Acute Pulmonary Embolism: Diagnosis and Treatment of Acute Pulmonary Embolism

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Why Care?

• PE is the most common preventable cause of death in hospitalized patients
• ~600,000 deaths/year
• 80% of pulmonary emboli occur without prior warning signs or symptoms
• 2/3 of deaths due to pulmonary emboli occur within 30 minutes of embolization
• Death due to massive PE is often immediate
• Diagnosis can be difficult
• Early treatment is highly effective
• YOU WILL TAKE CARE OF PATIENTS WITH PE!

Pathophysiology

Rudolph Virchow, 1858

Triad:
• Hypercoagulability
• Stasis to flow
• Vessel injury

Risk Factors

Hypercoagulability
- Malignancy
- Nonmalignant
- Pregnancy
- Postpartum status (<4 wk)
- Estrogen/OCP's
- Genetic mutations (Factor V Leiden, Protein C & S deficiency, Factor VIII, Prothrombin mutations, anti-thrombin III deficiency)

Venous Stasis
- Bedrest > 24 hr
- Recent cast or external fixator
- Long-distance travel or prolong automobile travel

Venous Injury
- Recent surgery
- Recent trauma (especially the lower extremities and pelvis)
Natural History of VTE

- 40-50% of pts with DVT develop PE, often “silent”
- PE presents 3-7 days after DVT
  - Fatal within 1 hour after onset of respiratory symptoms in 10%
  - Shock/persistent hypotension in 5-10% (up to 50% of patients with RV dysfunction)
- Most fatalities occur in untreated pts
- Perfusion defects completely resolve in 75% of all patients (who survive)

Diagnosis: Clinical Presentation

- Dyspnea, tachypnea, or pleuritic chest pain most common
  - Pleuritic pain = distal emboli → pulmonary infarction and pleural irritation
  - Isolated dyspnea of rapid onset= central PE with hemodynamic sequelae
  - Retrosternal angina = RV ischemia
- Syncope=rare presentation, but indicates severely reduced hemodynamic reserve
- Symptoms can develop over weeks
- In patients with pre-existing HF or COPD, worsening dyspnea may indicate PE

Diagnostic Test

- Imaging Studies
  - CXR
  - V/Q Scans
  - Spiral Chest CT
  - Pulmonary Angiography
  - Echocardiography
- Laboratory Analysis
  - CBC
  - D-Dimer
  - ABG’s
Diagnosis: Chest X-Ray

- Usually abnormal, but non-specific
- Study of 2,322 patients with PE:
  - Cardiac enlargement (27%)
  - Normal (24%)
  - Pleural effusion (23%)
  - Elevated hemidiaphragm (20%)
  - Pulmonary artery enlargement (19%)
  - Atelectasis (18%)
  - Parenchymal pulmonary infiltrates (17%)

Diagnosis Testing

Chest X-Ray Myth:

“You have to do a chest x-ray so you can find Hampton’s hump or a Westermark sign.”

Reality:

Most chest x-rays in patients with PE are nonspecific and insensitive

Chest X-ray Extra’s of PE

- Westermark’s Sign
  - A dilation of the pulmonary vessels proximal to the embolism along with collapse of distal vessels, sometimes with a sharp cutoff.
- Hampton’s Hump
  - A triangular or rounded pleural-based infiltrate with the apex toward the hilum, usually located adjacent to the hilum.

Radiology Findings in P.E.

V/Q Scan:

- Results: High, Intermediate, Low Probability
- Best if combined with Clinical Probability (PIOPED study):
  - High Clinical Prob + High Prob VQ= 95% likelihood of having a P.E.
  - Low Clinical Prob + Low Prob VQ= 4% likelihood of having a P.E.
CT Evidence of RV Dysfunction

- RV dilation
- RV/LV short axis >1= pulmonary hypertension
- RV/LV short axis >1.5= severe PE
- Leftward septal bowing

Diagnosis: ECG

- Usually non-specific ST/T waves changes and tachycardia
- RV strain patterns suggest severe PE
  - Inverted T waves V1-V4
  - QR in V1
  -Incomplete RBBB
  - S1Q3T3

Spiral CT

- Direct visualization of emboli.
- Both parenchymal and mediastinal structures can be evaluated.
- Offers differential diagnosis in 2/3 of cases with a negative scan.
BUT…
- Dye load and large radiation dose
- Optimally used when incorporated into a validated diagnostic decision tree
Pulmonary Angiography

• “Gold Standard”
  – Performed in an Interventional Cath Lab

• Positive result is a “cutoff” of flow or intraluminal filling defect

• Not easily accessible

Pulmonary Angiogram

Abrupt termination of ascending branch of right upper lobe artery confirming PE

S1Q3T3 and T Wave Changes
Diagnosis: Other tests

- Most patients with PE have a normal pulse oximetry
- A-a gradient is insensitive and non-specific

Lab Findings in P.E.

- **ABG:**
  - Hypoxemia
  - Hypocapnia
  - Respiratory alkalosis
  - Massive PE: hypercapnia, mix resp and metabolic acidosis (including lactic acid)
  - Patients with RA pulse ox readings <95% are at increased risk of in-hospital complications, respiratory failure, cardiogenic shock, death

Lab Findings in P.E.

- **BNP**
  - Insensitive test
  - Patient’s with PE have higher levels than patients without, but not ALL patients with PE have high BNP
  - **Good prognostic value measure:** if **BNP >90** associated with adverse clinical outcomes (death, CPR, mechanical vent, pressure support, fibrinolysis, embolectomy)

Lab Findings in P.E.

- **Troponin**
  - High in 30-50% of patients with moderate to large PE
  - Prognostic value if combined pro-NT BNP
  - Trop I >0.07 + NT-proBNP >600 = high 40 day mortality
  - Normal troponin has very high NPV (99-100%)
Diagnosis

• D-Dimer
  – Fibrin degradation product
  – ELISA tests are highly sensitive (>95%)
  – Non specific (~40%): cancer, sepsis, inflammation
  increase d-dimer levels

Hypercoagulability Work Up

– Protein C/S deficiency
– Factor V leiden deficiency
– AntiThrombin III deficiency
– Prothrombin 20210 mutation
– Antiphospholipid antibody
– High Homocysteine

Clinical Diagnosis of PE

• In summary, clinical signs, symptoms and routine tests do not allow for the exclusion or confirmation of acute PE but may increase the index of its suspicion
• Consider PE in cases of unexplained tachycardia or syncope

Treatment

• Anticoagulants
  – Heparin
    • Provides immediate thrombin inhibition, which prevents thrombus extension
    • Does not dissolve existing clot
  • Will not work in patients with antithrombin III deficiency
    – In this case use hirudins
  • Few absolute contraindications
Anticoagulation

- Heparin:
  - Lovenox: if hemodynamically stable, no renal function
    - 1mg/kg BID OR 1.5mg/kg QDay
  - Heparin gtt: if hypotension, renal failure
    - 80units/kg bolus then 18units/kg infusion
    - Goal PTT 1.5 to 2.5 times the upper limit of normal
- Coumadin:
  - Start once acute anticoagulation achieved
  - Start with 5mg PO qday OR 10mg PO q day
  - If start with 10mg then achieve therapeutic INR 1.4 days sooner
  - Complications and morbidity no different in 5mg or 10mg start
  - Goal INR 2 to 3

Fibrinolysis

- Considered once P.E. diagnosed
- If chosen, hold anticoagulation during fibrinolysis infusion, then resumed
- Associated with higher incidence of major hemorrhage
- Indications: persistent hypotension, severe hypoxemia, large perfusion defects, right ventricular dysfunction, free floating right ventricular thrombus, patent foramen ovale
- Activase (rtPA)

Embolectomy

- Surgical or catheter
- Indication:
  - Those who present severe enough to warrant fibrinolysis
  - In those where fibrinolysis is contraindicated or fails

Poor Prognostic Signs

- Hypotension (not caused by arrhythmia, sepsis, or hypovolemia)
  - SBP <90 mm Hg = 53% 90-day all cause mortality
  - SBP drop of 40 mm Hg for at least 15 minutes = 15% in–hospital mortality
- Syncope= bad
- Shock= really bad
Case Study

- 30 year old male had an ORIF of ankle fx 2 weeks ago, c/o sudden onset of chest pain.

What are the signs/symptoms of this disease? What are the risk factors for this disease?

PE Diagnosis

- Symptoms
  - SOB or dyspnea-
  - Chest pain (pleuritic)
  - Cough
  - Sudden onset

- Signs
  - Tachycardia > 100 beats per minute
  - Tachypnea > 20 breaths per minute
  - Hypoxia < 95% on RA

Case

- A 69 year old man presents to the ER with acute dyspnea. BP is 120/80 and pulse is 120 BPM. D-Dimer is positive.
- Spiral CT shows bilateral pulmonary emboli in >50% of arterial tree.
- Troponin 0.3 ng/ml
- Echocardiogram showed RV enlargement with septal bowing into LV.
- BP remained at baseline 130/80, persistent tachycardia 120 BPM.
- Risk of 30-day mortality estimated to be ~10%.
- Given the intermediate risk PE profile and lack of contraindications, after discussion with the patient it was decided that the benefits of rt-Pa administration>risks.
- Patient was admitted to the CCU and 100 mg rt-Painfused over 2 hours; dyspnea resolved over the course of the afternoon.
- Patient discharged to complete 6 months of warfarin, target INR 2.5.
- On f/u patient had no evidence of pulmonary hypertension or post thrombotic syndrome.
Pulmonary Embolus Risk Factors

• Hypercoaguability
  – Malignancy, pregnancy, estrogen use, factor V Leiden, protein C/S deficiency
• Venous stasis
  – Bedrest > 48 hours, recent hospitalization, long distance travel
• Venous injury
  – Recent trauma or surgery

D-dimer
– Very sensitive in low to moderate probability
– Not sensitive enough for high probability
– Not specific (Lots of false positives)

Spiral CT
– Current gold standard
  – Caution if impaired creatinine clearance

V/Q
– Many studies will be “indeterminate”

PE Diagnosis

PE Treatment

• IV fluid to maintain blood pressure
• Heparin (Limits propagation but does not dissolve clot)
  – Unfractionated: 80 u/kg bolus, 18 h/kg/hr
  – Fractionated (Lovenox): 1 mg/kg SC BID
• Fibrinolytics
  – Consider with large if patient is unstable