Bilateral Pulmonary Embolism

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Clinical History: A 60-year-old female with pelvic fracture developed shortness of breath with tachycardia and tachypnea

Clinical History: A 60-year-old female with pelvic fracture developed shortness of breath with tachycardia and tachypnea. Spiral CT of chest done with contrast to R/O PE.
Thrombus in bilateral main pulmonary arteries extending to upper and lower lobe arteries.
Lower lobe extension of thrombus.
Lower lobe extension of thrombus.
Lower lobe segmental extension of thrombus.
Lower lobe segmental extension of thrombus.
Lower lobe extension of thrombus.
Lower lobe segmental extension of thrombus.
Lower lobe sub segmental extension of thrombus.
Bilateral Pulmonary Embolism

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Diagnosis: Bilateral pulmonary embolism
Additional History: Patient had a fall and got a right acetabular fracture.

Discussion:
Pulmonary embolism is a common and potentially lethal condition. 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. As a cause of sudden death, massive pulmonary embolism is second only to sudden cardiac death.

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 event.
When a pulmonary embolism 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). 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 percent, evidence of recanalization within the thrombus is present, and an arterial web is present.

A pulmonary embolism 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 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.

Clinical signs and symptoms for pulmonary embolism are nonspecific; therefore, patients suspected of having pulmonary embolism—because of unexplained dyspnea, tachypnea, or chest pain or the presence of risk factors for pulmonary embolism—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 pulmonary embolism, although they may suggest another diagnosis. Pulmonary angiography remains the criterion standard for the diagnosis of pulmonary embolism, but with the improved sensitivity and specificity of CT angiography, it is now rarely performed.

Etiology:
Three primary influences predispose a patient to thrombus formation; these form the so-called Virchow triad, which consists of the following.
- Endothelial injury
- Stasis or turbulence of blood flow
- Blood hypercoagulability

Thrombosis usually originates as a platelet nidus on valves in the veins of the lower extremities. Further growth occurs by accretion of platelets and fibrin and progression to red fibrin thrombus, which may either break off and embolize or result in total occlusion of the vein. The endogenous thrombolytic system leads to partial dissolution; then, the thrombus becomes organized and is incorporated into the venous wall.

Pulmonary emboli usually arise from thrombi originating in the deep venous system of the lower extremities; 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.

The causes for pulmonary embolism are multifactorial and are not readily apparent in many cases. The causes described in the literature include the following:
- Venous stasis
- Hypercoagulable states
- Immobilization
- Surgery and trauma
- Pregnancy
- Oral contraceptives and estrogen replacement
- Malignancy
- Hereditary factors
- Acute medical illness
A study by Malek et al confirmed the hypothesis that individuals with HIV infection are more likely to have clinically detected thromboembolic disease.[6] The risk of developing a pulmonary embolism or DVT is increased 40% in these individuals.

History:
The challenge in dealing with pulmonary embolism is that patients rarely display the classic presentation of this problem, that is, the abrupt onset of pleuritic chest pain, shortness of breath, and hypoxia. Studies of patients who died unexpectedly from pulmonary embolism have revealed that often these individuals complained of nagging symptoms for weeks before death. Forty percent of these patients had been seen by a physician in the weeks prior to their death.[2]

The following risk factors can be indications for the presence of pulmonary embolism:
- Venous stasis
- Hypercoagulable states
- Immobilization
- Surgery and trauma
- Pregnancy
- Oral contraceptives and estrogen replacement
- Malignancy
- Hereditary factors resulting in a hypercoagulable state
- Acute medical illness
- Drug abuse (intravenous [IV] drugs)
- Drug-induced lupus anticoagulant
- Hemolytic anemias
- Heparin-associated thrombocytopenia
- Homocystinemia
- Homocystinuria
- Hyperlipidemias
- Phenothiazines
- Thrombocytosis
- Varicose veins
- Venography
- Venous pacemakers
- Venous stasis
- Warfarin (first few days of therapy)
- Inflammatory bowel disease

The PIOPED II study listed the following indicators for pulmonary embolism:
- Travel of 4 hours or more in the past month
- Surgery within the last 3 months
- Malignancy, especially lung cancer
- Current or past history of thrombophlebitis
- Trauma to the lower extremities and pelvis during the past 3 months
- Smoking
- Central venous instrumentation within the past 3 months
- Stroke, paresis, or paralysis
- Prior pulmonary embolism
- Heart failure
- Chronic obstructive pulmonary disease

Diagnostic Considerations:
The variability of presentation for pulmonary embolism sets the patient and clinician up for potentially missing the diagnosis. Such missed diagnoses occur in approximately 400,000 patients in the United States per year; approximately 100,000 deaths could be prevented with proper diagnosis and treatment.

The diagnostic challenge is that the "classic" presentation of the condition, with abrupt onset of pleuritic chest pain, shortness of breath, and hypoxia, is rarely seen. Studies of patients who died unexpectedly from pulmonary embolism have revealed that the patients 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.

The differential diagnoses are extensive, and they should be considered carefully with any patient thought to have pulmonary embolism. These patients also should have an alternative diagnosis confirmed, or pulmonary embolism should be excluded, before discontinuing the workup. Additional
problems to be considered include the following:

- Musculoskeletal pain
- Pleuritis
- Pericarditis
- Salicylate intoxication
- Hyperventilation
- Silicone pulmonary embolism
- Lung trauma
- Mediastinitis, acute

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References


Disclosures:

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