

ACUTE PULMONARY EMBOLUS ASSOCIATED WITH TRANSIENT VENTILATORY DEFECT: CASE REPORT

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In a patient with a clinical history highly suggestive of pulmonary embolism and angiographic evidence also indicative of embolism, the ventilation/perfusion pattern was that usually observed with parenchymal disease of the lung. The possible basis of this contradiction is discussed.

Since pulmonary perfusion scintigraphy lacks specificity, primary parenchymal disease can result in perfusion abnormalities that may not be distinguishable from those secondary to vascular disease such as pulmonary embolism. Specificity of the lung scan in the diagnosis of pulmonary embolism was enhanced when ^{133}Xe ventilation scintigraphy became available and was combined with perfusion scintigraphy (1).

In cases of suspected pulmonary embolism, the diagnostic method of choice at our institution is the noninvasive lung scan; pulmonary angiography is rarely employed. The purpose of this communication is to call attention to a class of V/Q scan findings that may mislead physicians by suggesting primary parenchymal or airways disease when a pulmonary embolus is present.

CASE REPORT

A 44-year-old male was admitted to the hospital for treatment of thrombophlebitis of the right calf that had been present for 3 days. His right calf was tender, the deep chord was palpable, and the circumference of the right calf measured 15½ in. compared to 14 in. for the left calf. No other abnormalities were detected by physical examination.

Heparin therapy, 1,200 U/hr, was begun immediately and 4 days later Coumadin, 5 mg/day, was added. Six days after admission the patient complained of right pleuritic chest pain that gradually increased in severity during the next 48 hr. He denied palpitation, shortness of breath, or tachypnea. Ex-

amination of the chest was unchanged and there was still no splinting, wheezing, or hemoptysis. The chest radiograph was normal with the exception of a calcified scar present on admission. Lung scans were obtained on the 8th, 11th, and 16th hospital days. A pulmonary angiogram was obtained 2 days after the last lung scan.

METHOD

Three millicuries of $^{99\text{m}}\text{Tc}$ -albumin microspheres was injected intravenously with the patient supine. Images were obtained in the anterior, posterior, and right and left lateral projections with a Searle Radio-graphics HP scintillation camera and a low-energy diverging collimator. The pulse height analyzer was set on a 140-keV photopeak with a 25% window. The camera was operated in the divided crystal mode, and the time required to obtain 150,000 counts from either lung on the anterior view was the time set for obtaining left and right lateral and posterior scintiphotos. The patient was subsequently ventilated in the upright position in a projection that demonstrated the most obvious perfusion defect. With the patient breathing tidally, Polaroid scintiphotos were obtained at 25-sec intervals and, after equilibrium had been reached, washout scintiphotos were obtained every minute until the lungs were clear of radioactivity. At that time a single breathhold scintiphoto was obtained with the patient inhaling xenon to total lung capacity.

RESULTS

The initial perfusion scan revealed a large perfusion defect seen best in the posterior projection. The 25-sec xenon washin study was normal (Fig. 1);

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however, the washout scintiphotos demonstrated marked delay in clearing the xenon from the right base. Because of marked ventilatory abnormality, the scan was interpreted as compatible with parenchymal disease. Since there was strong clinical suspicion of pulmonary embolus, the patient was scanned again 3 and 18 days later. The perfusion defect was unchanged (Fig. 2) but the delay in washout previously noted on the ventilation scan at the right base was no longer observed. The chest radiograph remained normal (Fig. 3A). Due to the atypical scan findings, pulmonary angiography was performed 2 days after the last scan (Fig. 3B) and demonstrated obvious cutoffs of the right lower lobe pulmonary arteries compatible with embolization. In addition, there was decreased flow to the lateral segmental artery of the right middle lobe, probably reflecting additional small emboli. The patient's chest and right leg pain completely resolved and he was discharged on oral Coumadin medication.

DISCUSSION

Experience with the combined ventilation/perfusion lung scan has identified two major patterns of diagnostic significance: (A) absent or reduced blood flow to the lungs with preservation of normal ventilation and (B) marked reduction of both perfusion and ventilation (1). The former pattern has consistently reflected primary vascular disease including thromboembolism while the latter pattern has been associated with the presence of primary parenchymal disease. Figure 1 clearly shows an abnormality on the washout phase of the ventilation scan but no abnormality can be detected on the washin or single breathhold phase. Regional areas of delayed xenon clearance following equilibrium breathing may be expected during the washout phase in patients with bronchoconstriction. Transient bronchoconstriction has been demonstrated in animals following experimental pulmonary embolus; this usually resolves within minutes (2,3) but may persist as long as 4

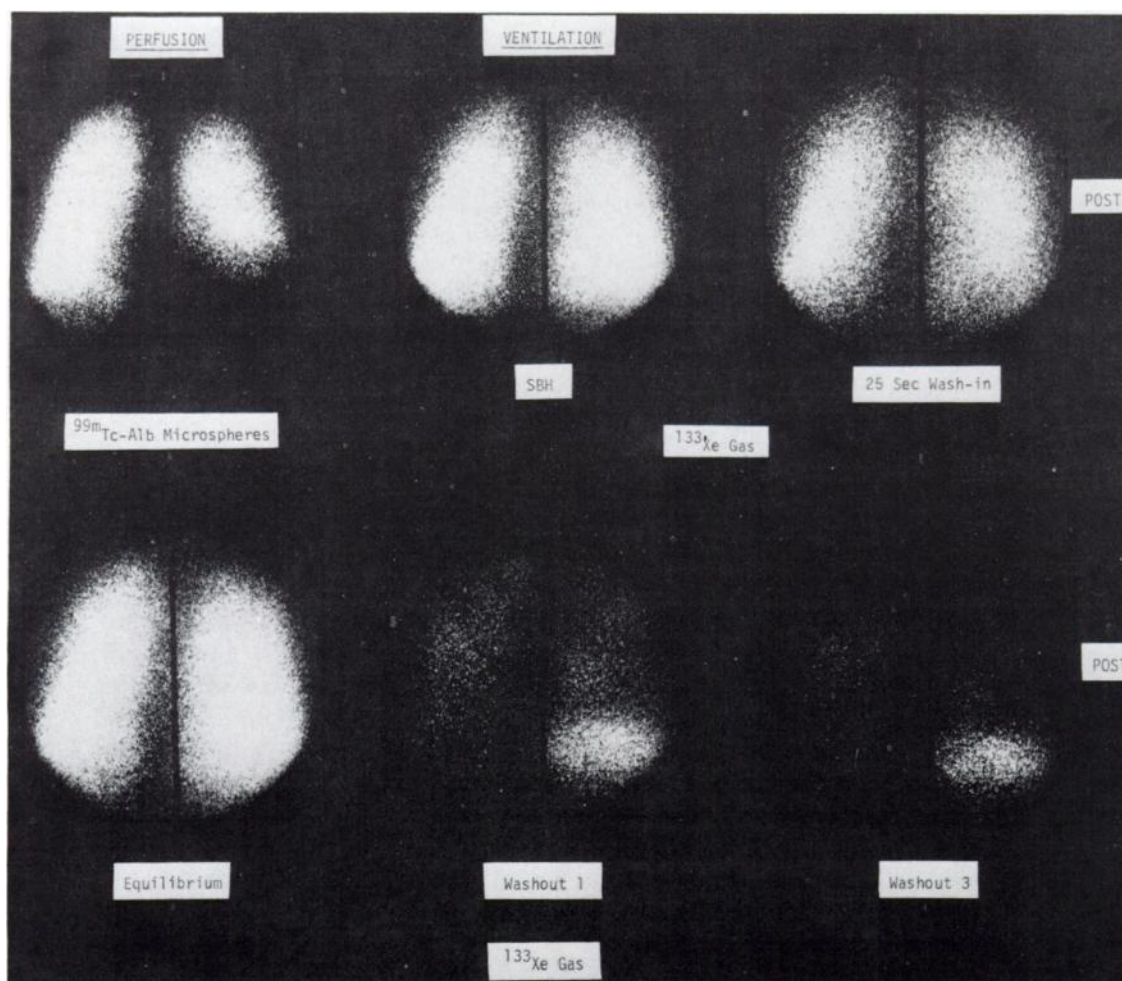


FIG. 1. Posterior V/Q scans taken on September 7, 1974. Perfusion scan (upper left) shows decreased perfusion involving right base. Ventilation scan shows normal pattern on single breathhold

and 25-sec washin scintiphotos but holdup of activity on washout phases of ventilation study (scintiphotos, lower right). V/Q pattern is consistent with parenchymal rather than embolic disease.

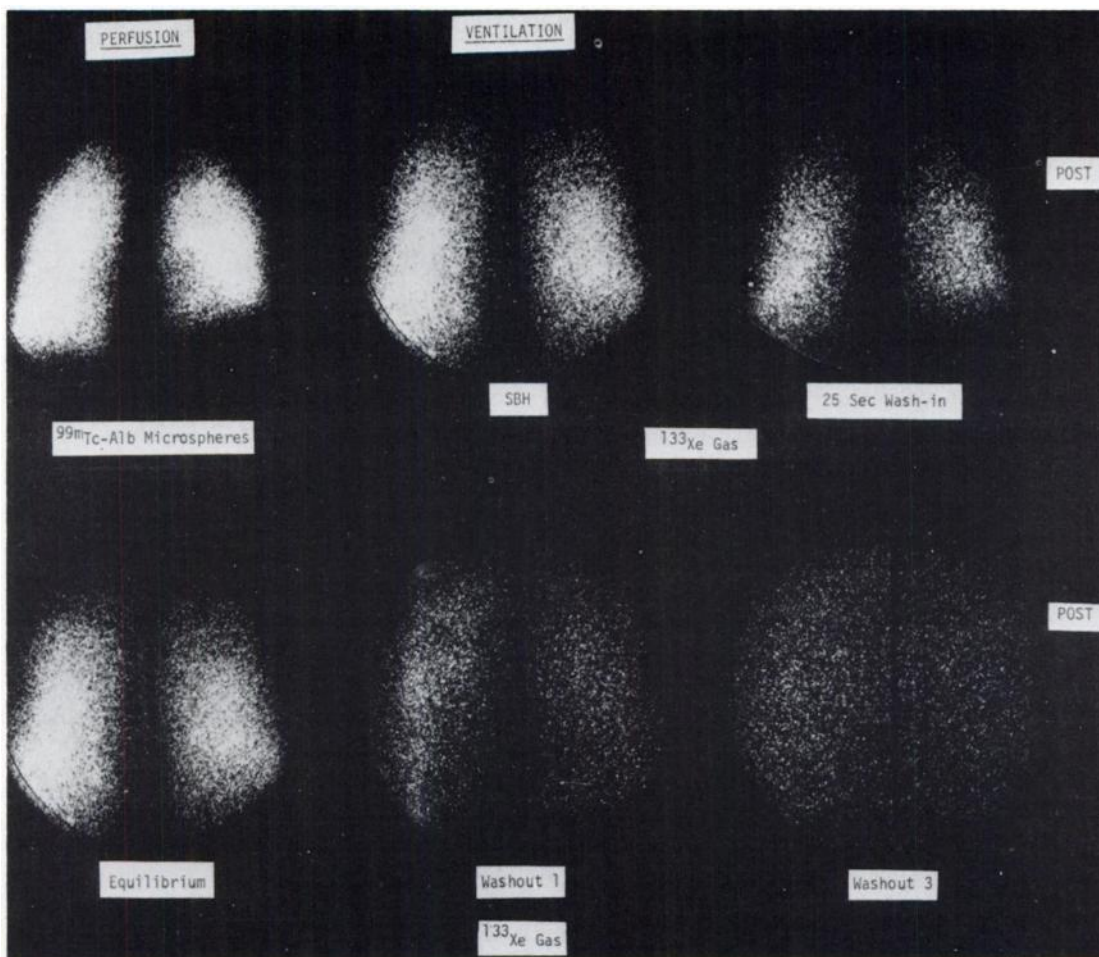


FIG. 2. Posterior V/Q scans taken on September 25, 1974. Perfusion scan again shows decreased counts of right base although there is slight improvement compared to study shown in Fig. 1.

Ventilation scan (washin—upper right; washout—lower right) is normal. V/Q pattern on this study is now consistent with pulmonary embolic disease.

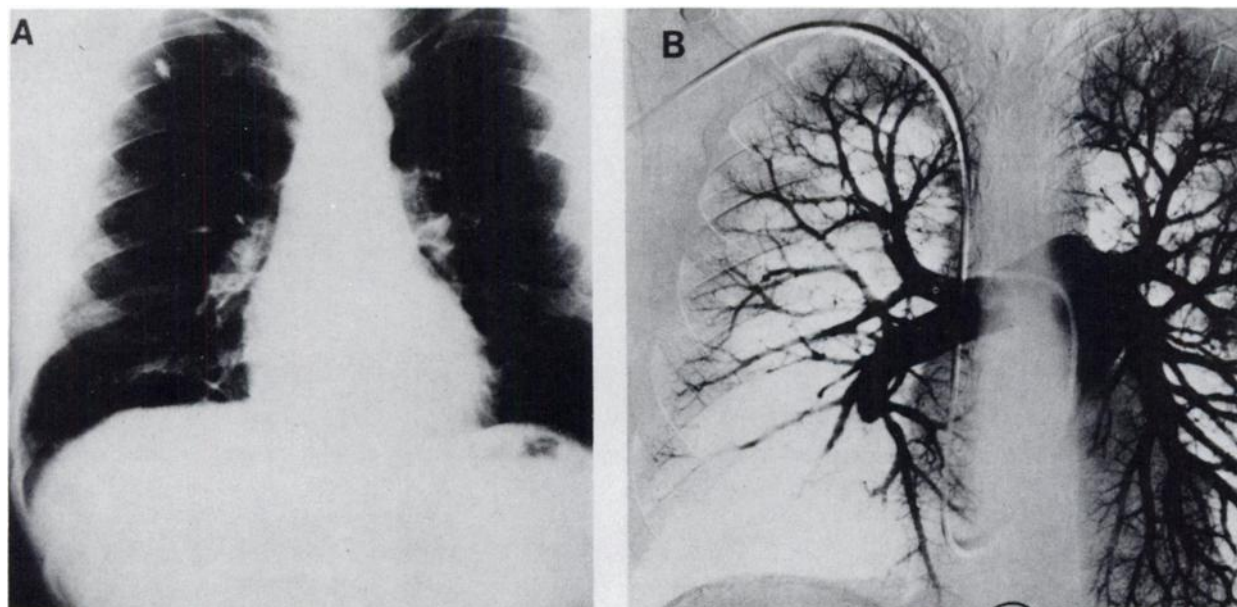


FIG. 3. (A) Anteroposterior chest roentgenogram taken on September 26, 1974. No infiltrates or regions of decreased vascularity are evident. Small calcified scar is noted in right upper lobe. (B)

Selected subtraction film from pulmonary angiogram shows cutoffs of right lower lobe pulmonary arteries. There is also decreased vascularity involving middle lobe.

hr (4) after the embolus. Stein has postulated that the bronchoconstriction observed in animal models may result from the release of humoral substances such as serotonin or histamine (5).

A recent case report showing a V/Q match at 5 hr after an acute embolic episode supports the experimental model and lends credence to the concept that similar mechanisms may function in man (6). It is more difficult to explain the case presented here in which a definite airways disease pattern was demonstrated 48 hr after the onset of clinical symptoms. It is conceivable, however, that multiple episodes of pulmonary emboli rather than a single occurrence might explain the scan findings, reflecting prolonged bronchoconstriction (7) with a concomitant ventilation abnormality observed days after the initial symptomatology. The possibility that the ventilation abnormality represented sustained bronchoconstriction following a single embolic event resulting in significant vascular compromise was also considered and supported by Boyer's statement: "In unanesthetized humans, bronchoconstriction may be more sustained than in the experimental model" (3).

We have no proven explanation for the matched V/Q abnormalities in this case. For clinical purposes, we suggest that pulmonary embolus cannot be excluded and that the diagnosis of parenchymal lung disease should be made with reservation in a patient who has a clinical history that strongly suggests pul-

monary embolus, has a clear x-ray, and has a single well-defined perfusion defect with matched abnormality on a ventilation scan. This is apparently true even in patients scanned days after the onset of clinical symptoms. These patients probably all deserve followup ventilation/perfusion scans to exclude vascular disease, and pulmonary angiography should be considered when there is a high index of suspicion.

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