Repeat Perfusion Imaging May Differentiate Airways Obstruction from Pulmonary Embolic Disease: Report of Two Cases

Lee W. Greenspon, Margaret M. LaManna, and Sandeep Dhand

The Medical College of Pennsylvania, Philadelphia, Pennsylvania

Two cases are presented in which patients with obstructive lung disease were considered to have a pulmonary embolism (PE). Emergency lung perfusion scans supported the diagnosis of PE in both cases. However, rapid resolution of the symptoms and perfusion defects by repeat ventilation–perfusion scanning at 24 hr suggested that PE was unlikely. In selected cases of wheezing patients, repeat perfusion scans may obviate the need for pulmonary angiography.

Acute bronchospasm (*1,2*) or mucous plugging (*3*) may mimic pulmonary embolic disease, especially when pleuritic chest pain and severe hypoxemia develop. When ventilation scans are not available for logistic reasons, a perfusion scan alone may be misleading and show striking segmental perfusion defects. Since bronchospasm or mucous plugging often responds dramatically to therapy, repeat perfusion scans within 24 hr may also show marked improvement. We report two cases in which repeat perfusion scans almost normalized by 24 hr. Review of the literature indicates that the rate of resolution of perfusion defects would have been much slower had pulmonary embolism occurred.

Case 1

A 56-yr-old woman complained of a sore throat and a dry cough. Repeated coughing precipitated shortness of breath. On the evening of admission she awoke with left-sided anterior chest pain, pleuritic in nature. Her past medical history was significant for hypertension for which she took 40 mg Furosemide daily. On physical examination her tempertaure was 99 °F, her pulse was 100 and regular, her respiratory rate was 32, and her blood pressure was 150/68. She did not have a paradoxical pulse. There was a grade II systolic ejection murmur with an S4 gallop present at the cardiac apex. There were diffuse expiratory wheezes. The white blood cell count was 10,000 with 53 segs, 12 bands, and 29 lymphs. The blood gases while breathing room air were: p_aO_2 = 53, p_aCO_2 = 31, pH = 7.43. The chest x-ray showed no pulmonary infiltrates (Fig. 1). The initial lung scan demonstrated multiple bilateral segmental and subsegmental perfusion defects (Fig. 2). Eight hours later, following bronchodilator therapy, a repeat ventilation–perfusion scan showed marked resolution of the previous segmental and subsegmental perfusion defects (Figs. 3A, 3B and Fig. 4).

For reprints contact: Margaret M. LaManna, MD, Nuclear Medicine, Deborah Heart and Lung Center, Brown Mills, NJ 08015.

Case 2

A 70-yr-old woman with known chronic obstructive pulmonary disease (FEV_1 = 1.5 L, FVC = 2.7 L, FEV_1% = 56%) and congestive heart failure was hospitalized with a 1-wk history of cough with mucopurulent sputum and dyspnea. Her condition improved initially on therapy with theophyllines, antibiotics, and lasix. About 10 days after admission she developed dyspnea, which worsened rapidly culminating in a respiratory arrest requiring mechanical ventilation. Mild wheezing was audible on physical examination. A perfusion lung scan showed essentially no perfusion to the left lung, and a chest radiograph demonstrated hyperinflation with bullous formation (Figs. 5 and 6). Bronchodilators, steroids, and intravenous heparin were administered, and she was able to be extubated 16 hr after the initial episode. A ventilation–perfusion lung scan was repeated 24 hr later (Fig. 7), demonstrating resolution of the left lung perfusion abnormality. Bronchoscopy, done 2 days later for unresolved atelectasis of the left lower lobe on the chest radiograph, demonstrated large amounts of thick mucopurulent secretions.

DISCUSSION

Patients presenting with the acute onset of wheezing, shortness of breath, and cough are likely to have obstructive airways disease. This diagnosis is often supported by a history of a recent upper respiratory tract infection and the presence of a normal chest radiograph. When the patient also complains of pleuritic chest pain, the differential diagnosis must be expanded to include pulmonary embolism (*4*). Sudden dyspnea, cough, and pleuritic chest pain are the most common symptoms of pulmonary embolism. Wheezing, on presentation, is relatively rare. Windebank et al. (*5*) reported wheezing in 11 of 250 patients with angiographically proven pulmonary emboli. A previous history of asthma was found in eight of these 11 subjects, and an allergic diathesis in six. All patients studied at the time of the pulmonary embolism had pulmonary function tests documenting obstructive lung disease with a forced expiratory volume to forced vital capacity (FEV_1/FVC) ratio of about 55%. As there is often poor correlation between radioisotope perfusion lung scanning and pulmonary angiography in patients with obstructive lung diseases, a pulmonary angiogram is often necessary when the diagnosis of pulmonary embolism is being considered (*6,7*).

In addition, ventilation–perfusion imaging is often difficult

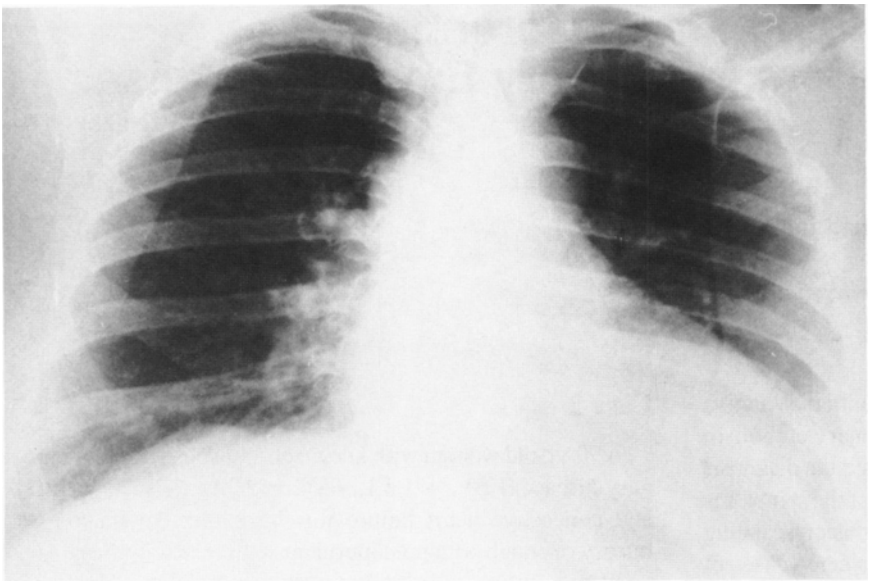

FIG. 1. The admission chest x-ray demonstrates no pulmonary infiltrates.

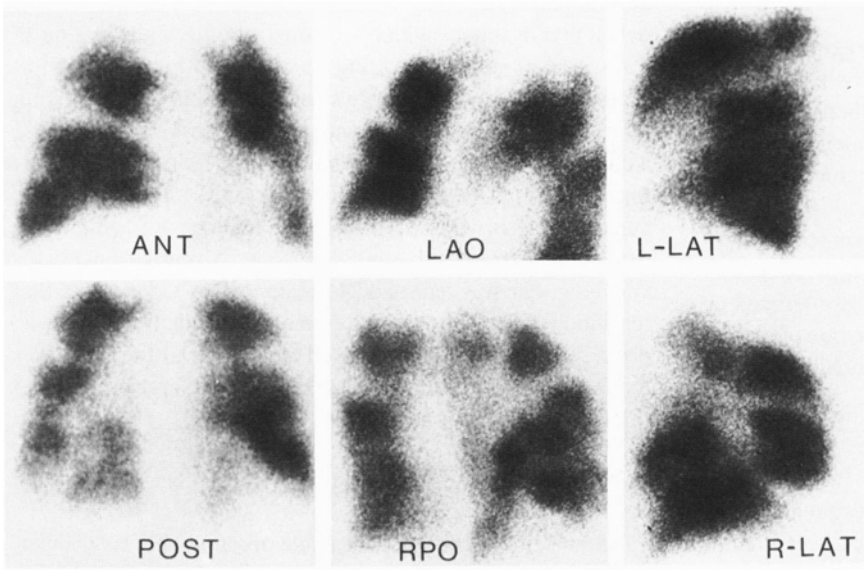

FIG. 2. After the intravenous injection of 4 mCi of ^{99m}Tc microspheres, images of the lungs were obtained in the anterior, posterior, oblique, and lateral projections. There are multiple segmental and subsegmental perfusion defects.

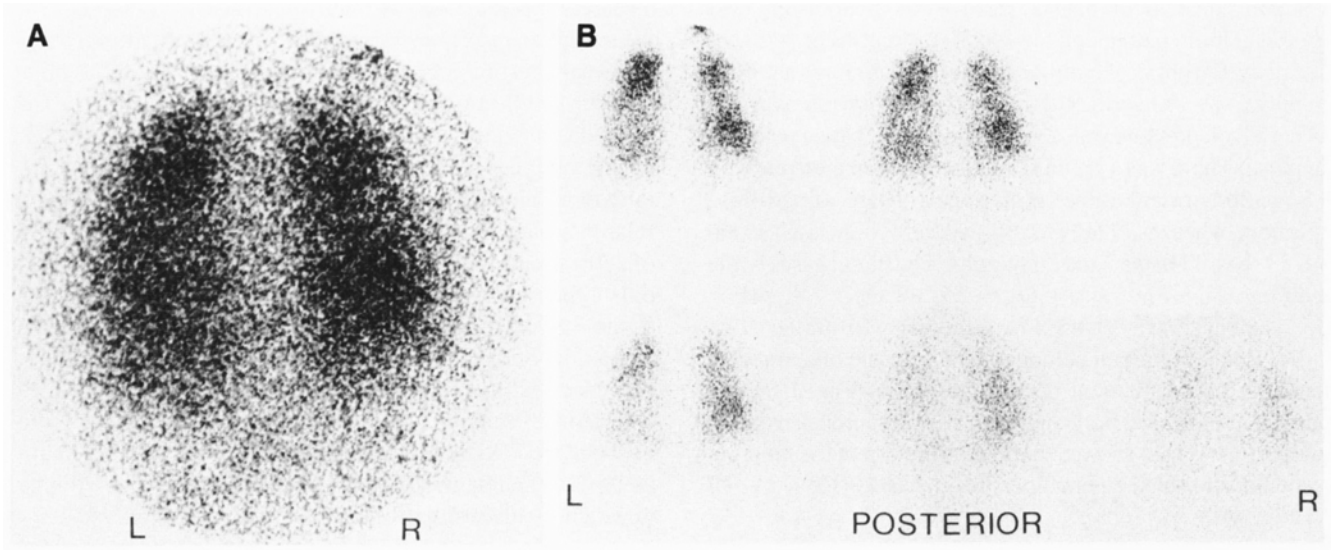

FIG. 3. After the inhalation of 10 mCi of ^{133}Xe, first breath (posterior) (A), equilibrium, and washout views (B) of the lungs were obtained in the posterior projection. There are patchy ventilatory abnormalities with diffuse xenon retention in keeping with obstructive physiology.

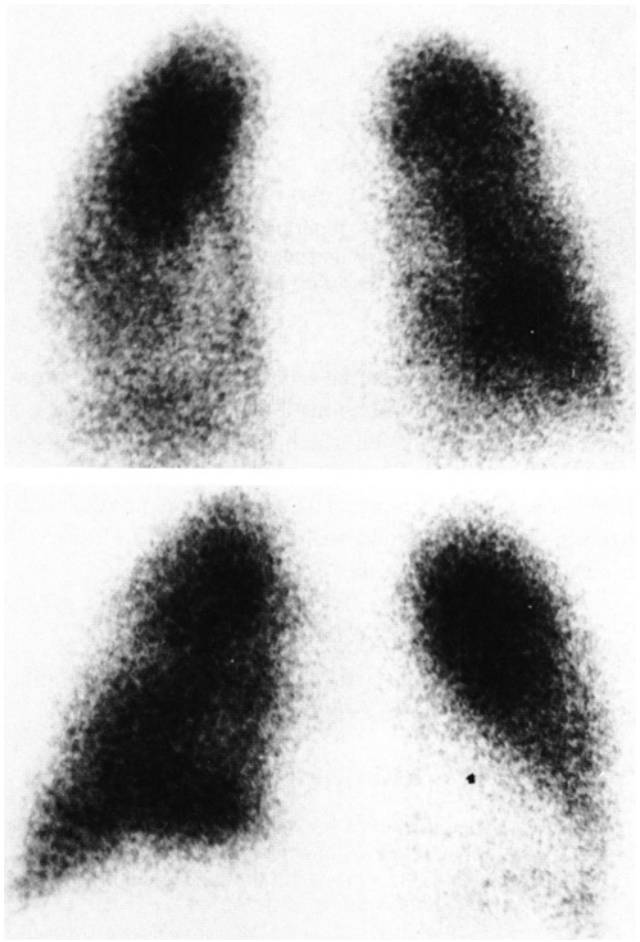

FIG. 4. The perfusion lung scan demonstrates marked resolution of the previous segmental and subsegmental perfusion defects. Top, posterior; bottom, anterior.

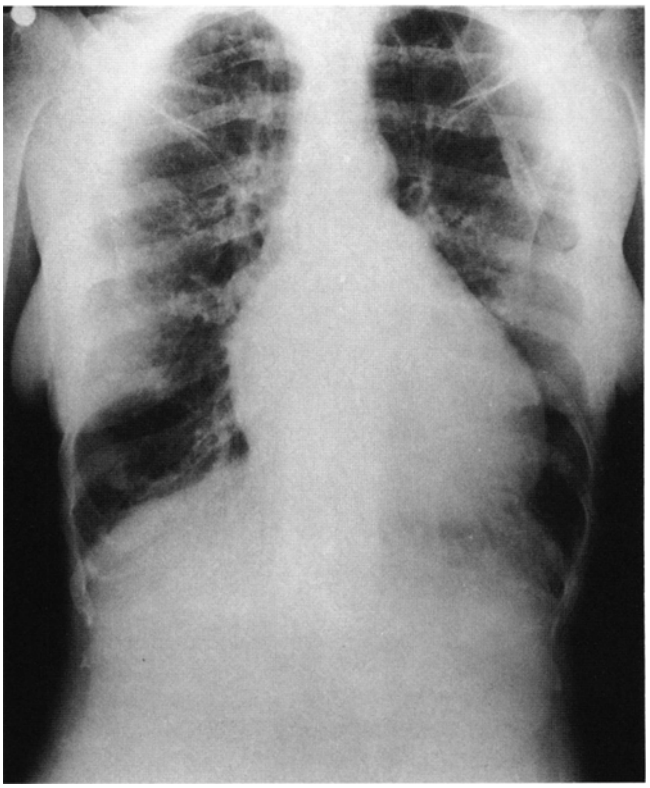

FIG. 5. Chest radiograph demonstrates hyperinflation with bullous formation. A.P. sitting.

to interpret in patients with obstructive lung disease (*8*). When airflow obstruction is significant, regional areas may be poorly ventilated, leading to alveolar hypoxia and local hypoxic vasoconstriction. During acute bronchial asthma multiple segmental perfusion defects may be observed and mimic pulmonary embolic disease. Combined ventilation perfusion imaging increases the specificity and accuracy of the diagnosis of PE because it permits the distinction of a primary ventilatory abnormality (ventilation–perfusion match) from a primary perfusion abnormality (ventilation–perfusion mismatch). Alderson et al. (*9*) studied 83 patients with obstructive lung disease for pulmonary embolism and compared ventilation–perfusion scans with arteriograms. All patients had at least one area of ventiation–perfusion match. The finding of a ventilation–perfusion mismatch in another area of the lung supported the diagnosis of pulmonary embolism with a sensitivity of 0.95 and a specificity of 0.95 when ventilation abnormalities involved less than 50% of the lung fields. In patients with obstructive lung disease xenon-133 (^{133}Xe) washout images are essential for detecting areas of ventilation–perfusion match defects. Taplin and Chopra (*10*) have used technetium-99m (^{99m}Tc)-DTPA aerosol inhalation images rather than xenon to increase the accuracy of ventilation imaging in making the diagnosis of pulmonary embolism in patients with obstructive lung disease.

In this report, the diagnosis of bronchial asthma was established in Case 1 by the rapid clinical improvement in dyspnea and wheezing after institution of bronchodilator therapy. The diagnosis was further supported by the rapid rate of resolution in the pulmonary perfusion defects. The resolution of pulmonary embolic obstruction has been studied in the dog (*11*). Early resolution (1–2 hr) may occur due to mechanical factors as the embolus location migrates and vascular obstruction becomes incomplete. The major mechanism, however, is in vivo fibrinolysis. Three hours after embolization in the dog the volume of the embolized clot is only 50% of the control thrombus. With heparinization, the volume of the clot at 3 hr is only 34%, suggesting heparin may enhance the resolution by preventing fibrin platelet aggregation on the surface of the embolus.

The rate of early resolution of pulmonary emboli in humans was assessed in the urokinase–streptokinase pulmonary embolism trial (*12*). Lung scans and pulmonary angiograms were repeated 12–18 hr after infusion of heparin in patients in whom acute pulmonary embolism was documented. Repeat lung scans demonstrated an average of 8.0% resolution at 24 hr and there was a decrease of about 20% in the embolic obstructive found on arteriography. Tow and Wagner (*13*) studied the rate of resolution of pulmonary embolism by lung scan. In patients with vascular obstruction of 16%–30% of the total lung area, the earliest improvement was seen in 6 days and the earliest

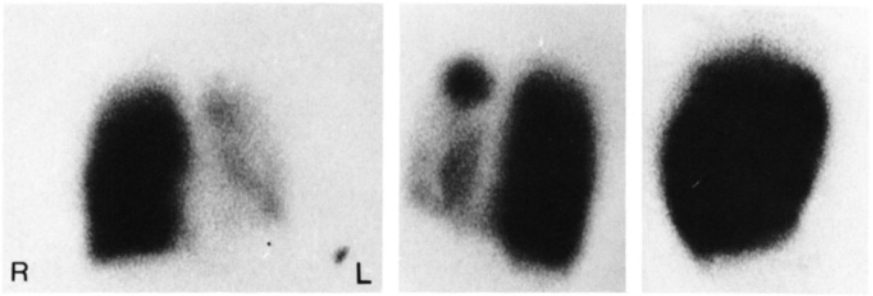

FIG. 6. A perfusion lung scan shows abnormal left lung perfusion. Far left, anterior; middle, posterior; far right, right lateral.

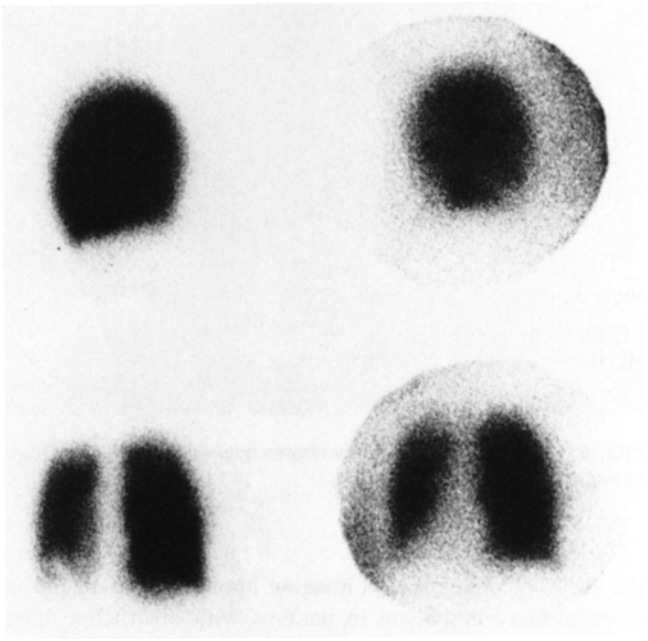

FIG. 7. Selected images from a krypton-81m ventilation study (upper right) and a perfusion lung scan demonstrate resolution of the left lung perfusion abnormality. Top, right lateral; bottom, posterior.

complete resolution was at 8 days. When the obstruction was 31%–50% of the total lung vasculature the earliest complete resolution was at 60 days.

In the cases described here, we found near complete resolution in the pulmonary perfusion defects after 8 hr and 24 hr respectively. This response is unlikely to occur in pulmonary embolic disease and most likely reflects the relief of hypoxic vasoconstriction after improvement in bronchial obstruction and normalization of alveolar ventilation.

In summary, wheezing is a rather rare complication of pulmonary embolic disease. When wheezing does occur, it usually is found in patients with underlying allergic or asthmatic histories. When perfusion imaging alone suggests the diagnosis of pulmonary embolism in these patients, heparin and bronchodilator therapy should be instituted. In selected cases, a repeat ventilation–perfusion scan by 24 hr may confirm suspicion of obstructive airways disease and save the patient a possible pulmonary arteriogram and the hazards of anticoagulation. However, more studies and well-designed clinical trails will be necessary to confirm this observation.

ACKNOWLEDGMENT

We wish to thank Karla Bechtel for her assistance in the preparation of this manuscript.

REFERENCES

1. Olazabal F, Roman-Irizarry LA, Oms JD, et al. Pulmonary emboli masquerading as asthma. *N Engl J Med* 1968;278:999–1001.

2. Webster JR, Saadeh GB, Eggum PR, et al. Wheezing due to pulmonary embolism. *N Engl J Med* 1966;274:931–933.

3. Dee M, Sarah PM, Bray ST, et al. Mucous plugging simulating pulmonary embolism in patients with quadriplegia. *Chest* 1984;85:363–366.

4. Sasahara AA, Carrilla JE, Morse RL, et al. Clinical and physiologic studies in pulmonary thromboembolism. *Am J Cardiol* 1967;20:10–19.

5. Windebank WJ, Boyd G, Moran F. Pulmonary thrombus-embolism presenting as asthma. *Br Med J* 1973;1:90–94.

6. Lippmann M, Fein A. Pulmonary embolism in patients with chronic obstructive pulmonary disease. *Chest* 1981;79:39–42.

7. Krumbolz RA, Burnham G, DeLong JF. Lung scan utilization in the diagnosis of pulmonary diseases. *Chest* 1972;62:4–7.

8. Wagner HN, Lopez I, Majano V. Radioactive xenon in the differential diagnosis of pulmonary embolism. *Radiology* 1968;91:1168–1174.

9. Alderson PO, Biello DR, Sachariah KG, et al. Scintigraphic detection of pulmonary embolism in patients with obstructive pulmonary desease. *Radiology* 1981;138:661–666.

10. Taplin GV, Chopra SK: Lung perfusion–inhalation scintigraphy in obstructive airway disease and pulmonary embolism. *Radiol Clin N Amer* 1978;14:491–513.

11. Moser KM, Guisan M, Bartimmo EE, et al. In vivo and post mortem dissolution rates of pulmonary emboli and venous thrombi in the dog. *Circulation* 1973;48:170–178.

12. The Urokinase Pulmonary Embolism Trials. A national cooperative study (phase I results). *Circulation* 1973;47(Suppl 2):1–108.

13. Tow DE, Wagner HN. Recovery of pulmonary arterial blood flow in patients with pulmonary embolism. *N Engl J Med* 1967;276:1053–1059.