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Radioisotope Pulmonary Scanning in the Pulmonary Vascular Bed Alterations

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肺血管床病変における肺スキヤニング

肺血流スキヤニングを肺の慢性閉塞性変化を有する17名の瀝青炭鉱夫、4例の逆シャントを有する心房中隔欠損症 (Eisenmenger's reaction) および2例の重症肺高血圧症を伴う僧帽弁狭窄症例に施行した。

シンチグラム上の肺葉ないし肺の縮少 ("shrunk lobe or lung sign") および "fissure sign" の原因となる辺縁での血流減少領域の拡大と肺辺

縁での放射線減少帯の不規則性が、各種原因による小血管床変化を有する全例で認められた。

肺スキヤニングは大血管による血流異常の診断に有用であるのみならず、小血管床病変の診断にも用いうる。放射性医薬品と装置の進歩改良によって、肺の小血管床での早期変化の診断が可能となり、これは患者の管理に大いに役立つであろう。

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With the recent advancement of cardiopulmonary hemodynamic knowledge, it is evident that the pulmonary vascular bed is receiving an increasingly large share of the attention of clinical and experimental investigation. However, the clinical diagnosis of the condition, especially in its earlier stages, is very difficult.

Since the development of pulmonary scanning with radioisotope labelled macroaggregated human serum albumin (MAA), scintigraphic studies of lungs are widely accepted and have become an essential tool in the diagnosis of abnormal pulmonary perfusion. There are numerous reports in the world literature concerning abnormal lung perfusion in pulmonary embolism, chronic obstructive lung disease, bronchial asthma, pulmonary infections and tuberculosis, pulmonary sarcoidosis, bronchogenic carcinoma, pulmonary mucoviscidosis, alpha-1-antitrypsin deficiency, congenital and acquired heart disease and pulmonary hypertension, demonstrated with radioisotope lung scanning. All these findings, however, only reflect gross regional blood flow defects.

Scintigraphic findings in pulmonary microembolism were recently reported experimentally by James *et al.*(9) and clinically by Eaton *et al.*(6, 7). They clearly showed that a diagnosis of pulmonary micro-

vascular bed alterations is feasible by the usual intravenous lung scan technique. It is the purpose of this paper to present certain characteristic scintigraphic abnormalities in known cases of pulmonary microvascular bed changes from various causes.

Materials and Methods

All the subjects in this study were adult and hospitalized in the West Virginia University Hospital (W.V.U.H.). Seventeen bituminous coal miners with chronic obstructive pulmonary disease, four cases of atrial septal defects with reverse shunts (Eisenmenger's reaction), and two patients with mitral stenosis having severe pulmonary hypertension were studied. All had dyspnea on exertion; however, no evidence of pulmonary embolism nor microembolism was noted.

All patients were prepared with 10 drops of oral Lugol's solution three to four hours prior to the lung scanning. Routine postero-anterior and left lateral chest roentgenograms at 72 inches were obtained in all cases. All patients received 300 microcuries of high specific activity microaggregated human albumin labelled with ^{131}I . This injection was given into an antecubital vein slowly during quiet respiration with the patient in the erect sitting position. An antero-posterior photoscan was begun 5 to 10 minutes after injection. In addition a right and left lateral scan was also obtained in all cases. A Pho/Dot scanner, Model 1735 Nuclear-Chicago Corporation, was employed for all examinations with a 19-hole focusing collimator.

Illustrative Cases

Case 1: C.T.L. This 59-year-old white male was admitted to West Virginia University Hospital for evaluation of exertional dyspnea, morning cough, and phlegm. He had worked in bituminous coal mines for 18 years, mainly in underground transportation as a motorman. He smoked approximately

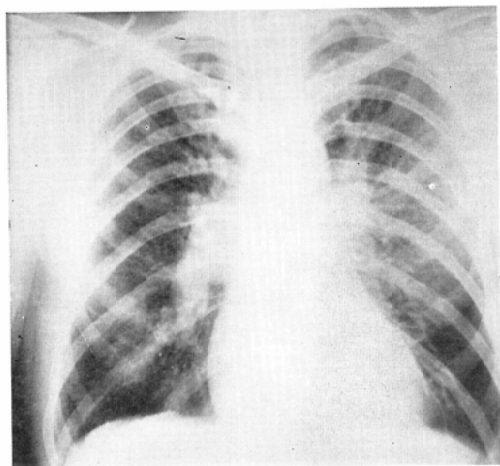


Fig. 1. Case 1. A bituminous coal miner with chronic obstructive pulmonary disease. Routine PA view of chest roentgenogram showing minimal generalized pulmonary emphysema and radiographic evidence of pulmonary arterial hypertension.

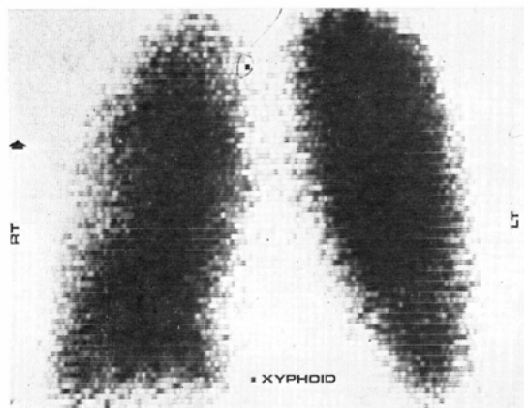


Fig. 2. Case 1. Frontal lung scan showing irregular curvilinear zone of decreased activity in the peripheral portion of the lungs, especially on the right. Increased activity in the left upper lung field suggesting pulmonary hypertension.

30 cigarettes per day for nearly 36 years.

Pulmonary function tests demonstrated severe obstructive airway disease. Total lung capacity was 143% and forced expiratory volume in one second was 40% of the predicted normal value. Residual volume occupied 68% of the total lung capacity compared with a predicted value of less than 40 per cent. Airway resistance was 5.4 cm H₂O/l/sec which is increased to nearly three times the predicted value of 1-2 cm H₂O/liter/second. Arterial blood gas analysis revealed severe hypoxemia with a compensated respiratory acidosis.

Right-sided cardiac catheterization at rest revealed severe pulmonary hypertension with a mean pulmonary artery pressure of 65 mm Hg, mean wedge pressure of 20 mm Hg, and a calculated arteriolar resistance of 10 units (800 dynes second cm⁻⁵).

The postero-anterior chest roentgenogram (Fig. 1) shows minimal generalized hyperinflation and linear fibrosis. Dilated proximal descending pulmonary arteries with peripheral narrowing in the lower lung fields and dilated upper lobe arteries can be seen. These findings are consistent with pulmonary arterial hypertension. The frontal lung scan (Fig. 2) shows an irregular curvilinear zone of decreased radioactivity in the peripheral portion of the lungs which is more pronounced on the right. There is increased radioactivity in the left upper lung field which is indicative of some pulmonary hypertension.

Case 2: S.R. This 59-year old white male was admitted to W.V.U.H. for evaluation of severe exertional dyspnea, cough, and phlegm. He had worked in a coal washing plant for 24 years and smoked approximately 20 cigarettes per day for nearly 25 years.

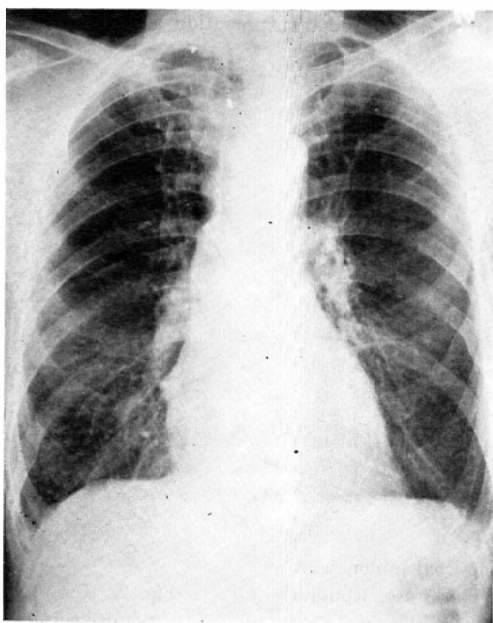


Fig. 3. Case 2. A bituminous coal miner with chronic obstructive pulmonary disease. Routine PA view of chest roentgenogram showing only minimal generalized pulmonary emphysema.

Pulmonary function tests demonstrated moderately severe obstructive airway disease. His total lung capacity was 156%, the forced expiratory volume in one second was 53% of predicted normal. Residual volume/total lung capacity ratio was 62%. Airway resistance was elevated to 3.6 cm H₂O/l/sec which is nearly twice the predicted value. Arterial blood gases showed moderate hypoxemia at rest without respiratory acidosis. The patient was unable to tolerate minimal exercise.

The postero-anterior chest film (Fig. 3) shows minimal generalized pulmonary emphysema. The pulmonary scan (Figs. 4A, B, and C) shows peripheral areas of hypoperfusion and an irregular, curvilinear zone of decreased activity around the circumference of the lung. There is also decreased

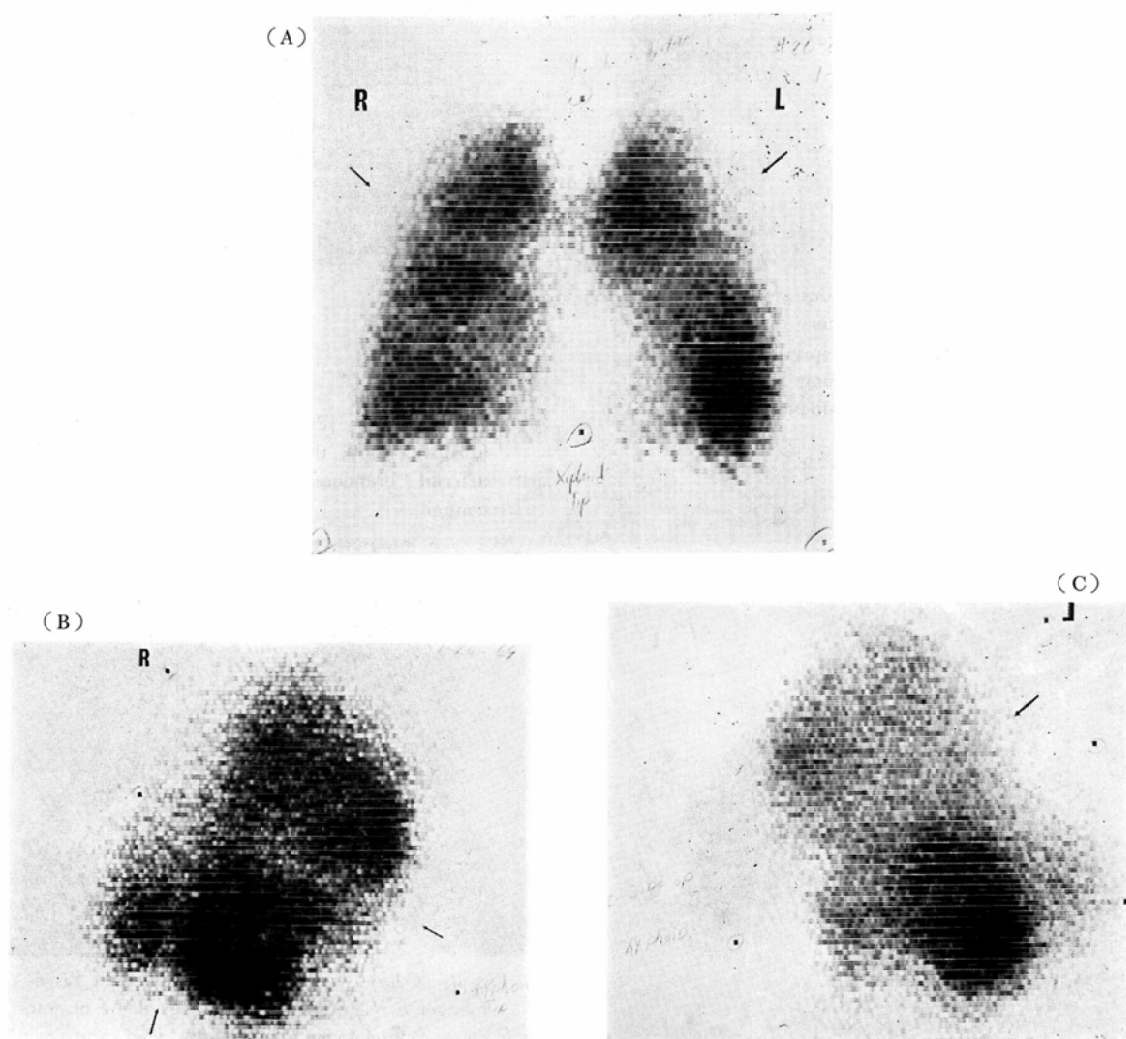


Fig. 4. Case 2. Frontal (A), right lateral (B), and left lateral (C) lung scans showing increased peripheral hypoperfusion areas, markedly irregular curvilinear zone of decreased activity around the circumference of the lung and circumferential decreased activity along fissures (arrows) producing "fissure sign."



Fig. 5. Case 3. A bituminous coal miner with chronic obstructive pulmonary disease. Routine PA view of chest roentgenogram showing essentially normal appearing lungs except for minimal left pleuro-phrenic adhesions.

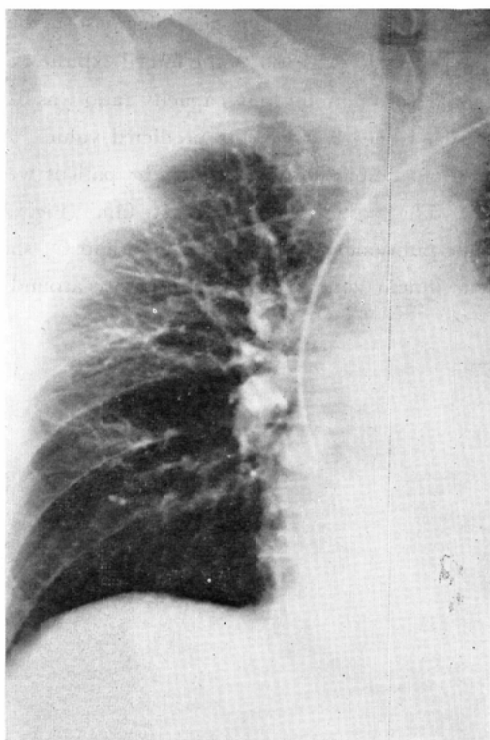


Fig. 7. Case 3. Pulmonary arteriogram shows abrupt cut-off the distal ends of pulmonary arterial branches suggesting vascular bed changes.

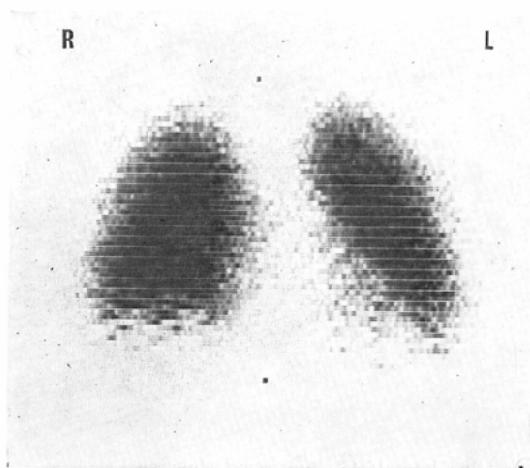


Fig. 6. Case 3. Frontal pulmonary scan showing increased and irregular peripheral hypoperfusion areas.

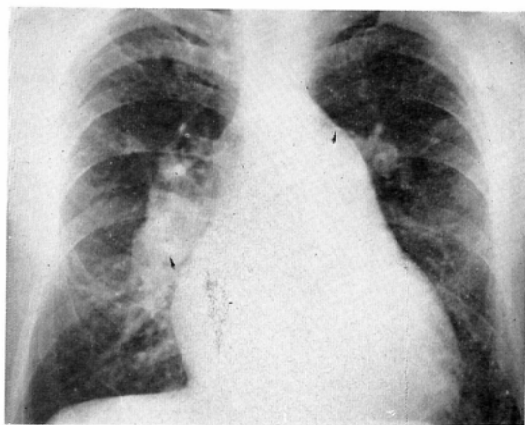


Fig. 8. Case 4. Atrial septal defect with Eisenmenger's reaction. Routine PA view of the chest roentgenogram showing marked right ventricular enlargement, markedly prominent main pulmonary artery segment with calcifications (arrow) and markedly dilated central pulmonary arteries with calcifications (arrow).

activity along the fissures producing the so-called "fissure sign" (Figs. 4 and 6).

Case 3: G.J.E. This 43-year old white male was admitted to W.V.U.H. because of exertional dyspnea, cough, phlegm, and wheezing. He had worked as a buggy operator in a bituminous coal mine for 12 years and had smoked approximately 20 cigarettes per day for 13 years. His symptoms had begun about two years prior to his admission to the hospital.

Pulmonary function tests showed moderately severe obstructive impairment. Total lung capacity was 155% and forced expiratory volume in one second was 65% of predicted normal. Residual volume/total lung capacity ratio was 65%. Airway resistance was elevated to 3.6 cm H₂O/l/sec which is nearly twice the predicted value. Arterial blood gases were normal at rest.

Right-sided cardiac catheterization revealed a mean pulmonary artery pressure of 20 mm Hg and a mean wedge pressure of 12 mm Hg.

The postero-anterior view of the chest film (Fig. 5) is normal except for minimal tenting deformity of the left diaphragm consistent with a pleurophrenic adhesion. The pulmonary scan (Fig. 6) shows irregular peripheral areas of decreased perfusion in both lungs. A pulmonary arteriogram (Fig. 7) also shows abrupt cut-off at the distal ends of arterial branches indicating alterations in the vascular bed.

Case 4: O.R. This 55-year old white male was admitted to West Virginia University Hospital for evaluation of severe dyspnea and cyanosis. At age 22 he was released from the Army Air Corps be-

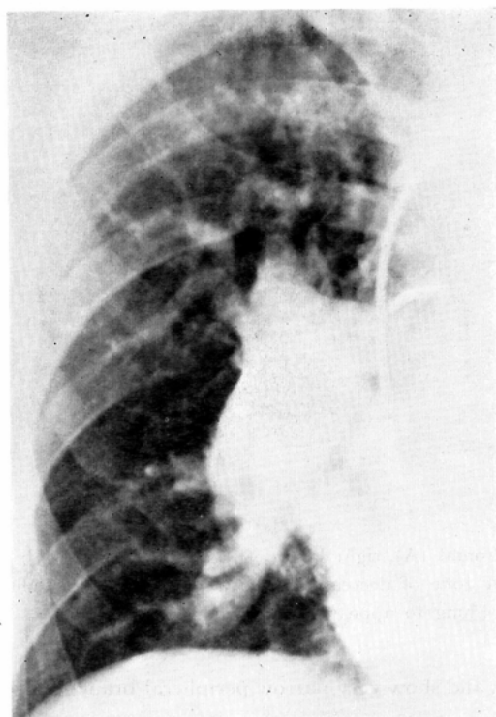


Fig. 9. Case 4. Pulmonary arteriogram showing pruned tree-like appearance of distal ends of arteries suggesting vascular bed changes. Note markedly dilated central pulmonary arteries with pulmonary arterial hypertension with atrial septal defect.

cause of heart disease although he had no symptoms at that time. He began noticing dyspnea on exertion associated with cyanosis of the lips, tongue, and fingernails in 1950.

Physical examination confirmed the presence of severe cyanosis of the lips, mucous membranes, and fingernails associated with moderate clubbing of the fingers. The pulmonary second sound was loud and heard prominently all across the anterior chest. An electrocardiogram showed marked right axis deviation and right ventricular hypertrophy.

Right-sided cardiac catheterization revealed marked pulmonary hypertension with a mean pulmonary artery pressure of 70 mm Hg, mean wedge pressure of 6 mm Hg, and a calculated pulmonary arterial resistance of 17 units ($1360 \text{ dyn/sec/cm}^5$).

A postero-anterior view of the chest film (Fig. 8) shows marked right ventricular enlargement, and a prominent main pulmonary artery segment which is calcified. Both descending pulmonary arteries

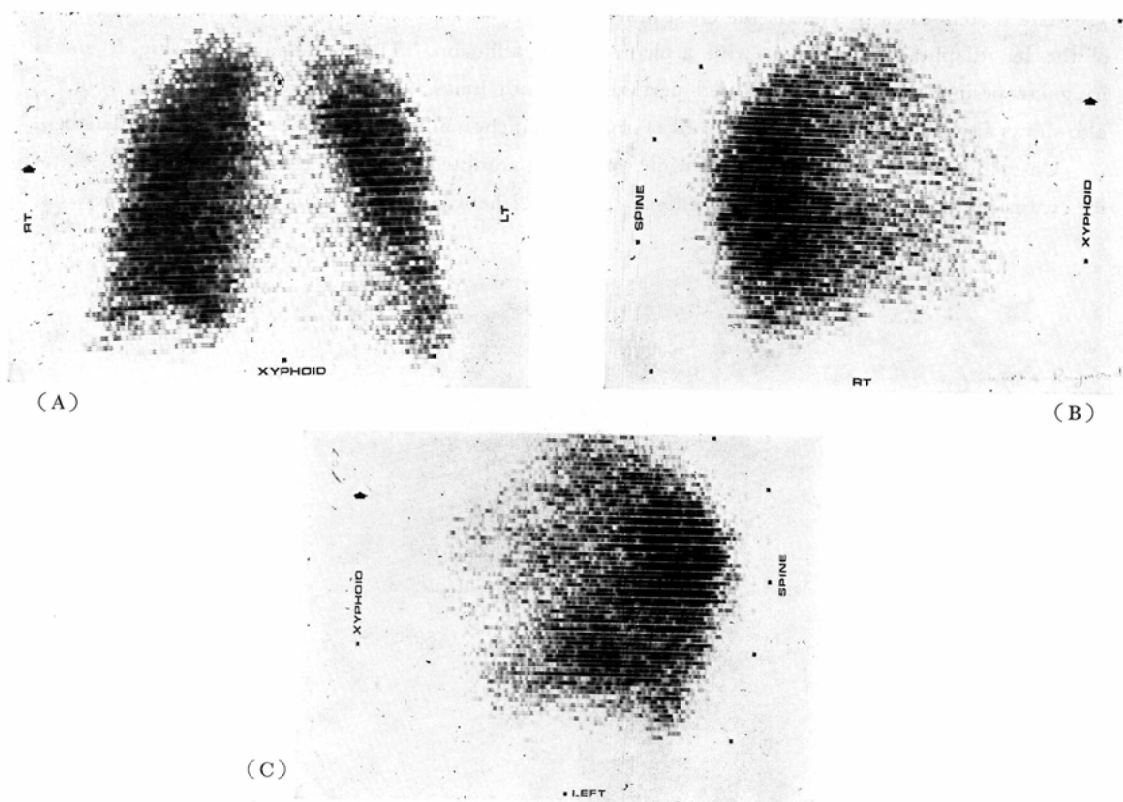


Fig. 10. Case 4. Frontal (A), right lateral (B), and left lateral (C) lung scans showing irregular curvilinear zone of decreased activity around the circumference of the lungs causing the lobes of lung to appear small in volume.

are markedly dilated, calcified, and show very narrow peripheral branches in the lower lobes. The upper lobe arteries are dilated. These findings are typical of marked pulmonary arterial hypertension. A pulmonary arteriogram (Fig. 9) shows the "pruned tree" appearance of the distal ends of arteries demonstrating the peripheral vascular bed as the site of vascular obstruction. The pulmonary scan (Figs. 10A,

B, and C) shows an irregular, curvilinear zone of decreased activity around the circumference of the lungs causing the lobes to appear smaller in volume than is evident from the postero-anterior chest film.

Case 5: G.S. This 50-year old white female was admitted to West Virginia University Hospital because of severe dyspnea. She had rheumatic fever many years ago. The symptoms of dyspnea began several years prior to this admission and were progressive. Physical examination revealed findings typical of mitral stenosis. Right-sided cardiac catheterization revealed a mean pulmonary artery pressure of 70 mm Hg, mean wedge pressure of 34 mm Hg, and a calculated pulmonary arterial resistance of 8 units ($640 \text{ dyn/sec/cm}^{-5}$).

The postero-anterior view of the chest film (Fig. 11) shows a characteristic mitral configuration of the heart and evidence of pulmonary arterial and venous hypertension. The pulmonary scan (Fig. 12) shows areas of peripheral hypoperfusion in both lungs. Increased activity in both upper lung fields

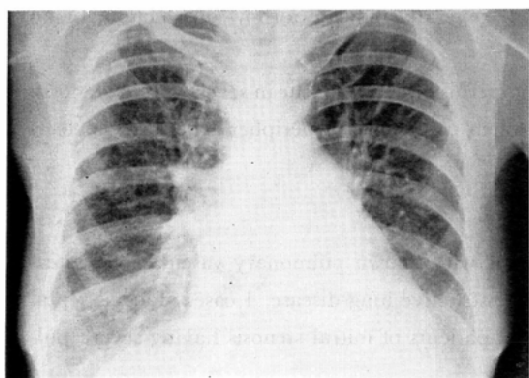


Fig. 11. Case 5. Mitral stenosis with severe pulmonary arterial and venous hypertension. Routine PA view of chest roentgenogram showing a characteristic mitral configuration of heart with evidence of pulmonary arterial and venous hypertension.

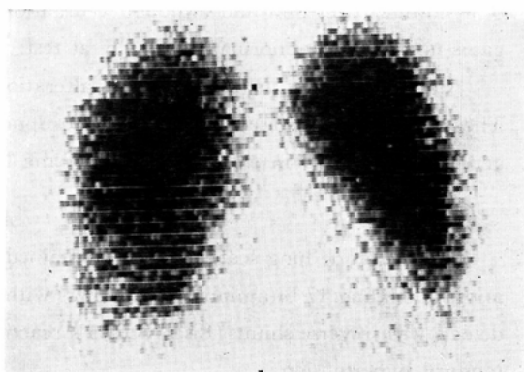


Fig. 12. Case 5. Frontal pulmonary scan showing increased and irregular peripheral hypoperfusion areas in both lungs. Note increased activities in both upper lung fields suggesting pulmonary hypertension.

is also evident, a finding which is frequently seen in cases of pulmonary hypertension.

Discussion

All our patients with abnormal pulmonary vascular bed changes even from different etiologies showed similar scintigraphic manifestations in lung scans.

Peripheral hypoperfusion areas are increased and showed irregular zones of decreased activity around the circumference of the lung causing the lobe or lung to appear small in volume, "shrunk lobe or lung sign" (Figs. 2, 4, 6, 10, and 12). There is also seen circumferential decreased activity along the fissure producing "fissure sign" (Fig. 4). These various scintigraphic manifestations probably reflect various severities of the disease and different anatomical sites of the involvements but also superimposed parenchymal scars.

These scintigraphic abnormalities concur with the findings in pulmonary microemboli which were

reported by James, *et al.* (9) and Eaton and his associates (6, 7). These were not only specific to the pulmonary microemboli but are most probably the scintigraphic reflection of microvascular bed changes of the lung.

Cases 1 and 4 illustrate abnormal perfusion patterns associated with marked pulmonary hypertension. In Case 1 these are the result of severe chronic bronchitis and emphysema; in Case 4 they are the result of vascular changes within the lungs induced by long exposure to excessive blood flow. Similar abnormalities in lung scans associated with pulmonary hypertension have been noted by others (4, 5, 8).

The lung scan in Case 2 shows a pattern similar to that seen by Eaton, *et al.* (6, 7) and James, *et al.* (9) in patients with pulmonary microembolism. However, in this case, the pulmonary function studies indicate that severe chronic bronchitis and emphysema are producing the perfusion defects.

Abnormal lung perfusion is seen in scans obtained from patients with asthma (2, 10) and is illustrated in Case 3 who has chronic obstructive pulmonary disease with asthma. This may occur in the presence of a normal chest film and although ventilation tests will generally indicate airflow obstruction, blood gases need not be abnormal, especially at rest.

Case 5 illustrates the more complex alterations in lung perfusion which occur in severe mitral stenosis. These consist of increased perfusion in the upper zones often coupled with peripheral perfusion defects generally occurring in the lower zones of the lungs.

Abstract

Radioisotope lung scannings were obtained in patients with known pulmonary vascular bed alterations including 17 bituminous coal miners with chronic obstructive lung disease, 4 cases of atrial septal defects with reverse shunts (Eisenmenger's reaction), and 2 patients of mitral stenosis having severe pulmonary hypertension.

Scintigraphic abnormalities consisting of increased width of peripheral hypoperfusion areas, irregular zones of decreased activity around the circumference of the lung causing the lobe or lungs to appear shrunken ("shrunken lobe or lung sign") or "fissure sign" were demonstrated in all our cases with known microvascular bed change of the lung from various causes.

Radioisotope pulmonary scanning is not only useful in the diagnosis of gross pulmonary perfusion abnormalities but it is also feasible to assess the pulmonary microvascular bed alterations. With improvement of radioactive scanning materials and instrumentations, the diagnosis of early changes in pulmonary microvascular bed may be possible and this will tremendously assist in the management of the patient.

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