Thrombosed Pulmonary Artery Aneurysm*  
A Rare Cause of a High-Probability Lung Scan

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The high-probability ventilation-perfusion lung scan is accepted as supportive of pulmonary embolism and often negates further diagnostic evaluation; however, there are processes that mimic the clinical presentation and radiographic findings of pulmonary emboli, including a unilateral segmental or greater perfusion defect. We present the findings in a patient whose presentation and ventilation-perfusion scans over a three-month course were suggestive of pulmonary embolism, yet pulmonary angiography revealed a thrombosed pulmonary artery aneurysm. The interpretation of a unilateral segmental perfusion defect as high probability does not secure the diagnosis of pulmonary embolism and should not preclude further evaluation for alternative etiologies. (Ches 1992; 102:1292-94)

For the past decade the ventilation-perfusion scan has played an important role in the evaluation of the patient with a suspected pulmonary embolism. By current criteria a scan with one or more segmental or greater perfusion defects and normal ventilation has been recognized a high probability for pulmonary embolism, and no further evaluation is indicated; however, this perfusion pattern, especially when unilateral in presentation, may be mimicked by nonthromboembolic processes that may necessitate further diagnostic evaluation. We present the findings in a patient in whom the clinical diagnosis of pulmonary embolism was suggested by multiple high-probability perfusion scans, yet who was found to have a ruptured pulmonary artery aneurysm. This process has not been reported previously as a cause of high-probability ventilation-perfusion scans.

CASE REPORT

A 36-year-old white woman was admitted with a one-week history of dyspnea. She denied chest pain, cough, or hemoptysis. The patient had quit smoking three months earlier after a 7-pack-year history. She denied illicit drug use. A chest roentgenogram revealed a right midfield infiltrate. An arterial blood sample measured a pH of 7.46, a PaO2 of 77.5 mm Hg, and a PaCO2 of 32.4 mm Hg, with an alveolar-arterial gradient of 31.7 mm Hg. The differential diagnosis included pulmonary embolism. A ventilation-perfusion scan was obtained and interpreted as high probability for pulmonary embolism (Fig 1). The patient received anticoagulatary therapy and was discharged on warfarin therapy.

Two months later, the patient was admitted to a different hospital with a three-day history of right-sided pleuritic chest pain, dyspnea, and a nonproductive cough. Although the patient claimed compliance with her warfarin sodium (Coumadin) therapy, her prothrombin time at admission was 11.5 s, with a control of 13.4 s. A chest roentgenogram displayed decreased vascularity in the right hemi-

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thorax, with a greater prominence of the right pulmonary artery. The diagnosis of acute pulmonary embolism prompted a ventilation-perfusion scan that revealed several segmental and subsegmental perfusion mismatches in only the right lung. The impression was that of high probability for pulmonary embolism. The patient again received anticoagulative therapy and was discharged on warfarin sodium (Coumadin) therapy.

Within two days of her discharge, the patient was admitted to a third hospital with worsening right-sided pleuritic pain, dyspnea, and cough. Her breath sounds were diminished in the right base. The chest roentgenogram now revealed a right lower lobe infiltrate with a right-sided pleural effusion. The differential diagnosis included recurrent pulmonary embolism vs an infectious process. A ventilation-perfusion scan was obtained. This time, a matched segmental defect in the right lower lobe was noted, consistent with a pneumonia and a pleural effusion. The scan was of low probability for pulmonary embolism. A computerized tomographic scan of her chest was obtained that suggested a saddle embolus in the right main pulmonary artery (Fig 2). A pulmonary angiogram confirmed an aneurysmal dilatation with distal thrombotic occlusion of the inferior division of the right main pulmonary artery with multiple intraluminal filling defects (Fig 3). The patient underwent successful surgical resection of the aneurysm.

**DISCUSSION**

The ventilation-perfusion scan has been an important tool in the evaluation of clinically suspected pulmonary embolism for more than a decade. Its utility is based upon the premise that clots significantly occlude the vessel's lumen, producing a perfusion defect without similar alteration in ventilation. A high-probability scan, denoted by one or more segmental perfusion defects unmatched by similar defects in ventilation, carries an 86 percent probability of pulmonary embolism, negating the need for angiographic verification.

Nevertheless, there are a variety of pathologic processes that not only mimic the clinical presentation of pulmonary embolism, but may also result in similar ventilation-perfusion mismatch. Among these are bronchogenic carcinoma, pulmonary artery hypoplasia or aplasia, bronchogenic cyst, sarcoid lymphadenopathy, dissecting aortic aneurysm, and misplaced pulmonary artery catheters. Not previously included in this list is pulmonary artery aneurysm. The differentiation between an embolic and nonthromboembolic origin of the perfusion defect becomes difficult and dependent upon the awareness of both the clinician and radiologist.

The unilateral presentation of a segmental perfusion defect should suggest that the precipitant may not be a pulmonary thromboembolism. Pulmonary emboli are a rare cause of one-sided defects. This rarity may be attributable to distal fragmentation of a large thrombus or to the

**FIGURE 1.** Perfusion lung scan demonstrating diminished perfusion in right lower lobe on anterior (Ant) and lateral projections. LAO, Left anterior oblique; and RAO, right anterior oblique.

**FIGURE 2.** Computerized tomography of chest suggests saddle embolus in lumen of right pulmonary artery.

**FIGURE 3.** Pulmonary angiogram demonstrates aneurysmal dilatation with thrombus occluding inferior division of right main pulmonary artery.
precipitous death of the patient in the face of massive occlusion. A perfusion defect that persists over a prolonged period with no evidence of a disastrous clinical outcome suggests an anatomic abnormality, rather than an embolic origin. In this instance, the next step in a diagnostic work-up would be pulmonary angiography. In the presence of a high-probability perfusion scan, the performance of pulmonary angiography is often met with some reluctance by the radiologist.

Other diagnostic interventions have been proposed. The role of two-dimensional echocardiography in the diagnosis of pulmonary artery thromboembolism has been anecdotal. Computerized tomography may be helpful in delineating nonvascular causes of unilateral perfusion defects, but this application is mentioned only sporadically. Pulmonary angiography remains the diagnostic gold standard.

Pulmonary artery aneurysms are rare. A 1947 retrospective review of postmortem reports found a 7.3 percent incidence in the general population. In the preantibiotic era, tuberculosis and syphilis were often causative agents. In recent years, congenital cardiac abnormalities have been associated with over 56 percent of the cases. Similar dilatations have been reported in patients with Marfan's syndrome and Behçet's syndrome.

As the clinical presentation of pulmonary artery aneurysm is nonspecific and may mimic that of a pulmonary embolism, a heightened awareness should prompt a more detailed evaluation. In over 95 percent of the reported cases, proximal pulmonary artery aneurysms may be detected as nodular densities on the chest roentgenogram. More distal lesions may not be as classic in appearance. With a suggestive ventilation-perfusion scan, pulmonary angiography becomes necessary for definitive diagnosis. Given the morbidity and mortality associated with peripheral embolization and rupture, the verification of the presence of a pulmonary artery aneurysm becomes imperative to allow prompt surgery.

The utility of the high-probability ventilation-perfusion lung scan remains invaluable in the correct clinical situation. Its acceptance as prima facie evidence for pulmonary embolism may be inappropriate with unilateral perfusion defects that may be attributable to nonthromboembolic causes. Evaluation for alternate etiologies should be considered when presented with this pattern.

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Tricuspid Valve Regurgitation following Blunt Thoracic Trauma*

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Valvular lesions following blunt thoracic trauma are uncommon. Tricuspid valve regurgitation occurs very rarely. We report a successful tricuspid valve reconstruction for rupture of the chordae tendineae in a young man nine years after a motor vehicle accident. The value of echocardiography and transesophageal echocardiography for the diagnosis and quantification of this valve lesion is stressed.

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| ECC | extracorporeal circulation | RA | right atrium | RV | right ventricle | TVR | tricuspid valve replacement |

In this time of high-speed motor vehicle accidents, blunt thoracic trauma is encountered with increasing frequency. Cardiac involvement is infrequent and consists largely of myocardial contusion of varying degree. Valvular lesions are uncommon and traumatic tricuspid valve regurgitation occurs very rarely. Up to now, some 70 cases have

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