RADIOLOGIC NOTES IN CARDIOLOGY

Lung Changes in Left Heart Failure

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SUMMARY

The lung changes in left heart failure are grouped into four main categories or stages: (1) pulmonary congestion, (2) interstitial edema, (3) alveolar edema, and (4) miscellaneous findings. Some patients, particularly those with mitral stenosis, develop these changes gradually, passing successively through the stages of pulmonary congestion → interstitial edema → alveolar edema. Other patients, particularly those with hypertension or myocardial infarction, often develop rapid left heart failure and tend to show alveolar edema. The roentgen diagnosis of incipient or early heart failure is important since pulmonary auscultatory findings may be minimal or absent.

Additional Indexing Words:
A lines Pulmonary congestion
B lines Pulmonary edema
C lines Pulmonary venous hypertension

A N ORDERLY classification is of value in analyzing the lung changes in left heart failure1 (table 1). Some patients, especially those with mitral stenosis, develop the changes gradually, passing successively through the stages of pulmonary congestion → interstitial edema → alveolar edema; sometimes these stages overlap. Other patients, particularly those with hypertension or myocardial infarction, often develop rapid left heart failure and tend to show alveolar edema, often with A lines.

Pulmonary Congestion

The significant roentgen finding of pulmonary venous congestion is distention of the upper lobe pulmonary veins on an upright chest film. A recumbent film is unreliable since the upper lobe veins are normally more prominent in the recumbent projection. Concomitant with the upper lobe venous distention, the lower lobe veins may be normal (fig. 1) or constricted (fig. 2). Thus there is a reversal of the normal situation in which the lower lobe veins are larger than the upper lobe veins.2

In the peripheral lung fields, the distended veins resemble a reindeer’s antlers or a TV antenna (fig. 1). In the hilar area, best perceived on the right side, the distended upper lobe vein crosses the upper portion of the hilum and produces a change in its contour. Normally, the vein crosses the hilum and produces a sharp angle (hilar angle) at the lateral margin of the hilum. Therefore, the lateral aspect of the normal hilum is somewhat concave. When the upper lobe vein is distended, it causes the lateral aspect of the hilum to appear convex. The upper portion of the hilum is enlarged, and the

Table 1

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Prominent pulmonary vein seen end-on may simulate a tumor (fig. 2). In spite of these changes, the hilar margins remain sharply outlined and are not hazy.

**Interstitial Edema**

Interstitial edema generally occurs when the pulmonary venous pressure exceeds 25 mm Hg, the plasma osmotic pressure. Interstitial edema consists of (1) septal edema, (2) perivascular edema, and (3) subpleural edema. There are usually no auscultatory findings of pulmonary edema.

**Septal Edema**

The roentgen appearance varies with the anatomic arrangement of the septa in different areas of the lung. These were described by Kerley as A, B, and C lines.

A *Lines*. A lines represent edematous interlobular septa in the upper lung fields. The lines are straight and nonbranching, measuring up to 5–10 cm in length. They run diagonally toward the hilar areas (fig. 3). They are more frequently seen in acute left ventricular failure, and are somewhat evanescent, occurring mainly in the first 24–48 hours. Although they may be seen in any type of left ventricular failure, they are most frequently noted soon after acute myocardial infarction. Other signs of interstitial edema are usually present.

B *Lines*. In heart disease, B lines represent dilated interlobular septa due to edema of the septa. B lines are shorter than A lines. They are also nonbranching and are generally seen at the periphery of the lower lung fields (fig. 4). They extend to the pleural surface and are typically perpendicular to the pleural surface. B lines usually disappear after cardiac compensation has been established. In some cases the B lines become permanent due to septal fibrosis.

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C. Lines. C lines represent small basilar interlobular septa superimposed in a random fashion. Occasionally, they are seen as short crisscrossing nonbranching lines in the basilar regions, resembling superimposed septa (fig. 5). However, in most cases a honeycomb or reticular pattern is produced (fig. 6). Roentgenograms of good quality are essential, since respiratory motion during film exposure will obscure the reticular pattern. For this reason, C lines are seldom seen in films made at the bedside.

Perivascular Edema

Perivascular edema represents interstitial fluid around the blood vessels. Roentgenologically it produces changes both in the hilar regions and the periphery of the lungs.

Hilar Haze. The hila appear large, with ill-defined margins (fig. 7). There is loss of the clear space normally present between the right hilum and the heart, although this may partly be due to central alveolar edema.

Peripheral Haze. Peripheral haze is often incorrectly called pulmonary “congestion” but actually it represents interstitial edema. The lungs show increased density due to a diffuse haziness (fig. 7). The margins of the pulmonary vessels are indistinct.

Subpleural Edema

Subpleural edema represents the extension of interstitial edema to the periphery of the lung and is usually seen where it lies against a pleural fissure. It appears as an elongated density, much like that of a thickened interlobar fissure (fig. 7). The pleural margin may be sharp, but the margin facing the lung is not well defined, thus distinguishing subpleural edema from interlobar effusion.
Figure 3

Interstitial edema—A lines. Straight nonbranching lines are present, representing A lines, one of which is indicated by arrow. Beginning hilar haze and peripheral haze are also present.

Alveolar Edema

Alveolar edema usually indicates acute left heart failure. When fluid spills into the alveoli, a familiar roentgen pattern results, i.e. the “butterfly” or “batwing” appearance (fig. 8). This central distribution of pulmonary edema may be due to differences in ventilation and lymphatic drainage of this portion of the lung. In many instances the distribution is atypical (fig. 9). Pulmonary edema may be symmetric or asymmetric, and unilateral or bilateral.

Alveolar edema may not only be atypical in distribution, but also in appearance. Most commonly, it produces large homogenous densities, but occasionally small miliarylike densities, medium-sized nodules (fig. 10), or even large nodules (cannonball appearance) may be present. Diffuse patchy densities simulating bronchopneumonia may occur; these patches of edema are most frequently seen following myocardial infarction.

Miscellaneous Findings

Several miscellaneous findings occur in left heart failure. They are frequently seen but are not necessarily pathognomonic of left heart failure.

Pleural Effusion

Pleural effusion is a common finding in left heart failure. It is often bilateral. When unilateral, the fluid is typically on the right.4 Unilateral fluid on the left is only rarely due to cardiac decompensation; usually some other etiology should be sought, particularly pulmonary infarction.

Pulmonary Hemosiderosis

Hemosiderosis develops from recurrent small venocapillary hemorrhage. Hemosiderin is picked
Figure 6

Interstitial edema—C lines. Magnified view of the right lung base in a patient with hypertensive heart disease. The C lines produced a fine reticular or honeycomb pattern which cleared rapidly after treatment for decompensation.1 (Courtesy of Charles C Thomas, Springfield, Illinois.)
Figure 7
Interstitial edema—multiple findings. Film of a 60-year-old man following myocardial infarction. A lines are seen as straight nonbranching lines, most pronounced in the right second anterior interspace and the left second, third, and fourth anterior interspaces. A few basilar B lines are faintly visualized. Lesser fissure appears thickened due to subpleural edema. Hilar haze and peripheral haze are also present.1
(Courtesy of Charles C Thomas, Springfield, Illinois.)
Figure 8

Alveolar edema. Typical butterfly appearance. In addition, a nodular patch of edema is present in the left upper lung field.
Figure 9

Alveolar edema—unilateral. The edema is unilateral on the right. In some areas the edema is confluent; in other areas a small miliary-nodular appearance is present.
Figure 10
Alveolar edema—atypical. Magnified view of right upper lung field. Changes were diffuse throughout both lungs. The edema is nodular, larger than miliary size. Cleared rapidly when compensation was established.
Figure 11
Hemosiderosis. Magnified view of right lower lung field showing numerous small nodular densities. The lesions resemble nodular edema, but they did not change after therapy.

Figure 12
Pulmonary bone nodules. View of the left lung in a patient with mitral stenosis. Osseous nodules of various sizes are present.
Figure 13

Aneurysmal dilatation of pulmonary veins. Teen-aged patient with rheumatic mitral disease, predominantly mitral insufficiency. Marked distension of pulmonary veins (small arrow) adjacent to the right hilum. The vascular nature of this shadow can be suspected because of the associated distention of the upper lobe veins. The left atrium (large arrow) also bulges into the right hilar region.

Pulmonary Fibrosis

Long-standing pulmonary venous hypertension may eventually produce pulmonary fibrosis. This is characterized by a progressive accentuation of the interstitial pulmonary markings as observed on serial roentgenograms.

Aneurysmal Dilatation of Pulmonary Veins

Aneurysmal dilatation of the pulmonary veins usually indicates mitral insufficiency, either congenital or rheumatic. The dilatation characteristically occurs at the junction of the pulmonary veins with the left atrium. The dilated veins occur on both sides but are usually best seen on the right (fig. 13). They appear as two nodular densities, at the upper and lower margins of the hilum. On the lateral projection they are contiguous with the posterior lobe veins.

Up by phagocytic cells. Clusters of these hemosiderin-laden macrophages are visible roentgenologically as small miliarylike nodules, which vary from 1 to 5 mm in diameter (fig. 11).

Pulmonary Ossification

Pulmonary bone nodules lie within the alveoli. They are not related to hemosiderosis. Their etiology is not clear, but they are believed to result from pulmonary venous hypertension and chronic pulmonary edema. They usually occur secondary to long-standing mitral stenosis and have been described with myxoma of the left atrium, constrictive pericarditis, or left ventricular failure. The ossific nodules vary somewhat in size and shape (fig. 12), which helps to distinguish them from old calcified histoplasmosis or tuberculosis.
Same case as figure 13, showing marked reduction in vein size after therapy for decompensation. This case also demonstrates combined venous and arterial hypertension. Venous hypertension: dilated upper and constricted lower lobe veins. Arterial hypertension: dilated central and constricted peripheral arteries. (Courtesy of Charles C Thomas, Springfield, Illinois.)

Secondary Pulmonary Arterial Hypertension

Patients with pulmonary venous hypertension may eventually develop pulmonary arterial hypertension. This is produced not only by the passive effect of increased venous pressure but also by reflex or reactive vasoconstriction, with eventual permanent thickening of the pulmonary arterioles.

Roentgenologically, the findings are those of combined pulmonary venous and arterial hypertension (figs. 13, 14). There is distention of the upper lobe veins (venous hypertension) plus dilated central arteries with abruptly constricted peripheral arterial vessels (arterial hypertension).

References
