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PULMONARY EMBOLISM: IMPROVING THE ODDS

Pulmonary embolism (PE) is the most commonly diagnosed pulmonary disorder in the inpatient setting, and at least one large autopsy series identified PE as the most common finding attributable to the cause of death.

Of the approximately 650,000 cases of PE in the United States each year, 10% die in the first hour. Although not much can be done for these patients, we must address ourselves to the half million survivors. The mortality rate for undiagnosed PE is 32%, but if the diagnosis is made and the patient is managed appropriately, mortality drops to 8%. Early diagnosis and institution of therapy can alter the unexpected and unwarranted outcomes of PE.

The source of PE in about 90% of cases is the lower extremities. Other less common sources are the pelvic veins, the right heart, and occasionally an in situ pulmonary thrombosis.

The peculiar physiologic response of the lungs to an embolic event is due to the dual blood supply. The pulmonary arterial supply and the bronchial circulation offer the advantage of an alternate system for the perfusor-supplied segment of the lung if one supply is occluded. In general, this system protects the lungs from pulmonary embolism, but in specific situations it can actually promote pulmonary infarction.

When a large pulmonary arterial occlusion occurs in a central site, the bronchial circulation can provide blood supply to this area. The vast pulmonary vascular bed easily resolves the influx of blood caused by a major arterial occlusion. Often, almost no roentgenographic evidence of the PE is present. However, with an obstruction in the peripheral or segmental pulmonary arteries, the influx of blood through the bronchial circulation creates a dilated bronchopulmonary anastomosis, leading to exudation of red blood cells into the supplied segment. In patients with no underlying cardiopulmonary disease, the presence of hemoptysis and the radiographic abnormality suggest pulmonary hemorrhage, which is usually resolved in a few days, leaving no roentgenographic evidence behind.

In patients with underlying cardiopulmonary disease, the elevated pulmonary venous pressure and its accompanying pressure gradients cause a defective resorption of the pulmonary hemorrhage and may lead to pulmonary infarction.

HEMODYNAMIC ALTERATIONS

Hemodynamic monitoring in patients with suspected PE is of extremely limited value. Hemodynamic alteration in PE depends on the underlying pulmonary status, the extent of vascular impairment, and the existing conditions present. The presence of a normal PO_2 should not lower the suspicion of pulmonary embolism. Data from three separate studies showed that, out of 54 patients with PE, 13% had normal PO_2 levels.

In patients with no underlying cardiopulmonary disease, the relationship between pulmonary vascular obstruction and mean increase in pulmonary arterial pressure is linear. However, regardless of the severity of obstruction, the pulmonary arterial pressure rarely goes above 40 mm Hg in the acute situation. In patients with underlying disease, the values are scattered and reflect the underlying status and not the extent of obstruction.

Hemodynamic monitoring in patients free of underlying disease can be used as a rough indication of the degree of PE severity. However, in patients with underlying disease it will not indicate the rate of lysis or the status of the pulmonary vascular bed.

PATIENT EVALUATION

Chest pain is reported by 80% to 90% of patients with PE. Tachypnea is present in 92% of patients, and dyspnea is also commonly present. These three symptoms are nonspecific; however, if a patient has none of these symptoms, the diagnosis of PE is unlikely. Electrocardiographic changes are also nonspecific for PE. Many patients have a normal sinus rhythm with tachycardia, and if electrocardiographic changes occur, they are usually ST-wave changes.

Chest roentgenography is equally nonspecific. Only 7% to 12% of patients in a large series had clear lung fields. About half of PE patients have pleural effusion,

which in most cases is an exudate. Chest roentgenography, electrocardiography, and serum enzymes are of greatest value in diagnosing mimics of PE, such as myocardial infarction, acute pulmonary edema, and pneumothorax. For more conclusive endpoints, diagnostic studies are needed. If PE is suspected, a ventilation/perfusion lung scan should be the first diagnostic test to be performed. In a study of 515 patients with suspected PE and normal lung scans (3- to 6-month follow-up), only one case of fatal PE was reported.

A normal lung scan therefore predicts a benign eventual course for an embolic episode and rules out clinically significant PE. A high-probability lung scan will lead most physicians to immediately start either anticoagulant or thrombolytic therapy. An indeterminate lung scan indicates the need for pulmonary angiography. A low-probability lung scan has to be interpreted in light of clinical suspicion. In some cases, a normal noninvasive study of the leg veins (Doppler or impedance plethysmography) may justify withholding therapy, while in others pulmonary angiography may be needed.

Pulmonary angiography remains the gold standard for the diagnosis of PE, yet this procedure has failed to gain widespread use due to a need for broader availability of adequate facilities, the invasive nature of the procedure, and its potential hazards. In competent hands this procedure is safe, with morbidity less than 1% and mortality less than 0.01%. Selective arteriography significantly enhances the test's diagnostic sensitivity. Positive findings indicative of PE are distinct filling defects, cutoffs, and oligemia. Although the interpretation of results may vary widely, and although selective injection does not always produce enough detail at the bases, pulmonary angiography is the best technique at present to establish the diagnosis of PE.

MANAGEMENT

Apart from supportive measures, the management of PE consists of anticoagulation therapy with or without prior fibrinolytic therapy. The use of and indications for fibrinolytic therapy before anticoagulation therapy is still controversial. Whether fibrinolysis decreases the mortality or morbidity from PE is not yet known; however, fibrinolysis costs more and has more bleeding complications than heparin therapy. The data that support thrombolytic therapy show a more rapid resolution of thromboembolism in the pulmonary circulation, and a more thorough removal of emboli from the microcirculation than heparin. Each patient should be evaluated individually. In the absence of absolute contraindications and under expert supervision, thrombolytic therapy is reasonable in patients with massive PE, or in patients whose underlying status makes them more susceptible to hemodynamic derangement.

ON THE HORIZON

In the future, the management of PE would be facilitated with more sensitive, less invasive imaging techniques. In particular, digital subtraction angiography with higher resolution would offer improved demonstration of PE. Indium 111 platelet scintigraphy has exciting potential in the diagnosis and management of deep vein thrombosis, but its role in the diagnosis of PE needs further investigation. Also, initial trials of angioscopy for localizing chronic embolic obstructions has been promising. The angioscope of the future could permit biopsy, injection, or even suctioning to expand its diagnostic and therapeutic capabilities.

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SUGGESTED READING

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ORDERING STOOL EXAMS: ROOM FOR IMPROVEMENT

Hospitalized patients with diarrhea can be divided into two groups: (1) those who present with diarrhea on admission or develop it within the first 3 hospital days, and (2) those who develop diarrhea after the third hospital day. The diarrhea in the first group of patients can have an infectious or iatrogenic etiology, whereas diarrhea occurring after the third day is nosocomial and has an iatrogenic etiology. Only rarely is this nosocomial diarrhea infectious, as in the case of food poisoning in the institutional setting, or inadequate decontamination of diagnostic equipment.