

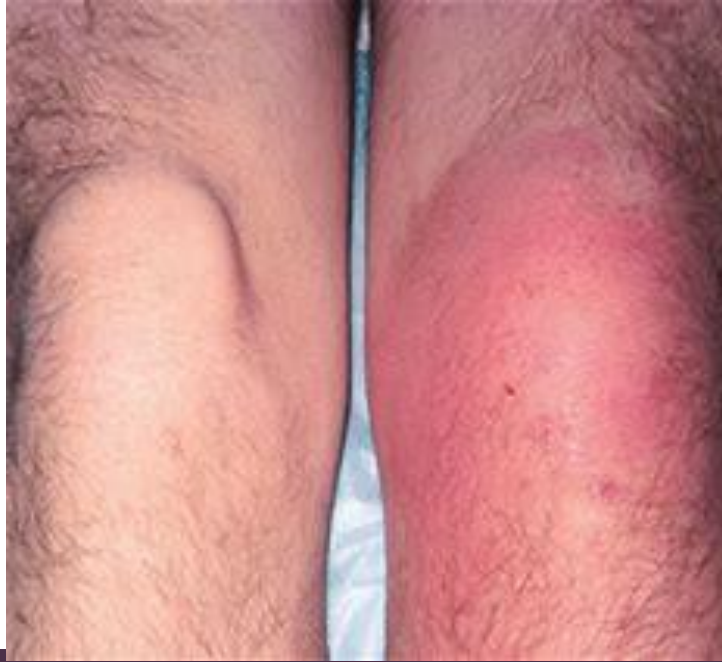
# Case Studies in Emergency Medicine with Primary Care Implications

*Robert D. Arnce, M.D. FACEP*



## Case #1

Patient is a 35-year-old Hispanic male with who presents the community health clinic complaining of left knee pain. He denies any history of trauma.



# History and Physical Exam

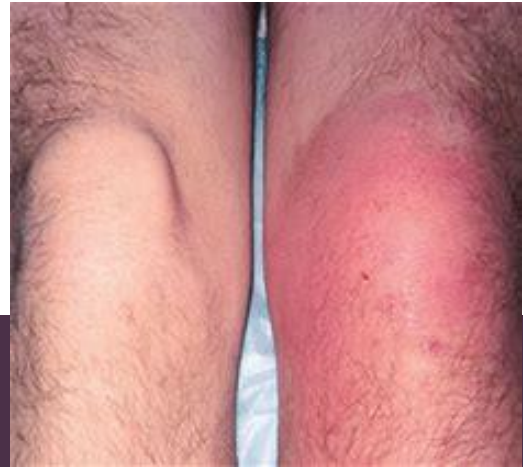
**Subjective:** CC: left knee pain

**HPI:** Patient is a 35-year-old Hispanic male who presents the ER complaining of left knee pain. Patient denies any history of trauma. He states his knee became red, swollen and painful two days ago and now the pain is so bad he will not bear weight on the left lower extremity. Patient states he's had a fever up to 102.3° F. He states He has had chills. He denies any other complaints.

- ROS: left knee pain otherwise negative
- PMhx: **Insulin-dependent diabetes (Type I)**
- PSHX: none
- Meds: Insulin; Lantus 15 units in the morning and 10 units at night. NovoLog on a sliding scale
- Allergies: none
- Shx: Smokes 1/2 pack of cigarettes a day, denies etoh, **IVDA (Last shot methamphetamine five days ago)**
- Fhx: denies

**Objective:** Vital signs: Temp: 102.5° F, heart rate 115, respiratory rate 18, blood pressure 110/68

- *LLE: The patient's left knee is erythematous, swollen, warm to the touch, and diffusely tender to palpation. The patient will not allow range of motion secondary to pain. He will not bear weight secondary to pain. The left lower extremity is neurovascular intact.*
- *The remainder the physical exam is unremarkable with the exception of resting tachycardia and fever.*



# Diagnostic Testing

- Knee X-ray = NAD
- WBC 18,000
- ESR 50
- CRP 7
- Chem: glucose 320
- Bicarb 20
- Lactic 1.8
- Joint Aspiration: 150,000 WBC's per HPF (mostly PMN's), Gram Positive Cocci present in clusters



# Septic Arthritis

- Epidemiology
  - *Responsible for 16,000 emergency department visits annually*
  - *Average LOS was 7 days*
    - 40% of patients were discharged home
    - 60% Discharge to a rehabilitation or nursing facility, longer hospital stay, and worse outcome correlated with:
      - *age >50 years, Medicaid, self-pay as primary payer, teaching hospital status, heart failure, and diabetes.*
- Pathophysiology
  - *Septic arthritis develops as a result of :*
    - hematogenous seeding
    - direct inoculation of bacteria into the joint
      - *Bites, trauma, arthroscopy, surgery, intraarticular injections*

# Risk Factors

- Advanced age
- Pre-existing joint disease
- Recent joint surgery or injection
- Skin or soft tissue infection
- Intravenous drug use
- Indwelling catheters
- Immunosuppression (including diabetes)

# Septic Arthritis

- The knee is involved in more than 50%
  - *wrists, ankles, and hips are also commonly affected*
- Joint is erythematous, swollen, warm and painful to the touch, ROM limited
- Diagnostic:
  - *plain film x-rays*
  - *Lab: elevated CBC, ESR, and CRP, BC's*
  - *Aspiration of Synovial Fluid*
    - Cell count, crystals, gram stain, C&S
    - Synovial fluid will show elevated WBC's 50k-150k/hpf and +/- bacteria
- Common Causes:
  - ***Staph, MRSA, Strep, Neisseria, lymes, mycobacteria***
- Treatment:
  - *Antibiotics; broad spectrum preferably after aspiration of synovial fluid and blood cultures*
    - Ex.: Vancomycin + cefepime
  - *Surgical washout of joint*

## Case #2

Patient is a 47-year-old white female seen in the Community Clinic by students for establishing care. Patient denies current complaint.





# History and Physical

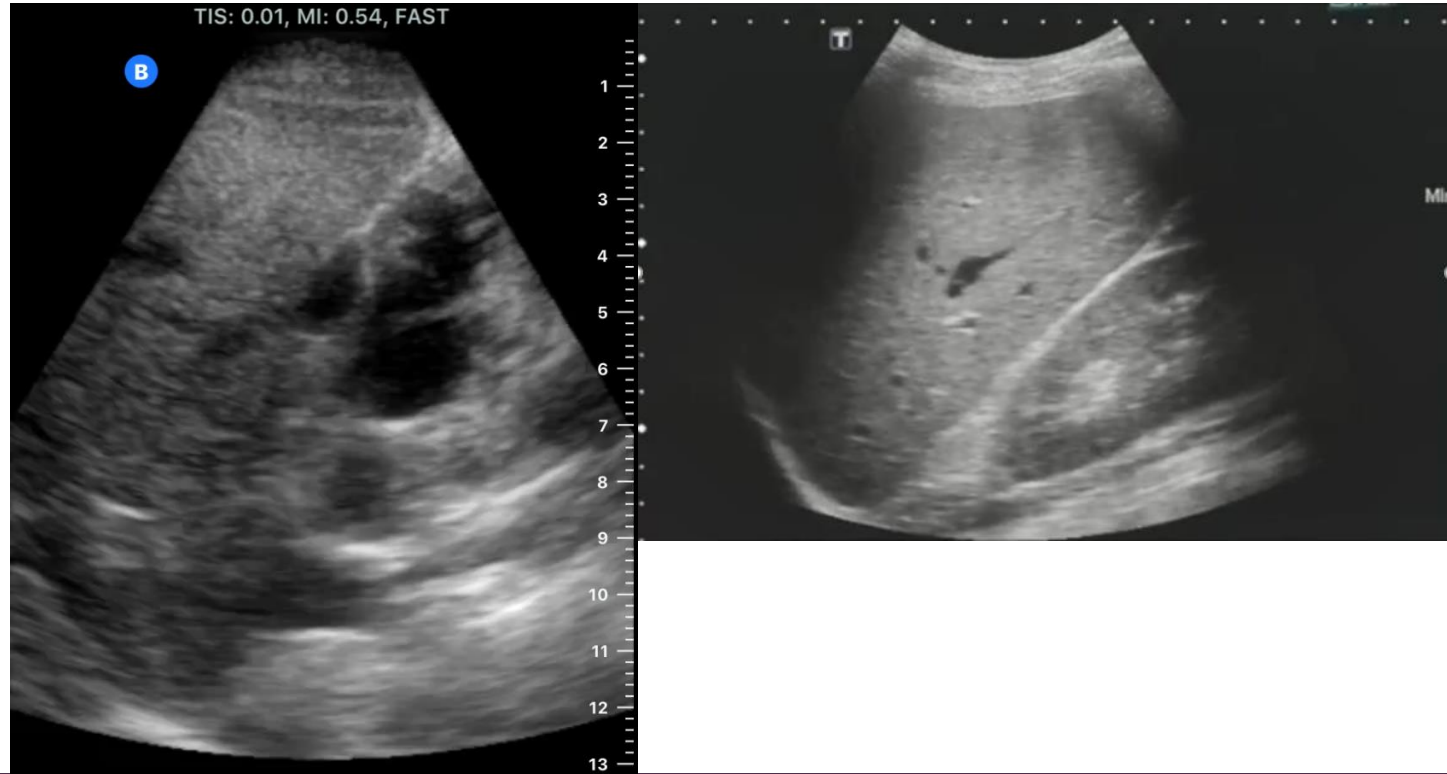
HPI: Patient is a 47-year-old white female who presents to the Community Clinic to establish care

- PMhx: HTN
- PSHX: none
- Meds: Lisinopril
- Allergies: none
- Shx: Smokes 1/2 pack of cigarettes a day, denies etoh,
- Fhx: “polycystic something”

Physical Exam: Bp 157/87 otherwise normal

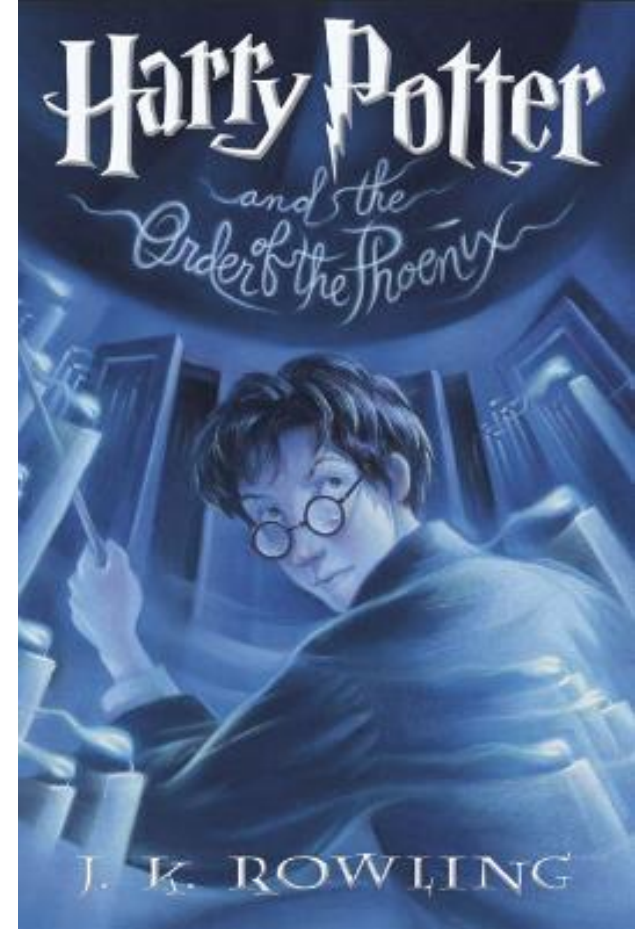
Lab: creatinine 1.8, GFR 35 otherwise nl

# Ultrasound performed by 2<sup>nd</sup> year KCUMB Student



# HOCUS POCUS

- POCUS (Point of Care Ultrasound)
- Done in office or at bedside by the provider
  - *To guide treatment of the patient*
- Done to answer a specific question
  - *“Is there a pericardial effusion?”*
  - *“Is there Intraperitoneal fluid?”*
  - *“Are there gallstones?”*
  - *“Is there hydronephrosis?”*
- Used to aid in clinical decision making
- Benefits patients and physicians by improving diagnostic accuracy while bringing the physician back to the bedside.



# Polycystic Kidney disease

- Epidemiology
  - occurring in approximately 1 in every 400 to 1000 live births
  - less than one-half of these cases will be diagnosed during the patient's lifetime as the disease is often clinically silent
  - Diagnosis based on the number of cysts per kidney on US, criteria is age dependent
  - Autosomal dominant polycystic kidney disease (ADPKD) is a common disorder
  - Autosomal recessive is less common but worse prognosis

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## CT scan showing polycystic kidney disease



Abdominal CT scan in a patient with polycystic kidney disease shows extensive cysts in both kidneys; the cysts have almost completely replaced the renal parenchyma.

# Hypertension in Polycystic Kidney Disease

- Hypertension
  - *a common early finding in ADPKD, occurring in 50 to 70 percent of cases before any significant reduction in glomerular filtration rate (GFR), with an average age onset of 30 years*
  - *Pathogenesis*
    - Increased activity of the renin-angiotensin system (RAS) and extracellular volume expansion are often present early in ADPKD (i.e., prior to elevation in the serum creatinine) and may play an important role in the rise in blood pressure
    - It has been suggested that cyst expansion, leading to focal areas of renal ischemia and enhanced renin release, is largely responsible for at least the initial rise in blood pressure
  - Treatment
    - **an ACE inhibitor should be the initial antihypertensive agent**
    - BP goal <130/80

## Case #3

Patient is a 62-year-old white female with PMhx sig for HTN, NIDDM, and hyperlipidemia who presents to her primary care physician's office complaining of 3 day h/o dyspnea on exertion and some mild intermittent chest pain.

- Normal stress test within the last year



# History and Physical Exam

**Subjective:** CC: DOE, SOA, CP

**HPI:** Patient is a 62-year-old white female who presents to her physician's office complaining of a three day history of dyspnea on exertion, shortness of breath, and intermittent chest pain. The dyspnea on exertion has gotten progressively worse. Patient states that she cannot walk more than a few steps without becoming short of breath. Her chest pain is intermittent, located in left precordium, radiates to the left shoulder, and is described as sharp and worse with inspiration. Patients had a nonproductive cough. She denies any fever, chills.

**PMHx:** NIDDM, HTN, Hyperlipidemia

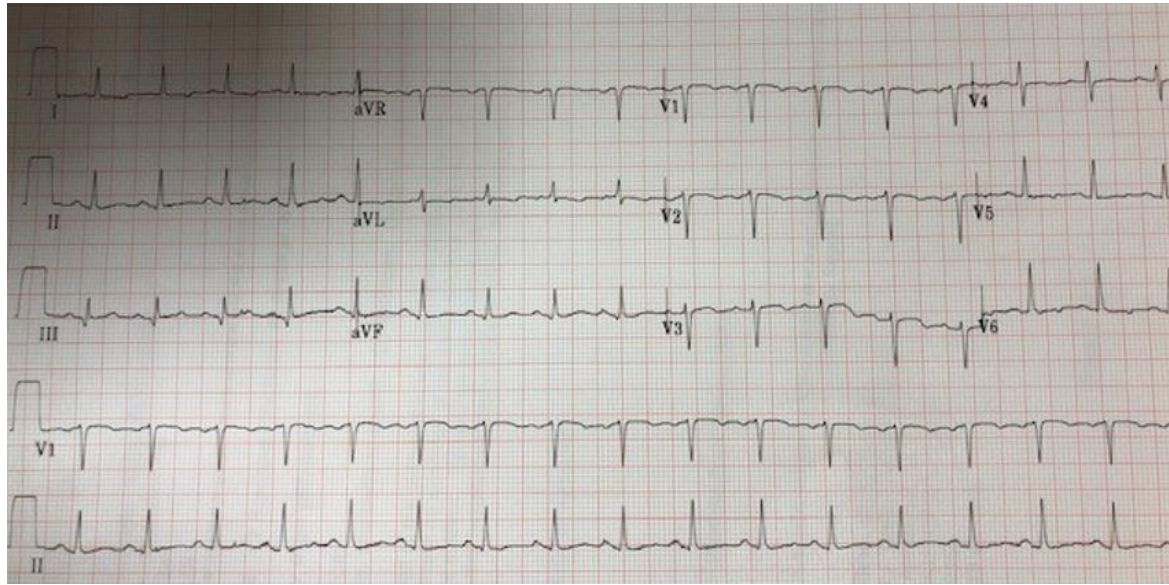
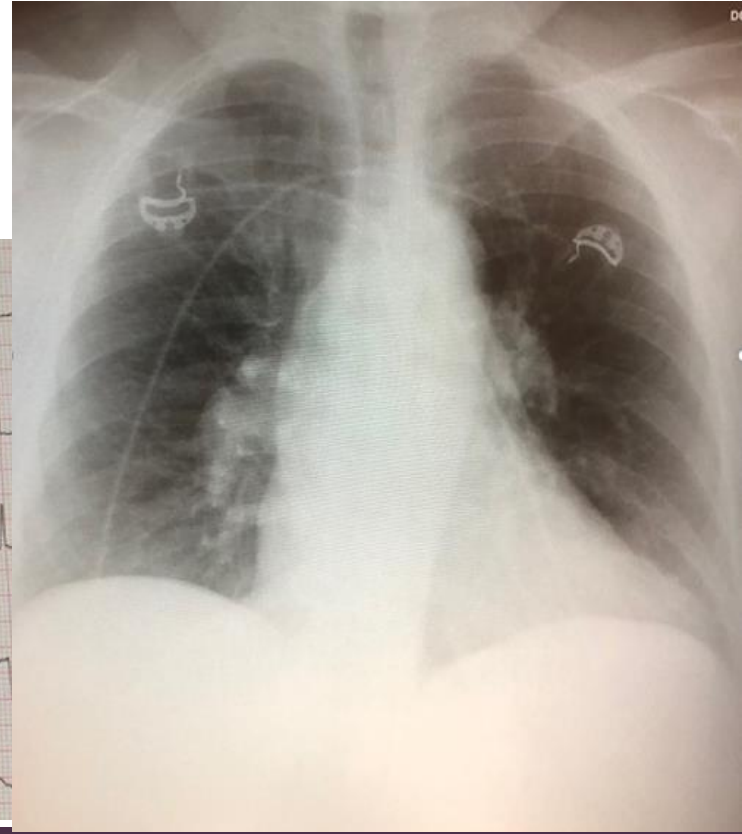
- PSHX: none
- Meds: metformin
- Allergies: none
- Shx: Smokes 1/2 pack of cigarettes a day, denies etoh
- Fhx: denies

**Objective:** Vital signs: Temp: 98.6° F, heart rate 105, respiratory rate 20, blood pressure 110/68, 94% on room air. However, when the patient walks down the hall. Her oxygen saturation drops to 84% on room air.

- *Physical exam is remarkable for mild tachypnea with respiratory rate of 22. And tachycardia with a resting heart rate of approximately 103-110. Lungs are clear auscultation bilaterally. There is no JVD were lower extremity edema, cardiac exam is regular rhythm without murmur, rub, or gallop*
- What is your working differential at this point?



# Diagnostic Testing



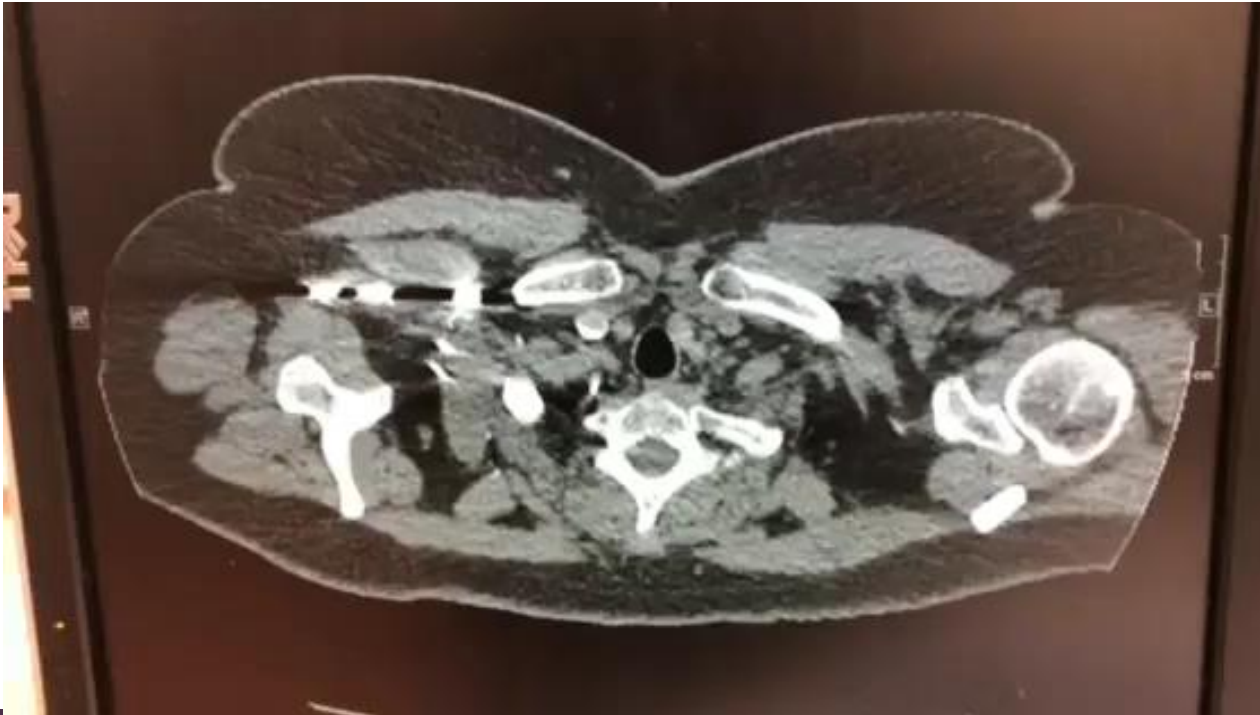


# Diagnostic Testing

08/13/19 14:20			
WBC	7.9	Sodium	137
RBC	4.59	Potassium	4.1
Hgb	12.4	Chloride	101
Hct	40.3	Carbon Dioxide	18 L
MCV	87.8	Plasma Anion Gap	18
MCH	27.0	BUN	13
MCHC	30.8	Creatinine	1.0
RDW	17.4 H	GFR Calculation	61
Plt Count	233	BUN/Creatinine Ratio	13
MPV	9.8	Glucose	138 H
Neut % (Auto)	59.4	POC Glucometer	143 H
Lymph % (Auto)	25.7	Calculated Osmolality	276
Mono % (Auto)	8.3	Plasma Calcium	9.3
Eos % (Auto)	5.2	Total Bilirubin	0.3
Baso % (Auto)	1.0	AST	29
Absolute Neuts (auto)	4.7	ALT	36 H
Absolute Lymphs (auto)	2.0	Alkaline Phosphatase	57
Absolute Monos (auto)	0.7	Creatine Kinase	69
Absolute Eos (auto)	0.4	CK-MB (CK-2)	0.75
Absolute Basos (auto)	0.1	Troponin I	0.032
		NT-Pro-B Natriuret Pep	961 H
		Total Protein	8.0
		Albumin	4.6
		Globulin	3.4
		Alb/Glob Ratio Alt Meth	1.4
		Procalcitonin	< 0.05 L

08/13/19 14:52	
D-Dimer, Quantitative	7159 H

# CTA Chest Axial View



# CTA of Chest Coronal View



# Pulmonary Embolism Epidemiology

- Estimates of the incidence of pulmonary embolism (PE) in the general population have increased following the introduction of D-dimer testing and computed tomographic pulmonary angiography in the 1990s
- The overall incidence of PE is approximately 112 cases per 100,000. PE is slightly more common in males than females and incidence increases with age. Deaths from PE account for approximately 100,000 deaths per year in the United States

# Pathophysiology

- **Virchow's triad**
  - venous stasis, endothelial injury, and a hypercoagulable state
  - Same process for DVT's
- **Most pulmonary emboli come from the proximal veins of the lower extremity**
  - iliac, femoral, and popliteal
- **Greater than 50 percent of patients with proximal vein deep venous thrombosis (DVT) have concurrent PE at presentation**
- Calf vein DVT's rarely embolize to the lungs
  - if untreated, one-third of calf vein DVT's extend into the proximal veins, where they have greater potential to embolize.

# Risk Factors

- similar to those for venous thromboembolism (VTE) in general
- Genetic hypercoagulable states ( ex. factor V Leiden)
- recent surgery, trauma, immobilization, initiation of hormone therapy, active cancer obesity, heavy cigarette smoking

# Clinical Presentation

- wide variety of presenting features, ranging from no symptoms to shock or sudden death
- The most common presenting symptom is dyspnea followed by chest pain (classically pleuritic in nature), cough, and symptoms of deep venous thrombosis.
- Hemoptysis is an unusual presenting symptom.
- Rarely do patients present with shock, arrhythmia, or syncope
- Many patients are asymptomatic or have mild or nonspecific symptoms.
- it is critical that a high level of suspicion be maintained such that clinically relevant cases are not missed.

# Diagnostic Work up

- D-Dimer if Appropriate based on wells score
- A score of  $<2.0$  and a negative D-dimer results in a PE rate of 1.5% (95% CI 0.4% to 3.7%).

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## Wells criteria and modified Wells criteria: Clinical assessment for pulmonary embolism

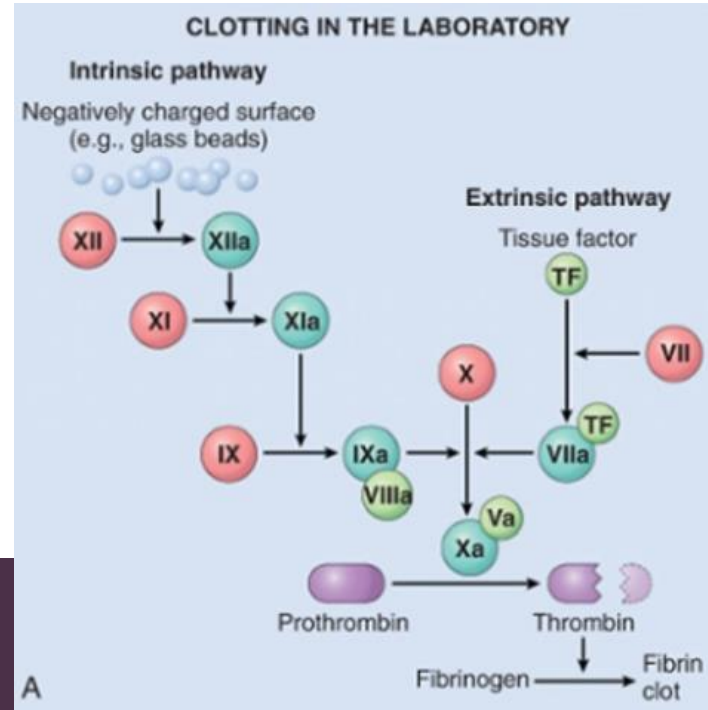
▪ Clinical symptoms of DVT (leg swelling, pain with palpation)	3.0
▪ Other diagnosis less likely than pulmonary embolism	3.0
▪ Heart rate $>100$	1.5
▪ Immobilization ( $\geq 3$ days) or surgery in the previous four weeks	1.5
▪ Previous DVT/PE	1.5
▪ Hemoptysis	1.0
▪ Malignancy	1.0
<b>Probability</b>	<b>Score</b>
<b>Traditional clinical probability assessment (Wells criteria)</b>	
High	$>6.0$
Moderate	2.0 to 6.0
Low	$<2.0$
<b>Simplified clinical probability assessment (Modified Wells criteria)</b>	
PE likely	$>4.0$
PE unlikely	$\leq 4.0$





# PE Treatment

- Hemodynamically Stable
  - *Heparin*
    - Unfractionated 80 units/kg IV bolus, 18 units/kg/hr.
    - Low molecular weight heparin (Lovenox) 1 mg/kg IV
  - *Xa inhibitors*
    - Xarelto (rivaroxiban)
    - Eliquis (apixaban)
  - *Other agents require initial heparin therapy and bridging*
    - Coumadin, Dabigatran, Edoxaban
- Hemodynamically Unstable
  - *Consider TPA*



# ANDEXXA

- Reversal agent for Xa inhibitors
- a recombinant modified human factor Xa protein indicated for patients treated with rivaroxaban or apixaban, when reversal of anticoagulation is needed due to life-threatening or uncontrolled bleeding.
- Currently, most patients will require 1 dose of Andexxa, which will generally **cost between \$25,000 and \$31,000** for one dose of the medication. May 9, 2019




# Case # 4

72 yo Hispanic male presents to the ER from physicians office for rash and spontaneous tongue bleeding



# Diagnostic Testing

	06/04/19 13:23
WBC	6.7
RBC	4.83
Hgb	11.9 L
Hct	41.1 L 
MCV	85.1
MCH	24.6 L
MCHC	29.0 L
RDW	22.1 H
Plt Count	< 3 L*
Neut % (Auto)	81.1 H
Lymph % (Auto)	15.3 L
Mono % (Auto)	2.5 L
Eos % (Auto)	0.4
Baso % (Auto)	0.4
Absolute Neuts (auto)	5.5
Absolute Lymphs (auto)	1.0
Absolute Monos (auto)	0.2
Absolute Eos (auto)	0.0
Absolute Basos (auto)	0.0
Platelet Estimate	Gr Dec

# ITP Epidemiology

- ITP is a common acquired bleeding disorder
- ITP incidence of approximately 1 to 5 per 100,000 adults
- one-fifth to one-third of ITP patients will be asymptomatic at the time of diagnosis (incidental finding of thrombocytopenia)
- The clinical manifestations of ITP are all mostly related to bleeding and/or thrombocytopenia; fatigue occurs in some patients. Bleeding may occur in up to two-thirds of patients; however, many are asymptomatic. Serious bleeding is seen most often in individuals with platelet counts  $<20,000/\text{microL}$

# Pathophysiology

- Results from: a combination of increased platelet destruction and impaired platelet production caused principally by antiplatelet autoantibodies.
- ITP is a diagnosis of exclusion, characterized by isolated thrombocytopenia without a clinically apparent condition responsible for the low platelet count; there are no reliable laboratory tests to confirm the diagnosis
- **Primary ITP** –not triggered by an associated condition.
- **Secondary ITP** – Secondary ITP is ITP associated with another condition (Lupus, CLL, HIV)

# ITP Treatment

- **Immediate treatment** — For patients with severe bleeding (eg, intracranial, gastrointestinal) and a platelet count  $<30,000/\mu\text{L}$ , we recommend immediate therapy that could include all of the following:
  - *Platelet transfusion*
  - *IVIg*
  - *Glucocorticoids*
    - Methylprednisolone
    - Dexamethasone

# Case #5

- Patient is a 68-year-old white male with past medical history significant for hypertension, diabetes, coronary artery disease, renal insufficiency, and prior partial amputation of the right foot and subsequent infection treated in the wound care clinic long-standing who presents to the ER from his physician's office complaining of infection of the right foot, and bad odor.



# History and Physical Exam

**Subjective:** CC: infected foot bad smell

HPI: Patient is a 68-year-old white male with past medical history significant for hypertension, diabetes, coronary artery disease, renal insufficiency, and prior partial amputation of the right foot and subsequent infection treated in the wound care clinic long-standing who presents to the ER from his physician's office complaining of infection of the right foot, and bad odor.

PMhx:, HTN, CAD, NIDDM, CRI

- PSHX: prior partial Right foot amputation, Left AKA
- Meds: multiple no blood thinners
- Allergies: none
- Shx: 1PPD smoker
- Fhx: denies

**Objective:** Vitals: BP 90/65, HR 112, RR 20, Sat 94% RA

PE: See picture otherwise unremarkable with the exception of tachycardia, and crepitus being present in the right foot

PE



# Diagnostic Testing



# Diabetic Neuropathy

- Peripheral and autonomic neuropathies are a major cause of morbidity in patients with diabetes mellitus
- 3 pronged approach to treatment
  - *Glycemic control*
  - *Foot care*
    - patients need to inspect their feet for the presence of dry or cracking skin, fissures, plantar callus formation, and signs of early infection between the toes and around the toe nails. Regular foot examinations by the physician to detect early neuropathy are also an essential component of the treatment of diabetic patients
  - *Treatment of pain*
    - The American Academy of Neurology
      - *Lyrica, gabapentin, amitriptyline, valproate, others*

# Gas Gangrene

- Clostridial myonecrosis (gas gangrene) is a life-threatening muscle infection that develops either contiguously from an area of trauma
- *Clostridium* species are widespread in nature due to their ability to form endospores. They are commonly found in soil and marine sediments as well as human and animal intestinal tracts.
- Traumatic wounds with vascular compromise (particularly deep penetrating injuries)
  - *knife wounds, gunshot wounds, and crush injuries create an anaerobic environment ideal for clostridia.*
  - *Traumatic injury accounts for about 70 percent of gas gangrene cases, and about 80 percent of these are caused by C. perfringens*

# Gas Gangrene

- Pain at a site of traumatic injury together with signs of systemic toxicity and gas in the soft tissue support the diagnosis of gas gangrene. Physical evidence of crepitus in the soft tissue is the most sensitive and specific finding on clinical examination
- Radiographic imaging can be especially useful for identifying gas in deep tissues. Gas within the soft tissue can be detected by radiography

## Case #6 “Interfering with Natural Selection”

- The patient is a 36 yo wm complains of right finger pain and his finger being stuck. He states that he was drunk and wrestling with his brother and hurt his finger a few days ago. He states that he was so drunk he fell asleep with a cigarette in his hand and that's how he burned his finger. He now presents complaining of continued deformity of his finger



# Physical Exam and Diagnostic Testing

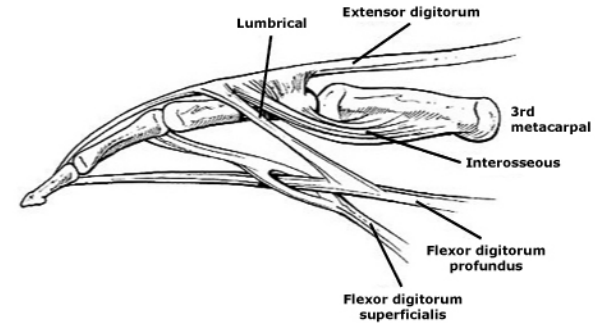




# Mallet Finger

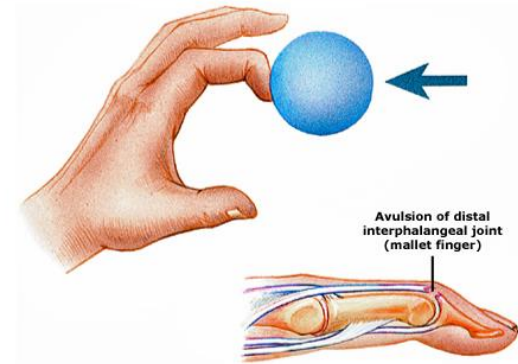
- A mallet finger injury is the most common closed tendon injury of the finger. The injury occurs most often in the workplace or during contact or ball-handling sports. It is most common in young to middle-aged males, which may reflect their higher rates of participation in such sports
- Traumatic disruption of the terminal slip of the extensor tendon at the distal interphalangeal (DIP) joint

## Finger tendons: Expanded view



This picture reveals the complex relationships among the tendons and bones of the finger and clearly shows how the flexor digitorum profundus travels through the flexor digitorum superficialis to insert on the distal phalanx.

## Mallet finger mechanism



Mallet finger injuries involve disruption of the insertion of the extensor tendon, usually caused by a direct blow to the fingertip.

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## Mallet finger



# Mallet Finger

- Mallet finger occurs most commonly during collision sports
- caused by a direct blow to the tip of the finger, which causes sudden, forceful flexion of the distal phalanx
- With mallet finger injuries, the tendon may be partially torn, completely ruptured, or associated with an avulsion fracture of the distal phalanx. Unopposed flexion leads to a fixed flexion deformity (called extensor lag) at the DIP joint if the injury remains untreated

# Case #7

- Patient is an Eight-year-old, undocumented, Hispanic female who presents complaining of today history of presents complaining of fever, headache, ear pain, myalgias, and facial swelling. Patient states that she started with right ear pain, followed by right facial swelling and then develop swelling of the left face and left ear pain.

# Physical Exam



# Mumps

- largely preventable via vaccination
- Prior to vaccinations (1967), about 186,000 cases were reported annually
- Since vaccinations, there has been a 99 percent decrease in mumps cases in the United States
- Resent resurgence secondary to lack of immunizations
- highly infectious and is transmitted by respiratory droplets, and direct contact

# Mumps Clinical Presentation and Treatment

- Parotitis occurs most commonly among children between two and nine years of age; tenderness, occasionally associated with earache, typically precedes parotid swelling. Parotitis may be unilateral or bilateral; initial unilateral involvement is followed by contralateral involvement a few days later in 90 percent of cases
- Typically, it begins with a few days of fever, headache, myalgia, fatigue, and anorexia, followed by parotitis; the illness is usually self-limited.
- Complications of mumps may include orchitis and neurologic manifestations (including meningitis, encephalitis, and deafness)
- Treatment is supportive

## Case #8

- 42-year-old white male presents to the ER for psychiatric evaluation for strange behavior. Patient is mildly confused and tachypnea. On physical exam. He states that he has had a sore ankle and is been taking an over-the-counter pain medication over the past several days. He states he has some nausea, loose stools and ring in his ears.



# Diagnostic Testing

Sodium	138	
Potassium	3.3 L	
Chloride	103	
Carbon Dioxide	13 L*	
Plasma Anion Gap	22 H	
BUN	47 H	
Creatinine	1.3 H	
GFR Calculation	64	
BUN/Creatinine Ratio	36 H	
Glucose	107 H	
Calculated Osmolality	288	
POC Arterial Lactate		
Plasma Calcium	9.6	
Magnesium		2.5 H
Total Bilirubin	0.5	
AST	34	
ALT	26	
Alkaline Phosphatase	106	
Creatine Kinase		244 H
CK-MB (CK-2)		4.71
Troponin I		
Total Protein		0.019

	08/13/19 11:51	08/13/19 17:22
Puncture Site	Right Radial	
POC ABG pH	7.446	
POC ABG pCO2	23.4 L	
POC ABG pO2	109 H	
POC ABG HCO3	16.1 L	
POC ABG Total CO2	17 L	
POC ABG O2 Sat	99 H	
POC ABG Base Excess	-6 L	
POC Modified Allen Test	POS	
POC VBG pH		7.457 H
POC VBG pCO2		33.2 L
POC VBG pO2		37
POC VBG HCO3		23.4
POC VBG Total CO2		24
POC Venous O2 Sat		74 H
POC VBG Base Excess		0
Patient Status	21.0	0.0
Respiration Rate	16.0	
O2 Delivery Method	Room Air	



# Serum Salicylate Level

- Therapeutic = 10 to 30 mg/dL
- above 40 mg/dL are associated with toxicity

	08/13/19 10:14	08/13/19 10:14	08/13/19 12:34	08/13/19 14:25
Salicylates		68.9 H*		59.5 H*
Urine Opiates Screen			NEGATIVE	
Ur Buprenorphine Scrn			NEGATIVE	
Ur Oxycodone Screen			NEGATIVE	
Urine Methadone Screen			NEGATIVE	
Urine Propoxyphene			NEGATIVE	
Acetaminophen		< 10 L		
Ur Barbiturates Screen			NEGATIVE	
Ur Tricyclics Screen			NEGATIVE	
Ur Phencyclidine Scrn			NEGATIVE	
Ur Amphetamine Screen			NEGATIVE	
Ur Methamphetamines Cmt			NEGATIVE	
U Benzodiazepines Scrn			NEGATIVE	
Urine Cocaine Screen			NEGATIVE	
U Cannabinoids Screen			NEGATIVE	
Alcohol, Quantitative	< 0.01			
Ethyl Alcohol mg/dL	< 10			

# Aspirin Effects

- Inhibition of cyclooxygenase
  - *contributes to platelet dysfunction and gastric mucosal injury.*
- Stimulation of the chemoreceptor trigger zone in the medulla causes nausea and vomiting.
- Activation of the respiratory center of the medulla results in hyperventilation and respiratory alkalosis.
- Interference with cellular metabolism (eg, Krebs cycle, oxidative phosphorylation) leads to metabolic acidosis.

# Clinical Presentation of Aspirin Toxicity

- tinnitus, vertigo, nausea, vomiting, and diarrhea
- With severe intoxication, altered mental status (ranging from agitation to lethargy), hyperpyrexia, noncardiac pulmonary edema, and coma.
- Hyperpnea is often observed in salicylate overdose and is an early clinical finding that helps establish the diagnosis
- pay particular attention not only to the rate, but also to the depth of respiratory effort.

# Treatment

- Alkalinization of urine
- Treat hypokalemia aggressively
  - *Hypokalemia interferes with efforts at urinary alkalinization*
- Dialysis

Thank you!