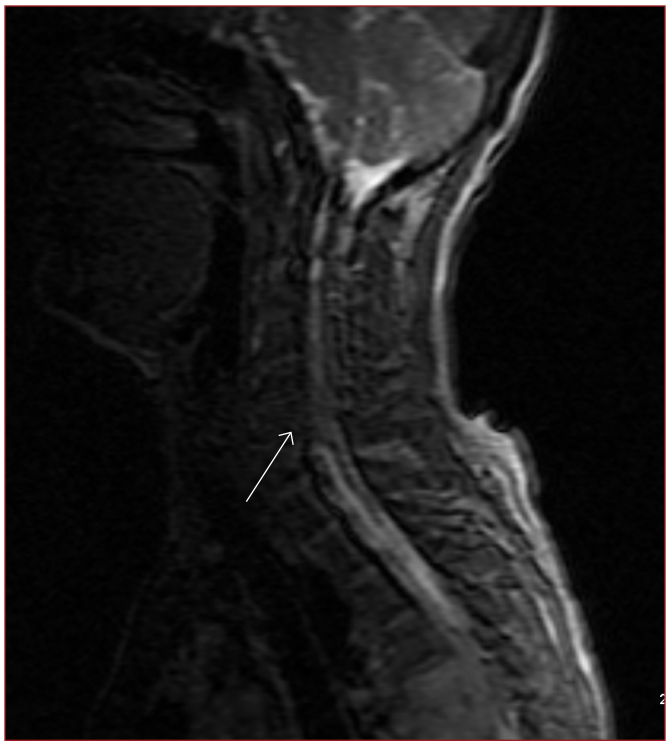
Temp
97.5
Heart Rate
65
Resp Rate
27
BP
193/95
O2 Sat
100%

Physical Exam

General: Well developed, well-nourished unkempt white female in NAD, able to converse in full sentences
HENT: Normocephalic, atraumatic; moist mucous membranes; poor dentition
Eyes: PERRL; sclera anicteric without conjunctival pallor or hemorrhage
Neck: Supple, no meningismus; trachea midline
Pulmonary: CTA bilaterally with good air movement. No rhonchi, wheezes, or rales; no respiratory distress
Cardiac: RRR with no murmurs, rubs, or gallops
Abdomen: Soft, non-tender, non-distended, without rebound or guarding. Active bowel sounds and no costovertebral angle tenderness
Musculoskeletal: No obvious deformities. Diffuse tenderness to palpation over cervical and thoracic spine and paraspinal muscles no palpable step offs, swelling, or overlying erythema
Vascular: Warm, well perfused. 2+ peripheral pulses
Skin: No rashes or lesions. No cyanosis; no splinter hemorrhages
Neuro: A&O x 4, Strength 5/5 flexion and extension bilateral hips, knees, ankles. Sensation intact to light touch bilateral lower extremities without saddle anesthesia. Reflexes 2+ bilateral patellar and Achilles; gait narrowing and steady. Negative Romberg

ED & Hospital Course

A CTPA of the chest was done to evaluate for pulmonary embolus and subsequently found a lesion concerning for discitis. When further evaluation with MRI confirmed the finding, neurosurgery was consulted in emergency department and did not suspect spinal cord involvement. The patient was placed on spinal precautions and a Minerva brace when out of bed. Empirical antibiotics were withheld initially. Eventually it was determined that the risks of obtaining a tissue culture outweighed the benefit of definitive speciation, therefore patient was started on Vancomycin and ciprofloxacin for coverage of Pseudomonas as recommended by infectious disease. Transthoracic echocardiogram demonstrated no vegetations concerning for endocarditis. Blood and urine cultures were negative for any growth. Although the patient developed decreased sensation to pin prick below the level of T5/T6 during hospitalization, there were no surgical interventions performed because repeat MRI demonstrated no change in canal stenosis. The patient was discharged to a skilled nursing facility with a PICC line in place to complete 6 weeks of antibiotic therapy.



MRI of T-spine: Discitis/osteomyelitis at T5-6 level with diffuse edema, paravertebral soft tissue and epidural enhancement and phlegmon. No definite epidural abscess is identified. There is mild to moderate cord compression with suggestion of focal cord edema at T5/T6 level.

Discussion

Discitis is an infection involving the intervertebral disc space most commonly found in the lumbar spine followed by cervical then thoracic spine. It has a bimodal distribution, peaking in early childhood (generally < 5 years of age) and around the sixth decade of life with an incidence ranging from 0.4 to 2.4 per 100,000 each year.[1,2] The overall mortality rate is between 2% to 11% and approximately one third of patients have residual disability.[1,3]

The symptoms suggestive of discitis are nonspecific (Table 1), and thus the diagnosis is often delayed an average of 2-6 months after onset of symptoms. Due to this non-specific presentation, approximately 70% of patients have more than 1 ED visit before the correct diagnosis is made.[3,] Fever is an inconsistent finding, seen in only 50% of patients.[4] The most common complaint is back pain, often with an insidious onset, that is worse at night and with focal tenderness. Other nonspecific symptoms include anorexia, lethargy, weight loss, and vomiting. Focal neurological deficits such as limb weakness, dysthesias, radicular pain, gait disturbance, bowel incontinence or urinary retention are concerning but uncommon. If present, an epidural abscess or mass should be considered. In children, it occurs it almost exclusively in the lumbar region, typically presenting with a progressive limp, back pain, or refusal to walk.[2] The differential includes musculoskeletal, degenerative or metastatic spinal disease, disc herniation, vertebral compression fracture, osteomyelitis, spinal tumors, and inflammatory spondyloarthropathies.[1,2,4]

Many comorbidities can increase the risk of discitis; these include diabetes (11% to 31%), age, injection drug use, indwelling vascular device, malignancy, alcoholism, cirrhosis, chronic kidney disease, immunosuppression, HIV/AIDS, and history of spinal trauma or fracture.[1,3] In addition, invasive spinal procedures account for 20-30% of cases of discitis.[1,3]

Findings in Discitis

FEVER	ANOREXIA
BACK PAIN	LETHARGY
WORSE AT NIGHT	WEIGHT LOSS
FOCALLY TENDER	VOMITING

Table 1 - Neurologic findings are rare and should prompt consideration of mass / compressive lesions

Pathogens can infect the spine via hematogenous spread, direct inoculation including trauma or spinal surgery, and contiguous spread from adjacent infected tissues. Common sources include the genitourinary tract, endocarditis, skin/soft tissues (IVDU, infected catheters, post-operative infections), respiratory tract, and dental infections. The vertebral endplate is typically the primary infection site with secondary contiguous extension into the disc and often the vertebral body as well, called spondylodiscitis. The vertebral endplate is a thin layer of hyaline cartilage positioned above and below the disc's nucleus and most of the annulus. It functions as a semipermeable membrane allowing nutrients and metabolites to diffuse into the disc from capillary blood. In children, vascular channels exist in this cartilaginous region of the disc space. It is postulated these vascular channels in kids and the metaphyseal arteries supplying the vertebral endplate in adults can become infected from septic emboli causing both bone infarction and secondary infection of the disc. This is because the nucleus and most of the annulus of the disc is dependent on diffusion of nutrients and metabolites across the endplate.[1,2,4] Contiguous spread of infection can then result in infection of the paravertebral tissues, vertebral bodies, and epidural space.

Staphylococcus aureus is the most common causative organism, followed by gram-negative bacilli and streptococci /enterococci.[1,,2,3,4] Of note, gram negative bacilli (E. coli, Proetus and Pseudomonas) are often associated with immunosuppression/immunocompromised, diabetes, IVDU, and infections of gastrointestinal and genitourinary tracts.[1] Blood cultures are only

Continued on page 14

#interntakeover

B Pod Case

Sickle Cell Disease

and getting to know its complications

Julie Teuber, MD
University of Cincinnati R1

case originally seen by Julie Teuber, RI and Dina Gozman R4

History of Present Illness

The patient is a 41 year-old female who presents to the emergency department with a four-day history of shortness of breath. She was seen at an urgent care facility, where she recieved breathing treatments and had wheezing on exam at that time. She reported continuation of her symptoms leading her to come to emergency department tonight. She also reports a 24-hour history of increased pain consistent with her sickle cell pain crises. She currently takes methadone and Dilaudid and reports these medications are not covering her pain. Patient reports pain is all over her body which is consistent with her prior episodes of pain. She denies chest pain and says this shortness of breath is consistent with prior episodes of asthma exacerbation.

Past Medical History

Sickle cell disease, asthma, aspergillus sensitivity syndrome, recurrent sinusitis, port infections

Past Surgical History

Cholecystectomy, tonsillectomy

Medications

Albuterol, Symbicort, Vitamin D3, Klonopin, Exjade, Benadryl, Colace, Flonase, Lasix, Dilaudid, Hydroxyurea, Ibuprofen, Levocetirizine, Methadone, Singular, Zofran

Allergies

Vancomycin, Penicillins, Linezolid, Cephalosporins, Latex

Social History

Unremarkable

Temp
99.1
Heart Rate
108
Resp Rate
27
BP
115/51
O2 Sat
84% RA

Physical Exam

General: Patient is thin, surgical mask in place and appears as if she doesn't feel well
HEENT: Head atraumatic, pupils equal round and reactive to light, extraocular movements intact, significant scleral icterus, mucus membranes moist, oropharynx nonerythematous
Neck: Supple, no lymphadenopathy, trachea midline
Pulmonary: Diffuse rhonchi, crackles and mild wheezing bilaterally
Cardiac: Regular rate and rhythm, normal S1S2, no murmurs, rubs, or gallops
Abdomen: Soft, nontender, nondistended, no rebound, no guarding
Musculoskeletal: No obvious deformities, no tenderness to palpation, 2+ radial pulses bilaterally, 2+ dorsalis pedis pulses bilaterally
Neuro: Alert and oriented x4, speech is clear and intact without dysarthria, gait intact

Labs:
Lactate 3.6
WBC 41.5
Hg 5

ED & Hospital Course

The patient's oxygen saturation was noted to be 84% upon arrival to the emergency department and she was placed on 4L oxygen via nasal cannula and subsequently saturated in the high 90s. Labs were drawn to evaluate the state of her sickle cell disease which included a CBC, BMP, reticulocyte count, LDH. Given her hypoxia and shortness of breath, a chest x-ray was ordered (Figure 1). The team was concerned that clinically the patient appears to have acute chest syndrome. She was started on antibiotics to cover for hospital acquired pneumonia given she was recently admitted for a sickle cell pain crisis. She was also given 2 L of normal saline and packed red blood cells were ordered to correct her hemoglobin of 5. The patient was admitted to medical step down unit for treatment of her acute chest syndrome.

Discussion

Sickle cell disease (SCD) is an autosomal recessive disorder of hemoglobin S that results from the substitution of valine for glutamic acid as the sixth amino acid of the beta globin chain which produces a poorly soluble hemoglobin tetramer in low oxygen states. These poorly soluble tetramers align into fibers that result in distortion of the red blood cell creating the classic sickle shape.[1]

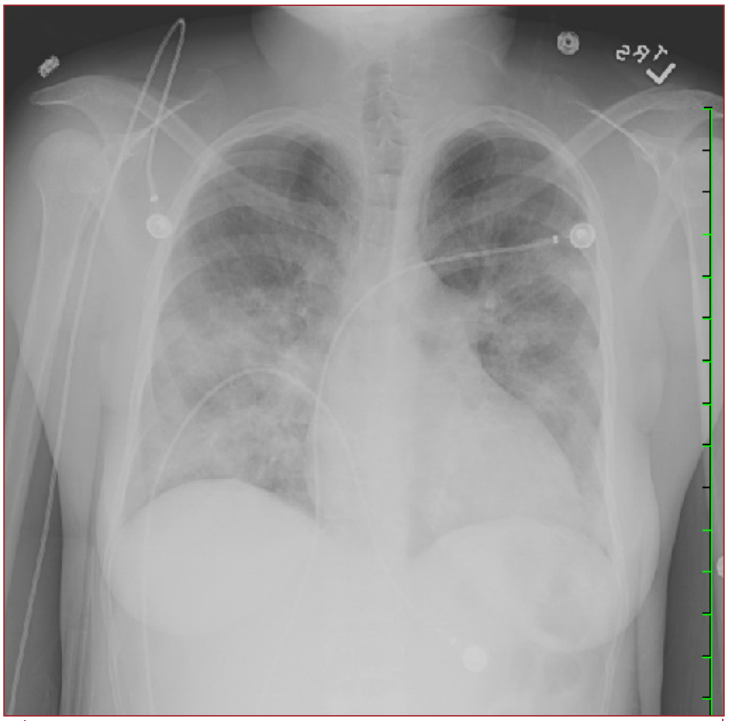


Figure 1: CXR demonstrating multifocal opacities concerning for pneumonia

Complications of Sickle Cell Disease by System

Acute Painful Episodes

Known as a sickle cell crisis, this is the most common type of vasoocclusive event and the most common complication for which patients seek medical attention. These episodes peak between ages 19-39. The frequency of crises is indirectly related to mortality. 40% of patients report no painful episodes, however 1% report 3-10 crises per year. These pain crises may be precipitated by dehydration, infection, stress, menses, or alcohol consumption--however, the majority of these episodes have no identifiable cause.[1]

Neurologic Complications

Unfortunately, 24% of patients will have a clinically significant stroke by age 45.[1] There is an increased rate of stroke with hydroxyurea use.[4] There is also a 2-3x increase in epilepsy above the general population.[1] Other neurologic complications include neurocognitive decline, spinal cord compression, and intracerebral hemorrhage.

Cardiac Complications

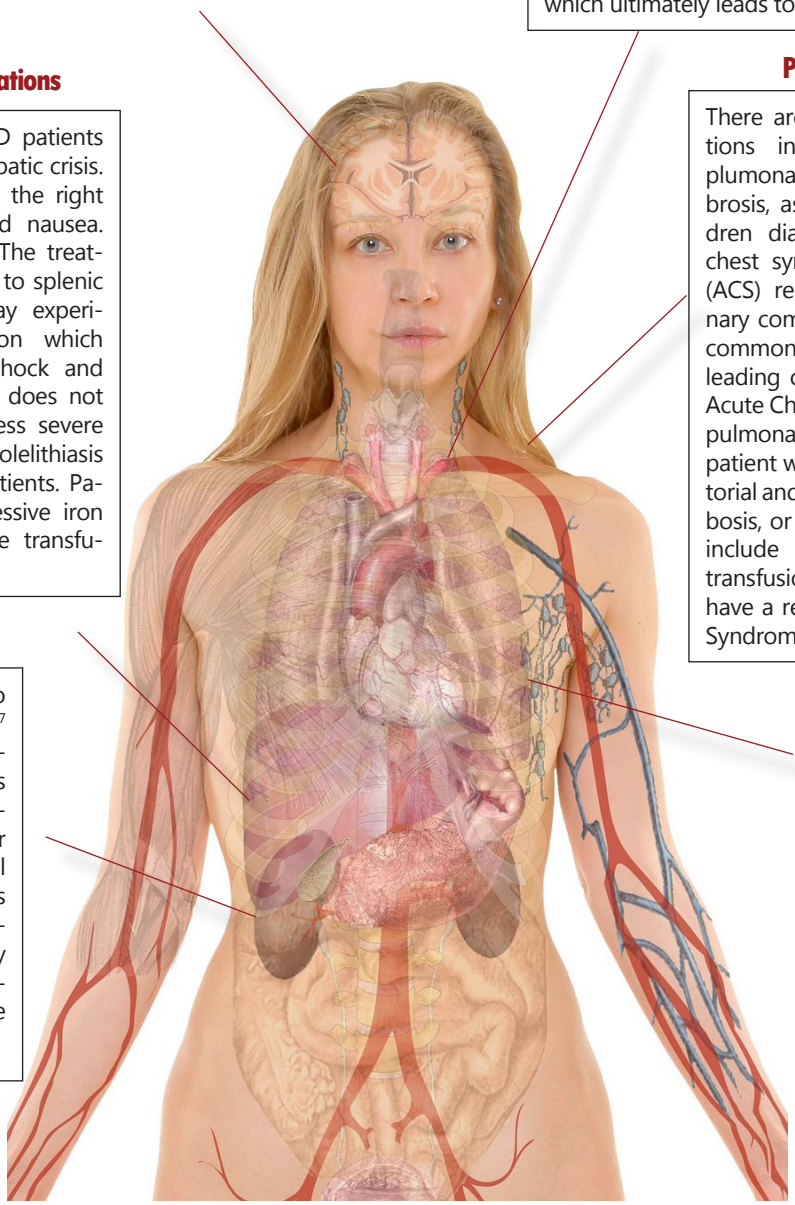
Acute myocardial infarction occurs in approximately 10% patients due to an increased oxygen demand exceeding their limited oxygen-carrying capacity. Many patients also have an increased cardiac output secondary to chronic hypoxia which ultimately leads to cardiomegaly. [1]

Hepatobiliary Complications

Approximately 10% of SCD patients will experience an acute hepatic crisis. Symptoms include pain in the right upper quadrant, fever and nausea. Transaminitis is common. The treatment is supportive. Similar to splenic sequestration, patients may experience hepatic sequestration which leads to acute anemia, shock and ultimately death if patient does not receive blood products. Less severe complicaitons include cholelithiasis found in nearly 70% of patients. Patients may also have excessive iron stores if receiving multiple transfusions for their anemia. [6]

Pulmonary Complications

There are multiple pulmonary complications including pulmonary embolism, plumonary hypertension, pulmonary fibrosis, asthma, which occurs in 2/3 children diagnosed with SCD,[5] and acute chest syndrome. Acute chest syndrome (ACS) represents 30-50% of all pulmonary complications. It is the second most common cause of hospitalization and the leading cause of death for SCD patients. Acute Chest Syndrome is defined as a new pulmonary infiltrate on chest x-ray in a patient with SCD. The etiology is multifactorial and may occur with infection, thrombosis, or pulmonary fat emboli. Therapies include oxygen, antibiotics, and blood transfusion. Patients taking hydroxyurea have a reduced incidence of Acute Chest Syndrome.[1]



Renal Complications

Renal failure is seen in up to 18% of patients with SCD.[7] Other common renal complications include painless hematuria, proteinuria, renal infarction, renal tubular acidosis, focal segmental glomeruloscleros that leads to ESRD, and renal medulary carcinoma that is nearly seen exclusively in black patients with HbSC or sickle cell trait. [8]

Splenic Complications

These complications are secondary to repeated episodes of splenic infarction during sickling of red cells within the spleen. Splenic infarction usually occurs between age 2-4yo. Asplenism makes patients susceptible to infection from encapsulated organisms (including S. pneumoniae, H. influenzae type b).[1]

Continued on page 14

Pharm Consult

COPD Update

Treatment Strategies and Controversy Surrounding Antibiotic Therapy

Desiree Kosmisky, PharmD
University of Cincinnati

Jessie Winter, PharmD, BCPS
University of Cincinnati

Chronic Obstructive Pulmonary Disease (COPD) is a chronic and preventable disease characterized by ongoing airflow limitation caused by a mixture of obstructive bronchiolitis of the small airways and parenchymal destruction (emphysema).[1] As pulmonary disease is the fourth leading cause of death in the world, the burden is great with COPD exacerbations. They account for more than 600,000 hospitalizations annually, with in-hospital mortality associated with hypercapnic exacerbation with acidosis in approximately 10-25%.[1-3]

In the emergency department, treatment of COPD exacerbation ideally follows Global Initiative for Chronic Lung Disease (GOLD) Guidelines, last updated in 2014. Standard therapy for hospitalized patients includes symptom assessment (blood gases, chest radiograph); supplemental oxygen and consideration of mechanical ventilation; identification and treatment of associated conditions (heart failure, arrhythmias); monitoring of fluid balance and nutrition; and consideration of thromboembolic prophylaxis. Drug therapy consists of short-acting bronchodilators and oral or intravenous steroids. Antibiotic therapy is currently recommended in patients with high risk of infection.

Short-Acting Bronchodilators

Short-acting beta$_2$-agonsists, which relax bronchial smooth muscles by stimulating the beta$_2$ receptors include albuterol sulfate and levalbuterol. Anticholinergic agents such as ipratropium bromide block the effect of acetylcholine at parasympathetic sites in the bronchial smooth muscles, leading to vasodilation. No significant FEV$_1$ difference has been shown in modality of delivery, although nebulizers may be more easily used in patients who are more ill. Therefore, doses and/or frequencies of short-acting bronchodilators should be increased (Level of Evidence B) and delivered using nebulizers or spacers (Level of Evidence C).

Oral or Intravenous Steroids

Glucocorticoid therapy has been shown to have a beneficial effect on clinical outcome, hospital length of stay, and FEV$_1$ recovery.[1] Prednisone 40 mg daily for 5 days, which is equivalent to 32 mg of intravenous methylprednisolone, is the most commonly recommended strategy for treatment of exacerbations (Level of Evidence B). Although the GOLD guidelines also state that there are insufficient data to provide firm conclusions regarding optimal duration.[1] The newest recommendations are based largely on the Reduction in the Use of Corticosteroids in Exacerbated COPD (REDUCE) trial. This randomized, multicenter, non-inferiority trial included 311 patients presenting to the emergency department with acute exacerbation. Patients were randomized to a five or 14 day course of methylprednisolone 40 mg IV on day one followed by prednisone 40 mg daily. A 5 day course of therapy was found to be noninferior to a 14-day course of treatment for the primary outcome of COPD exacerbation in six months (35.9% vs. 36.8%, HR 0.95 90% CI 0.70-1.29, p=0.006). There were no significant differences in time to re-exacerbation, time to death, the composite endpoint of exacerbation, death, or both, and recovery of lung function.[5]

Antibiotic Therapy

Between 50 and 70% of COPD exacerbations have an infectious etiology, including bacterial and viral causes. Bronchoscopic studies have indicated that at least 50% of patients have bacteria present in the lower airways during exacerbations, but these bacteria may be difficult to discern from colonization.[1,6] The guidelines suggest that antibiotic therapy should be based on local resistance patterns and recommend initial empiric treatment with an aminopenicillin with or without clavulanic acid, a macrolide (most commonly azithromycin), or a tetracycline.[1] A study assessing the pathogens isolated in 188 sputum samples during acute exacerbation of 111 patients with severe or very severe COPD over a one year period showed the most commonly isolated pathogen to be P. aeruginosa (n = 54), followed by H. influenzae (n = 37), S. pneumoniae (n = 31), M. catarrhalis (n = 29) and Staphylococcus aureus (n = 12). H. influenzae was present most commonly with a single exacerbation (33% vs. 16%, p = 0.006), whereas Enterobacteriaceae were present most commonly with repeated exacerbations (33 vs. 16%, p = 0.006).[7] Thus, in patients with more severe disease at baseline, particularly with repeated exacerbations, broader-spectrum antibiotics with antipseudomonal

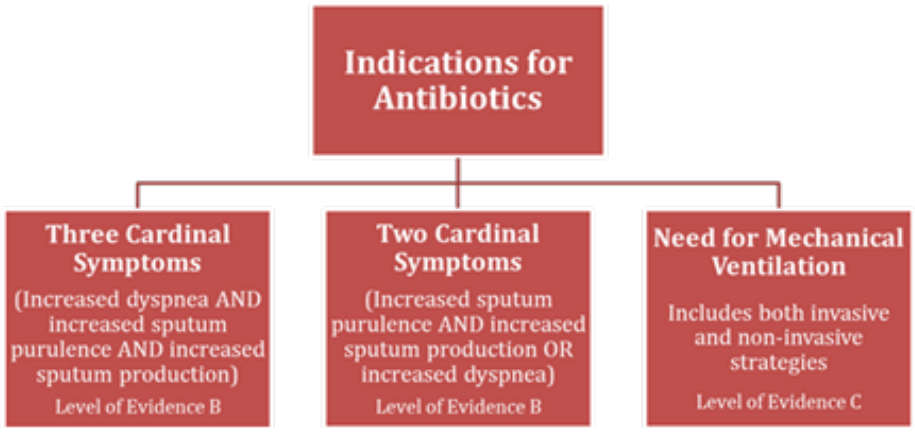


Figure 1. Indications for Antibiotic Therapy

coverage may be warranted.

The use of antibiotics in COPD is controversial. Current GOLD guideline recommendations are listed in Figure 1. Recommended length of therapy is usually five to ten days (Level of Evidence D).[1]

One of the first trials to show a benefit associated with antibiotic therapy was conducted in 173 outpatients who underwent 362 exacerbations over a 5 year period. Patients were randomized to placebo or broad-spectrum antibiotic therapy for 7 to 10 days based on presence of cardinal symptoms of exacerbation. A treatment success was defined as a resolution of all symptoms exacerbation within 21 days. In the placebo arm, 55% of patients had treatment success in comparison to 68.1% in the antibiotic arm ($p < 0.001$). The authors concluded that patients presenting with all three cardinal symptoms or two cardinal symptoms (if one symptom was increased sputum purulence) would likely benefit from antibiotic therapy.[8]

A prospective, randomized, placebo-controlled trial was conducted by Nouria and colleagues in 93 patients with acute COPD exacerbations requiring mechanical ventilation for at least six hours, with the majority of patients ultimately requiring intubation. Patients were treated with oral ofloxacin 400 mg once daily for 10 days or placebo. Cardinal signs on presentation were not recorded, although pneumonia was ruled out on chest radiograph. In-hospital mortality was decreased in the ofloxacin arm (4% vs. 22%; absolute risk reduction 15.7%; p=0.01). Those receiving ofloxacin were less likely to develop nosocomial pneumonia, which the authors determined to be the largest contributor to reduced mortality. Duration of mechanical ventilation (6.4 vs 10.6 days; $p = 0.04$) and both ICU (9.4 vs. 14.5 days; $p = 0.02$) and hospital length of stay (LOS) were shorter in the ofloxacin arm.[4]

A multicenter, retrospective cohort of 84,621 patients aged 40 years or older hospitalized for acute COPD exacerbation compared the composite outcome of treatment failure (defined as initiation of mechanical ventilation after the second hospital day), inpatient mortality, readmissions for acute exacerbations of COPD within 30 days of discharge, LOS, and hospital costs. At least 2 consecutive days of antibiotic therapy were administered to 79% of patients. Antibiotic use was associated with an odds ratio of 0.87 (95% CI 0.82-0.92) for treatment failures and statistically significant reductions were observed in all of the endpoints which comprised the primary outcome except for LOS. Those who received antibiotic therapy had a statistically significantly higher risk of allergic reactions, Clostridium difficile diarrhea, and readmissions within 30 days for diarrhea.[2]

In contrast, Daniels and colleagues conducted a randomized controlled trial enrolling 223 hospitalized patients with 265 COPD exacerbations. Patients were randomized to doxycycline 100 mg twice daily for 7 days or placebo in addition to an extended course of systemic glucocorticoids to assess the effect of antibiotic therapy independent of glucocorticoid use. On day 10, clinical success was significantly higher in the intention-to-treat doxycycline group in comparison to placebo (80% vs. 69%; OR 1.9; 95% CI 1.1-3.3; $p = 0.03$), but this difference was not observed at 30 days (61% vs. 53%; OR 1.3; 95% CI 0.8-2.0; $p = 0.32$). The authors concluded that the effect of antibiotic therapy may be transient when combined with glucocorticoids.9

A 2012 Cochrane review of antibiotics for COPD exacerbations included 16 trials and 2068 patients.[10] In outpatient exacerbations, there was a statistically significant reduction in reduction of treatment failures (RR 0.75; 95% CI 0.60-0.94; I2= 35%), but the evidence was low quality and this difference was not observed when studies were restricted to currently available drugs (RR 0.80; 95% CI 0.63-1.01; I2= 33%). High quality evidence showed a statistically significant reduc-

Continued on page 11

Primary Author	Study Design	Antibiotics	Patient Population	Outcome	With vs Without Abx	p
Anthonisen (1987)	Prospective, randomized, placebo-controlled trial	7 to 10 days of broad-spectrum antibiotics or placebo	173 outpatients with 362 exacerbations over 5 years	Treatment Success (resolution of all defining symptoms in 21 days)	68.1 % vs. 55%	< 0.001
Nouria (2001)	Prospective, randomized, placebo-controlled trial	Oral ofloxacin 400 mg once daily for 10 days or placebo	93 ICU patients with acute exacerbations requiring mechanical ventilation for at least 6 hours	In-hospital mortality	4% vs. 22%; absolute risk reduction 15.7%	0.01
Rothberg (2010)	Multicenter, retrospective cohort	At least 2 consecutive days of antibiotic therapy were administered to 79% of patients	84,621 patients > 40 years old hospitalized for acute COPD exacerbation	Treatment failure*	9.8% vs. 11.8% odds ratio of 0.87 (95% CI 0.82-0.92)	< 0.001
Daniels (2010)	Prospective, randomized, placebo-controlled trial	Doxycycline 100 mg twice daily for 7 days or placebo	223 hospitalized patients with 265 COPD exacerbations	Clinical success on Day 10	80% vs. 69% OR 1.9 (95% CI 1.1-3.3)	0.03
				Clinical Success on Day 30	61% vs. 53%; OR 1.3 (95% CI 0.8-2.0)	0.32

*composite of initiation of mechanical ventilation after second hospital day, inpatient mortality, readmissions for exacerbations within 30 days of discharge, length of stay, hospital costs

GME Funding

Medicine's Looming Conundrum

Dan Axelson MD
University of Cincinnati R2

It is no secret that the nation faces an ever-growing shortage of doctors. As it stands today we have 62,000 too few physicians treating America's ill, and the AAMC estimates that by 2025 the shortage will grow to over 130,000 physicians.[1] Paradoxically, health care expansion may serve to make the problem worse. The impact that physician shortages will have on quality and timeliness of care is not hard to imagine but the solution to the problem is less clear.

So how did we get here? Population growth, the aging baby boomer generation, and expansion of health coverage have all caused increased demand for physician services. The nation's medical schools have responded with a 30% increase in enrollment since 2002, in part by opening 18 new medical schools over the past decade as well as increasing seats in existing ones.[2] But this doesn't address the bottleneck. Residency training is funded through Medicare, the nation's healthcare fund for the elderly, which provides roughly $9.5 billion a year in residency funding to teaching hospitals.[3] This level of financial support has been capped in Congress since 1997. Thus far, all that the increase in medical school enrollment has served to accomplish is to decrease the proportion of foreign medical graduates in US residency positions as more and more US graduates are available to fill the stagnant number of residency spots. The consequences and merits of this phenomenon can be debated, but from the perspective of an American physician shortage this is not a bad thing. American medical school graduates are presumably more likely to stay in the US to practice, so our collective rate of residency attrition to other countries would decrease as more residents are US medical school graduates. But this doesn't entirely make up for the shortage. In short, more residency positions are needed.

The source of funding for more residency spots remains elusive. In 2013 there were proposals out of both the House and Senate to increase the number of residency positions supported by Medicaid, but the fate of such legislation remains clouded. A recent Institute of Medicine report[4] has called in to question whether the government should be involved in supporting GME. Opponents argue that the federal government does not fund undergraduate medical education or other health professions in any similar way. Furthermore the government does not fund other important societal professions like computer scientists, engineers, or mathematicians. What makes physicians different, asks the Institute Of Medicine? It is an interesting historical point to note the way in which Medicare funding for residency began. At the onset of diagnosis related groups (DRGs, the blocks of money payed to hospitals via Medicare for treatment bundles based on diagnosis) teaching hospitals grew concerned that these sums were not adequate to reimburse the particularly complex and severely ill patient's they felt they were seeing. After much back-and-forth regarding this reimbursement, Congress arbitrarily doubled what teaching hospitals saw in reimbursement per DRG and set that higher amount as Indirect Medical Education funding, i.e. funding to be contributed to residency spots.[5] In an era of increasing budgetary constraints, it will be ever harder to justify an increase in these funds.

> "As it stands today we have 62,000 too few physicians treating America's ill and the AAMC esitmates that by 2025 the shortage will grow to over 130,000 physicians."

States are innovating to help fill the gap. Some states are directly increasing their budgetary contributions to GME funding. Others are setting up fast-tracked primary care or other badly-needed clinical pathways for trainees.[5] Others push hospitals directly to help shoulder the burden of funding more residents. Thus far there has been no silver bullet. But the physician shortage grows and policy thought-leaders as well as physicians themselves will need to address this topic urgently, drawing upon the best innovations of various states.

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Epic TIPS

We sat down with the architect himself Dr. Frank Fernandez - here are the tips to make your charting that much easier and more effective...

Insert smartblocks (the click through exams) into your note templates, examples
.edphysicalexambyage

Use .procdoc for easier procedure documentation that also generates the right billing

Right click on menus to accept them (ex. the CXR indication menu) instead of clicking the accept button

Use .edus for ultrasound documentation

Use t+90 in the order time to order a test 90 minutes from now

Make your community referrals easy, place these in your discharge instructions
.eduhdental
.eduhcommunity

Discharge better - use this as a discharge instruction template
.edhealthliteracy

Patient needs a place to stay? Give them a list of shelters
.edhomelessmen
.edhomelesswomen

Your patient needs to get home?
.edtransportation

Got MRSA? Use this for bleach bath instructions
.bleach

Pharm Consult
Continued from page 9

tion of treatment failures in hospitalized patients (non-ICU) with severe exacerbations (RR 0.77; 95% CI 0.65-0.91; I2= 47%), regardless of restriction to currently available medications. Evidence from trials conducted in inpatients (non-ICU) showed no effects of antibiotics on mortality (n = 4), length of stay (n = 4), or re-exacerbations (n = 1). The only evidence available for ICU patients was the previously mentioned Nouria and colleagues trial, which the authors concluded was strong evidence for the use of antibiotics in this patient population.[4]

In conclusion, antibiotics should be used judiciously in patients with COPD exacerbations. There is evidence that these agents have a beneficial role in exacerbations, particularly in patients with evidence of infection or with severe disease requiring mechanical ventilation. The paucity of large, randomized, controlled trials and the publication of the majority of data prior to the establishment of standards of care with the GOLD guidelines in 2001 represent significant limitations to current recommendations. As with all antibiotic therapy, it is vital to balance selection of appropriate coverage and patient outcomes with the development of resistance and likelihood of increased adverse effects.

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List of Submitted B Pod Cases

	R4	R1
Septic Joint	Bohanske	
STEMI refused cath lab (ethics?)	Bohanske	Shah
Dilantin Tox	Gozman	Thompson
HA with aneurysm, bloody tap	Cousar	Titone
Massive hemetemasis with varices	Redmond	Polsinelle
Flexor tenosynovitis	Redmond	Polsinelle
Transverse Myelitis	Bohanski	Donnelly (IM)
Isopropol in E-cigs	Moschella	Shah
Pain with defecation, bone in rectum	Redmond	
TTP vs HUS	Verzwyvelt	Miller (ortho)
Acute Chest Syndrome	Gozman	Teuber
Pancytopenia	Redmond	Plash
Bacterial Meningioencephalitis	Bohanske	
SVC syndrome, new neoplasm	Bohanske	Riney
Lyme disease	Cyrus	Maika
IVDU with discitis	Bohanske	Dang
Fungal PNA	Cousar	Renne

Annals of B Pod is looking for YOU to submit your interesting cases of B Pod - There is a composition book at the R4 desk - annalseditors@gmail.com

Send to cath lab... Or not?

Aalap Shah, MD
University of Cincinnati R1

case originally seen by Aalap Shah R1 and Mike Bohanske R4

History of Present Illness

The patient is a 52 year-old female with history of diabetes, hyperlipidemia, hypertension, CAD status post multiple stent placement currently on aspirin/clopidigrel presenting for chest pain and shortness of breath of several hour duration. She states that her chest pain and shortness of breath began acutely around 4 AM and woke her up from sleep. The pain feels like a squeezing pain in the center of her chest, which radiates towards her back between her shoulder blades. This pain is associated with shortness of breath. She has had similar symptoms of pain in the past with exertion, however it had previously remitted with rest. She states her pain today has been constant since waking up, and she's been unable to find any relief. Upon talking to the patient she states "this feels like my other heart attacks."

Past Medical History

Hypertension, diabetes, hyperlipidemia, coronary artery disease (s/p multiple stents to left anterior descending, left posterior descending, and obtuse marginal), and depression

Medications

Apirin, atorvastatin, citalopram, clopidogrel, fenantyl patch, furosemide, isosorbide mononitrate, lisinopril, lorazepam, metoprolol, moprhine, nitroglycerin, pantoprazole, potassium, ventolin

Social History

Currently ~1 pack per day smoker

Allergies

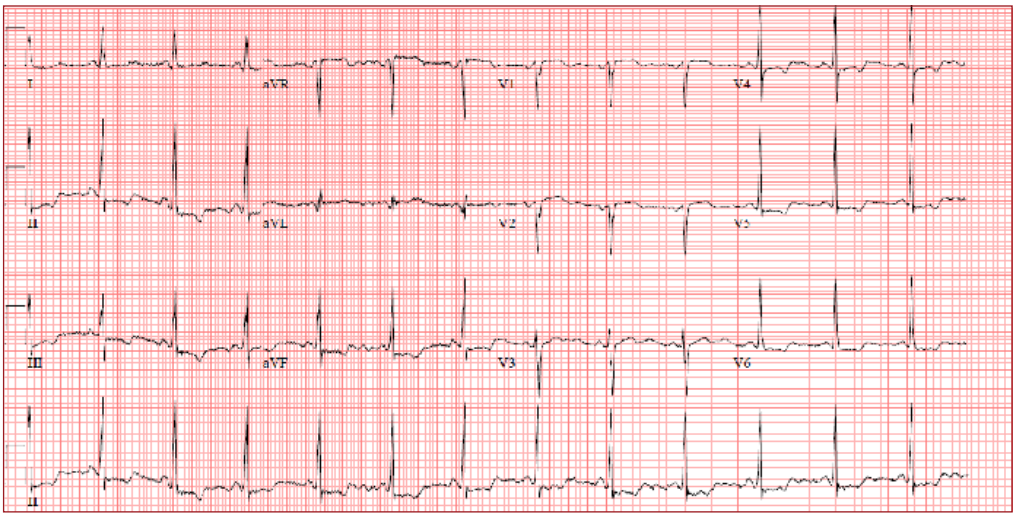
Amitriptyline, doxycycline, Ranexa, latex

Physical Exam

General: Middle aged female appearing stated age, obese, lying in bed, appears anxious
HEENT: Head atraumatic, pupils equal round and reactive to light, extraocular movements intact, sclera clear, mucus membranes moist, oropharynx nonerythematous
Neck: Supple, no lymphadenopathy
Pulmonary: Clear to auscultation bilaterally, no wheezes, rhonchi, or rales
Cardiac: Tachycardic with a regular rhythm, normal S1S2, no murmurs, rubs, or gallops
Abdomen: Soft, nontender, nondistended, no rebound and no guarding
Musculoskeletal: No obvious deformities, no tenderness to palpation
Vascular: 2+ radial/PT pulses bilaterally
Skin: Warm, dry, well perfused, no rashes
Neuro: AAOx4. CN 2-12 intact. Sensation intact. Strength grossly equal and symmetric

Vital Signs

Temp
98.2
Heart Rate
133
Resp Rate
21
BP
188/93
O2 Sat
98%



EKG 1: concerning for ST elevation MI with ST elevations of 1-1.5mm in avL, V2, avR, with diffuse ST depressions, most prominent in leads II, III, avF, V5, V6.

ED and Hospital Course

The patient recieved an EKG (Figure 1) on arrival and was immediately uptriaged to the SRU. Chest x-ray revealed no evidence of acute cardiopulmonary disease or mediastinal widening. Upon discussion with patient regarding potential care options, she stated that she did not want any intervention done for her symptoms. She specifically refused cardiac catherization because she had catheterizations in the past and did not want any more invasive procedures. Therefore, the cath lab was not activated. The cardiology fellow was consulted and evaluated the patient at bedside. She recieved labetolol for her hypertension and tachycardia, pain medication, as well as integrellin, and a heparin drip. After significant discussion regarding alternative modalities of treatment of her condition the patient agreed to treatment with tenecteplase and received 45mg in the ED. 20 minutes after administration, the patient had a repeat EKG (Figure 2) performed and the patient had symptomatic relief. She was admitted to the CVICU for further management. The patient's cardiac enzymes remained normal. On hospital day 1, the patient's condition was improved and she was discharged at her request. She was to follow up with her well established outpatient cardiologist.

Discussion

Unlike the typical emergency department patient, the patient in this case was not a diagnostic dilemma. She had known CAD with multiple past interventions, presented with chest pain and a STEMI. However, the situation was atypical in that the patient continued to refuse any intervention. As physicians we are often reluctant to accept patient's wishes when we feel their decisions are detrimental to their overall health. This poses an ethical dilemma, placing our desire and duty to treat the patient's illness in opposition to our duty to stay true to the patient's wishes.

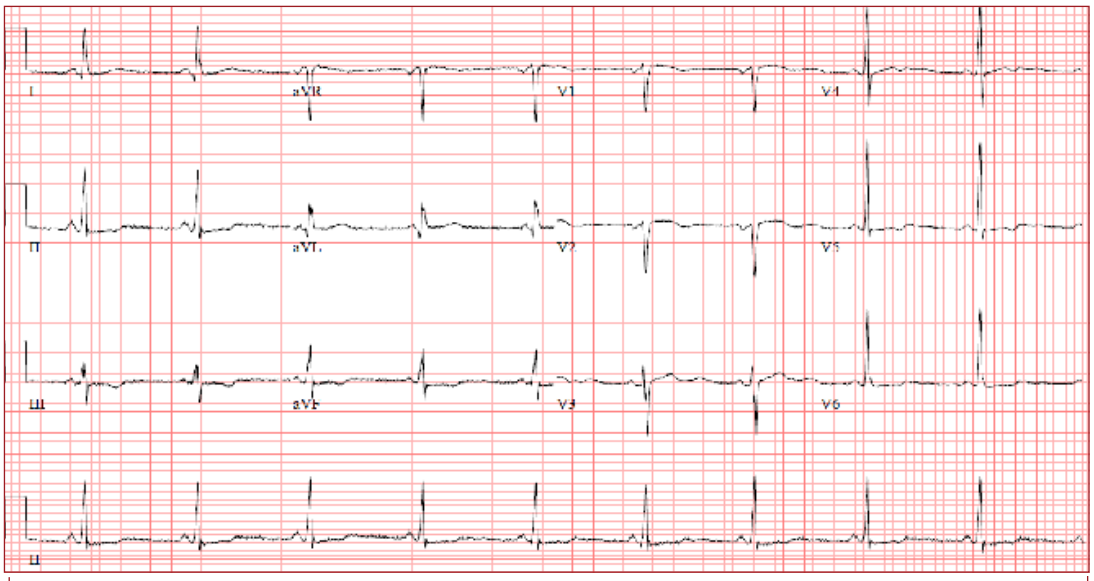


Figure 2: Post-intervention EKG shows resolution of STEMI with mild persistent ST depression

In the event of a patient refusing treatment, there are multiple points that need to be addressed. First and foremost is the patient's capacity to make decisions. Notably, this is a distinct entity from competency, which is a legal determination of mental state and can only be determined by a court. Capacity, on the other hand, is an individual's ability to make an informed decision, and can be determined by any licensed physician. Multiple approaches have been suggested for determining capacity, however in order to avoid provider bias, an 'algorithmic' approach should be taken. (Figure 3)

In this case, our patient had adamantly denied any invasive intervention from the start of her ED course, even prior to the EKG. She was found to be alert and oriented without clouding of sensorium, evidence of delirium, substance intoxication, or underlying psychiatric disturbance. She had discussions with multiple providers including the ED intern, senior resident, attending provider and cardiology team, all of whom spoke with her regarding risks and benefits of proposed treatment, alternative therapies or no treatment. She was able to adequately restate the major points of discussion, risks/benefits, and was able to give reasoning behind her decision. A complicating factor in this case was her overall anxiety, pain and history of depression. After her pain was addressed, however, she stated she had been preparing for a future heart attack and had decided long prior to this episode that she would not have intervention. She had made this clear to her friends and family. She further stated that she did not intend to die, and denied any history of suicidal ideation, however was willing to accept this risk as a consequence of avoiding invasive procedures.

Capacity Checklist

- ☐ Patient is free from conditions which impair consciousness, thought process, or memory
- ☐ Patient understands their current medical condition, preposed treatment, and potential alternatives
- ☐ Patient is able to determine personal importance of various risks and benefits
- ☐ Patient can rationally integrate all information to come to a decision
- ☐ Patient's choice is stable over time

Figure 3: Essential elements of a capacity evaluation.

Within the model for determining capacity is the assumption of adequate informed consent. The way we as physicians present the different risks and benefits as well as the possibility of alternative treatments has a large impact on the patient's decision making process. As physicians, we often have our own biases regarding optimal treatment. It is often seen as a failure on the part of the physician if we are unable to provide optimal treatment as a result of patient refusal.

Research shows there is often a large gap between the theory and the actual practice of informed consent. This reaserch showed patients often have poor understanding of not only the procedure in question but also acceptable alternatives. Similarly, the concept of patient autonomy, as well as the importance from the consent being free from significant coercion or undue influence is often viewed as flexible as doctors generally operate with a 'best interest' principle.[2] Similarly, as current literature favors PCI vs thrombolysis or medical management of acute MI, we rarely consider other treatment options for our patients with this presentation.[3] However, in this case, alternatives to treatment became an important consideration, as the patient was not amenable to the proposed initial treatment. Ultimately the patient did agree to alternative treatment with Tenecteplase in the ED, with significant subsequent improvement.

It is important to remember that the concepts of patient capacity and in-

Continued on page 15

Sickle Cell Disease
Continued from page 7

Infectious Complications

There are increased infection rates by encapsulated organisms in asplenic patients. Bacteremia by S. pneumoniae is the most common infection. Sepsis may lead to bone marrow suppression, DIC, and meningitis and it has an associated mortality rate of 20-50%.[9] The most common bacterias associated with SCD pneumonia are Mycoplasma pneumoniae, Chlamydia pneumoniae, and Legionella.[1]

Multi-Organ Failure

These most often occur during severe pain episodes and can be reversed by prompt exchange tranfusion therapy. This complication is potentially life threatening although the pathogensis is unknown.[1]

Hematologic Complications

Acute severe anemia may occur along with the chronic anemia requiring transfusions. An aplastic crisis is defined as a transient arrest of erythropoiesis leading to decrease in Hg, decreased reticulocytes in peripheral blood and is associated with human parvovirus B19. These patients require acute transfusion therapy. Another grave and rare complication includes the hyperhemolytic crisis. This is a sudden exacerbation of anemia with reticulocytosis from multiply transfused patients. Experts report this is consistent with delayed transfusion reaction where both the transfused cells and native cells are destroyed.[1]

Bone Complications

Osteomyelitis occurs from infection that occurs in infarcted bone. The most common bacteria associated with osteomyelitis in patients with SCD are Salmonella species. Bone infarction and avascular necrosis occur most commonly at the femoral head secondary to multiple sickling episodes. This painful complication occurs in approximately 40% of SCD patients. Bone marrow infarction may occur causing reticulocytopenia, leukoerythroblastic blood, pancytopenia and pulmonary fat embolism which require exchange transfusion, heparin and steroids to treat.[1]

Discitis
Continued from page 5

positive in 50% of patients.[1,3,4] Therefore, tissue biopsy is often necessary to identify causative organism. The diagnostic work up includes CBC with diff, ESR, CRP, and blood cultures. CRP and ESR elevation is seen in over 90% of discitis patients and leukocytosis is seen in less than 50% of patients.[1,3] In addition, work up should also include a BMP and urinalysis as there is a higher incidence of discitis in patients with diabetes. In addition, urinary tract infections are frequently missed sources of bacteremia and possible sources of hematogenous spread. A PPD and or chest xray should be considered in patients at risk for TB.

The imaging modality of choice in suspected discitis is MRI with a sensitivity of 96% and specificity of 94%.[3] Radiologic changes on plain films lag clinical symptoms and typically require 10-14 days to become apparent, therefore are often normal. CT scans are sensitive in the detection of endplate and bone destruction and contrast-enhanced scans can identify inflammatory changes as well. CT is not sensitive in the detection of bone marrow changes or epidural involvement, therefore MRI is the preferred image modality.

Treatment is typically non-operative and requires 4 to 8 weeks of parenteral antibiotics and immobilization. Surgery is indicated in cases of spinal cord compression, instability, abscess, or severe persistent pain. Administration of antibiotics should be withheld until biopsy to identify organism in adults. If the patient is hemodynamically unstable, has a neurologic deficit, or evidence of epidural abscess then empiric antibiotics should be administered (Table2).

The symptoms of discitis are very non-specific, therefore it should be included in the differential for those presenting with back pain. A high clinical suspicion is warranted in those with risk factors such as diabetes, those who are IVDU, and those who are immunocompromised because if not readily diagnosed, it carries a high morbidity and mortality.

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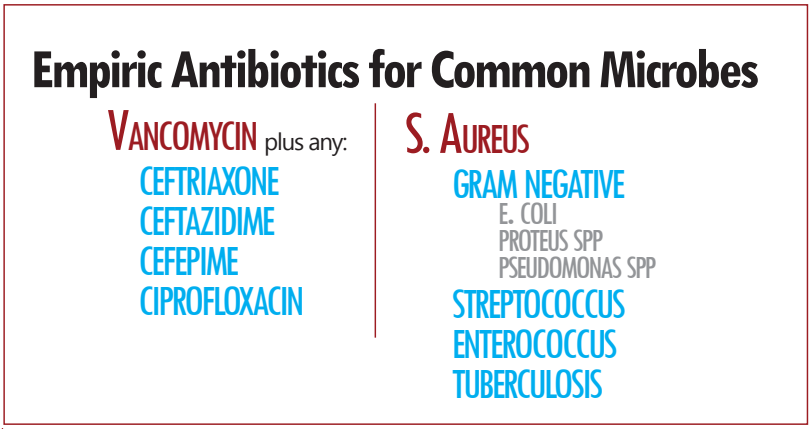

Table 2- Microbes and antimicrobials essential to discitis

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STEMI Refusal
Continued from page 13

formed consent are in place to protect the patient and to assure autonomy in decision-making. It is the duty of the physician to present an unbiased picture of potential options. It can be difficult to allow a patient to refuse a treatment that the provider feels will be beneficial, however it is important not to let the opinion of the provider affect the patient's ability to make a decision. Rather the provider and patient should share in a discussion of different options, reservations to treatment if any, and ultimately, a plan which takes into account both the provider's expertise and medical knowledge and patients informed decision. In difficult situations, the provider should not view their role in terms of failure or success in treating a particular medical condition but rather as fulfilling their duty to the patient.

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Quick Hit Hypokalemia

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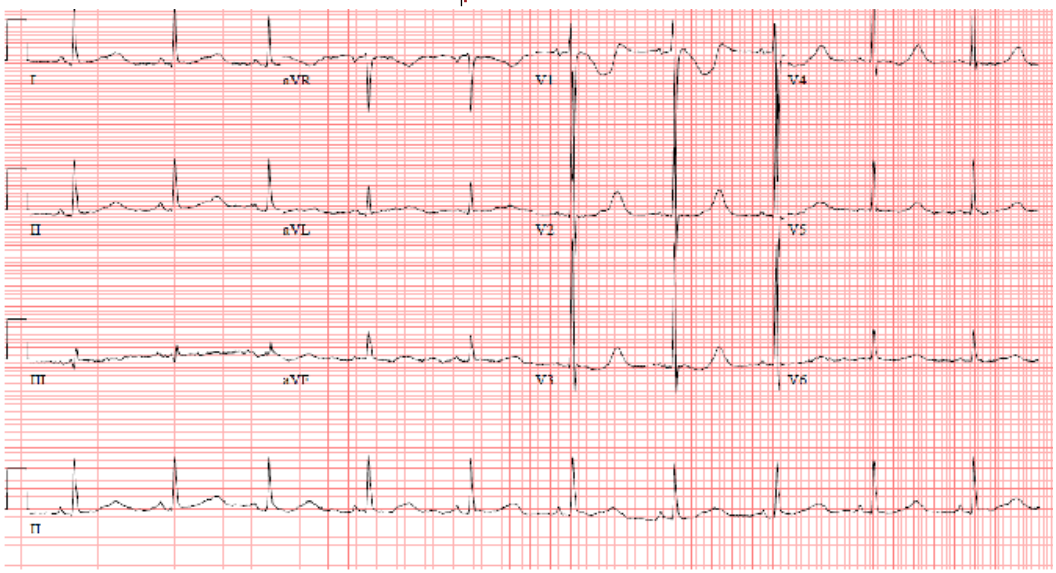
University of Cincinnati R2

Case

21yo male with a history of multiple GI surgeries on chronic TPN who presents with 3 days of nausea and vomiting. Labs revealed a K of 2.0.



EKG from a patient with a K of 2.0 showing U waves, and long QT.

EKG Changes in Hypokalemia

K < 2.7 mmol/L

- Increased Amp & Width of P Wave
- Prolongation of PR
- T wave flattening & Inversion
- ST depression
- Apparent long QT due to fusion of T & U waves

K < 2.4 mmol/L

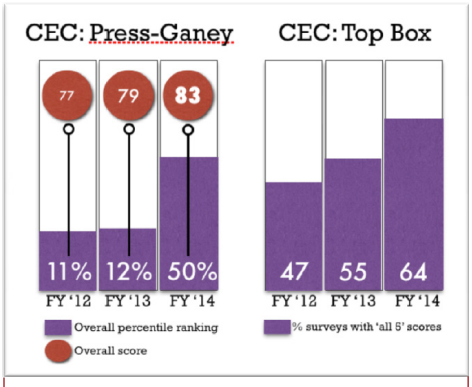
- Frequent SVT & ventricular ectopics
- AFib, A flutter, Atrial Tachycardia
- Ventricular Arrhythmias: Torsades de Pointes

CEC Update
Continued from Back Cover

lection of objective metrics such as turn-around-time of consultative services. In addition, this past month, new ASCOM phones have been disseminated throughout the ED to nearly all physicians, MLPs, and nurses to facilitate standardized and efficient communication. These phones will now use seven digit numbers with the prefix 688-, with the exception of the R3 Tele Phone and A Pod attending which will transition in the fall. Nursing leaders have also worked diligently to improve clinical operations, including the trial of in-person nurse-to-nurse handoff at bedside between the ED and inpatient nurses for patients admitted to 5NW or the MICU.

This proves to be an exciting and dynamic time for clinical operations on both the systems and resident level. With expansion of interest from the residents, including participation in the Operations Leadership Academy and independent elective projects, there are an increasing number of resident driven quality improvement projects in the works. Look out in the future for the end products of resident initiatives including use of sub-dissociative doses of ketamine for pain control, ED frequent-user utilization review and care plan formation, as well as MRI utilization. Also, CPQE protocols continue to evolve, so look for new updates to protocols for Malignancy with SIRS criteria, and Cardiac PET scanning in high-risk chest pain to name a few.

We will continue to evolve and improve to provide the best clinical environment for our primary missions: resident education and patient care.

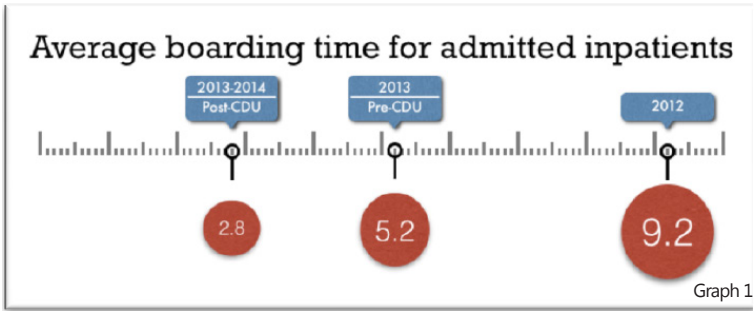


Graph 4

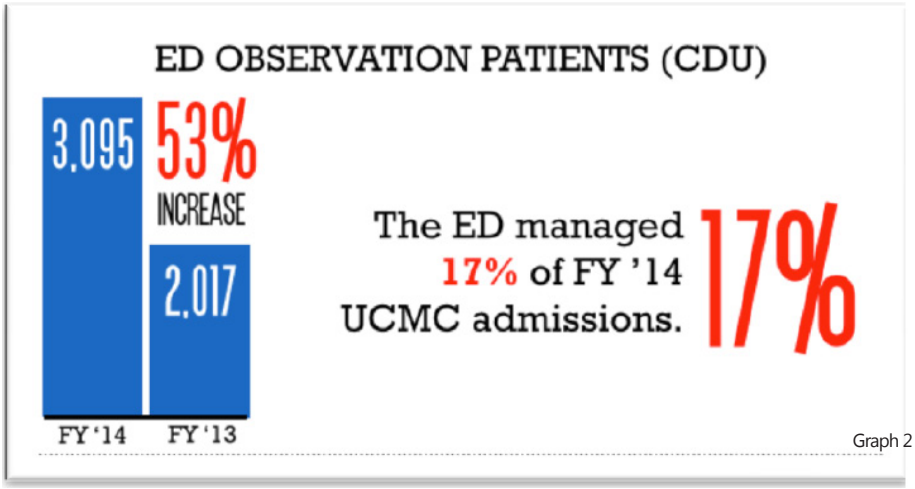
CEC: STATE OF THE DEPARTMENT

A Residency Assistant Medical Director Perspective

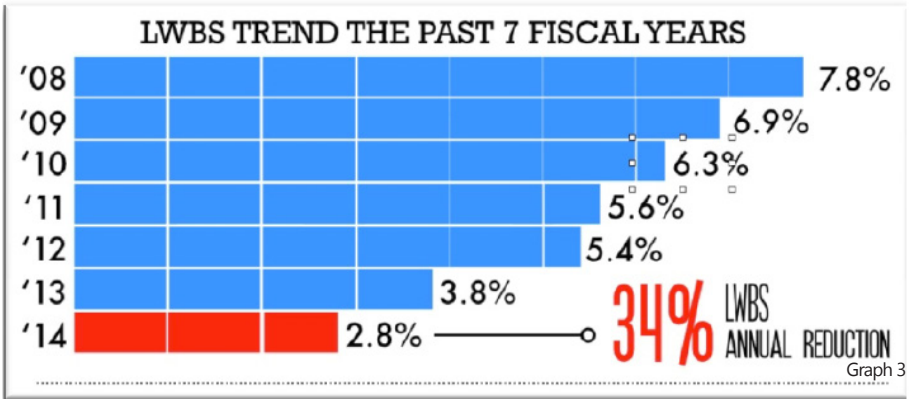
Megan Redmond, MD
University of Cincinnati R4



Graph 1



Graph 2



Graph 3

When looking at numbers from fiscal year 2014, it's clear that continued process improvements and initiatives in the operations arena have made a significant impact in the delivery of high quality, efficient, patient-centered clinical care at UCMC CEC. As demonstrated by the included graphs, the clinical operations team eclipsed goals and was able to boast all-time best performance metrics for the fourth consecutive year. These include, but are not limited to, a left-without being seen (LWBS) rate of 2.9%, patient experience overall rating percentile of 83.1, a record-high "top box" performance, and a door-to-physician time on average of 27 minutes.

Through the collaboration and efforts of system-level physician leadership in operations, administration and information technology, UCMC CEC has launched effective initiatives to streamline patient flow and progression, and enhance the patient experience. In March 2013, the clinical decision unit was created. Its utilization has expanded rapidly since inception: we have provided care for 6,000 patients (9 ED observation patients per day). Through the utilization of protocols, dedicated personnel and care coordination, the CDU is able to provide extended observation, diagnosis, and treatment to safely disposition 17% of inpatient admissions (Graph 2). This process also greatly contributes to improved patient flow, decreased 24-hour hospital admissions, and decreased ED boarding of admitted patients (Graph 1).

While metrics from fiscal year 2014 are promising, the dynamic and challenging atmosphere of the UCMC CEC continues to drive process improvement as additional challenges are identified that serve as barriers to patient care, flow and efficiency. After over a year of planning and production, the final stages of the CEC Front End Redesign construction are almost complete. This process has involved a staged renovation of minor care, intake, and the main entrance/lobby to support patient-family centered care, facilitate patient intake, optimize privacy, and improve the patient-staff experience. Such renovation included the expansion of minor care to 12 rooms (with a surge to 15 rooms when needed) including a designated procedure room, expanded eye room, and a dedicated physician dictation area with lockers. Also, three "flex rooms" have been created (D14-16) that can flex geographically between acute care beds and minor care beds as dictated by patient volume and acuity. In addition, the UCMC CEC triage process continues to evolve based on best practices, with increased attention to "direct-to-bed" triage when possible. In this process, patients will be brought back directly to an available room and receive initial vitals and RN assessment at bedside to streamline the triage process. This process echoes current national triage practices.

In addition, this past February, multiple initiatives have gone live after months of planning and optimization. First, the CEC implemented an "ED Contact Order" to track consultant call-back times and provide data-driven feedback to our clinical partners. Through this process, all consultant calls are facilitated and time-stamped through the unit HUC, which allows for the col-

Continued on page 15