

General Practice

Radiological Features of Pulmonary Oedema

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SUMMARY

A clinical classification embracing most of the causes of pulmonary oedema is given, as well as a radiological classification, and the different ways in which pulmonary oedema may present radiologically are briefly described.

S. Afr. med. J., 55, 792 (1979).

Pulmonary extravascular fluid balance, like extravascular fluid balance anywhere in the body, depends on the following factors: (a) hydrostatic pressure in the capillaries and in the interstitial tissue; (b) osmotic pressure in the capillaries and in the interstitial tissue; (c) permeability of the capillaries; (d) capacity of the lymph vessels to drain interstitial fluid. In addition, surfactant and intra-alveolar gas pressure in the lungs have an influence on extravascular fluid balance.

Pulmonary oedema may develop whenever there is a sufficiently severe derangement in any of these mechanisms. The lung has, however, a big reserve, and pulmonary capillary pressure may increase from the normal of about 7 mmHg to 20-25 mmHg, and pulmonary lymphatic drainage may increase manyfold before overt pulmonary oedema develops.

Many conditions can derange one or more of these normal controlling mechanisms to such a degree that pulmonary oedema develops. In some conditions the mechanism of production of pulmonary oedema is simple, but in most conditions it is complex and in some it is ill-understood.

Whatever the cause or mechanism of pulmonary oedema there is a wide variety of radiological appearances spanning a wide spectrum. Although there are certain clues and certain associations, in the individual case the cause or mechanism of pulmonary oedema cannot be deduced from the radiological appearance.

CLINICAL CLASSIFICATION

Because of the complexity of the mechanisms of production of pulmonary oedema, the causes of pulmonary oedema are best approached from the clinical point of view and grouped as follows:

Cardiac causes. These include left ventricular failure, mitral valve disease, left atrial myxoma, cor triatriatum

and pulmonary venous obstruction, either congenital or acquired. These causes operate mainly by raising the pulmonary venous and pulmonary capillary pressure.

Renal causes. These include acute nephritis and chronic renal failure as well as renal involvement in lupus erythematosus, polyarteritis nodosa, Goodpasture syndrome, etc. These causes work mainly through disturbed water and electrolyte balance, damage to the pulmonary capillary membrane and perhaps alterations in the plasma proteins.

Central nervous system causes. These include head injury, intracranial haemorrhage, epileptic attack, and brain tumour. These probably work through the adrenergic nervous system with liberation of circulating catecholamines which increase pulmonary capillary pressure.

Pulmonary causes. These include irritation due to inhalation of irritant gases such as ozone, nitrous oxide, wood smoke, chlorine or phosgene, or aspiration of irritant fluids such as gastric contents (Mendelson syndrome) or water (drowning in salt or fresh water); overwhelming infections, especially those due to viruses such as influenza viruses, but also with bacteria such as Friedländer's bacillus; trauma to the chest with pulmonary contusion; prolonged inhalation of oxygen at high concentration causing pulmonary oxygen intoxication; pulmonary arteritis in, for example, lupus erythematosus and polyarteritis nodosa; asphyxia with damage to the pulmonary capillaries by anoxia; rapid aspiration of pleural fluid or a pneumothorax; and pulmonary embolism, either thrombo-embolism, fat embolism or amniotic fluid embolism.

Hypo-albuminaemia. This may be a main cause or a contributing factor in renal disease, hepatic disease, protein-losing enteropathy or severe malnutrition such as kwashiorkor.

Diverse causes. These include 'shock lung' (adult respiratory distress syndrome, post-traumatic pulmonary insufficiency); high altitude pulmonary oedema; allergic reactions, for example due to nitrofurantoin, busulphan or penicillin; overdosage with narcotics, especially heroin; overtransfusion with fluid or blood; circulating 'toxins', for example alloxan or snake venom or toxins in 'septic shock' and eclampsia; and can also occur during pregnancy or after confinement (possibly amniotic fluid embolism).

Depending on the cause of pulmonary oedema, there may or may not be cardiac enlargement and/or evidence of raised pulmonary venous pressure.

RADIOLOGICAL CLASSIFICATION

Pathologically and radiologically, pulmonary oedema can be classified as: (i) interstitial pulmonary oedema; (ii) intra-alveolar pulmonary oedema; (iii) mixed interstitial and intra-alveolar pulmonary oedema.

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Interstitial Pulmonary Oedema

The radiological features of interstitial pulmonary oedema are as follows: oedematous interlobular septa with associated dilated lymph vessels become radiologically recognizable as Kerley A, B and C lines and Kreeel D lines (Fig. 1). Kerley B lines are the best known and are most commonly seen. They appear as short, thin sharply defined hairline shadows situated horizontally and extending perpendicularly to the pleural surface and they are most numerous in the lower lung fields, especially in the costophrenic sulci. They are due to oedematous interlobular septa and dilated lymph vessels in the lung periphery.

Kerley A lines are longer, somewhat angular hairline opacities best seen in the upper and mid-lung zones and extending towards the hili. They are caused by oedematous interlobular and intersegmental septa and dilated lymph vessels situated more centrally in the lungs. Kerley C lines are only rarely seen and appear as a fine reticular pattern probably due to superimposition of fine oedematous interlobular septa. Kreeel D lines are thicker and longer than Kerley A, B or C lines, being up to 2 mm in width and 5-7 mm in length. They are often sharply angulated and are more often seen in the anterior parts of the lungs as viewed on lateral radiographs. They also are due to oedematous interlobular and intersegmental septa.

Oedematous interstitial tissue adjacent to the visceral pleura and on both sides of the interlobar fissures becomes radiologically visible as so-called thickened pleura, thickened interlobar fissures or lamellar pleural effusions.

Interstitial oedema extending from the hilar regions into the lung fields imparts a slight 'ground-glass' veiling to the hilar regions and obliterates the sharp demarcation of the hilar pulmonary vessels, so that these vessels now become poorly defined and blurred in outline. This is the so-called hilar or perihilar clouding (Fig. 2).

As the interstitial oedema extends further into the lung fields, the slight 'ground-glass' veiling and the blurring of the pulmonary vessels extend further out from the hilar regions producing so-called pulmonary clouding.

The oedematous perivascular and peribronchial sheaths cause blurring of vessel outlines and thickening of bronchial walls; the latter can, however, only be recognized when a bronchus is seen end on.

Intra-alveolar Pulmonary Oedema

The radiological features of intra-alveolar pulmonary oedema are as follows: as fluid accumulates in the alveoli the hilar and basal lung regions assume a finely granular appearance. This soon becomes patchy, giving poorly defined, irregular, blotchy, coalescent opacities in the lung fields. Together with these opacities the pulmonary vessels become indistinct or invisible and if the opacities are extensive enough an air bronchogram may become visible (Fig. 3).

The pulmonary opacities vary in size from tiny nodules with a fine miliary pattern to big confluent opacities filling most of the lung fields. The commonest picture is probably that of medium-sized opacities symmetrically and uniformly distributed throughout both lungs, but sparing the

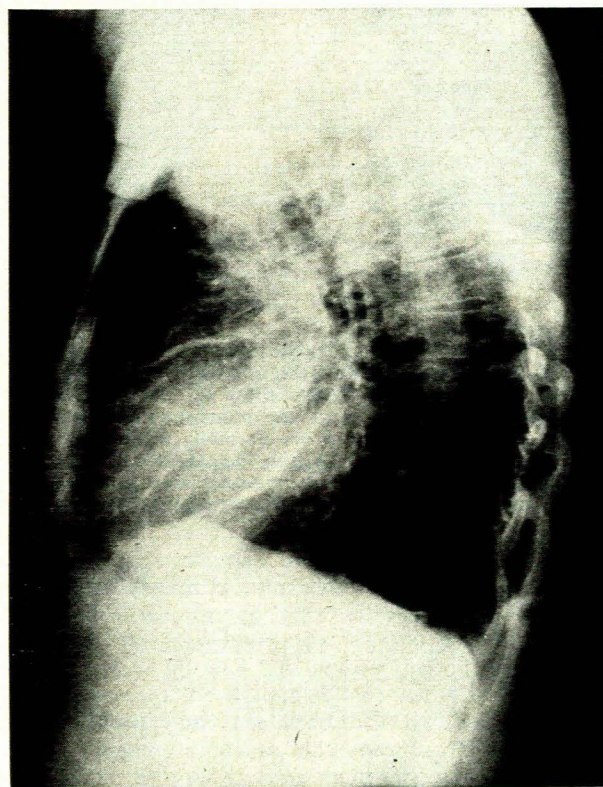
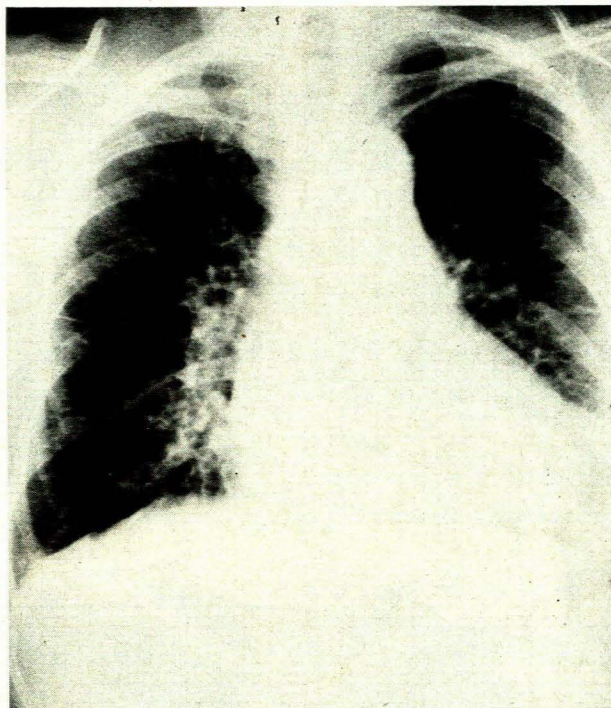


Fig. 1. Interstitial pulmonary oedema due to left ventricular failure. Top: Kerley B lines are seen as thin, horizontal, hairline opacities at the lung bases; bottom: Kerley D lines are seen as long, angulated, strandlike opacities in the anterior lung fields. Note also the oedematous horizontal and oblique interlobar fissures.

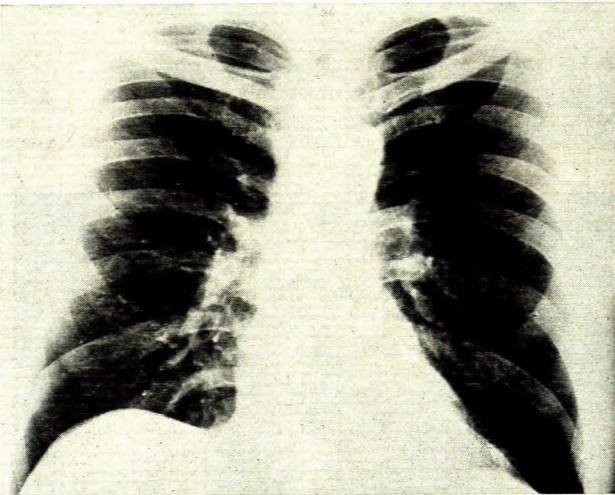
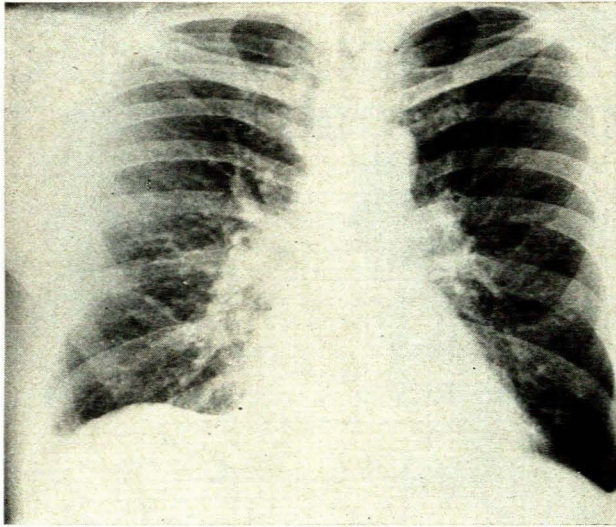


Fig. 2. Interstitial pulmonary oedema due to left ventricular failure. Top: the hilar regions show 'ground-glass' veiling and the sharp demarcation of the pulmonary vessels is obliterated; bottom: the pulmonary oedema has cleared up, the hilar regions are normally translucent and the pulmonary vessels are sharply demarcated. Note also how the engorged upper lobe vessels have returned to normal and how the heart has decreased in size.

extreme pulmonary apices and extreme lung periphery. When these opacities are concentrated mainly around the hilar areas, a so-called 'batwing' appearance is produced. The opacities are not always symmetrical or widespread and they may be confined to one lung or even one lobe. These opacities often change rapidly in appearance, size and distribution and with treatment they may clear in 24-48 hours. The speed with which this change occurs is often the most important or only way to differentiate pulmonary oedema from pulmonary infection.

Mixed Intra-alveolar and Interstitial Oedema

Intra-alveolar and interstitial pulmonary oedema often

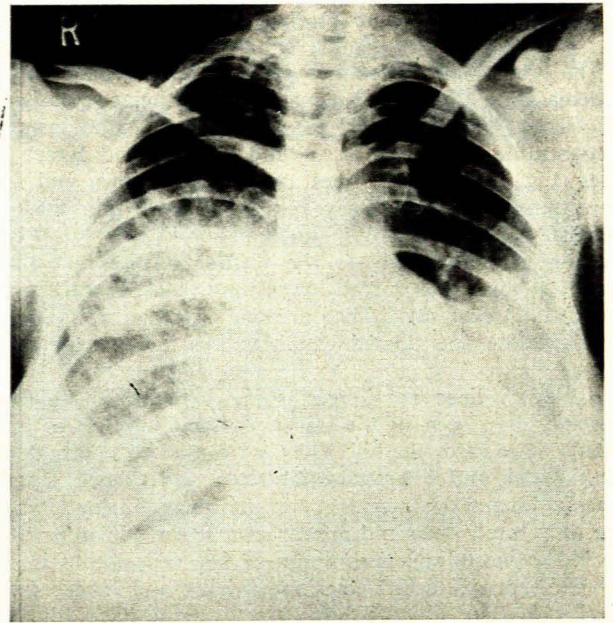


Fig. 3. Intra-alveolar pulmonary oedema in mitral stenosis. Note the poorly defined coalescent opacities which obscure the pulmonary blood vessels.

occur together and then the changes of intra-alveolar oedema mask or completely obliterate the changes of interstitial oedema.

Unusual distributions of pulmonary oedema can occur when certain factors either prevent or precipitate oedema in a certain part of the lung. Unilateral pulmonary oedema may occur in the dependent lung in unconscious or ventilated patients who lie for a long time on one side; when gastric contents are aspirated into one lung; when pulmonary contusion or veno-occlusive disease affects one lung only; after rapid aspiration of a large pleural effusion or pneumothorax (Fig. 4); when there is hypoplasia of one pulmonary artery or a big unilateral pulmonary embolus; or when one lung is severely underperfused as in Macleod's syndrome (Fig. 5) or in the scimitar syndrome.

Pulmonary oedema from whatever cause diminishes pulmonary compliance and this in turn causes elevation of both domes of the diaphragm. Together with pulmonary oedema, especially if it is of long standing, there may be unilateral or bilateral pleural effusions.

CONCLUSIONS

No specific pattern of pulmonary oedema can be attributed to any specific cause, but certain clues and certain generalizations can be made:

1. Dilated upper lobe vessels or so-called upper lobe blood diversion will indicate raised pulmonary venous pressure most likely due to a mitral valve lesion or to left ventricular failure.

2. An enlarged heart shadow will probably indicate a cardiac or renal cause.

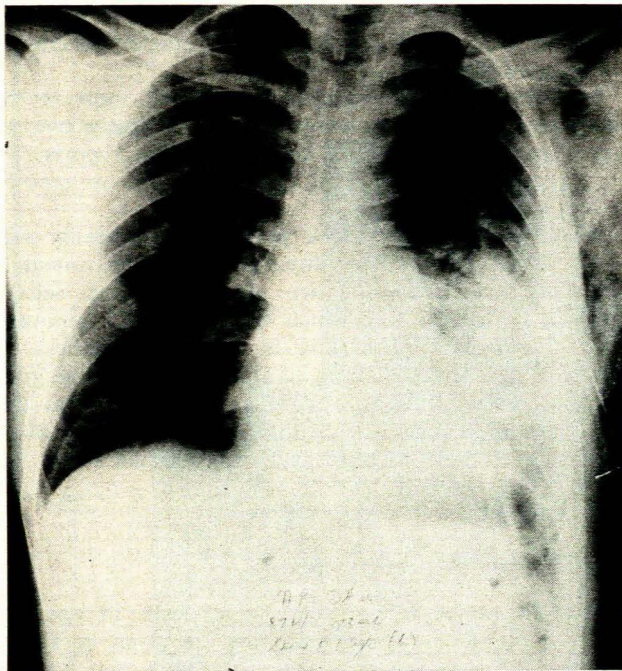
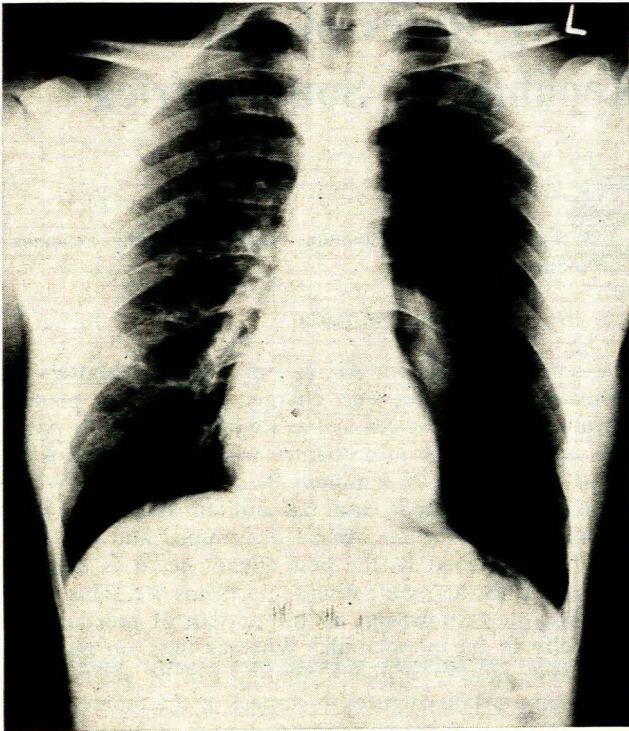


Fig. 4. Top: a big left-sided pneumothorax; bottom: pulmonary oedema involving the left lung after aspiration of the pneumothorax.

3. Slowly increasing pulmonary capillary pressure tends to produce predominant interstitial pulmonary oedema, and this is well seen in mitral valve lesions and left

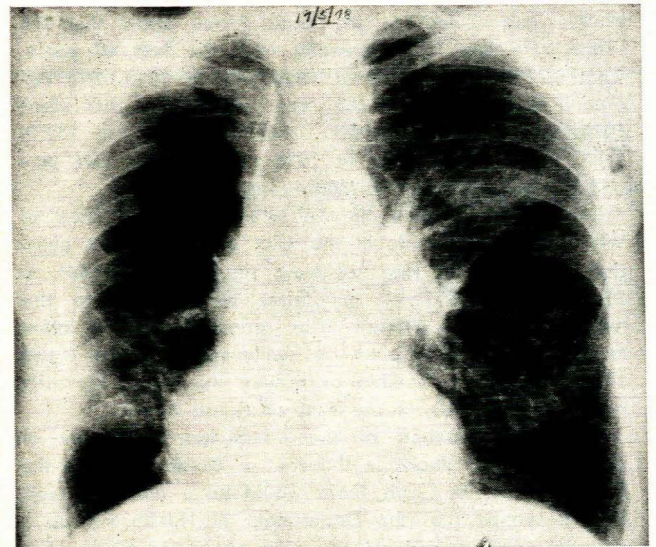
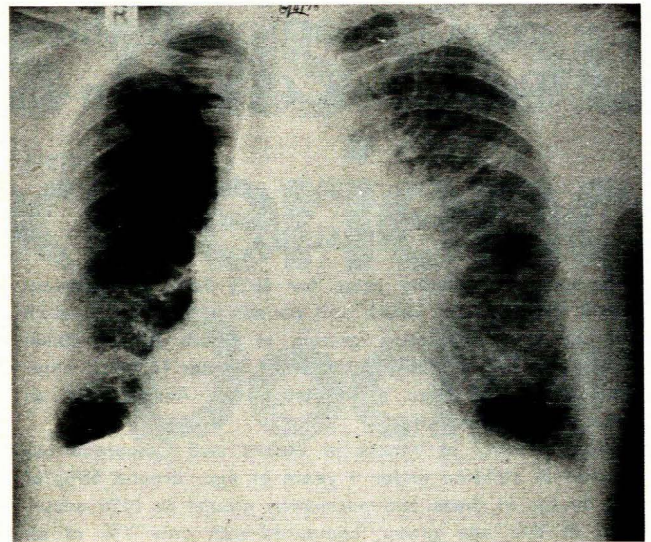


Fig. 5. Top: interstitial pulmonary oedema involving the normal left lung in a patient with a Macleod syndrome of the right lung; bottom: the pulmonary oedema has largely cleared from the left lung. Note that the hyperlucent, underperfused, abnormal right lung remains unchanged.

ventricular failure when the latter develops slowly.

4. Acute incidents tend to produce predominant intra-alveolar pulmonary oedema as seen in coronary thrombosis, acute left ventricular failure or acute glomerulonephritis.

5. The 'batwing' type of pulmonary oedema tends to occur in renal failure, acute left ventricular failure, lupus erythematosus and polyarteritis nodosa.

An awareness of the numerous causes and the wide spectrum of radiological appearances of pulmonary oedema will help in the diagnosis of obscure pulmonary opacities.