

Diagnosis of pulmonary embolism in trauma patients

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SUMMARY

Pulmonary embolism (PE) is a common complication in the trauma patient. The clinical presentation is often subtle and the diagnostic workup can be impeded by frequently held misconceptions. In addition, circumstances unique to the trauma population add complexity to the evaluation. We present a literature review of the pathophysiology, clinical presentation, and diagnostic workup of PE.

Key words: Pulmonary embolism. Trauma patient. Pathophysiology.

INTRODUCTION

Pulmonary embolism (PE) remains a significant problem in hospitalized trauma patients. These patients are at increased risk of deep vein thrombosis (DVT), one of the most common causes of PE. DVT complicates as many as 60% of trauma cases, and when PE occurs, it carries a high mortality rate¹⁻³. The mortality rate increases among patients with right ventricular dysfunction, age >60, and comorbid disease⁴. PE is the most common cause of preventable hospital death, and it remains one of the most difficult to diagnose. Seventy percent of all diagnoses are made on postmortem examination⁵⁻⁷. The diagnosis of PE is complicated in trauma populations for the following reasons: 1) communication may be hampered by head injury, intoxication, or intubation and 2) patients, especially those with chest trauma, often have other physical reasons for their symptoms. PE may occur early in the posttraumatic period, sometimes within only several days of injury³. As discussed in the following sections, there are many commonly held misconceptions about the presentation and diagnostic workup of PE.

PATHOPHYSIOLOGY

Pulmonary embolism is the end of a spectrum of thromboembolic disease. When a clot forms in the deep venous system, it can break off or embolize completely to the pulmonary vasculature. Thigh and pelvic thrombi usually pose the highest risk, but calf thrombi propagate and embolize in 20% of cases and upper extremity thrombi can embolize as well^{8,9}. Once a thrombus embolizes to the pulmonary vasculature, a number of sequelae are possible. Generally, a ventilation-perfusion mismatch and hypoxia develop. A variety of neurohumoral factors can lead to bronchospasm and wheezing. Large clots can cause right ventricular dysfunction and lead to acute cor pulmonale and hypotension¹⁰.

Risk factors. For a variety of reasons, trauma patients (especially those with head injury, spinal cord injury, pelvic fracture, or leg fracture¹) are at particularly high risk of thromboembolic complications. Their risk factors relate to Virchow's triad—intimal damage, venous stasis, and hypercoagulability¹¹—which can be identified in nearly 90% of trauma patients. Forty percent of the cases of thromboembolic disease can be attributed to those three factors induced by trauma and/or surgery⁴.

Each risk factor can be exacerbated by pre-existing or co-existing practices and conditions. Intimal damage can be caused by intravenous drug abuse and the use of central lines. Venous stasis can result from the

immobilization of fractured limbs in casts, by stroke, and by inactivity related to prolonged travel. Hypercoagulable states are found in many patients with malignancy or sepsis, in pregnant women, and in association with the use of oral contraceptives¹². The complex hypercoagulable state in trauma patients relates to endothelial cell injury and the release of procoagulant factors¹³. Hematologic conditions such as protein C and S deficiency, the presence of lupus anticoagulant, and disorders of fibrinogen and plasminogen also cause hypercoagulable conditions¹⁴. Age >60 and co-existing medical conditions such as cardiac disease, congestive heart failure, nephrotic syndrome, systemic lupus erythematosus, and other autoimmune conditions place the patient at increased risk for thromboembolism⁷. One of the most important risk factors is a history of thromboembolic disease¹¹.

Symptoms. The classic presentation of pulmonary embolism is a sudden onset of pleuritic chest pain and dyspnea; however, the symptom complex is usually more varied. Dyspnea and pleuritic chest pain actually only occur in 60% to 73% of cases, while cough occurs in 37% to 50%^{12,15}. Apprehension is also fairly common, occurring in more than 60% of cases. Apprehension and dyspnea may be the only symptoms and may mimic panic attack or anxiety disorder¹⁶. Less common symptoms include hemoptysis (20%), wheezing (9%), palpitations (10%), and syncope (10%).¹⁷

Physical examination. Although the physical exam is nonspecific in pulmonary embolism, patients frequently exhibit abnormal findings. Tachypnea (respiratory rate >20) is the most common finding, being present in up to 70% of patients. Despite common belief, tachycardia and clear chest exams are not typical of pulmonary embolism. Tachycardia occurs in only 30% of patients and up to 50% exhibit crackles on lung exam¹⁵⁻¹⁷. Since PE is a complication of deep vein thrombosis, it may seem logical that evidence of DVT would be present on physical exam. However, fewer than one third of patients with PE exhibit lower extremity pain and swelling¹⁵⁻¹⁷. In one surveillance study, which documented a 57% incidence of DVT in the posttraumatic period, fewer than 2% of patients had symptoms of DVT prior to study¹.

Many of the signs and symptoms of PE are variable and nonspecific, underscoring the difficulty in making the diagnosis. The combination of dyspnea, tachypnea, and pleuritic chest pain is present in 95% of patients with PE¹⁷. It is important to view the entire clinical complex and not rely on any one clinical feature to arrive at a diagnosis.

DIAGNOSTIC TESTS

Initial tests. The chest film, electrocardiogram (ECG), and arterial blood gas measurements are used as initial screening studies in patients with suspected PE. Although they may provide important information about a patient's condition and prompt more in-depth workup, they are seldom diagnostic for PE¹⁸.

The chest film is important in excluding other causes of a patient's symptoms but, despite common belief, is abnormal in more than 80% of patients with PE^{15,16}. The most common abnormalities noted are atelectasis and parenchymal infiltrates (in more than two thirds of patients). Small pleural effusions are also noted in 50% of patients with PE. The "classic" findings of Hampton's hump (a wedge-shaped pleural-based infiltrate) and Westermark's sign (a prominent pulmonary artery with decreased pulmonary vascularity) are rare and poorly associated with PE¹⁹.

The purpose of the ECG, like the chest film, is to exclude other pathology, specifically pericarditis and acute myocardial infarction. The most common abnormality is the presence of nonspecific ST-T wave changes, which are noted in up to 50% of PE patients²⁰. The "classic" S₁Q₃T₃ and evidence of right ventricular strain, right bundle branch block, and right axis deviation are present in fewer than one third of PE patients. Other findings such as atrial fibrillation and premature atrial or ventricular contractions are noted in fewer than 5% of PE cases. (Recall that sinus tachycardia is present in only 30% of patients.) Despite the nonspecific abnormalities noted, only 6% of patients have a normal ECG²⁰.

Arterial blood gas measurement is a useful means of assessing a patient's oxygenation and ventilation status but again is not helpful in the definitive diagnosis of PE. Although many patients with PE are hypoxic, the room air PO₂ is >80 mmHg in 25% and >90 mmHg in 10%^{14,16}. The A-a gradient is an equally unsatisfying

screening test. As many as 10% of patients with angiographically proven PEs have a normal A-a gradient, and the PIOPED study found no significant difference between patients with and those without PE^{15,16,18}. Although the chest film, ECG, and arterial blood gas may support or challenge a diagnosis of pulmonary embolism, most patients require further testing. The ventilation perfusion scan (V/Q) is the usual next step in the workup for PE.

Ventilation-perfusion scan. In preparation for a perfusion scan, radioactive macroaggregated albumin is injected and then lodges in the pulmonary vasculature. Images are taken using a gamma camera. Emboli larger than 2 mm can be detected. The ventilation scan adds specificity to the study by detecting “mismatched defects.” Scans are interpreted as normal, low probability, intermediate probability, and high probability for pulmonary embolism. Unfortunately, there is poor interrater reliability between radiologists¹⁸. It is more helpful to think of V/Q scans as diagnostic or nondiagnostic²¹. Diagnostic readings are normal and high probability. Nondiagnostic readings are low and intermediate probability. It is also helpful to combine the physician’s assessment of the pretest probability for PE as low or high clinical suspicion²¹. Using this approach, 90% of patients with high probability scans have angiographically proven evidence of PE and 95% of patients with normal scans have negative angiograms¹⁸. When V/Q scans are read as intermediate probability, 16% of patients with low clinical suspicion and up to 66% of patients with high clinical suspicion may have angiographically documented PE²². Even low probability scans are associated with a 40% chance of PE when clinical suspicion is high and 4% when clinical suspicion is low²². It is evident that nondiagnostic V/Q scans are associated with an intolerably high incidence of PE. In these cases, it will be necessary to pursue further diagnostic testing.

Pulmonary angiography. Pulmonary angiography is the gold standard in the diagnosis of PE. Most physicians are reluctant to utilize angiography because of its invasive nature. However, compared with the risk of unnecessary anticoagulation or a missed diagnosis of PE, the risk of angiography is relatively small. The mortality rate is less than 0.5%; death generally occurs in critically ill patients. Other complications occur in only 2% to 6% of patients and include transient elevations in serum creatinine, bleeding, and dye reaction²³.

Evaluation for DVT. Since PE is a complication of thromboembolic disease, it seems logical to pursue a diagnosis of DVT when evaluating patients for PE. Unfortunately, fewer than 60% of patients with PE have DVT that can be documented by ultrasound or venography²⁴. The most efficient use of venous evaluation may be as an angiography-sparing modality. One study documented a 50% reduction in the need for angiograms in patients with intermediate probability V/Q scans who were identified as having lower extremity DVT on ultrasound²⁵. Thus, in patients with nondiagnostic V/Q scans, it is reasonable to pursue an ultrasound study before proceeding to angiography.

D-dimer assay. When a thrombus forms, the endogenous fibrinolytic system is activated. The breakdown of fibrin produces a number of degradation products, including the D-dimer moiety. This fragment can be measured by latex agglutination assays and enzyme-linked immunoassays (ELISA). The ELISA is more accurate but requires more time to run. Recent studies show the ELISA assays to be 98% sensitive but only 40% specific for PE.²⁶ Although more studies are required, one can see the utility for a test with such a high sensitivity for thrombosis. The D-dimer has been shown to be useful in the trauma patient²⁷. When placed in the full clinical context, a patient with a low suspicion for PE, low probability V/Q, and negative D-dimer may need no further workup.

Alveolar dead space analysis. Alveolar dead space, which increases when emboli occlude the pulmonary vasculature, can be measured easily with the use of an arterial blood gas measurement, an end-tidal CO₂ detector, and the following equation: $V_D/V_T = (PaCO_2 - P_{et}CO_2)/PaCO_2$, where V_D/V_T represents dead space and $P_{et}CO_2$ represents end-tidal CO₂. When the dead space is less than 0.4 in conjunction with a normal spirogram, the negative predictive value for PE is 96%²⁸.

Transesophageal echocardiogram. Transesophageal echocardiography (TEE) can be a useful modality during evaluation of an unstable patient, because the patient does not need to be moved to a distant site. TEE can be up to 85% sensitive for large pulmonary emboli. Abnormalities usually are related to right ventricular dysfunction and include increased right ventricular end diastolic volume, abnormal septal movement, tricuspid regurgitation, and abnormal right ventricular wall motion²⁹.

MANAGEMENT

After appropriate resuscitation and stabilization, the mainstay of therapy in pulmonary embolism is anticoagulation. Heparin activates antithrombin III and prevents the conversion of fibrinogen to fibrin. Patients should receive a loading dose of 5,000 to 10,000 U heparin and then an initial infusion rate of 1,000 U/h. The partial thromboplastin time (PTT) should be checked in 6 hours, with a target value of 1.5 to 2 times control. Oral anticoagulation therapy with coumadin is usually initiated on the first day, as it takes several days to achieve therapeutic levels³⁰. The International Normalized Ratio (INR) is now preferred as the standard by which to measure the therapeutic response to coumadin. The target INR for treatment of thromboembolic disease is 2 or 3. Unfortunately, some patients in the postinjury period are not candidates for anticoagulation: Specifically, patients with head injury and those being observed for nonoperative management of hepatic and splenic injuries are better candidates for vena cava filters.

Given the high incidence of thromboembolic disease in the posttraumatic period, with its attendant morbidity and mortality, much attention has been paid to prevention^{1-3,13}. Pneumatic compression devices, subcutaneous heparin, low-molecular-weight heparin, and vena caval interruption have all been used with varying results^{2,13}.

RESUMEN

El tromboembolismo pulmonar es una complicación común en el paciente traumatizado. A menudo la presentación clínica es insidiosa y el diagnóstico puede dificultarse debido a concepciones erróneas. Además, las circunstancias únicas que rodean a la población traumatizada agrega complejidad a la evaluación. Con base en una revisión de la literatura se presenta la fisiopatología, presentación clínica y diagnóstico del tromboembolismo pulmonar.

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