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# Pulmonary Embolism

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# Background

- Pulmonary Embolism (PE) is often one of the most difficult and deadly diagnosis to make
- Even in our modern era of testing, patients often have fatal PE's even though they were recently seen by a physician
- This diagnostic difficulty is due to the range of symptoms
- Difficult to know when to consider it.

# Definition and Pathophysiology

- PE refers to obstruction of the pulmonary artery or one of its branches by material (eg, thrombus, tumor, air, or fat) that originated elsewhere in the body
- PE can be classified as acute or chronic. Patients with acute PE typically develop symptoms and signs immediately after obstruction of pulmonary vessels. In contrast, patients with chronic PE tend to develop slowly progressive dyspnea over a period of years due to pulmonary hypertension

# Definition

- Acute PE can be further divided into massive and submassive
  - Massive refers to an embolus that results in hypotension
    - It is a catastrophic entity that frequently results in acute right ventricular failure and death.
    - When death occurs, it is often within one to two hours of the event, although patients remain at risk for 24 to 72 hours

# Definition

- Acute PE:
  - Submassive encompasses every other occurrence not meeting criterion as massive
  - Includes saddle PE's
    - is a PE that lodges at the bifurcation of the main pulmonary artery
    - . Most saddle PE are submassive. In a retrospective study of 546 consecutive patients with PE, 14 (2.6 percent) had a saddle PE. Only two of the patients with saddle PE had hypotension.

# Pathophysiology

- Most PE arise from thrombi in the deep venous system of the lower extremities
- However, they may also originate in the right heart or the pelvic, renal, or upper extremity veins.
- Iliofemoral veins are the source of most clinically recognized PE.
- Estimated that 50 to 80 percent of iliac, femoral, and popliteal vein thrombi (proximal vein thrombi) originate below the popliteal vein (calf vein thrombi) and propagate proximally.



# Pathophysiology

- Most calf vein thrombi resolve spontaneously and only 20 to 30 percent extend into the proximal veins if untreated.
- Most lower extremity thrombi develop at sites of decreased flow, such as valve cusps or bifurcations.
- After traveling to the lung, large thrombi may lodge at the bifurcation of the main pulmonary artery or the lobar branches and cause hemodynamic compromise.
- Smaller thrombi continue traveling distally and are more likely to produce pleuritic chest pain, presumably by initiating an inflammatory response adjacent to the parietal pleura.

# Pathophysiology

- Only about 10 percent of emboli cause pulmonary infarction, usually in patients with preexisting cardiopulmonary disease.
- Most pulmonary emboli are multiple, with the lower lobes being involved in the majority of cases

# Pathophysiology

- Impaired gas exchange due to PE cannot be explained solely on the basis of mechanical obstruction of the vascular bed and alterations in the ventilation to perfusion ratio. Gas exchange abnormalities are also related to the release of inflammatory mediators, resulting in surfactant dysfunction, atelectasis, and functional intrapulmonary shunting.

# Pathophysiology

- Hypotension is due to diminished cardiac output (CO)
  - which results from increased pulmonary vascular resistance (PVR) impeding right ventricular outflow and reducing left ventricular preload.
  - PVR is increased from physical obstruction of the vascular bed with thrombus and vasoconstriction
  - the vasoconstriction due to the effects of inflammatory mediators and hypoxia.

# Risk Factors

HyperCoagulability	Venous stasis
Malignancy	Bedrest > 48hrs
Non malignant Thrombophilia	Cast or Extrenal fixator
Pregnancy	Recent Hospitalization
Postpartum status (< 4wks)	Long distance automobile or air travel
Estrogens	Venous Injury
Antiphospholipid antibodies	Recent surgery requiring GA
Genetic Mutation <ul style="list-style-type: none"> <li>Factor V leiden mutation</li> <li>Prothrombin mutation</li> <li>Factor VIII mutations</li> </ul> Functional/antigenic Protein C deficiency <ul style="list-style-type: none"> <li>Functional/antigenic Protein S deficiency</li> </ul>	

# Signs and Symptoms

- Specific symptoms and signs are not helpful diagnostically because their frequency is similar among patients with and without PE.
- In the Prospective Investigation of Pulmonary Embolism Diagnosis II (PIOPED II), the following frequencies of symptoms and signs were noted among patients with PE who did not have preexisting cardiopulmonary disease:
  - The most common symptoms were dyspnea at rest or with exertion (73 percent),
  - pleuritic pain (44 percent), cough (34 percent)

# Signs and Symptoms

- >2-pillow orthopnea (28 percent)
- calf or thigh pain (44 percent), calf or thigh swelling (41 percent), and wheezing (21 percent).
- The onset of dyspnea was usually within seconds (46 percent) or minutes (26 percent).

# Signs and Symptoms

- The most common signs were tachypnea (54%), tachycardia (24 percent), rales (18%)
- decreased breath sounds (17 percent)
- An accentuated pulmonic component of the second heart sound (15 percent)
- Jugular venous distension (14 percent).
- Symptoms or signs of lower extremity deep venous thrombosis (DVT) were common (47 percent). Included edema, erythema, tenderness, or a palpable cord in the calf or thigh.



# Mortality Factors

- Untreated PE has a mortality rate of 30%
- However recurrent emboli are the most common cause of mortality
- There are certain findings during the initial phases that put the patient at increased risk of death

# Mortality Factors

- Right Ventricular Dysfunction
  - In the patients with PE who are normotensive or hypotensive, this finding carries a 2 fold increase in mortality
  - More accurate in the hypotensive population
- Right Ventricle Thrombus
- Elevated serum troponins

# Outcomes

- Effective anticoagulant therapy decreases the mortality rate from approximately 30 percent to 2 to 8 percent.
- Among survivors, some degree of pulmonary hypertension and exercise limitation appears to be common.
- Six months after their acute PE, approximately 50 percent of the patients had persistent or worsened elevation of their right ventricular systolic pressure (suggests pulmonary hypertension).
- This was frequently accompanied by dyspnea at rest and/or exercise intolerance.