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Acute Pulmonary Emergencies: Pulmonary Embolism, Pulmonary Edema and Parapneumonic Effusions

UMHS PEM Conference Series

April 2010

Michele M. Nypaver, MD

Case Presentation

- A 13y/o African American female presents to the peds ED with complaints of chest discomfort and SOB, worse with exercise and now even walking. Had URI symptoms several weeks ago (as did others in family), seemed to resolve until day of presentation when CP/SOB became suddenly worse. No fever, occasional dry cough, no V/D, no runny nose, sore throat.
- No meds/allergies/imm's UTD

More Info?

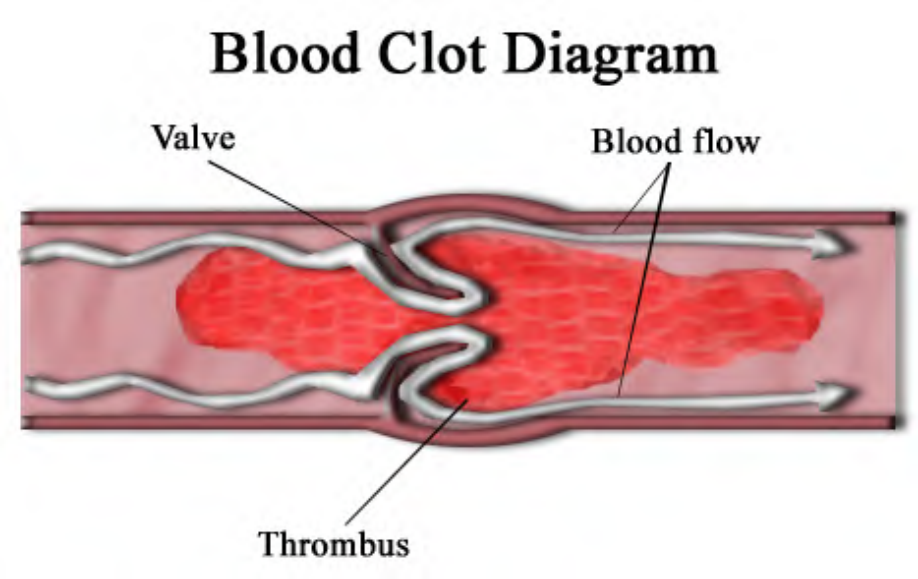
- What other questions?
- Past Medical History
- Significant for deep venous thrombus in the right calf in November 2006. She received anticoagulation with Lovenox until follow-up Doppler scan demonstrated resolution of the DVT. Currently on no anticoagulation.
- No bruises, weight loss, No oral contraceptives, no other sx's on ROS

Pediatric Pulmonary Embolism

- Review Pathophysiology
- Suspecting the diagnosis
- Adult versus pediatric PE
- Evaluation
- Treatment

image removed

Please see original image of DVT/Pulmonary Embolism at http://www.activeforever.com/t-deep-vein-thrombosis-article.aspx



 Persian Poet Gal, "Blood Clot Diagram (Thrombus)", Wikimedia Commons

Please see original image of Pulmonary Embolism at http://www.riversideonline.com/source/images/image_popup/r7_pulmonaryembolism.jpg

Please see original image of Pulmonary Embolism at http://www.ncbi.nlm.nih.gov/pubmedhealth/PMH0001189/

PE Pathophysiology

- Embolic clot size / location determines presentation
- Cardiac Effects:
  - Clot obstructs RV outflow
  - Sudden increased RV dilatation and pressures
  - RV pressure can affect (reduce) LV fxn
  - If PFO, R to L shunt can occur
  - Vasoconstriction of Pulm Vasculature:
    - Increased Pulm Vasc Resistance
    - Release of neural/humoral mediators which increase pulmonary vasculature resistance
- **DECREASED CO, VQ mismatch**
- **Sudden, unpredictable cardiovascular collapse**

Pulmonary Embolus

- Lung Effects
  - Clot prevents diffusion of oxygen from alveoli to circulation
  - Overall increases dead space of some portion (or all) of the lung
  - Affected area becomes atalectatic
  - Overall
    - Increase in pulm vascular resistance
    - Decreased alveolar availability for gas exchange

Adult PE

- 600,000 cases/yr
  - Traditional risk factors:
    - Bed rest (> 3 days)
    - Heart dz
    - Malignancy
    - Prior DVT/PE
    - Surgery (in last 3 months)
    - Estrogen RX
    - Pregnancy
    - Hypercoaguable states
    - Recent travel
    - 20-25% Have NO identifiable risk factors on presentation

Approach to Diagnosis

- Adult clinical symptoms
  - Determine pre test probability for the likelihood of PE
  - Direct testing that reflects likelihood of disease while minimize risk to patient and overuse of invasive procedures
  - Caveat: None are 100% sensitive or specific
  - Gold Std: Angiography

Wells Clinical Prediction Rule for Pulmonary Embolism

Clinical feature	*Points*
Clinical symptoms of DVT	3
Other diagnosis less likely than PE	3
Heart rate greater than 100 beats per minute	1.5
Immobilization or surgery within past 4 weeks	1.5
Previous DVT or PE	1.5
Hemoptysis	1
Malignancy	1

Risk score interpretation (probability of PE):

- >6 points: high risk (78.4%);
- 2 to 6 points: moderate risk (27.8%);
- <2 points: low risk (3.4%)

Wells PS et al. Ann Intern Med. 1998;129;997

Clinical Presentation of PE (Adult)

- Tachypnea
- Rales
- Tachycardia
- 4th Heart Sound
- Accentuated S2
- Dyspnea
- Pleuritic chest pain
- Cough
- Hemoptysis

Girard P. et al. Am J Respir Crit Care Med. 2001;164:1033
Prospective Investigation of Pulmonary Embolism Diagnosis Study (PIOPED).

Canadian childhood Thrombophilia Registry (N=405)

- **Incidence of Pulm Embolism 0.07/10,000**
- **Pediatric PE Risk Factors (one or more)**

Central Venous Catheter	60%
Cancer/Bone marrow transplant	25%
Cardiac surgery	19%
Surgery (other)	15%
Infection	12%
MVA/trauma/Burn	10%
Oral contraception	4%
Obesity	2%
Congenital / acquired pro-thrombotic disorder	2%
SLE	1.5%

Ped Emerg Care 2004: 20 (8) Green et al. Chest 1992:101;1507

Pediatric PE

- Neonatal considerations
  - Peri-partum asphyxia
  - Dehydration
  - Sepsis
  - Most PE's due to Catheters
    - Aorta, pulmonary, renal
- Special considerations
  - Renal disease: Nephrotic syndrome (altered levels of antithrombin and increased other coag proteins).
  - Klippel-Trenauay
  - Hemangiomas
  - Anti phospholipid antibodies (Lupus)
- Non thrombotic emboli:
    - Foreign bodies
    - Tumor emboli
    - Septic emboli
    - Post traumatic fat emboli

Pediatric PE and DVT

- Incidence of pediatric PE in children with documented DVT 30%
- DVT in children may obviate the need to evaluate the chest
- Pediatric DVT often in upper venous system
- PE can originate from intracranial venous sinus thrombosis
- Mortality from DVT/PE in children may be lower than adults (2.2% from Canadian Registry; all deaths were from emboli to upper venous system and were catheter related).

Clinical Presentations in Pediatric PE

- **Similar to adults BUT**
- **Children have better physiologic reserve**
- **Less prominent respiratory rate or heart rate changes compared to adults**
- **Other signs/symptoms associated with PE**
  - **Pleuritic chest pain**
  - **Hemoptysis**
  - **Cyanosis**
  - **RHF**
  - **Hypoxia**
  - **Hypercarbia**
  - **Pulm hypertension**
  - **Rare cases of paradoxical embolism/stroke (venous to arterial emboli due to cardiac defect or pulm av malformation.**

Case KA

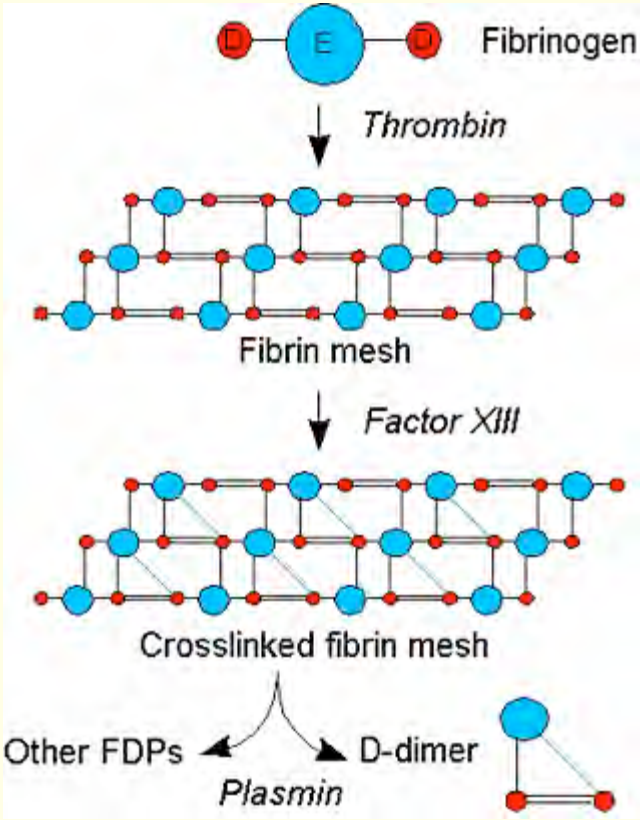
- Physical Exam:
- Vitals: Temp 98.0, pulse 120, respiratory rate 36, blood pressure 122/76, pulse ox 95% on room air, weight 60.4 kilograms
- She has a normal S1 and physiologically split loud S2. She has a 2/6 systolic ejection murmur audible at the right and left upper sternal borders.

Case KA

- Initial Evaluation
  - CBC
  - ABG
  - EKG
  - Other labs: anticardiolipin antibody, a lipoprotein A level, a homocysteine level, factor V Leiden mutation screening, and prothrombin 20210 mutation.
  - Hemodynamic/respiratory monitoring

Evaluation of Children with Suspected PE

- **Toolbox**
  - **ABG**: Low paO2; low paCO2 initially, ominous rise in paCO2
  - **EKG**: Sinus tachycardia, RAD, RVH, RBBB
  - **Increased a-A gradient**
  - **D-Dimer**: Highly sensitive in adults coupled with clinical evaluation to r/o PE; Few data in children and false positive in presence of infection/ malignancy.



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A-a gradient = PAO2 − PaO2

Aa Gradient = [FiO2*(Patm-PH2O)-(PaCO2/0.8)] - PaO2

Aa Gradient = (150 - 5/4(PCO2)) - PaO2

EKG & PE

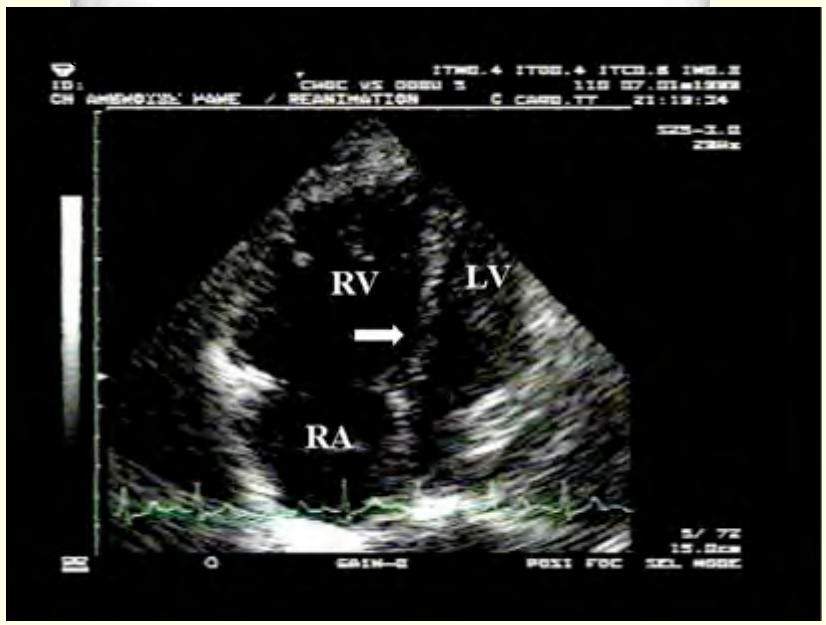
- ECG features in PE lack specificity and sensitivity
- Value of ECG for the diagnosis of PE is debatable
  - ECG can be normal in pulmonary embolism, and other recognised features of PE include sinus tachycardia (heart rate >100 beats/min), negative T waves in precordial leads, S1 Q3 T3, complete/incomplete right bundle branch block, right axis deviation, inferior S wave notch in lead V1, and subepicardial ischaemic patterns.
- The mechanism for these ECG changes is acute right heart dilatation, such that the V leads that mostly represent the left ventricle now represent the right ventricle (RV). The presence of inverted T waves on precordial leads suggests massive PE.

EKG & PE

- “S1 Q3 T3” - prominent S wave in lead I, Q and inverted T waves in lead III
- Right bundle branch block (RBBB), complete or incomplete, often resolving after acute phase
- Right shift of QRS axis
- shift of transition zone from V4 to V5-6
- ST elevation in VI and aVR
- generalized low-amplitude QRS
- sinus tachycardia, atrial fibrillation/flutter, or right-sided PAC/PVC
- T wave inversion in V1-4, often a late sign.

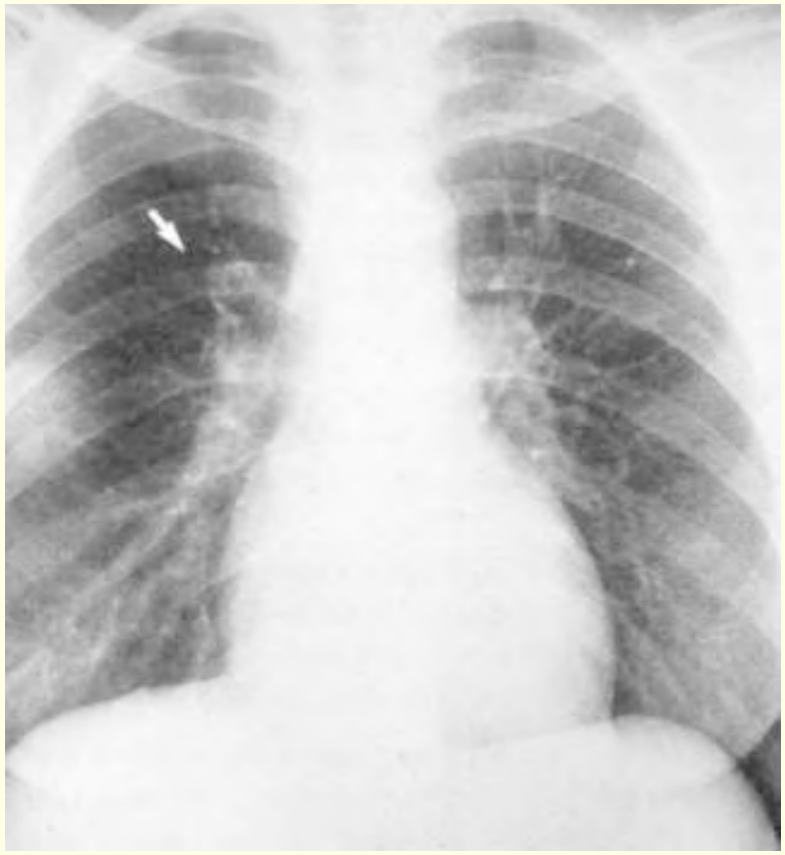
Evaluation of Children with Suspected PE

- **CXR:** infiltrates, atelectasis, unilateral pleural effusion, hypovascularity in lung zone (Westermark's sign) & pyramid shape infiltrate with peak directed to hilus (Hampton's hump). Chronic PE can result in findings of RH enlargement, enlarged PA's.
- **Echo**: not helpful for distal clots, better to assess pulm hypertension and massive PE with central position.



Plain film radiography **Chest X-ray**

- **Westermark sign** – Dilatation of pulmonary vessels proximal to embolism along with collapse of distal vessels, often with a sharp cut off.



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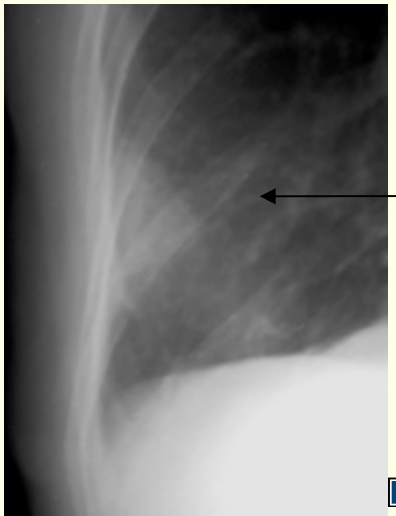

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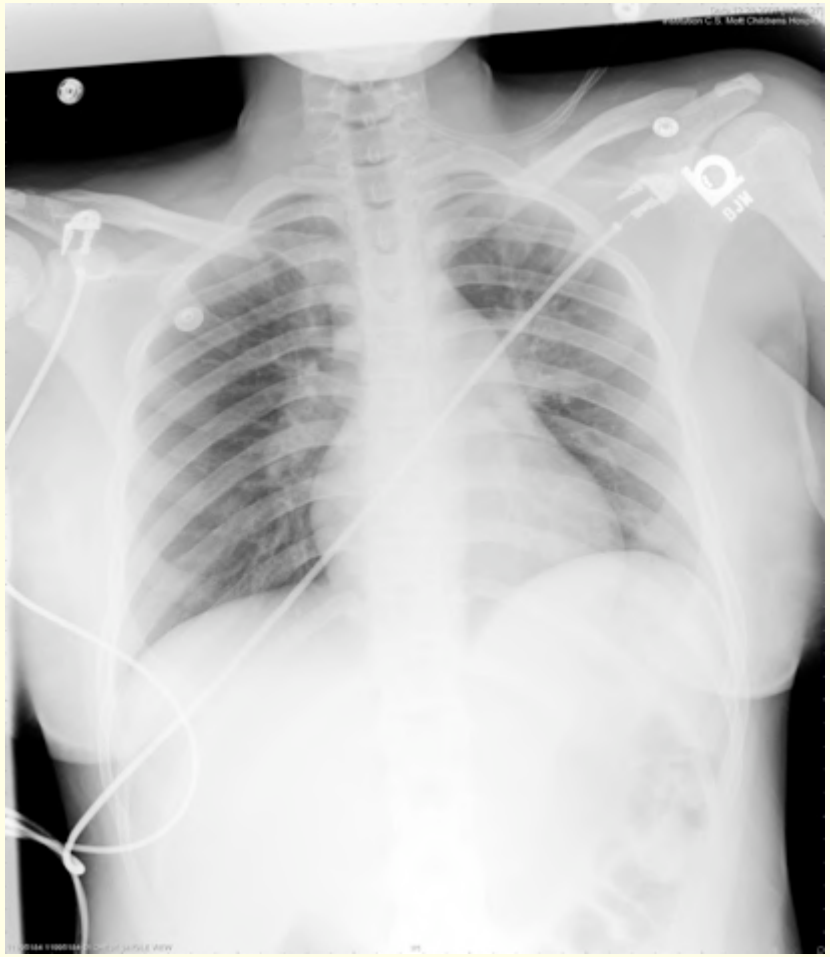
CASE KA from initial presentation

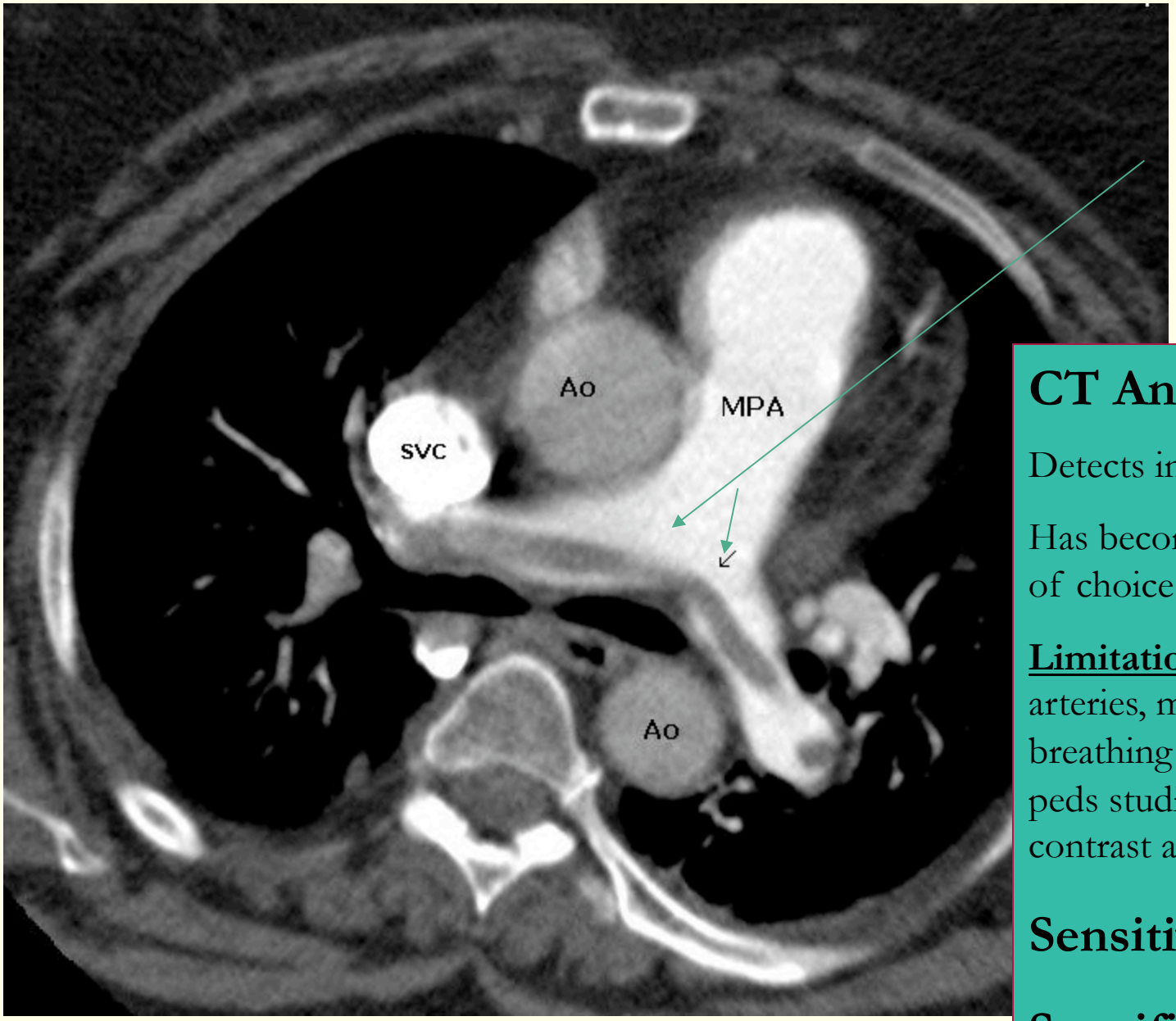


Evaluation of PE

- DVT : Looking for a source
  - Duplex US: Good for lower extremities
    - Detects echogenic thrombi
    - Absence of flow
    - Non-compressibility of veins
    - Used in series to detect thrombus organization
    - Limitations: Not useful for pelvic DVT, thoracic inlet (vessels beneath the clavicles or within the chest); ok for jugular vein clots versus venography.

V/Q Scan evaluation for children with suspected PE

- **V/Q scan primary** screening tool **for PE in children**
- **Safe, sensitive and relatively low radiation**
- **Limited to children beyond infancy/toddlers who can cooperate**
- **Results:**
  - High Probability
  - Intermediate Probability
  - Low Probability
  - Very Low Probability
  - Normal
- **Limitations in interpretation:**
  - Poor inter-observer agreement
  - Underlying pulm pathology
  - Use in CHD with R-L shunt
  - Sensitivity/specificity using PIOPED criteria for V/Q studies in children are not clear



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Saddle type pulmonary embolus

CT Angio

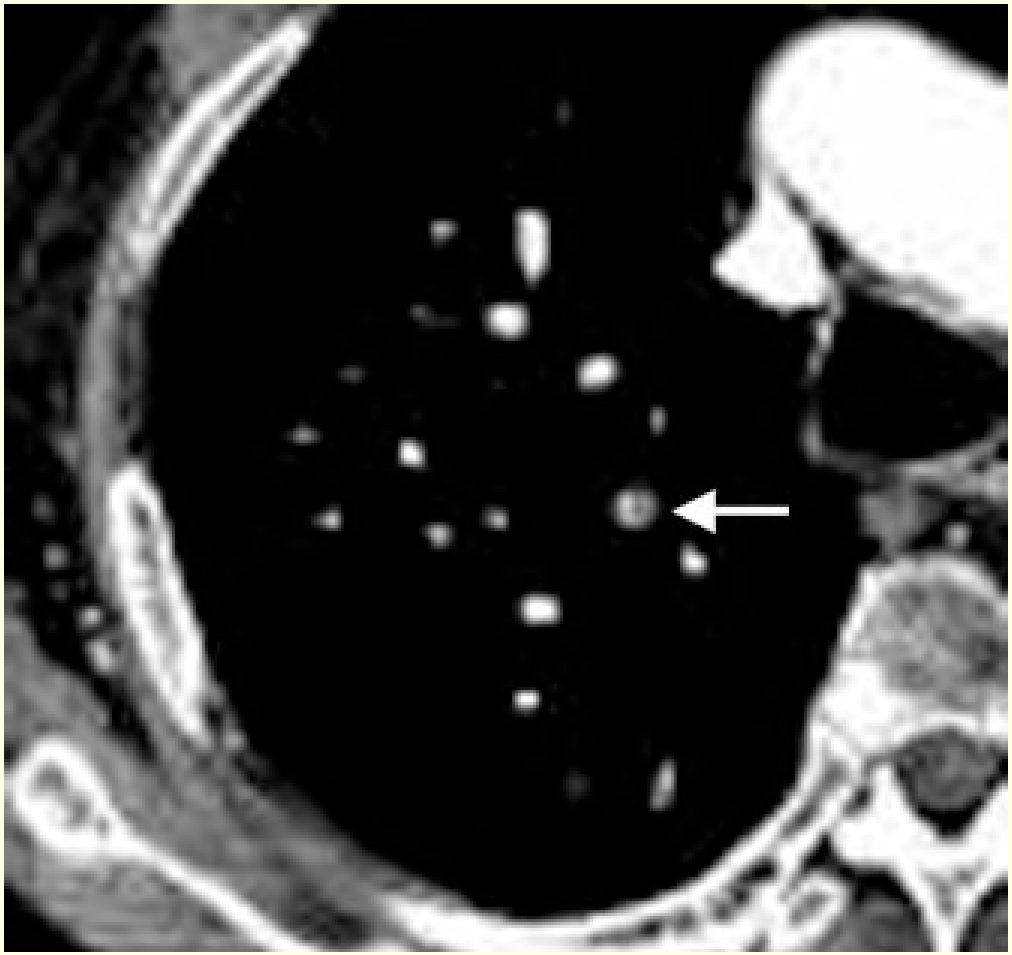
Detects intra-luminal defects

Has become the imaging test of choice in adults BUT

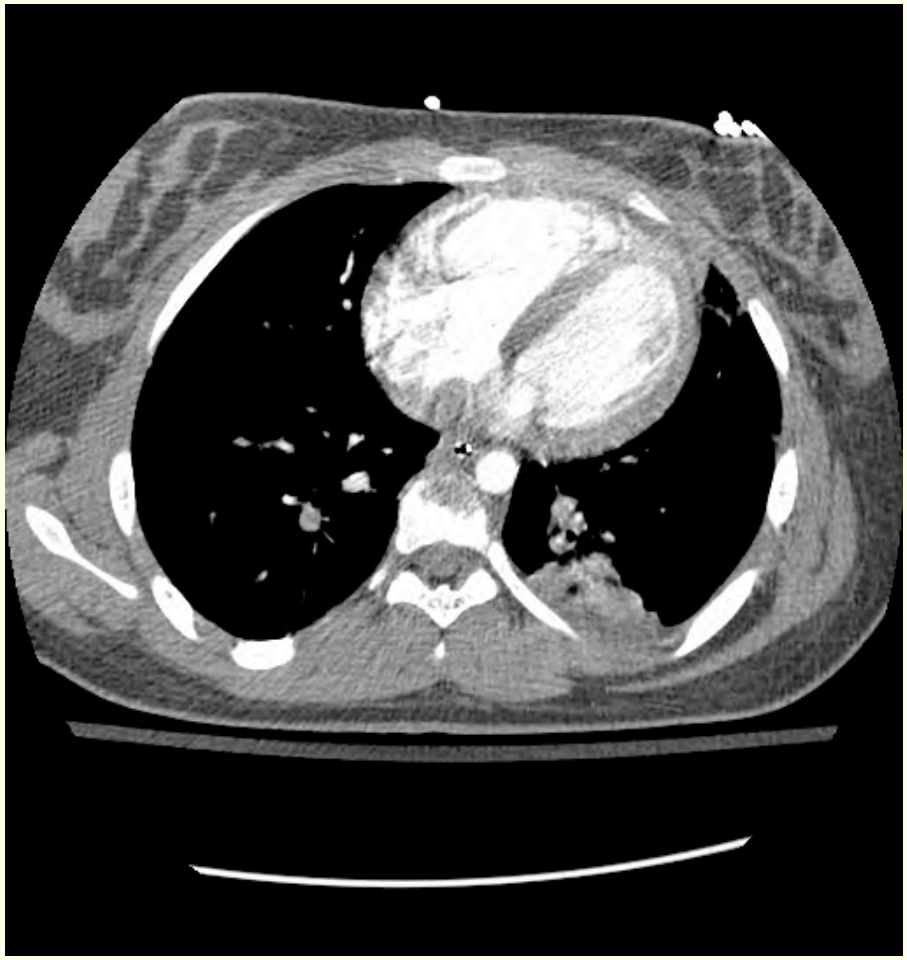
Limitations sub-segmental arteries, movement or breathing that might occur in peds studies, requires Iodine contrast and **radiation.**

Sensitivity 50-100%

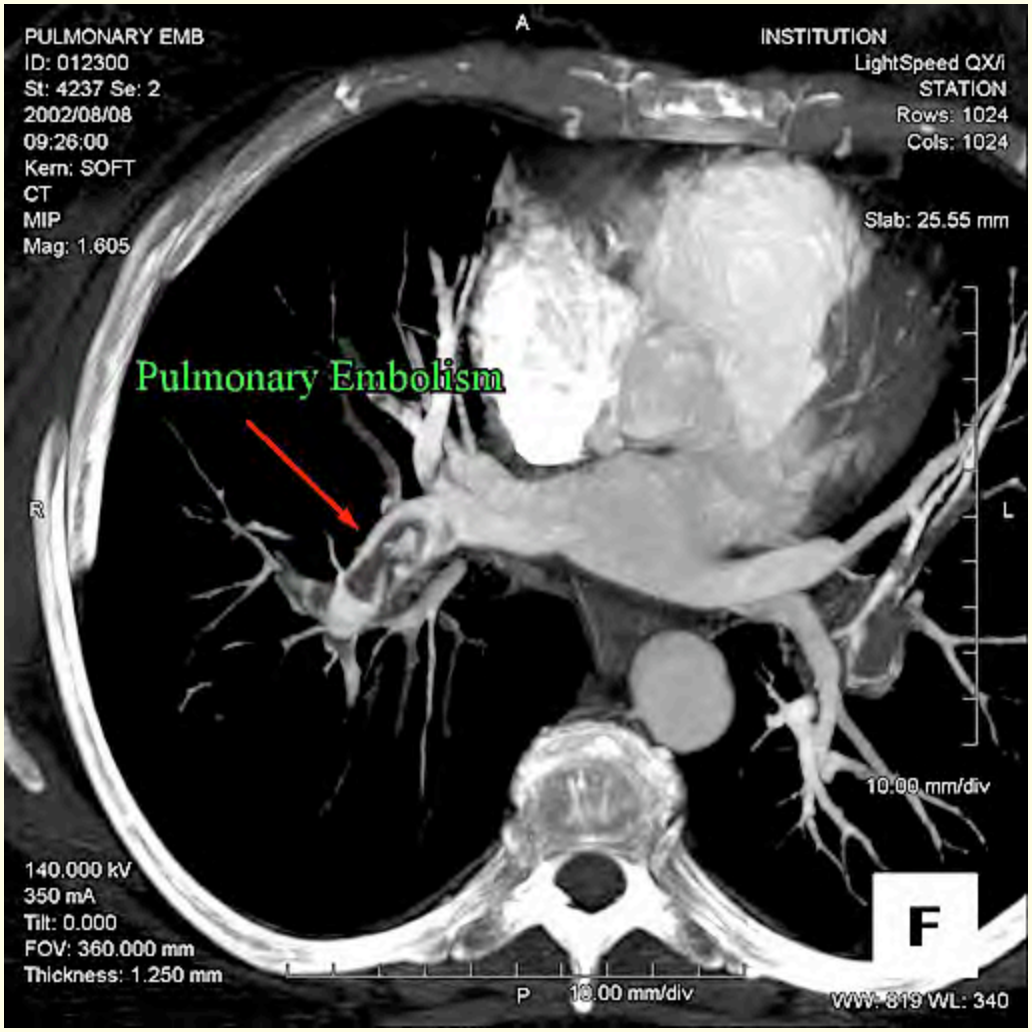
Specificity 81-100%



Case KA from initial presentation



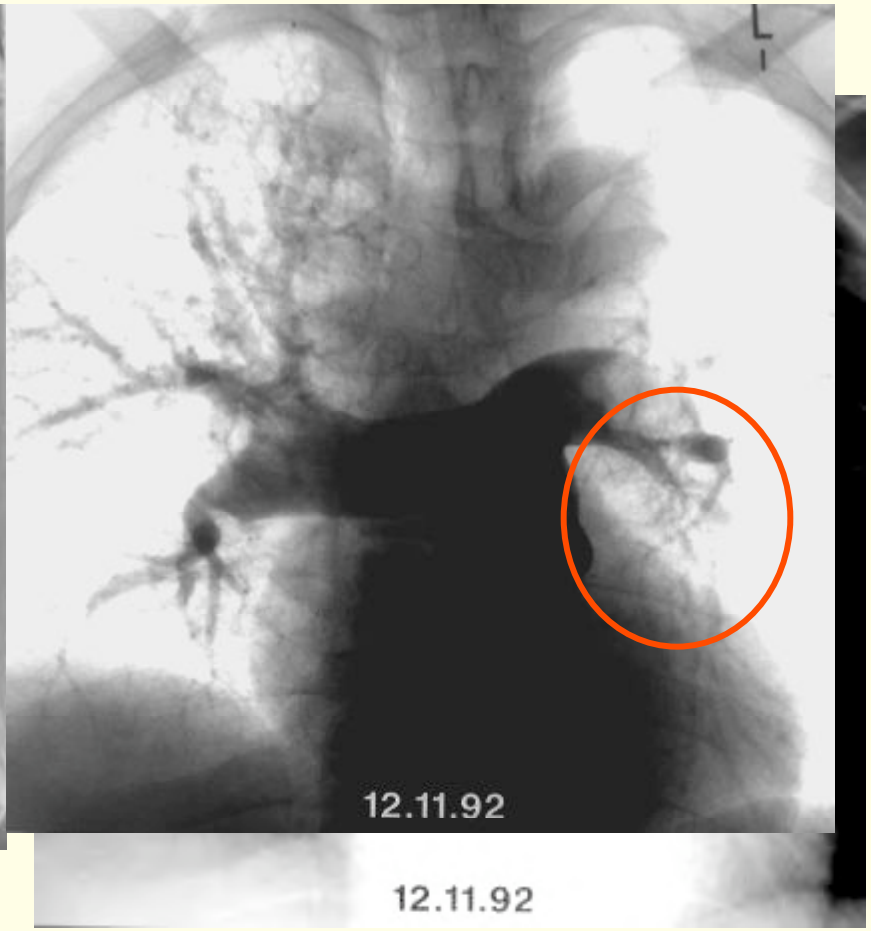
MRI evaluation for PE



- MRI
  - Recent ability to visualize the pulmonary arteries
  - Limitations
    - Acutely ill patients
    - Long test times
    - Patient monitoring
    - Subsegmental PE's
    - Sedation requirement in kids
    - Availability of resources
    - Sensit 68-88%
    - Specificity > 95%

Pulmonary Angiogram

PD-INEL



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PE Management

ABC's

Oxygen

Airway support

If intubation required, beware of hypotension

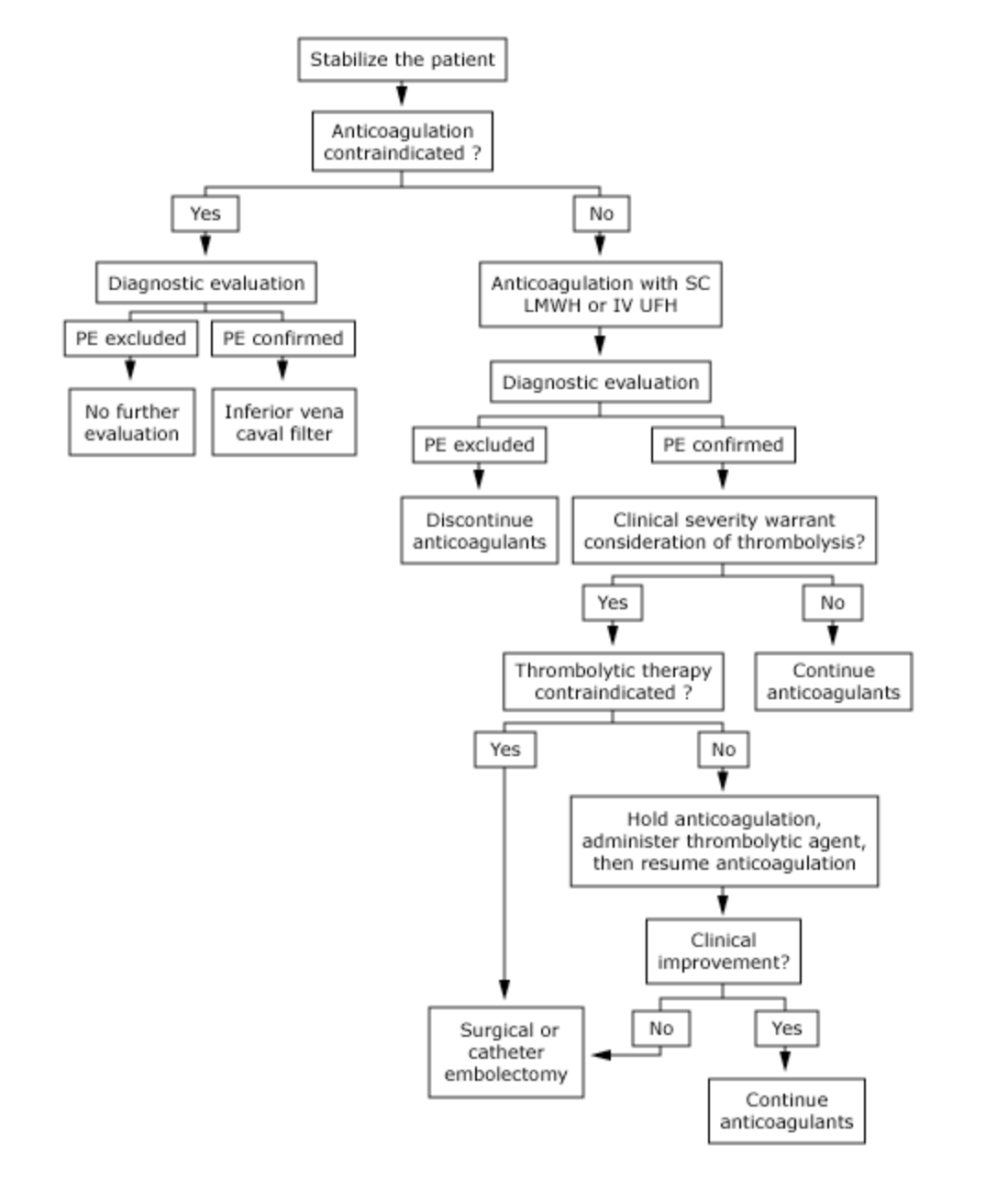
IV access

RV strain in PE necessitates CAREFUL fluid admin

Early vasopressors (norepi) if BP low/ unresponsive to judicious fluids

Anticoagulation: Heparin

Other options: Thrombolytics, Filter, Embolectomy, Surgical embolectomy



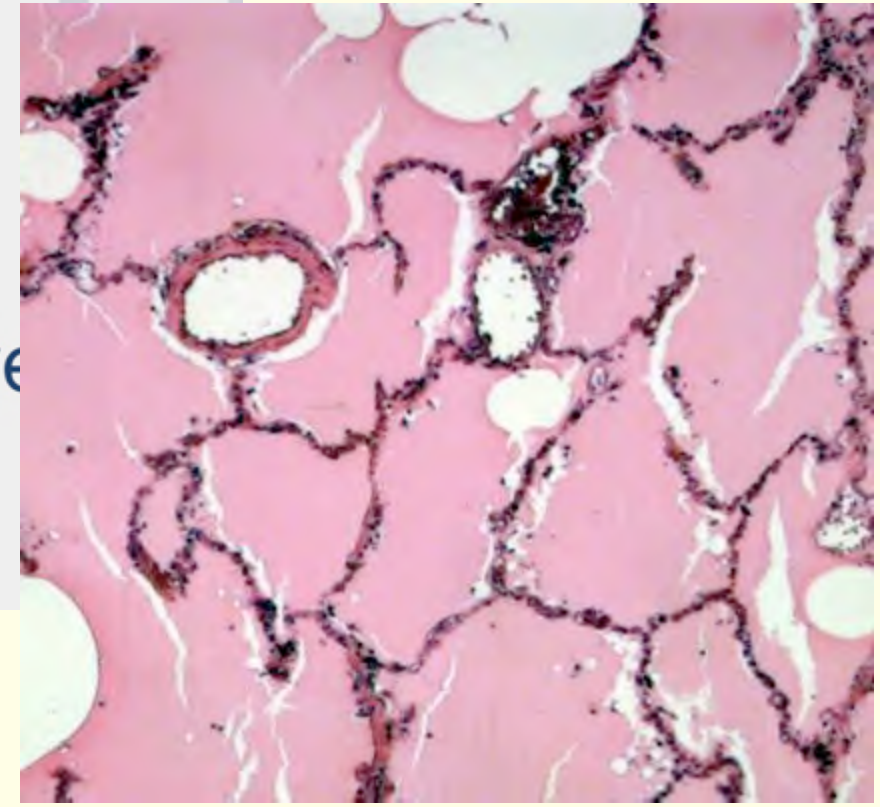
Pulmonary Embolism: Treatment

- **IV Heparin vs Low Molecular Weight Heparin (LMWH)**
- **IV Un-fractionated (UF) Heparin: Hypotension, massive PE, RF**
  - 75-100 units/kg bolus over 10 minutes
  - Infusion 20 units/kg/h
  - Maintain Prothrombin time (PTT) 60-85 seconds
  - Oral or LMWH follow up to Heparin
- **LMWH Dosing: For hemodyanamically stable pts**
  - **Enoxaparin**
    - > 2mo/age: 1mg/kg SQ BID
    - < 2mo/age: 1.5 mg/kg SQ daily
  - **Reviparin**
    - > 5kg: 100 U/kg SQ BID
    - < 5kg: 150 U/Kg SQ BID

Pediatric Pulmonary Edema

image remove

Anatomical drawing of Pulmonary Edema removed.



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Drawing of alveoli in pulmonary edema removed.

Pulmonary Edema Pathophysiology 101

Net filtration = (Lp x S) x (hydraulic pressure — oncotic pressure)

Pulmonary Edema in children

- Clinical presentation
  - Poor feeding/poor weight gain
  - Tachypnea/Dyspnea/grunting
  - Tachycardia
  - Cough
- Evaluation
  - History & Exam: pallor, diaphoresis, Inc RR
  - CXR
  - EKG
  - Other monitors as indicated: Pulmonary Artery Catheterization

Pediatric Pulmonary Edema

- Negative pressure pulmonary edema
  - Post obstructive pulmonary edema (POPE)
- Non Cardiogenic pulmonary edema
- Cardiogenic pulmonary edema

CXR Findings in pulmonary edema

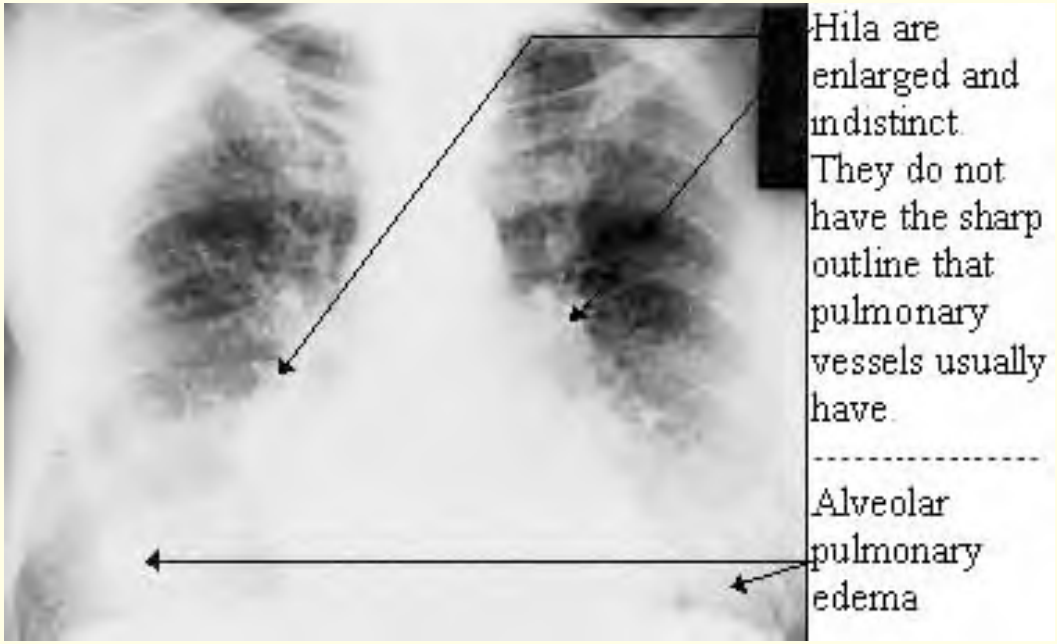
- Increased heart size -- cardiothoracic ratio >0.50.
- Large hila with indistinct margins
- Prominence of superior pulmonary veins; cephalization of flow
- Fluid in interlobar fissures
- Pleural effusion
- Kerley B lines
- Alveolar edema
- Peribronchial cuffing
- Limitations:
  - Edema may not be visible until amount of lung water increases by 30% or more
  - Edema produces similar radiographic findings as other materials that may fill the alveoli (pus, blood etc).

Table 1. Radiographic Features That May Help to Differentiate Cardiogenic from Noncardiogenic Pulmonary Edema.*

Radiographic Feature	Cardiogenic Edema	Noncardiogenic Edema
Heart size	Normal or greater than normal	Usually normal
Width of the vascular pedicle†	Normal or greater than normal	Usually normal or less than normal
Vascular distribution	Balanced or inverted	Normal or balanced
Distribution of edema	Even or central	Patchy or peripheral
Pleural effusions	Present	Not usually present
Peribronchial cuffing	Present	Not usually present
Septal lines	Present	Not usually present
Air bronchograms	Not usually present	Usually present

* Data are from Milne et al.[28] and Aberle et al.[31]

† The width of the vascular pedicle is determined by dropping a perpendicular line from the point at which the left subclavian artery exits the aortic arch and measuring across to the point at which the superior vena cava crosses the right mainstem bronchus. A vascular-pedicle width greater than 70 mm on a portable digital anteroposterior radiograph of the chest when the patient is supine is optimal for differentiating high from normal-to-low intravascular volume.[32]



Example X-Ray of Kerley B Lines

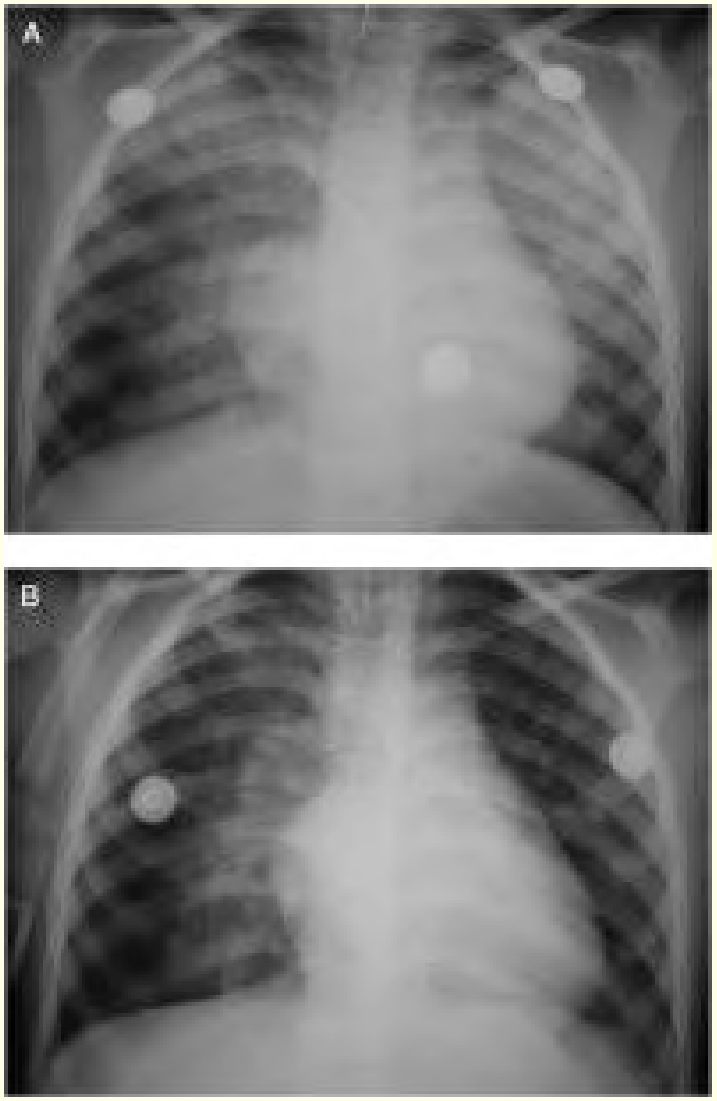
Kerley B lines are caused by peri-vascular edema, with a base on the pleural surface of the lung and extending horizontally a variable, but usually short, distance toward the center of the chest.

Correlation of CXR with Pulmonary Capillary Wedge Pressure

- Pulmonary Cap Wedge Pressure and CXR Findings:
  - 5-12 mmHg Normal
  - 12-17 mmHg Cephalization of pulm vessels
  - 17-20 mmHg Kerley lines
  - > 25 mmHg Frank pulmonary edema

Negative Pressure Pulmonary Edema

- Etiology
  - Can be associated with any upper airway obstruction
    - Croup, epiglotitis, FB, post op T & A, tumor, hanging, intubation for non airway procedures
- Clinical Presentation
  - Rapid onset, short lived course
  - Pulmonary edema occurs once obstruction is relieved
  - SOB, cough (frothy pink fluid)
- Treatment
  - Most require ETI/CPAP/PEEP
  - Diuretics, Inotropic support and invasive hemodynamic monitoring is usually not needed if dx is clear
  - Sx usually resolve in 12-24 hours



A case of negative pressure pulmonary edema

A: Acute pulmonary edema

B: Resolving pulmonary edema

Large negative intrapleural pressure

▼

Increase lymph flow and interstitial edema

▼

Pulmonary edema

Cardiac Effects: Neg intrapleural pressures also increase venous return to rt heart and pooling of bloood in the pulmonary venous system during inspiration. Increased VR to rt ventricle causes pressure on LV (reduced LV compliance)

Post obstructive pulmonary edema (POPE)

Type I POPE

- **Postextubation laryngospasm**
- **Epiglottitis**
- **Croup**
- **Choking/foreign body**
- **Strangulation**
- **Hanging**
- **Endotracheal tube obstruction**
- **Laryngeal tumor**
- **Goiter**
- **Mononucleosis**
- **Postoperative vocal cord paralysis**
- **Migration of Foley catheter balloon used to tamponade epistaxis**
- **Near drowningIntraoperative direct suctioning of endotracheal tube adapter**

Type II POPE

Post-tonsillectomy/ adenoidectomy

Post-removal of upper airway tumor

Choanal stenosis

Hypertrophic redundant uvula

Cardiogenic pulmonary edema

- Usually due to congenital heart disease
  - Left to Rt shunt lesions (PDA, VSD)
  - LV filling/emptying defects (Aortic Stenosis)
  - Total Anomalous Pulmonary Veins (TAPV) (obstruct emptying of pulm veins)
- Arrhythmia
- Cardiomyopathy
- Pneumonia/pulmonary infection
- High output states
- Iatrogenic

Pediatric Cardiogenic Pulmonary Edema: Etiology by presentation time

- First week of life
  - Ductal dependant congenital heart lesions, (pre ductal coarctation) and lesions causing pulmonary venous obstruction to vent filling (cor triatriatum)
- 2-4 weeks of life
  - Left to right shunting lesions (VSD) as pulmonary vascular resistance decreases
- > 6 months of life
  - Usually specific diagnosis

Non Cardiogenic Pulmonary Edema

- Definition:
  - Noncardiogenic pulmonary edema is defined as the radiographic evidence of alveolar fluid accumulation without hemodynamic evidence to suggest a cardiogenic etiology (ie, pulmonary wedge pressure 18 mmHg). The accumulation of fluid and protein in the alveolar space leads to decreased diffusing capacity, hypoxemia, and shortness of breath.
- Most common Cause:
  - ARDS
  - High Altitiude pulmonary edema
  - Neurologic pulmonary edema
  - Reperfusion pulmonary edema
  - Reexpansion pulmonary edema

Pulmonary edema and ARDS

- Damaged alveolar capillary membrane (permeability pulmonary edema)
- Allows leakage of fluid and protein from intravascular to interstitial and ultimately into alveolar spaces
- Presentation:
  - SOB
  - Pulm infiltrates/hypoxemia
- Etiology: Many
- DX: pulmonary artery wedge pressure less than 18 mmHg favors acute lung injury (> 18mmHg doesn't always exclude lung etiology). Other: plasma brain natriuretic peptide (BNP) high in cardiogenic causes.

Non Cardiogenic Pulm Edema

- Treatment
  - No known treatment to correct capillary permeability
  - Supportive measures while lung recovers
    - Airway/ventilatory management
    - Nutrition
    - Fluid: Diuresis/fluid restriction improve lung function and positively affect patient outcome
    - Cardiac management
    - Others: Prostacycline, nitrous oxide, steroids, beta agonists
  - Surfactant

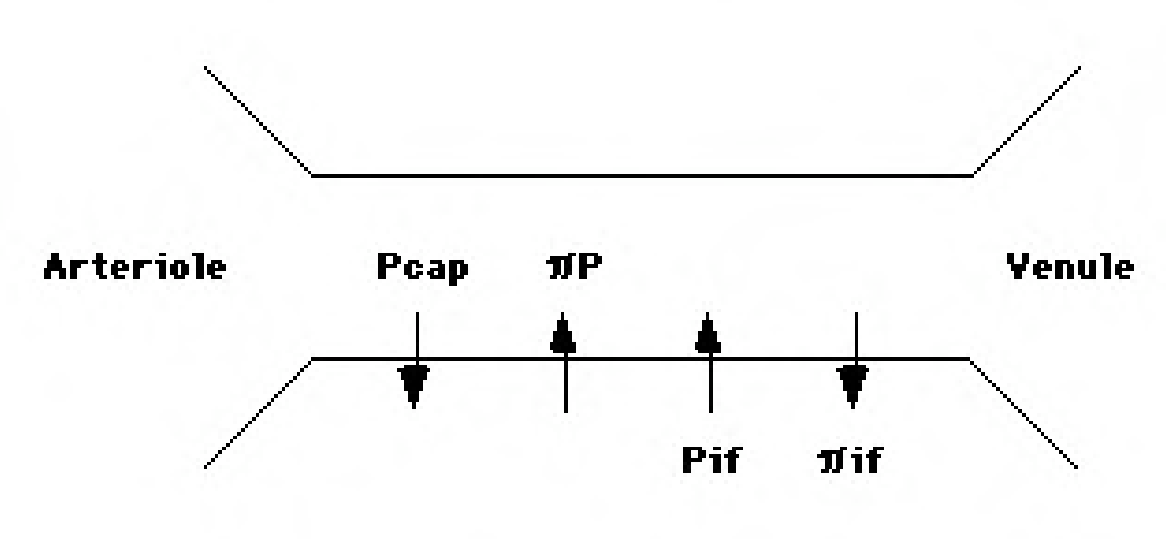
JAMA. 2005;293:470-476. **Effect of Exogenous Surfactant (Calfactant) in Pediatric Acute Lung Injury (ALI)**

Neurologic Pulmonary Edema

- Exact cause unknown
- Medulla/nuclei of solitary tract/hypothalamus
- CNS conditions associated with Neuro pulm edema:
  - Trauma
  - Infection
  - Seizure
  - Cervical spine injuries
- Clinical: Onset of SOB minutes/hours after neurologic insult
- DX: Setting, Hemodynamic measurements, including blood pressure, cardiac output, and pulmonary capillary wedge pressure are normal.

Neurologic Pulm Edema: Lab theories

- Pulmonary venoconstriction can occur with intracranial hypertension or sympathetic stimulation and can elevate capillary hydrostatic pressure and produce pulmonary edema without affecting left atrial, systemic, or pulmonary capillary wedge pressures. Constriction of the pulmonary veins of rats follows head trauma, and can be attenuated with alpha adrenergic antagonists



Neurologic Pulm Edema

- Treatment
  - Airway support
  - Alpha adrenergic drugs
  - Beta adrenergic antagonists

High Altitude Pulmonary Edema (HAPE)

- More kids are going places!
- Rapid ascension to > 12,000 feet
- Some children are more at risk:
  - Downs
  - Kids who LIVE at altitude…go to lower areas then reascend. Children more predisposed to reascent HAPE
- Pathophysiology:
  - Accentuated hypoxemia
  - abnormally pronounced degree of hypoxic pulmonary vasoconstriction
  - Release of mediators
  - Leaky endothelium?

HAPE

- Clinical presentation
  - Variable: Hours, days, explosive onset
  - SOB, cough, sputum (frothy, pink)
- RX:
  - Oxygen
  - Descent
  - Bedrest
  - Dexamethesone for emergencies
  - Hyperbaric chambers for rescue removals
  - Education: Slow descents
  - Prevention: Nifedipine

Emergency Department Therapy of Acute Pulmonary Edema in Children

- Assessment
  - Etiology: Likely cardiac vs non cardiac?
- Oxygen
- CXR/Exam: Determination of pump status
- Diuresis
- Inotropic support?
- Directed evaluation

Parapneumonic Effusions

- Pleural effusion associated with lung infection
  - Infection may (rarely) be spread from remote places: retropharyngeal, abd, vertebral, retroperitoneal spaces to pleura
  - Pleural inflammation—leak
    - Proteins
    - Fluid
    - WBC's
  - Initially sterile—subsequently may become infected=EMPYEMA
    - Presence of grossly purulent fluid in pleural cavity

Parapneumonic Effusions

- Increasing incidence
  - USA & UK
  - Li et al. Pediatrics 2010
  - Roxburgh et al. Arch Dis Child 2008
- Rise coincident with rise in antibiotic resistance, despite pneumococcal vaccine: serotypes not covered?
- Mortality highest children < 2y/o
- Spring/Winter 2X greater than summer/fall
- Male = Females

Changing Etiology

- Before 1945: Pneumococci/Staph
- After PCN/Sulfa: Staph aureus
- 1980's: H. influenza/Pneumococci/Staph
- 1990's: H. flu disappears! (except adults)
- 1980's and up: increase in Bacteroides, Fusobacterium
- Now: St. pneumonia w resistance patterns (pcn non susceptable) and or pcn susceptable strains in certain communities
- MRSA
- Coag (-) St. aureus, Strep viridans, Grp A strep, Alpha hemolytic strep, Actinomyces species.
- GRP A Strep, TSS and Empyema?

Stages of Parapneumonic Effusion

- Exudative
  - Normal Glucose
  - Normal ph
  - Low cell count
- Fibrinopurulent
  - PMN invasion
  - Bacterial/fibrin deposition on pleura
  - Thickened exudate and loculations
  - pH/glucose=decrease
  - LDH = increase
  - Don’t layer out on xray
  - Lasts 7-10 days

Stages of Parapneumonic Effusion

- Organizational
  - Pleural Peel formation
    - Fibroblasts grow on parietal/visceral pleura
    - Restricts lung reexpansion
    - Impairs function
    - Persistent pleural space
    - Dry tap

Complications

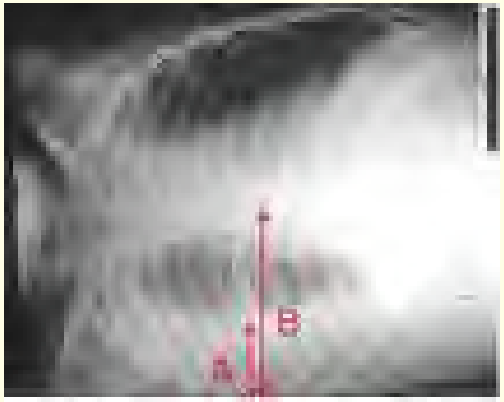
- Infrequent in children
  - Bronchopleural fistula
  - Lung abcess
  - Empyema necessitatis (perforation thru chest wall)

Clinical presentation

- Fever
- Malaise
- Low appetite
- Cough
- Chest pain
- Dyspnea/Tachypnea: Shallow to minimize pain
- Splint side
- Not usually toxic appearing
- Rarely present as septic shock

Clinical Presentation

- Mediastinal shift
- Hypoalbinemia
- Thromobcytosis
- Hypoxia
- Radiology:
  - Plain films
  - Decubitus films
  - Ultrasound
  - Chest CT



Effusion Analysis

- Layering > 1cm: Easier target
- Who to tap: Better to find the organism
- If small and abx already begun, reasonable to wait/see response to abx
- Thoracentesis w/w/out US guidance
- Dry tap: Consider sterile 5-10cc fluid/reaspirate
  - Cx
  - Pneumococcal Antigen Latex agglutination and PCR
  - Sensitivity/specificity 90/95% respectively
  - pH, glucose, LDH, cell count & differential
  - Specimens must be on ice and tightly capped
- Other tests: Blood cx in all pts
- Misc if indicated: Sputum/tracheal asp, TB, Titers (mycoplasma, ASO, Resp viruses), CBC, CRP, sLDH (to compare to pl sample)

ED EVAL

- ABC's
- Assessment for effects on respiratory status
- RR, pulse ox, VBG and or ABG
- CXR (decubitus?)
- US
- To tap or not to tap
- Treatment:
  - Directed at most likely etiology
  - Goals: Sterilize the pleural space, drain as necessary, reexpand the lung

Hospitalization & Surgical issues

- Most will require hospitalization
- Surgical intervention is controversial
  - Drain and debride (VATS) Video Assisted Thorocostomy versus
- Fibrinolytic therapy urokinase, streptokinase, and alteplase (tissue plasminogen activator, tPA). PLUS Chest tube
- Outcome in children w normal lungs is excellent
  - Early VATS decreases hosp stay and drainage
  - Outcome similar VATS vs Fibrinolytic Rx

One approach

- Drain acutely (no more than 10-20cc/kg) then observe (w Abx) w/o chest tube
- If reaccumulates---VATS/w chest tube
  - (Texas Childrens)

- Antibiotics (IV until resolution of fever):
  - Clindamycin alone
  - Clindamycin + Cefotaxime
  - Life Threatening: Vanco + Cefotaxime

Who Gets a Chest Tube?

- Large amounts of free flowing pleural fluid
- Evidence of fibrinopurulent effusions (eg, pH <7.0, glucose <40 mg/dL [2.22 mmol/L], LDH >1000 IU [16.67 kat/L], positive gram stain, frank pus)
- Failure to respond to 48 to 72 hours of antibiotic therapy
- Compromised pulmonary function (eg, severe hypoxemia, hypercapnia)

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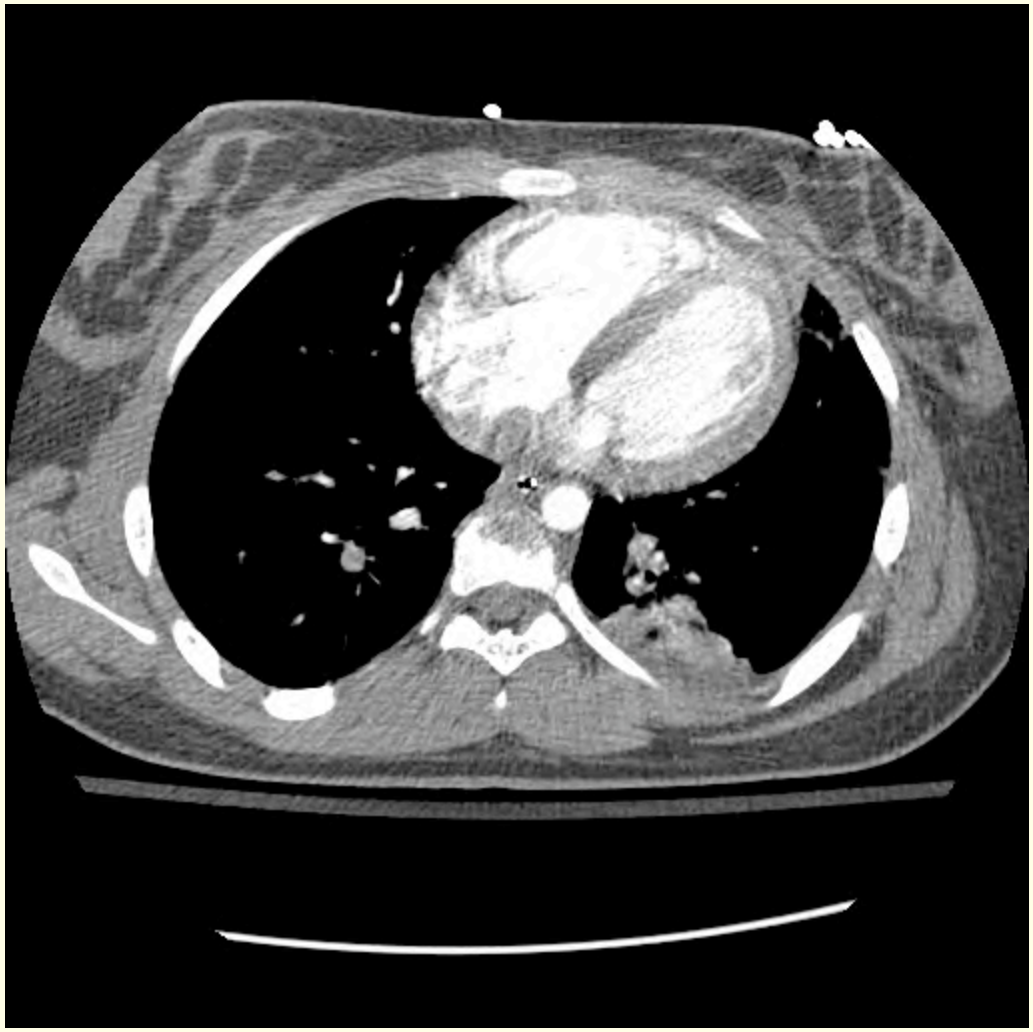

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Slide 7, Image 2: Please see original image of DVT/Pulmonary Embolism at http://www.activeforever.com/t-deep-vein-thrombosis-article.aspx

Slide 8, Image 1: Please see original image of Pulmonary Embolism at http://www.riversideonline.com/source/images/image_popup/r7_pulmonaryembolism.jpg

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Slide 38, Image 1: Anatomical drawing of Pulmonary Edema removed.

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