NCBI Bookshelf. A service of the National Library of Medicine, National Institutes of Health.

StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 Jan-.

Acute Pulmonary Embolism

Authors

Vrinda Vyas[1]; Abdulghani Sankari[2]; Amandeep Goyal[3].

Affiliations

[1] SUNY Upstate Medical University
[2] Wayne State University
[3] University of Kansas Medical Center

Last Update: December 11, 2024.

Continuing Education Activity

Acute pulmonary embolism (PE) occurs when a blood clot that has arisen from another area obstructs the pulmonary arteries. PE typically originates as a deep vein thrombosis (DVT) in the lower extremities. Together, PE and DVT form the spectrum of venous thromboembolism (VTE). Symptoms of PE vary and are often nonspecific, including dyspnea, chest pain, cough, and syncope. Severe cases may involve hemodynamic instability and signs of right ventricular strain. Diagnosis combines clinical probability scoring systems like the Wells and Geneva criteria with tests such as D-dimer levels, CT pulmonary angiography, and ultrasound. Hemodynamically unstable patients may require bedside imaging for rapid diagnosis.

Management of PE includes supportive measures, anticoagulation as the mainstay of treatment, and reperfusion strategies for severe cases. Thrombolysis, catheter-directed therapy, and surgical embolectomy are options for hemodynamically unstable patients. Long-term anticoagulation is essential to prevent recurrence, with duration tailored to individual risk factors. Recognizing and promptly treating PE is critical to reducing its high mortality and morbidity rates. This activity for healthcare professionals is designed to enhance the learner's competence in promptly recognizing the clinical features of PE, performing the recommended evaluation, and implementing an appropriate interprofessional management approach to improve patient outcomes.

Objectives:

- Identify the clinical features of acute pulmonary embolism.
- Select the most appropriate diagnostic imaging modality for a patient with a suspected acute pulmonary embolism.
- Differentiate between acute pulmonary embolism and other cardiopulmonary conditions.
- Apply interprofessional team strategies to improve care coordination and outcomes in patients with acute pulmonary embolism.

Access free multiple choice questions on this topic.

Introduction

Acute pulmonary embolism (PE) is a life-threatening condition that occurs when a blood clot that has arisen from a different area obstructs the pulmonary arteries. Most commonly, PE originates from a deep vein thrombosis (DVT) in the lower extremities. PE usually occurs when a part of this thrombus breaks off and enters the pulmonary circulation. PE rarely occurs from embolizing other materials into the pulmonary circulation, eg, air, fat, or tumor cells. [1] Together, PE and DVT comprise venous thromboembolism (VTE), a significant cause of morbidity and mortality

worldwide. Risk factors for PE include genetic predispositions like thrombophilia and acquired conditions, including prolonged immobility, surgery, and malignancy.

Despite advancements in diagnostic tools and treatment options, the nonspecific symptoms of PE (eg, dyspnea, chest pain, and syncope) often overlap with other cardiovascular and respiratory conditions, making timely diagnosis challenging for clinicians. Prompt recognition and management of PE are critical, as delayed intervention can lead to severe complications, including hemodynamic instability, right ventricular failure, and sudden death. Risk stratification tools such as the Wells criteria and Geneva score, alongside diagnostic imaging, are essential for accurate diagnosis. However, the underutilization of these tools, coupled with variations in therapeutic approaches, underscores the need for standardized clinical protocols. Clinicians can improve patient outcomes through enhanced knowledge and proficiency in identifying risk factors, employing diagnostic strategies, and initiating evidence-based treatments.

Etiology

Risk Factors for Pulmonary Embolism

Most PEs originate as lower extremity DVTs. Hence, PE risk factors are the same as DVT. The Virchow triad of hypercoagulability, venous stasis, and endothelial injury provides an understanding of these risk factors.

Risk factors can be classified as genetic and acquired. Genetic risk factors include thrombophilia, eg, factor V Leiden mutation, prothrombin gene mutation, protein C deficiency, protein S deficiency, and hyperhomocysteinemia. Acquired risk factors include immobilization for prolonged periods (eg, bed rest of longer than 3 days, any type of travel for >4 hours), recent orthopedic surgery, malignancy, indwelling venous catheter, obesity, pregnancy, cigarette smoking, and oral contraceptive pill use.[2][3][4][5] Smoking is a risk factor for all causes of pulmonary infarction, including those associated with PE.[6] Paradoxically, younger age (peaking at 40) and increased height are associated with an increased likelihood of developing a PE complicated by pulmonary infarction, while obesity is associated with a reduced likelihood.[7]

Other predisposing factors for VTE include:

- Fracture of lower limb
- Hospitalization for heart failure or atrial fibrillation/flutter within the previous 3 months
- Hip or knee replacement
- Major trauma
- History of previous venous thromboembolism
- Central venous lines
- Chemotherapy
- Congestive heart failure or respiratory failure
- Hormone replacement therapy
- Oral contraceptive therapy
- Postpartum period
- Infection (specifically pneumonia, urinary tract infection, and HIV)
- Cancer (highest risk in metastatic disease)
- Thrombophilia

- Bed rest longer than 3 days
- Obesity
- Pregnancy

Cancer carries a high risk for thrombus formation and, hence, PE. Pancreatic cancer, hematological malignancies, lung cancer, gastric cancer, and brain cancer carry the highest risk for VTE.[8] Infection anywhere in the body is a common trigger for VTE.[9] Myocardial infarction and congestive heart failure increase the risk of PE. Patients with VTE have an increased risk of subsequent stroke and myocardial infarction.[10][11]

Types of Pulmonary Embolism

Categorizing PE based on the presence or absence of hemodynamic stability is crucial. Hemodynamically unstable PE, previously called massive or high-risk PE, is PE that results in hypotension as defined by systolic blood pressure <90 mm Hg or a drop in systolic blood pressure of ≥40 mm Hg from baseline or hypotension that requires vasopressors or inotropes), the old term "massive" PE does not describe the size of the PE but rather the hemodynamic effect. Patients with a hemodynamically unstable PE are more likely to die from obstructive shock (ie, severe right ventricular failure).

Hemodynamically stable PE is a spectrum ranging from small, mildly symptomatic, or asymptomatic PE (low-risk PE or small PE) to PEs that cause mild hypotension that stabilizes in response to fluid therapy or those that present with right ventricular dysfunction (submassive or intermediate-risk PE) but are hemodynamically stable.

Epidemiology

Acute PE incidence ranges from 39 to 115 per 100,000 population annually; for DVT, the incidence ranges from 53 to 162 per 100,000 people.[12] After coronary artery disease and stroke, acute PE is the third most common type of cardiovascular disease.[13] Furthermore, the incidence of acute PE is higher in males than females.[14] PE complicated by pulmonary infarction occurs at a rate of 16% to 31%.[7]

Overall, PE-related mortality is high, and in the United States, PE causes 100,000 deaths annually.[14] However, the mortality rates attributable to PE are challenging to estimate accurately because many patients with sudden cardiac death are thought to have had a thromboembolic event like PE. The case-fatality rates of PE have decreased, likely due to improved diagnostic methods, early interventions, and therapies.

Pathophysiology

PE occurs when a blot clot enters the pulmonary circulation. Multiple emboli are typically involved within the lower lung lobes more frequently than the upper lobes; bilateral lung involvement is also more common.[15] Large emboli tend to obstruct the main pulmonary artery, causing saddle embolus with deleterious cardiovascular consequences. In contrast, smaller-sized emboli block the peripheral arteries and can lead to pulmonary infarction, manifested by intra-alveolar hemorrhage.

PE leads to impaired gas exchange due to obstruction of the pulmonary vascular bed, leading to a mismatch in the ventilation-to-perfusion ratio because alveolar ventilation remains the same. Still, pulmonary capillary blood flow decreases, leading to dead space ventilation and hypoxemia. Also, mediators, eg, serotonin, are released, which cause vasospasm and further decrease pulmonary flow in unaffected lung areas. Local accumulation of inflammatory mediators alters lung surfactant and stimulates respiratory drive, resulting in hypocapnia and respiratory alkalosis.[16]

In PE, pulmonary vascular resistance increases due to the mechanical obstruction of the vascular bed with thrombus and hypoxic vasoconstriction. Pulmonary artery pressure increases if thromboembolic occlusion is >30% to 50% of the total cross-sectional area of the pulmonary arterial bed. Increased pulmonary vascular resistance increases the right ventricular afterload, which impedes right ventricular outflow and causes right ventricular dilation and flattening or

bowing of the interventricular septum. Developing the right bundle branch block may increase the desynchronization of the ventricles. The decreased right ventricular outflow and concomitant right ventricular dilation reduce left ventricular filling, compromising cardiac output.[17]

As a result, left ventricular filling is reduced in early diastole, reducing cardiac output and causing systemic hypotension and hemodynamic instability. Right ventricle failure due to acute pressure overload is the primary cause of death in severe PE. Given the above pathophysiological considerations, clinical symptoms and signs of overt right ventricular failure and hemodynamic instability indicate a high risk of early (in-hospital or 30-day) mortality.

Additionally, the early literature suggested that patients with underlying cardiac disease were at the greatest risk for developing a pulmonary infarction associated with acute PE, as poor collateral circulation, in combination with pulmonary thromboembolism, was thought to result in infarction.[18] However, recent studies suggest the opposite. Specifically, younger patients without cardiopulmonary disease were found to be more likely to suffer a pulmonary infarction secondary to PE.

Experts hypothesize that longstanding local tissue hypoxia from chronic cardiopulmonary disease states leads to more robust bronchial vascular collateralization, protecting parenchyma from infarction.[7] The lung parenchyma receives its oxygen supply from 3 nonredundant sources: deoxygenated blood from pulmonary arteries, oxygenated blood from the bronchial circulation, and direct oxygen diffusion from alveoli.[19] A sufficient impedance from one of these sources can cause infarction and subsequent tissue necrosis. Inflammatory mediators from ischemic parenchyma can further limit gas exchange following the resultant vasoconstriction and bronchoconstriction.[20] When ischemia of lung tissue is not reversed promptly, infarction ensues. A unilateral infarct occurs in 77% to 87% of pulmonary infarctions, with the strongest predilection for the right lower lobe. Multiple studies show a stark predominance of pulmonary infarction in the lower lobes relative to the upper lobes, thought to be due to gravity's influence on the unique relationship between alveolar, pulmonary, and bronchial arterial pressure.[7][21]

History and Physical

Clinical History

A timely diagnosis of PE is crucial because of the high associated mortality and morbidity, which may be prevented with early treatment. Notably, 30% of untreated patients with PEs die, while only 8% die after timely therapy.[22] [23] Unfortunately, diagnosing PE can be difficult due to the variety of nonspecific clinical signs and symptoms in patients with acute PE. The most common symptoms of PE include dyspnea, pleuritic chest pain, cough, hemoptysis, presyncope, and syncope. Dyspnea may be acute and severe in a central PE, whereas mild and transient dyspnea is observed in small peripheral PE. In patients with preexisting heart failure or pulmonary disease, worsening dyspnea may be the only symptom. Chest pain is a frequent symptom usually caused by pleural irritation due to distal emboli causing pulmonary infarction.[24] In central PE, chest pain may be from underlying right ventricular ischemia and needs to be differentiated from an acute coronary syndrome or aortic dissection.

Less common presentations include arrhythmias (eg, atrial fibrillation), syncope, and hemodynamic collapse. [25] Hemodynamic instability is a rare but essential form of clinical presentation, as it indicates central or extensive PE with severely reduced hemodynamic reserve. Syncope may occur and may be associated with a higher prevalence of hemodynamic instability and right ventricular dysfunction.[26] Recognizing that patients with large PE may sometimes be asymptomatic or have mild symptoms is crucial. PE may often be asymptomatic or discovered incidentally during diagnostic workup for another disease. Therefore, in addition to PE symptoms, clinicians should look for VTE risk factors to determine the clinical probability of PE.

Physical Examination Findings

On examination, patients with PE might have tachypnea and tachycardia, which are common but nonspecific findings. Other examination findings include calf swelling, tenderness, erythema, palpable cords, pedal edema, rales, decreased