Update On Pulmonary Embolism

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Pulmonary embolism (PE) is a common and lethal condition causing mostly from thrombi in iliac and femeral veines. Most patients who succumb to pulmonary embolism do so within the first few hours of the event. Despite diagnostic advances, delays in pulmonary embolism diagnosis are common and represent an important issue (1) As a cause of sudden death, massive pulmonary embolism is second only to sudden cardiac death in US. In patients who survive a pulmonary embolism, recurrent embolism and death can be prevented with prompt diagnosis and therapy. Unfortunately, the diagnosis is often missed because patients with pulmonary embolism present with nonspecific signs and symptoms. If left untreated, approximately one third of patients who survive an initial pulmonary embolism die from a subsequent embolic episode. After 2 years suffered from PE, 3.8% of the patients develop chronic thromboembolic pulmonary arterial hypertension (CTEPH) (2).

95% pulmonary emboli arise from thrombi originating iliac and femeral veines (3); however, they may rarely originate in the pelvic, renal, or upper extremity veins or the right heart chambers. After traveling to the lung, large thrombi can lodge at the bifurcation of the main pulmonary artery or the lobar branches and cause hemodynamic compromise. Smaller thrombi typically travel more distally, occluding smaller vessels in the lung periphery. These are more likely to produce pleuritic chest pain by initiating an inflammatory response adjacent to the parietal pleura. Most pulmonary emboli are multiple, and the lower lobes are involved more commonly than the upper lobes.

When a PE is identified, it is characterized as acute or chronic. In terms of pathologic diagnosis, an embolus is acute if it is situated centrally within the vascular lumen or if it occludes a vessel (vessel cutoff sign) (see the first image below). Acute

pulmonary embolism commonly causes distention of the involved vessel. An embolus is chronic if it is eccentric and contiguous with the vessel wall, it reduces the arterial diameter by more than 50%, evidence of recanalization within the thrombus is present, and an arterial web is present, finally becomes CTEPH

A PE is also characterized as central or peripheral, depending on the location or the arterial branch involved. Central vascular zones include the main pulmonary artery, the left and right main pulmonary arteries, the anterior trunk, the right and left interlobar arteries, the left upper lobe trunk, the right middle lobe artery, and the right and left lower lobe arteries. A pulmonary embolus is characterized as massive when it involves both pulmonary arteries or when it results in hemodynamic compromise. Peripheral vascular zones include the segmental and subsegmental arteries of the right upper lobe, the right middle lobe, the right lower lobe, the left upper lobe, the lingula, and the left lower lobe.

The risk factors for forming veinous thrombi are one of the most important points to suspect patients having PE. They include heritable (dificiencies of antithrombin, plasminogen, protein C, S, factor V leiden,…) acquired (reduced mobility/immobility, cast, advanced age, acute medical illness, trauma, major surgeries, oral contraceptives/HRP, obesity, central venous catheterization,…) and protable (elevated levels of homosystin, factors VIII, IX, XI, fibrinogen,…) factors (4,5)

The variability of presentation sets the patient and clinician up for potentially missing the diagnosis. The challenge is that the "classic" presentation with abrupt onset of pleuritic chest pain, shortness of breath, and hypoxia is rarely seen. Studies of patients who died unexpectedly of pulmonary embolism revealed that the patients had complained

of nagging symptoms, often for weeks, before dying. Forty percent of these patients had been seen by a physician in the weeks prior to their death (6)

The most important conceptual advance regarding PE over the last several decades has been the realization that pulmonary embolism is not a disease; rather, pulmonary embolism is a complication of venous thromboembolism, most commonly deep venous thrombosis (DVT). Virtually every physician who is involved in patient care encounters patients who are at risk for venous thromboembolism, and therefore at risk for PE

Physical examination findings are quite variable in PE and, for convenience, may be grouped into 4 categories as follows: massive PE, acute pulmonary infarction, acute embolism without infarction, multiple PE or thrombi

The presentation of pulmonary embolism may vary from sudden catastrophic hemodynamic collapse to gradually progressive dyspnea. (Prior poor cardiopulmonary status of the patient is an important factor leading to hemodynamic collapse.) Most patients with PE have no obvious symptoms at presentation. In contrast, patients with symptomatic DVT commonly have PE confirmed on diagnostic studies in the absence of pulmonary symptoms. Four common symtoms of PE are dyspnea (73%), pleuritic chest pain (66%), cough (37%), hemoptysis (13%) (7).

Clinical signs and symptoms for PE are nonspecific; therefore, patients suspected of having PE because of unexplained dyspnea, tachypnea, or chest pain or the presence of risk factors for PE must undergo diagnostic tests until the diagnosis is ascertained or eliminated or an alternative diagnosis is confirmed. Further, routine laboratory findings are nonspecific and are not helpful in PE, although they may suggest another diagnosis.

Ventilation/Perfusion (V/Q) scan is the most sensitive modality for screening PE and CTEPH. Pulmonary angiography historically was the criterion standard for the diagnosis of PE, but with the improved sensitivity and specificity of CT angiography, it is now rarely performed. In the case, without V/Q scan, CT angiography with multidetetor can use instead of it, but can not rule out PE or CTEPH completely.

Therapy: All patients with PE require rapid risk stratification. Thrombolytic therapy should be used in patients with acute PE associated with hypotension (systolic BP<90 mm HG) who do not have a high bleeding risk (8). Do not delay thrombolysis in this population because irreversible cardiogenic shock can develop. Thrombolytic therapy is suggested in select patients with acute PE not associated with hypotension and with a low bleeding risk whose initial clinical presentation or clinical course after starting anticoagulation suggests a high risk of developing hypotension. In patients with acute PE, immediate full anticoagulation is mandatory for all patients suspected to have DVT or PE, anticoagulation with IV UFH, LMWH, or fondaparinux is preferred over no anticoagulation (8). Most patients with acute PE should receive LMWH or fondaparinux instead of IV UFH. In patients with PE, if concerns regarding subcutaneous absorption arise, severe renal failure exists, or if thrombolytic therapy is being considered, IV UFH is the recommended form of initial anticoagulation (8). Diagnostic investigations should not delay empirical anticoagulant therapy. Long-term anticoagulation is critical to the prevention of recurrence of DVT or PE. The general consensus is that a significant reduction in recurrence is associated with 3-6 months of anticoagulation

Prognosis: As a cause of sudden death, massive PE is second only to sudden cardiac death. Massive PE is defined as presenting with a systolic arterial pressure less

than 90 mm Hg. The mortality for patients with massive PE is between 30% and 60%, depending on the study cited (9,10,11). Autopsy studies of patients who died unexpectedly in a hospital setting have shown approximately 80% of these patients died from massive PE. The majority of deaths from massive PE occur in the first 1-2 hours of care, so it is important for the initial treating physician to have a systemized, aggressive evaluation and treatment plan for patients presenting with pulmonary embolism.

Nonmassive PE is defined as having a systolic arterial pressure greater than or equal to 90 mm Hg. This is the more common presentation for PE and accounts for 95.5-96% of the patients 8 (10,12).

Hemodynamically stabile PE has a much lower mortality rate because of treatment with anticoagulant therapy. In nonmassive PE, the death rate is less than 5% in the first 3-6 months of anticoagulant treatment. The rate of recurrent thromboembolism is less than 5% during this time. However, recurrent thromboembolism reaches 30% after 10 years (13).

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