Differential Imaging of pulmonary oedema Tips for junior Radiologists

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Learning objectives

Our aim is to point out to junior radiologists tips; how careful evaluation of certain radiographic features improve ability to determine the cause of any given case of pulmonary oedema
Background

Development of pulmonary oedema (increased extravascular lung water) is a common and potentially life-threatening clinical problem, particularly in critical-care unit patients. There are three principal varieties: cardiac, commonly resulting from myocardial or valvular heart disease; overhydration, usually caused by excess saline infusion or renal failure with retention of salt and water; and capillary permeability, which can be caused by a wide variety of pathologic, traumatic, and infective conditions resulting in injury to the pulmonary microvasculature. The three principal mechanisms of oedema formation are (1) increased hydrostatic pressure gradient across the capillary wall, (2) diminished osmotic pressure gradient across the wall, and (3) increased capillary permeability (damage to the endothelialcell junctions, which permits both fluid and large molecules to leak out of the vessels). Change in plasma oncotic pressure is usually a contributory rather than a primary cause of pulmonary edema. A fourth, and often neglected factor, is the ability of the lymphatics to remove excess extravascular lung water (EVLW).

Clearly in many instances the history, signs and symptoms, and clinical course of the patient's disease will be sufficient to establish the cause of pulmonary oedema, but remains a large proportion of cases, particularly in critical care units, where the cause of the oedema may be very difficult to be determined. The chest radiograph is the first line practical method of detecting pulmonary oedema at an early stage and following its evolution accurately, allowing us also to determine the cause of pulmonary oedema in a high percentage of cases. Plain chest film is performed in all patients with pulmonary oedema. In certain cases additional CT images are needed as oedema mimics other diseases or occurs as unsuspected finding in patients having CT scan for other reasons.
Imaging findings OR Procedure details

On the basis of past clinical experience review of chest X-ray and CT images of patients with radiographic evidence of pulmonary oedema in association with clinical and physiologic documentation of the cause of their oedema, several features have been identified, that permit the cause of oedema to be determined correctly in high percentage of cases.

A. Capillary permeability oedema

Capillary permeability oedema can be associated with or without diffuse alveolar damage (DAD). ARDS is the commonest type of capillary permeability oedema with DAD, and is used for various acute or subacute, diffuse pulmonary lesions that cause severe hypoxemia. DAD may be the direct result of a local precipitating factor or may occur secondary to some systemic condition. Primary or direct injuries to the alveolar and vascular endothelium of the lung usually result from the exposure of these cells to chemical agents, infectious pathogens, gastric fluid, or toxic gas, which destroy or severely damage the cells. Secondary damage is due to a systemic biochemical cascade creating oxidizing agents, inflammatory mediators, and enzymes, which also harm these endothelial cells during sepsis, pancreatitis, severe trauma, or blood transfusion. Permeability oedema without DAD as the name implies, refers to pulmonary oedema in which permeability changes are not primarily associated with DAD.

Permeability edema is distinctive by virtue of patchy, frequently peripheral, non-gravitational distribution of oedema, often with small intervening unaffected patches of lung, usually sparing the costophrenic angles. Patients have usually normal heart size, vascular pedicle and flow distribution. Septal lines are never found in a case of pure capillary permeability oedema. Similarly, peribronchial cuffing and pleural effusions are much rarer in capillary permeability oedema than in the other two varieties. In contrast, air bronchograms are very common in capillary permeability oedema. (Fig 1,2,3,4)

B. Cardiogenic pulmonary oedema

Cardiogenic pulmonary oedema (CPE) is defined as pulmonary oedema due to increased capillary hydrostatic pressure secondary to elevated pulmonary venous pressure. It reflects the accumulation of fluid with a low-protein content in the lung interstitium and alveoli as a result of cardiac dysfunction. The usual causes
of systolic and diastolic left ventricular dysfunction are coronary artery disease, myocarditis, valvular disease, cardiomyopathy, hypertension, and congenital heart diseases.

In cardiac failure the even (homogeneous from chest wall to heart), principally basal pattern of distribution is by far the commonest, whereas the perihilar component is a common pattern as well. The distribution of oedema is clearly affected by gravity and that is why usually appears as increased density at the lowest visualized part of the lungs and the costophrenic angles. Patients have an enlarged heart, and an inverted pulmonary flow. In cardiac failure, in the absence of obstructive lung disease, lung volumes are reduced, reflecting the diminished compliance of a wet lung. Peribrochial cuffing, septal lines and pleural effusions are quite common. Most patients have an increased vascular pedicle width indicating increased systemic blood volume. (Fig 5,6,7,8,9)

C. Increased hydrostatic pressure oedema

Hydrostatic oedema is secondary to elevated capillary hydrostatic pressure, with the preservation of normal selective permeability of the capillary endothelium and alveolar epithelial barriers. The commonest causes are renal disease, volume overload. Two pathophysiologic and radiologic phases are recognized in the development of pressure oedema: interstitial oedema and alveolar flooding or oedema. The first visible site of accumulation of fluid is in the loose connective tissue around the blood vessels and airways. This loose connective tissue space is continuous around the vessels and bronchi, the interlobular septa and the subpleural space. Fluid in the spaces is shown on the chest radiograph as peribronchial cuffing, Kerley lines, and subpleural fluid accumulation. If the quantity of extravascular fluid continues to increase, the oedema will migrate centrally with progressive blurring of vessels, first at the lobar level and later at the level of the hilum. Lung radiolucency decreases markedly, making identification of small peripheral vessels difficult. Clearly, as fluid accumulates within the perivascular space, a point must be reached at which the sheath is maximally distended and the pressure increases abruptly (a process which could be called "interstitial tamponade.") At this point, alveolar oedema occurs. Central oedema predominates with balanced blood flow distribution, while the costophrenic angles are usually spared. The overhydration patients show a high percentage of widened vascular pedicles. (Fig 10, 11,12,13)

Neurogenic Pulmonary Oedema

Finally it is worth mentioning few words about neurogenic pulmonary edema (NPE), a kind of mixed edema, that is usually under-diagnosed in acute neurologic injuries. Neurogenic
pulmonary oedema is defined as an acute pulmonary edema occurring shortly after a central neurologic insult. It often presents without pre-existing cardiovascular or pulmonary pathology—pathology that could explain the edema. Its pathophysiology remains poorly understood. Two different mechanisms seem to coexist. A hemodynamic factor, which is mainly the effect of the adrenergic response to the cerebral insult inducing intense pulmonary vasoconstriction, that results in an increase in pulmonary hydrostatic pressure, followed by an increase in the permeability of pulmonary capillaries. An 'inflammatory' mechanism also induces an increase in the permeability of pulmonary capillaries. (Fig 14,15)
Fig. 1: Diffuse patchy ground glass consolidations, not gravity dependent within each lobe, in a 78 years old patient with ARDS hospitalized with sepsis. Note absence of pleural effusions.

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Fig. 2: Diffuse patchy ground glass consolidations, within in a 65 years old patient with ARDS

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Fig. 3: Air bronchogram sign in a 65 years old patient with ARDS

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**Fig. 4:** Normal vascular pedicle width (< 60mm) and normal heart size in a patient with capillary permeability oedema

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Fig. 5: Even distribution of ground glass opacities as a result of alveolar edema, and bilateral pleural fluid in a 41 years old patient with cardiogenic pulmonary edema

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Fig. 6: Redistribution of pulmonary blood flow in a 58 years old patient with chronic heart failure presented with cardiogenic pulmonary edema

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Fig. 7: Acute cardiogenic pulmonary edema. Note the enlarged heart size

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**Fig. 8:** Kerley B lines in a 62 years old patient with cardiogenic pulmonary edema

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Fig. 9: Increased vascular pedicle width >60mm in a patient with CHF presented with pulmonary edema

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Fig. 10: Alveolar edema mainly with perihilar distribution, and small pleural effusions in a 45 years old patient under hemodialysis

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Fig. 11: Alveolar oedema with central distribution in a hospitalized patient, as a result of overhydration

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Fig. 12: A 60 years old patient under hemodialysis presented with dyspnoea. Note that central edema predominates with balanced blood flow distribution, while the costophrenic angles are spared.

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Fig. 13: Perihilar oedema in a patient with Chronic renal failure (bat's wing sign)

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**Fig. 14:** Diffuse patchy alveolar infiltrations in a young woman patient, that was admitted to our hospital after a car accident under drug influence. At first the lesions were thought to be lung contusions.

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Fig. 15: Same patient. On second look the presence of septal thickening made the diagnostic difference. The final diagnosis was neurogenic oedema.

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Conclusion

Chest radiograph and sometimes CT scan are practical methods of detecting pulmonary oedema at early stage. Proper evaluation of certain radiographic features can hint at the underlying cause which would lead to more rapid and definitive treatment.
References