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A Clinical Presentation of Acute Pulmonary Embolism

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Abstract

Pulmonary embolism, despite being common, often remains elusive as a diagnosis, and clinical suspicion needs to remain high when seeing a patient with cardiopulmonary symptoms. Once suspected, diagnosis is usually straightforward; however, optimal treatment can be difficult. Risk stratification with clinical scores, biomarkers and imaging helps to refine the best treatment strategy, but the position of thrombolysis in intermediate risk (submassive) pulmonary embolism remains a grey area. Pulmonary embolism response teams are on the increase to provide advice in such cases.

Keywords: Diagnostic algorithms; pulmonary angiography; pulmonary emboli; risk stratification

Introduction

Pulmonary embolism (PE) is the third most frequent acute cardiovascular syndrome. Annual PE incidence and PE-related mortality rates rise exponentially with age, and consequently, the disease burden imposed by PE on the society continues to rise as the population ages worldwide. Recently published landmark trials provided the basis for new or changed recommendations included in the 2019 update of the European Society of Cardiology Guidelines (developed in cooperation with the European Respiratory Society). Refinements in diagnostic algorithms were proposed and validated, increasing the specificity of pre-test clinical probability and d-dimer testing, and thus helping to avoid unnecessary pulmonary angiograms.

Acute pulmonary embolism: magnitude of the problem

Venous thromboembolism (VTE), clinically presenting as deep vein thrombosis (DVT) or acute pulmonary embolism (PE), is the third most frequent acute cardiovascular syndrome after myocardial infarction and stroke. Annual incidence rates for PE lie between 39 and 115 per 100,000 population; for DVT, the rates are 53–162 per 100,000 . The incidence of VTE is almost eight times higher in individuals aged 80 years or older than in the fifth decade of life.
Diagnosis of PE during pregnancy can be challenging as symptoms frequently overlap with those of normal pregnancy. Furthermore, d-dimer levels continuously increase during pregnancy and it has been reported that levels can be above the 500 µg/L threshold in up to 25% of pregnant women in the third trimester. Moreover, registry data suggested that d-dimer testing might also be of limited sensitivity in this setting. Aiming to clarify the situation, a multinational prospective management trial included 441 pregnant women presenting to emergency departments with clinically suspected PE.

Types of Pulmonary Embolism

It is extremely crucial to divide PE based on the presence or absence of hemodynamic stability.

Hemodynamically unstable PE (previously called massive or high-risk PE) is PE which results in hypotension (as defined by systolic blood pressure (SBP) less than 90 mmHg or a drop in SBP of 40 mm Hg or more from baseline or hypotension that requires vasopressors or inotropes), the old term "massive" PE does not describe the size of the PE but describes its hemodynamic effect. Patients with hemodynamically unstable PE are more likely to die from obstructive shock (i.e., severe right ventricular failure).

Pathophysiology

PE occurs when deep venous thrombi detach and embolize to the pulmonary circulation. Pulmonary vascular occlusion occurs and impairs gas exchange and circulation. In the lungs, the lower lobes are more frequently affected than the upper, with bilateral lung involvement being common. Larger emboli wedge in the main pulmonary artery, while smaller emboli occlude the peripheral arteries. Peripheral PE can lead to pulmonary infarction, manifested by intra-alveolar hemorrhage. Pulmonary infarction occurs in about 10% of patients without underlying cardiopulmonary disease. Obstruction of the pulmonary arteries creates dead space ventilation as alveolar ventilation exceeds pulmonary capillary blood flow.

Clinical Presentation

Prompt recognition of a PE is crucial because of the high associated mortality and morbidity, which may be prevented with early treatment. Failure to diagnose PE is a serious management error since 30% of untreated patients die, while only 8% succumb with effective therapy. Unfortunately, PE may be asymptomatic or present with sudden death. Characteristic signs and symptoms such as tachycardia, dyspnea, chest pain, hypoxemia, and shock are non-specific and are present in many other conditions, such as acute MI, congestive heart failure, or pneumonia. In the Prospective Investigation of Pulmonary Embolism Diagnosis II (PIOPED II) trial, patients with PE had a range of signs and symptoms. Common signs were tachypnea (54%) and tachycardia (24%). The most common symptoms were dyspnea, usually of onset within seconds, at rest or with exertion (73%), pleuritic pain (44%), calf or thigh pain (44%), calf or thigh swelling (41%), and cough (34%). With only 24% of patients presenting with tachycardia, the majority of patients lacked one of the most common findings.

Treatment

In hemodynamically stable patients, without contraindications to systemic anticoagulation, parenteral anticoagulation with subsequent conversion to vitamin K antagonists is the mainstay of therapy. Early initiation is paramount as patients may quickly decompensate. Patients who present to the emergency room with acute PE have decreased mortality if anticoagulation treatment commences in the emergency room, rather than waiting until after admission. Supportive care of hypoxemia and hemodynamic instability should be instituted. Hemodynamically unstable patients may benefit from fibrinolytic therapy. However, the role for fibrinolysis is limited, with significant bleeding occurring in up to 13% of patients. The use of bolus thrombolytics during cardiopulmonary arrest may have some benefit when PE is strongly suspected and the patient does

not respond to resuscitation. Mechanical thrombolysis with catheter-directed embolectomy and fibrinolytic therapy can also be used. Systemic heparin, either in the form of unfractionated heparin or low-molecular-weight heparin (LMWH), is the mainstay of treatment. LMWH is advantageous in ease of administration, monitoring, lower potential for heparin-induced thrombocytopenia.

Anticoagulation

It is vital to remember that the mainstay of treatment of acute PE is anticoagulation.

It is important to note that either low-molecular-weight heparin (LMWH) or fondaparinux or unfractionated heparin (UFH) can be used for anticoagulation in acute PE. LMWH and fondaparinux are preferred since they have a less incidence of inducing major bleeding and heparin-induced thrombocytopenic. UFH is usually only used in patients with hemodynamic instability in whom primary reperfusion treatment might be required, or in patients with renal impairment. Newer oral anticoagulants (NOACs) and vitamin K antagonists (VKA) can also be used for anticoagulation in PE.

Conclusion

An interventional approach to managing both acute LE-iliofemoral DVT and massive and submassive PE has great promise. There remains a paucity of robust long-term evidence, particularly addressing safety outcomes in therapies utilizing drugs and delivery systems that can result in bleeding complications.

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