Current Approach to Radiologic Evaluation of Suspected Pulmonary Embolism

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Agenda

• Background of Pulmonary Embolism
• Menu of tests
• Index patient - A.B.
• Companion patients
• Conclusions
Background: Definition

- Pulmonary Embolism (PE) refers to obstruction of the pulmonary artery (or one of its branches) by material (thrombus, air, fat, or tumor) originating from elsewhere in the body
**Pathophysiology: Embolus course**

- Deep proximal lower-extremity veins are the most common site of thrombus formation leading to PE
- Thrombus travels through the inferior vena cava to the right atria, right ventricle, and enters the pulmonary arterial system, where it can cause obstruction


Diagram of embolus course
Pathophysiology: Venous thrombus development

- Risk factors for the development of PE are related to Virchow’s Triad: (1) endothelial injury, (2) venous stasis, (3) blood hypercoagulability
- Conditions associated with increased risk of thrombus formation
  - Primary (genetic): mutations in factor V Leiden, Protein C or S deficiency, Antithrombin III deficiency
  - Secondary (acquired): prolonged bed rest/immobilization, surgery, lower extremity trauma, malignancy, DIC, systemic lupus erythematosus, oral contraceptive pills, stroke, smoking, pregnancy/post-partum state
Pathophysiology: Consequences of pulmonary embolism

• PE causes obstruction of flow and vasospasm from platelet release of vasoactive agents (serotonin, thromboxane A2), causing increased pulmonary vascular resistance

• Increased pulmonary vascular resistance causes right ventricular strain, which can lead to RV dilation, dysfunction, and possibly death from right heart failure

• Mortality rate approaches 30% without treatment, usually due to recurrent embolism

• Mortality is reduced to 2-8% with appropriate anticoagulation
Typical symptoms and signs of Pulmonary Embolism

• Presentation is variable and nonspecific
• Symptoms include:
  – Acute-onset dyspnea
  – Pleuritic chest pain
  – Cough
  – Hemoptysis
• Signs include:
  – Tachycardia
  – Tachypnea
  – Hypoxia
  – Signs of right ventricular dysfunction (bulging neck veins, left parasternal lift, accentuated pulmonic component of second heart sound)
Laboratory studies and ECG changes associated with Pulmonary Embolism

- Labs may demonstrate elevated serum D-dimer (sensitive, nonspecific)
- ECG frequently demonstrates tachycardia; may also see T-wave inversions in anterior leads (V1-V4) or S1Q3T3
• We have reviewed the pathophysiology of PE as well as the typical history, physical exam findings, lab abnormalities, and ECG changes.
• These findings are variable and non-specific.
• Therefore, radiologic studies are critical to aid in the diagnosis so that appropriate treatment can be initiated!
Menu of Radiologic Tests

- Chest X-ray
- Chest CT (CT angiogram)
- V/Q scan
- Venous ultrasound
- Less commonly-used studies
  - Pulmonary Angiography
  - Echocardiography
  - MR Angiogram
Chest X-ray: Introduction

- 1st imaging study used to evaluate new-onset dyspnea, pleuritic chest pain, cough
- PA and lateral views are obtained
- CXR is ideal to help identify alternative diagnoses for acute symptoms (e.g. pneumonia, pulmonary edema), possibly avoiding additional studies
Chest X-ray: Findings in Pulmonary Embolism

- Findings in PE are nonspecific, ranging from normal to abnormal. Example findings include:
  - Atelectasis
  - Pleural effusion
  - Parenchymal opacities
- Classic PE abnormalities:
  - Westermark sign (central pulmonary artery prominence with decreased pulmonary markings more peripherally)
  - Hampton’s hump (peripheral wedge-shaped opacity)
- Remember: A patient with hypoxemia and dyspnea with a normal CXR has a PE until proven otherwise.
Chest CT: Introduction

- CT angiogram (CTA), CT Pulmonary Angiography (CTPA), Multidetector CT (MDCT), and Helical CT are CT studies with IV contrast used to evaluate for the lungs for suspected PE.
- CTPA has become the imaging study of choice to diagnose PE.
- Sensitivity of 83-99% and specificity 89-98% for PE.
- Findings: emboli demonstrated as intraluminal filling defects (areas of low attenuation) in pulmonary artery branches.
Chest CT: Advantages and disadvantages

• Advantages:
  – Fast
  – Accurate
  – Allows for direct visualization of thrombus as well as other lung abnormalities that may account for symptoms (e.g. pneumonia, pulmonary masses)
  – High interobserver agreement

• Disadvantages: radiation exposure, limited by contraindications to IV contrast administration (e.g. contrast allergy or renal insufficiency)
V/Q Scan: Introduction

- Previously the imaging study of choice, now largely replaced by CT
- Nuclear Medicine study, sensitive for PE
- Ventilation (V) scan: Tc-99m labeled microaerosol agents are inhaled via a nebulizer and deposit on bronchoalveolar lining, demonstrating areas of ventilated lung.
- Perfusion (Q) scan: Tc-99m labeled albumin is injected, which lodge in precapillary arterioles, demonstrating areas of perfused lung.
V/Q Scan: Interpretation

• Need baseline CXR to evaluate underlying lung disease to aid in interpretation of confusing scans
• Areas of mismatch, i.e. abnormal perfusion with normal ventilation strongly suggests PE
• Matched defects suggest underlying lung disease
• A study can be classified as normal, low probability, intermediate probability, and high probability of PE
• A normal V/Q scan excludes PE; a high probability scan combined with a high clinical probability of PE= 95% chance of PE
• Limitations:
  – Many studies are nondiagnostic
  – It is challenging to evaluate scans in patients with underlying airway disease (e.g. COPD)
V/Q Scan: Possible future directions

• Studies using a hybrid gamma-camera/Multidetector CT system to conduct *simultaneous* V/Q SPECT (single photon emission computed tomography) and MDCT angiography to evaluate for PE have been conducted with good results
  – Results in improved sensitivity and specificity
  – Uses low-dose CT, decreasing radiation exposure from CT
  – A non-contrast study, avoiding contraindications to IV contrast

• Use in clinical practice remains to be seen
Venous Ultrasound

- In patients who remain undiagnosed, venous U/S may prove useful to diagnose PE
- Most patients with PE have concomitant DVT
- A DVT identified on Doppler U/S with venous compression increases suspicion for PE
- Limitations: Negative U/S does not exclude PE
Echocardiography

- Not usually used in workup of suspected PE
- Can help evaluate the risk of right heart failure in patient with massive PE
- Findings may include increased right ventricle (RV) size, decreased RV function, RV thrombus
- Cannot exclude PE
MR Angiogram

- Similar to CTA, MR angiogram (MRA) provides a noninvasive evaluation of pulmonary arteries.
- Not commonly used due to long examination time, high cost, and technological limitations.
- In some institutions, occasionally used in patients who have contraindications to contrast administration.
- May have expanded role in the future with technological advances.
Pulmonary Angiography

- Pulmonary angiography (PA) was previously the “gold standard” for diagnosing PE
- Rarely performed today due to advances in detection on CT
- Current role remains unclear-- possibly for use in those patients who remain undiagnosed with less invasive methods who require definitive diagnosis
Summary of Radiologic algorithms used to evaluate for Pulmonary Embolism

• Previous radiologic algorithm for evaluating PE: 1) CXR  2) V/Q scan  3) PA
• Current algorithm: 1) CXR  2) CTPA  3) V/Q scan if CT is equivocal or there are contraindications to CT  4) Venous U/S, Echo, MRA, PA as appropriate to clinical situation if diagnose not established
• Future technologies may combine imaging modalities, such as simultaneous V/Q scan with CT to improve the sensitivity and specificity
We have reviewed the menu of radiologic tests available to diagnose PE and are ready to apply this knowledge to patients.
Our Patient A.B.: Clinical presentation

- 48 year old woman with no PMH presents with 6 days of right lower extremity pain and swelling and dyspnea on exertion
- History of recent plane travel; no other immediately identifiable risk factors for PE
- Physical exam:
  - BP 120/66, Pulse 100 at rest (120 with ambulation), RR 16, O2 sat 97% on room air (at rest and with ambulation)
  - No acute distress
  - Normal heart and lung exam
  - Right lower extremity with non-pitting edema and tenderness to palpation
- ECG demonstrated sinus tachycardia
- DVT was identified by venous ultrasound at an outside hospital
- CT Angiogram was performed on suspicion of PE
Our Patient A.B.: corresponding axial and coronal views on CTA

Axial C+ CTPA

Coronal C+ CTPA

Do you notice any abnormalities?
Our Patient A.B.: Thrombus on CTA

There is evidence of bilateral thrombi!

Axial C+ CTPA

Review your anatomy on the following labeled CT axial slice.
Our Patient A.B.: Anatomy

Axial C+ CTPA

RA- Right atrium, RV- Right ventricle, LV- Left ventricle, AA- Ascending aorta, DA- Descending aorta, SVC- superior vena cava, PV- Pulmonary veins, AV- Azygous vein, *- THROMBI= low-attenuation filling defect in high attenuation vessels
Patient A.B.’s CTPA demonstrates multiple bilateral segmental and subsegmental filling defects. (Remember the right and left pulmonary arteries branch into lobar, segmental, and subsegmental arteries).
Our Patient A.B.: Evaluate ventricles on CTA

Is there evidence of right-heart strain?

Axial C+ CTPA
Our Patient A.B.: No evidence of right-heart strain on CTA

Normal right and left ventricle size. No interventricular septal bowing.

Axial C+ CTPA
Companion patient #1: Right-heart strain on CTA

Interventricular septum is bowing into the left ventricle.
Right ventricle > Left ventricle

Axial C+ CTPA
Companion patient #2 and #3: Railway sign on CTA

Axial C+ CTPA

Coronal C+ CTPA

High-attenuation contrast seen outlining thrombus, creating parallel lines, similar to railroad tracks in appearance.
Companion patient #4: Saddle embolus on CTA

Saddle embolus: large, low-attenuation filling defect seen straddling the contrast-enhanced right and left pulmonary arteries.
Companion patient #5: Corresponding findings on CXR and CTA

What is this finding called?
Hampton’s Hump!
Companion Patient #6: Hampton’s hump on CXR and CTA

Peripheral wedge-shaped opacity on CXR. On CTA, we see that the lung tissue downstream from the thrombus is infarcted, creating the CXR abnormality. This CXR was originally interpreted as a RLL pneumonia. Remember, an alveolar opacity on CXR can be due to: fluid, blood, protein, and pus.
Companion patient #7: Chronic PE on CTA

Chronic PE: eccentric, lining the vessel wall, with rounded edges. It is seen here as a high-attenuation defect due to calcifications.
Companion patient #8: Normal V/Q scan

The lung is uniform perfused and ventilated.

Multiple projections of V/Q scan
Companion patient #9: High probability of PE on V/Q scan

This patient was unable to do a CT with contrast due to worsening renal insufficiency.

Uniform ventilation

Hypoperfusion in RUL, RLL, LUL, most of LLL

Multiple projections of V/Q scan

Courtesy of Dr. Donohoe
Our Patient: A.B:

- Patient was treated with anticoagulation to prevent thrombus extension and recurrence
  - Low molecular weight heparin until INR was therapeutic on Coumadin
  - Depending on clinical presentation, treatment may include: thrombolytics, embolectomy, anticoagulation, IVC filter placement
- Optimal duration of anticoagulation is controversial for this patient, given relatively unprovoked (no risk factors) nature of DVT/PE
  - There is a higher risk of recurrence off of anticoagulation in unprovoked DVT/PE
- Patient went on to have age-appropriate malignancy screening and possible thrombophilia work-up
Conclusions

• PE can be life-threatening and the presentation is variable and non-specific.
• Radiologic imaging plays a KEY role in the diagnosis of PE!
• CTPA is the current imaging study of choice, with emboli presenting as intraluminal filling defects in the contrast enhanced pulmonary artery branches.
• Early detection and treatment, usually with anticoagulation, is key to reducing PE-associated mortality.
References

- Thompson, B., Hales, C. Diagnosis of acute pulmonary embolism. *UpToDate*. 2009 June.
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