

Unilateral pulmonary edema

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None

Unilateral pulmonary edema has been reported in the literature, but it is a very rare condition that can easily be confused with other diagnoses. Acute unilateral pulmonary edema can be either ipsilateral or contralateral to the causative process. An understanding of the underlying pathophysiologic mechanism is essential if we are to diagnose this condition correctly and prescribe appropriate treatment. We report a case of left-sided pulmonary edema in a 57-year-old man who had been diagnosed 4 days earlier with right basilar pneumonia. The absence of any signs of sepsis or left ventricular failure plus the asymmetric presentation of edema led us to suspect unilateral pulmonary embolism in the left lung. The diagnosis was confirmed by spiral computed tomography. [Emergencias 2009;21:309-311]

Key words: Acute contralateral pulmonary edema. Pulmonary embolism. Pneumonia.

Introduction

Unilateral pulmonary edema has been reported in the literature, but it is a very rare condition that can easily be confused with other diagnoses. Acute unilateral pulmonary edema can be either ipsilateral or contralateral to the causative process. An understanding of the underlying pathophysiological mechanism is essential for the correct diagnosis and appropriate treatment of this condition.

Case report

We report the case of a 57 year-old-man, smoker of 1 pack/day, with a history of bilateral renal stones, treated varicose veins in both lower limbs, episodes of superficial thrombophlebitis without usual treatment. He consulted our department for right chest pain with pleuritic characteristics and cough with haemoptysis of 24 hours evolution. On examination the patient was eupneic, normoperfused and afebrile, with a basal oxygen saturation of 96% and without jugular engorgement (JE). Respiratory auscultation showed right basal rales; blood tests only showed leucocytosis (12.8×10^9 leukocytes/l, 81% segmented

and 15% lymphocytes). Chest X-ray showed a condensation in the right lower lobe. After diagnosis of community-acquired pneumonia, Fine index stage I, he was treated with levofloxacin 500 mg/24 h and paracetamol 1 g/6 h.

Four days later he returned for worsening general condition, fever up to 38.5°C, increase of right chest pain, continuous cough with haemoptysis, and progressive dyspnoea. On examination, the patient had a temperature of 37.7°C, heart rate of 104 beats per minute, respiratory rate of 34 breaths per minute, central cyanosis and blood pressure of 120/80 mm Hg. Examination of the head and neck showed normal carotid pulses without YE, tachycardia heart rhythm of 104 beats per minute without murmurs; pulmonary auscultation showed hypoventilation and rales in the basal and middle right fields. Abdominal examination showed no findings of interest; the varicose lower limbs showed bilateral post-phlebotic sequelae but no swelling or other signs of deep vein thrombosis (DVT). Electrocardiogram (ECG) showed sinus rhythm of 98 bpm, a normal axis and no change in repolarization. Chest X-ray (Figure 1) revealed a right basal consolidation, right pleural effusion and left alveolar-interstitial edema with butterfly wing appearance. Basal

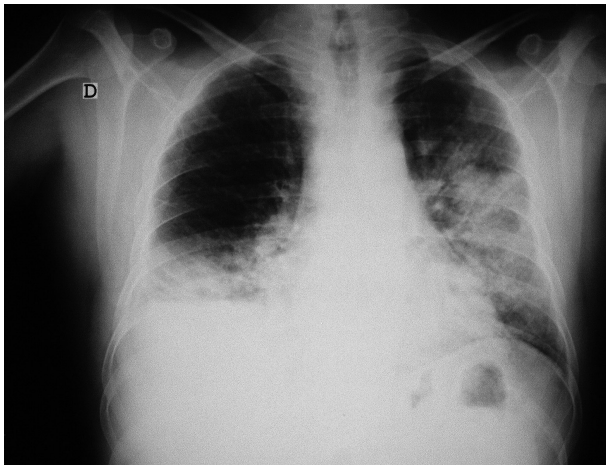


Figure 1. Chest radiograph in which we see a consolidation right basal pleural effusion and an interstitial pattern alveolar in the left lung.

blood gases (FiO₂ 21%) showed: pO₂ 43 mmHg and pCO₂ 36 mmHg. Haemogram showed 10.4 x 10⁹ leukocytes/l (92% segmented, 6% lymphocytes), and the rest was normal. Laboratory D-dimer (turbidimetric method) was 2.72 µg/ml (normal 0-0.5). Quick time was normal. Helicoidal CT scan (CT) showed right pulmonary thromboembolism (PTE), right effusion, left mediastinal adenopathy and pulmonary edema, without evidence of PTE in the left lung. The echocardiogram showed the right ventricle to be smaller than the left without paradoxical wall displacement and with good ventricular contraction. There were no signs of valve dysfunction or diastolic ventricular dysfunction.

Discussion

We report a case of unilateral left pulmonary edema in a patient previously diagnosed with right basal pneumonia due to pleuritic chest pain, expectoration with haemoptysis and chest X-ray evidence of image of condensation. After 4 days on antibiotic treatment he suffered worsening of clinical condition, with progressive dyspnoea being added to the symptoms reported. After re-assessment in the ED, diagnosis was established as pulmonary embolism with right-sided pleural effusion and pulmonary edema in left butterfly wing, without any apparent sign of left heart failure.

In this regard it would have been helpful to count on the determination of natriuretic peptide (BNP) to interpret whether the dyspnoea and pulmonary edema were due to cardiac causes or not. Morrison et al¹ showed that BNP is of great help

for this distinction. Because BNP concentration presents good correlation with pulmonary capillary pressure, it is used to differentiate between acute pulmonary edema (APE) of cardiogenic or non-cardiogenic origin. When BNP is less than 100 pg/ml, the possibility of heart failure is very low (negative predictive value 90%). When BNP is greater than 500 pg/ml, the possibility of heart failure is very high (positive predictive value 90%). Finally, when the value of BNP is between 100 and 500 pg/ml, one should consider other possibilities such as stable cardiac dysfunction without acute decompensation, congestive impairment (neither of which were the case in our patient), and right heart failure due to chronic obstructive lung disease (COPD) with *cor pulmonale* or pulmonary embolism. This intermediate elevation of BNP in the case of pulmonary embolism may be related with dilation and right ventricular dysfunction due to pressure overload and, in the case of this disease, higher concentrations are correlated with higher mortality.

Unilateral APE^{2,3} may be ipsilateral to the process which produces it (systemic-pulmonary shunt, bronchial obstruction, unilateral occlusive disease, unilateral aspiration, pulmonary contusion, prolonged lateral decubitus, rapid thoracostomy) or contralateral (absence or Swyer-James pulmonary artery hypoplasia, PTE, localized emphysema, lobotomy or pneumonectomy and pleural disease).

In PTE the embolisms capable of producing clinical symptoms are multiple and large, manifesting in haemorrhage or infarction (haemorrhage plus necrosis). This presents with pleuritic pain, dyspnoea, haemoptysis, and sometimes pulmonary edema of unaffected areas.

Pulmonary edema in the absence of left heart failure is rarely reported as a complication in the picture of angiographic pulmonary embolism. In the PIOPED study, pulmonary edema, without distinguishing between unilateral or bilateral, was observed in 10% of patients presenting with suspected PTE (confirmed by angiography), compared with 20% in the case of the suspicion not being confirmed. Pulmonary edema and vascular redistribution in left hemithorax occurs more significantly in patients with suspected but not confirmed PTE, compared to those where PTE was confirmed⁴. Similarly, most cases where pulmonary edema is present also show previous cardiopulmonary disease⁵.

One mechanism explaining the asymmetry of left pulmonary edema is an imbalance in pulmonary perfusion secondary to unilateral right

PTE. This causes hyper-flow to the contralateral lung, the left in this case, and results in a left interstitial-alveolar radiological pattern in the absence of other causes^{6,7}. It seems that a blockage of at least 50-75% of the pulmonary vascular bed is necessary for the pulmonary edema to appear⁸.

The possible mechanism underlying localized pulmonary edema due to hyperperfusion is probably the result of a combination of various factors. Thus, in experimental animals subjected to blockage of the right lung artery and two branches of the left lower lobe pulmonary artery, with maintenance of cardiac output, the authors observed protein-rich hemorrhagic pulmonary edema in the left upper lobe (the area not occluded)^{6,9}.

It is also noteworthy that the pathophysiology of hyperperfusion edema resembles edema altitude pulmonary, but does not explain fully¹⁰. In the altitude pulmonary edema, hypoxic pulmonary vasoconstriction associated the existence of microthrombi, is responsible pulmonary hypertension, but this is unevenly distributed. Pulmonary capillaries occluded territories are not subject to high pressure levels that damage their walls and followed by an inflammatory reaction with hyperpermeable secondary to the release of serotonin, histamine, prostaglandins, free radicals oxygen and probably cytokines, as observed in the bilateral pulmonary edema associated to pulmonary emboli minimum⁸⁻¹¹.

You can put up for discussion if the final result has been a fact in the evolution of intercurrent pneumonic process or whether the diagnosis of this latter was not correct. It is well known that a TEP causing pulmonary infarction or stroke effusion, may clinically and radiographically similar

one pneumonia and lead to early diagnosis and treatment wrong. Moreover, taking into account the mentioned the PIOPED study observations, in particular, the fact that the presence of pulmonary edema vascular redistribution in the contralateral hemithorax more common in patients suspected of having PTSD was not confirmed by angiography in the who do show the presence of edema in our patient would be against the TEP. However, the experience of the current case, in the presence of a unilateral pulmonary edema, contralateral to the lesion original, should be considered as the cause PTSD most likely the edema, especially in the absence signs of left ventricular failure.

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Edema pulmonar unilateral

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El edema de pulmón unilateral está descrito en la literatura médica, pero es una situación muy poco frecuente y de fácil confusión en su diagnóstico. El edema agudo de pulmón unilateral puede ser ipsilateral al proceso que lo produce o contralateral al mismo. Es importante comprender el mecanismo fisiopatológico que lo produce para llegar a un correcto diagnóstico y adecuado tratamiento. A este respecto presentamos un caso de edema pulmonar izquierdo en un paciente de 57 años, diagnosticado de neumonía basal derecha 4 días antes, que reaccude por empeoramiento de su situación clínica. La ausencia de signos de sepsis, de fallo ventricular izquierdo y la asimetría del edema permitió sospechar de tromboembolismo pulmonar unilateral izquierdo, que se confirmó con tomografía computarizada helicoidal. [*Emergencias* 2009;21:309-311]

Palabras clave: Edema agudo del pulmón contralateral. Tromboembolismo pulmonar. Neumonía.