Right lower lobe oedema as a rare sign of left heart failure

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Two cases of right lower lobe oedema are presented as a supposedly very rare sign of left heart failure after myocardial infarction in the presence of acute mitral regurgitation. Causes of unilateral oedema and its clinical relevance in the diagnostic and therapeutic perspectives are discussed, and a review of the literature on this topic is presented. (*Neth Heart J* 2006;14:225-28.)

Keywords: oedema, right lower lobe, regurgitation, mitral valve

Even though the ICT revolution has delivered accelerated enhancement of imaging techniques, a look back at basic roentgenology dealing with everyday practical issues is not amiss. What could be more basic than a plain X-ray of the thorax. Still, one can argue about the interpretation of distinct phenomena, such as the significance of unilateral oedema. Bilateral pulmonary oedema is a well-known sign of left heart failure. Unilateral oedema as a clinical presentation of this pathological condition seems to be far more uncommon and has been addressed only by a few articles in recent years, the majority of which described oedema of all the right lobes or solely the upper one.[1-9] Our intention is to present two cases with right lower lobe oedema as a supposedly very rare sign of left heart failure after myocardial infarction in the presence of mitral regurgitation. In addition a short review of the literature on this topic is presented.

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Case 1

A 78-year-old woman with a history of rectal bleeding of unknown origin, a capsular transient ischaemic attack due to a saccular aneurysm of the arteria carotis interna, arterial hypertension, a percutaneous transluminal coronary angioplasy following mild myocardial infarction, and implantation of a VDD pacemaker in the presence of a third degree AV block was admitted to our cardiac care unit suffering a limited myocardial infarction. Echocardiography in the past revealed a normal systolic LV function without mitral regurgitation and there was no history of any pulmonary disease noted. This diagnosis was mainly based on past medical history and on cardiac enzymes (maximum levels of CPK 603, LDH 241, CK-MB 95, and troponin I 9.8) as ECG interpretation was inconclusive due to VDD pacemaker rhythm with a left bundle branch block (LBBB) configuration. In accordance with international guidelines, low-molecular-weight (LMW) heparin, acetylsalicylic acid and nitrates were administered. Less than 36 hours later she developed severe dyspnoea, and an arterial blood gas sample demonstrated a state of respiratory acidosis due to hypercapnia (pH 7.12, pCO_2 80.1 mmHg, base excess –5.6, pO_2 61 mmHg and SO_2 82%), requiring urgent intubation and respiratory support. Chest radiography showed an extensive right unilateral opacity (figure 1).

Pulmonary infection was ruled out and transthoracic echocardiography revealed a high-velocity regurgitant jet directed directly towards the orifice of the right lower pulmonary vein, confirming the diagnosis of acute mitral regurgitation. At that time LV and LA parameters were within normal limits and the LV function was preserved despite asynchronic anteroseptal wall motion in LBBB conduction. As this diagnosis was very clear, no transoesophageal echocardiography was performed at this time. Mechanical ventilation with initially high positive end-expiratory pressure was started along with intravenously administered diuretics, vasodilatory therapy and low-dose inotropics. The oedema gradually cleared and the patient could be weaned from mechanical ventilation within two days. Coronary angiography then

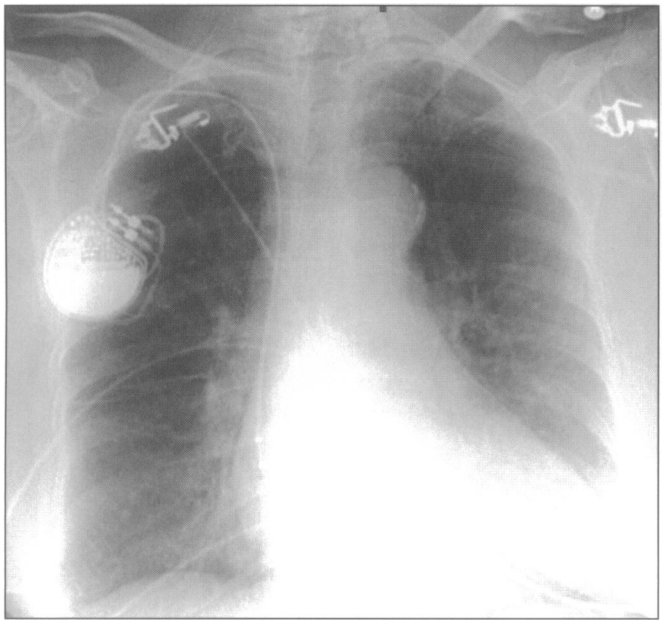

Figure 1. Standard chest X-ray showing distinct features with right lower lobe congestion.

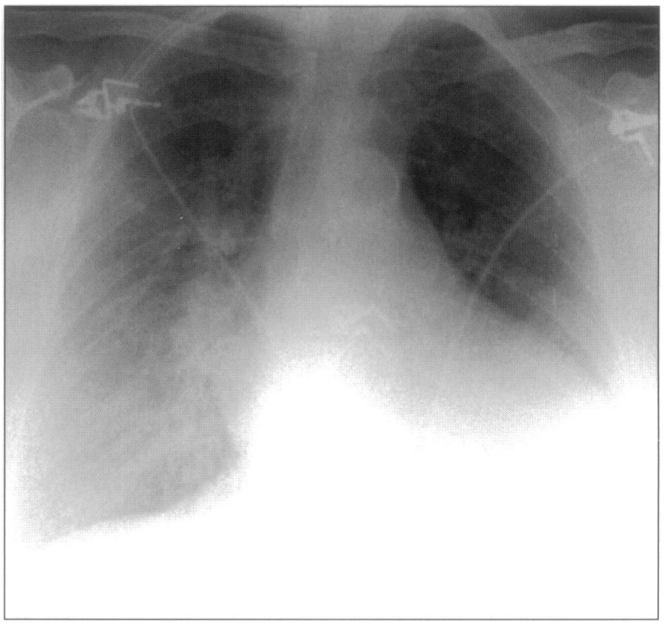

Figure 2. Standard chest X-ray showing quadrant pulmonary oedema in the right lower and middle lobes respectively.

revealed three-vessel disease with lesions in the left anterior descending (LAD), the smaller right coronary artery (RCA) and the distal right circumflex (RCX). The proximal RCX showed a subtotal occlusion with thrombus formation and therefore could be regarded to be the culprit lesion. Subsequently she was referred to a cardiovascular centre for coronary surgery and mitral valve repair as well. During surgery, some two weeks after the initial symptoms, a dilatation of the mitral valve annulus was noted as a result of persisting mitral regurgitation. The subvalvular apparatus seemed to be intact. The further clinical course was uneventful and at follow-up she is doing well.

Case 2

Whilst taking a shower, a 72-year-old woman suddenly experienced nausea before passing out for a few minutes. After regaining consciousness she suffered a dull pain in the chest, radiating to both her jaws. Bearing in mind a myocardial infarction some 20 years earlier, causing left ventricular dysfunction with a residual ejection fraction of no more than 37%, she was admitted to the hospital for further evaluation. Her history also included type II diabetes, severe obesity, cholecystectomy and multiple transient ischaemic attacks. Electrocardiography revealed a possible inferior wall infarction in the presence of an LBBB configuration, confirmed by cardiac enzymes (maximum levels of CPK 624, LDH 249, CK-MB 107, and troponin I 17.8), and she was transferred to our cardiac care unit for treatment with LMW heparin, acetylsalicylic acid and nitrates. In the presence of limited ECG changes and poor general health neither thrombolysis nor PTCA were performed. Chest radiography showed oedema of the right lower and middle lobes (figure 2), so diuretics were added. As a systolic murmur was heard on physical examination, transthoracic echocardiography was performed and revealed mild mitral regurgitation. Due to severe obesity it was not possible to specify the regurgitant jet or the status of the subvalvular apparatus. Global systolic LV function seemed to be normal at that time, without significant dilatation. Pulmonary disease such as infection or aspiration was ruled out with the help of laboratory tests and clinical signs at follow-up. After stabilisation her clinical course was uneventful.

Discussion

As shown in table 1, a unilateral pulmonary opacity can be caused by infection, neoplasm, lung infarction, atelectasis, aspiration, bronchial obstruction or pulmonary oedema due to left heart failure.[1] In this article we restrict ourselves to the phenomenon of unilateral pulmonary oedema. Pulmonary oedema can be described as a pathological state in which liquid accumulates in the lungs, mostly because liquid is being filtered through the pulmonary endothelial surface faster than it can be removed.[4] Its causes are shown in table 2.

Radiographic findings of pulmonary oedema in left heart failure usually consist of a bilateral symmetrical opacity, preferably in the central zones of the lungs, commonly known as 'butterfly shadow'.[2,3] Unilateral pulmonary oedema is regarded to be an unusual complication, largely seen in the same clinical settings as bilateral oedema and specifically in re-expansion pulmonary oedema, upper airway obstruction, pro-

Table 1. Causes of unilateral pulmonary opacities.

Unilateral pulmonary opacities
Infection
Neoplasm
Lung infarction
Atelectasis
Aspiration
Bronchial obstruction
Pulmonary oedema

Table 2. Causes of bilateral pulmonary oedema.

Bilateral pulmonary oedema
Elevation of pulmonary capillary hydrostatic pressure
Decreased plasma oncotic pressure
Impaired lymphatic drainage
Disruption of alveolar epithelial-endothelial integrity

longed lateral decubitus position, left-to-right intracardiac shunt, obstruction to pulmonary venous drainage, congenital or acquired one-sided pulmonary perfusion defect and unilateral sympathectomy (table 3).[2,3,5,6] It is usually seen in the right lung and differences in the lymphatic draining capacity are thought to play an important role for that matter: fluids in the right lung drain through the small right broncho-mediastinal trunk, which sometimes forms the right lymphatic trunk, whereas fluids in the left lung drain through the much larger thoracic duct. This means that fluids in the left lung are much more easily drained, whilst on the right, fluid overload can more easily exceed the capacity of the broncho-mediastinal trunk, causing right pulmonary lung oedema.[2,3]

In the case of left ventricular failure with papillary dysfunction and mitral regurgitation one could expect to find bilateral pulmonary oedema, since the left atrial overload is in fact divided among all four or preferably the upper pulmonary veins. A retrograde blood flow directed towards the posterior left atrial wall is indeed often seen in severe mitral regurgitation and reversal of blood flow in both the pulmonary venous systems may then cause bilateral pulmonary oedema.[1] It is, however, thought to be less common that the retrograde blood flow is selectively directed towards the right pulmonary venous system.[1-9] The regurgitant jet targets the right superior pulmonary vein in most of these cases, causing oedema of the right upper lobe.[1] Our patient, on the other hand, showed oedema of the right lower lobe, caused by regurgitation straight into the orifice of the right inferior pulmonary vein. This is an even more unusual phenomenon as in all previous cases 73.33% showed oedema of the right upper pulmonary lobe.[1] It should be noted, however, that

Table 3. Causes of unilateral pulmonary oedema.

Unilateral pulmonary oedema
Re-expansion pulmonary oedema after thoracocentesis or pneumothorax
Upper airway obstruction
- Acute bronchial obstruction by foreign body or tumour
- Intubation of right main-stem bronchus
Aspiration pneumonitis
Unilateral sympathectomy
Left-to-right intracardiac shunt
- e.g. ventricular septal rupture
Obstruction to pulmonary venous drainage
- Compression of a pulmonary vein (e.g. aneurysm of adjacent aorta or pulmonary artery)
- Mitral valve regurgitation
Congenital or acquired one-sided pulmonary perfusion defect
- Pulmonary arterial compression
- Misplacement of central venous pressure catheter
- Congenital absence or hypoplasia of a pulmonary artery
- Lung resection (lobectomy or pneumonectomy)
Old poliomyelitis
Epilepsy
Intrapleural nitrogen mustard therapy
Swyer-James syndrome
Postoperative pulmonary vein thrombosis
Prolonged lateral decubitus position
Surgical or traumatic injury

Lesieur et al. diagnosed their cases in an eight-month period, suggesting that this condition may be far more common than generally assumed, as is confirmed by others.[1,8,9] Even more so, in 131 retrospectively reviewed chest radiographs of patients with severe mitral regurgitation, 117 showed signs of oedema and in 12 of these cases the oedema involved predominantly the right upper lobe.[7] Left unilateral oedema caused by mitral regurgitation, however, seems to be the least usual as, to our knowledge, it has been reported only once in English literature.

Conclusion

In a period of no more than two months we encountered two patients with oedema of the right lower lobe. As only very few of these cases have been reported in the last three decades, we are under the impression that this condition is frequently missed and may thus be far more common than generally assumed, as has been suggested by others.[1,8,9] Considering the fact that our first patient eventually developed extensive oedema of the three right lobes, we assume that unilateral oedema may be an isolated feature in the early onset of bilateral oedema, especially in the acute form of mitral regurgitation (related to valve anatomy and

pathology) and that timing of radiography could well be a key factor in detection. Nevertheless, these findings are clinically relevant and could serve as a tool in the diagnostic and therapeutic work-up, especially since right lower lobe oedema due to mitral regurgitation represents a rarely reported and frequently overlooked phenomenon. These distinctive radiographic patterns could well be regarded to be a diagnostic hint for acute mitral insufficiency prompting supplementary investigation. Other possibilities and causes of unilateral pulmonary opacity should of course be excluded at this time. Radiographic signs of left atrial enlargement could be helpful in the diagnostic process but nowadays transoesophageal echocardiography is regarded to be the best way to reveal mitral valvular anatomy and incompetence.[1-4,7] Transthoracic ultrasound investigation has to be regarded as a second-best optional technique in these cases but can nevertheless be of value in critically ill patients. ■

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