Testing For Pulmonary Embolism

To the Editor:

We read with great interest the paper, "Clinical Validity of a Normal Perfusion Lung Scan in Patients with Suspected Pulmonary Embolism", by Hull et al (Chest 1990; 97:23-26).

Their study was designed to evaluate the accuracy of a normal perfusion lung scan in excluding the diagnosis of pulmonary embolism. Our department of respiratory medicine has a special interest in venous thromboembolism. We believe that a perfusion lung scan is most valuable when it is normal and, therefore, can exclude the possibility of pulmonary embolism—which is in agreement with Hull et al. However, among the group of patients involved in this study, there is not even a single patient in whom respiratory symptoms highly suggestive of pulmonary embolism persisted in the presence of a normal perfusion scan and no alternative explanation. The majority of patients in this study were outpatients and, therefore, likely to have less severe respiratory symptoms. Moreover, in one-third of patients with normal perfusion scan, no alternative diagnosis was made. Duration and severity of symptoms in this group of patients has not clearly been mentioned.

We have documented massive pulmonary embolism on pulmonary angiography in four patients who had normal perfusion lung scans. Pulmonary angiography was performed only because clinical suspicion of pulmonary embolism was strong and persistent. Previous studies have shown that this situation can arise in cases of partial occlusion, recanalization of the clot, or with saddle embolus.

Pulmonary angiography is the only diagnostic test in such situations.

Isotope lung scanning is our first-line investigation in patients suspected of pulmonary embolism. If perfusion scan is normal, we stop anticoagulation therapy and look for an alternative diagnosis. However, if a strong clinical suspicion of pulmonary embolism persists, then we always perform pulmonary angiography—the gold standard for diagnosis of pulmonary embolism. We recommend that this policy be considered in such situations.

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REFERENCES


To the Editor:

Drs. Shaheen and Windbank have made an assumption with their statement: "among the group of patients involved in this study, there is not even a single patient in whom respiratory symptoms highly suggestive of pulmonary embolism persisted in the presence of a normal perfusion scan and no other alternative explanation." It is not possible to make this conclusion from the data in our article:

the number of patients with persistent respiratory symptoms is not described. Our objective was to test the safety of withholding anticoagulant treatment in symptomatic patients with suspected pulmonary embolism and normal perfusion scans, regardless of clinical manifestations. Our study did so. We studied a broad spectrum of patients with a wide range of severity of symptoms.

We agree that the perfusion lung scan does not have perfect sensitivity for pulmonary embolism. Two findings of our study support this conclusion: 1) the finding of proximal-vein thrombosis at presentation in 1 percent of patients (five of 493 patients), and 2) the finding of venous thromboembolism on follow-up in 0.6 percent of patients (three of 515 patients). Our findings are supported by the observations of Kipper et al.

Which patients with normal perfusion scans should have pulmonary angiography? This remains clinical judgement. We would consider performing pulmonary angiography in a patient with a normal perfusion scan if the clinical findings suggested massive saddle embolism. Such patients are rare! Indeed, venous thromboembolism occurred in only eight of 515 patients (1.5 percent) in our study. This outcome compares favorably with the 2 percent rate of pulmonary embolism reported by others on follow-up in patients with negative pulmonary angiograms. Thus, pulmonary angiography is unlikely to provide added benefit if the perfusion scan is normal, other than in the exceptional patient.

Both Moser and Secker-Walker have suggested the most important concept underlying the modern approach to venous thromboembolism is that pulmonary embolism is not a disease; rather, embolism is merely a complication of deep venous thrombosis. Recent data in patients with pulmonary embolism suggest that if proximal-vein thrombosis is absent, clinically-evident, recurrent venous thromboembolism is rare. An alternative to performing pulmonary angiography is to perform objective testing for proximal-vein thrombosis. Noninvasive objective tests for proximal-vein thrombosis (such as impedance plethysmography or B-mode venous ultrasound) are increasingly available and can be performed serially. If proximal-vein thrombosis is detected, therapy is indicated. If serial testing is negative and the patient has adequate cardiorespiratory reserve, anticoagulant therapy can be safely withheld.

Communications

References


Pulmonary Alveolar Phospholipoproteinosis Associated with Amyloidosis

To the Editor:

Pulmonary alveolar phospholipoproteinosis (PAP) is a rare disorder characterized by deposition of lipoprotein material in the alveoli. 1 The disease has been associated with a variety of infections, hematologic malignancies, and exposure to certain dusts and chemicals.2 In the case presented here, pathologic features characteristic of PAP have been documented in a patient with renal amyloidosis. To our knowledge, this association has never been reported before. A 37-year-old man was admitted to the hospital because of severe abdominal pain of acute onset in the right upper quadrant, fever, chills, and asthenia of three days duration. The abdominal pain radiated to the anterior costal area and increased with cough and inspiratory movements. Forty-five days pre-admission, the patient had a short episode of fever accompanied by ankle edema that resolved with nonspecific therapy. Temperature was 39°C. On admission, an x-ray film of the chest showed consolidation in the middle and inferior right lobes and lower left lobe. Urinalysis revealed heavy proteinuria (5.5 g per day). Escherichia coli was consistently isolated in repeated blood cultures taken at 12-h intervals. Liver function tests disclosed normal aminotransferase levels and HBsAg-positive serum; anti-HBc antibodies were detected. Fever subsided after four days of antibiotic therapy with cephalosporins and aminoglycosides. Three months later, images of pulmonary consolidation persisted. Descending urography was normal. Proteinuria ranged from 2.8 to 4.7 g per day. Tuberculin skin test (5 and 10 TU of PPD-T) was negative. Acid-fast bacilli in sputum smears were not identified. A percutaneous liver biopsy showed auromine-negative granulomas without caseous necrosis. Because chest roentgenograms did not reveal changes, a thoracot-omy/lung biopsy was performed. The most characteristic microscopic feature was the filling of large groups of alveoli with a granular and proteinaceous material that yielded a positive periodic acid-Schiff reaction, but negative to stain with alcian blue. A diagnosis of PAP was established. Renal biopsy included ten glomeruli infiltrated with eosinophilic, amorphous deposits which were positive with Congo red stain and revealed apple-green birefringence under polarized light. Deposition of amyloid, particularly in the vascular pole, mesangium, and in some capillary loops was found in all glomeruli. Similar depositions were also observed in the subintima of arterial vessels. The diagnosis of AA-type amyloidosis was made. Neither the liver nor lung parenchyma showed amyloid deposits. The patient underwent four therapeutic bronchoalveolar lavages, but the lung infiltrates did not tend to disappear. Lavages were discontinued upon the patient's request. The nephrotic syndrome improved progressively; ten months after admission there was a proteinuria <1 g per day. Corticosteroids were not given. The chest roentgenogram abnormalities vanished in a parallel manner. Complete roentgenographic resolution was evident six years after diagnosis of PAP. At present (eight years after admission), the patient has normal pulmonary and renal function.

The pathogenesis of PAP remains obscure.3,4 Defective macrophage function is a major feature of PAP. It has been suggested that impaired phagocytic activity of pulmonary macrophages may result from altered cell-mediated immunity.5 The clinical spectrum of the disorder varies between lack of symptoms and major restrictive pulmonary dysfunction. Patients with PAP may show spontaneous resolution, response to therapeutic lavage, or may die as a result of progressive respiratory failure or opportunistic infections. The case presented here illustrates an interesting occurrence of PAP and renal amyloidosis. Apparently the patient showed no response to therapeutic lavage and continued to have evidence of chronic interstitial infiltrates for several years; spontaneous resolution was noticed in the eighth follow-up year. Both amyloid disease and PAP have common disturbances in immunoregulatory mechanisms as an important step in the pathogenesis. Whether the intra-alveolar accumulation of phospholipids is a nonspecific response to a wide range of factors or represents an unknown type of immune disorder is open to question.

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