Case Report

Unilateral Lobar Pulmonary Edema

Garvi J Pandya[*], Amartya Mukhopadhyay[**], Chua Ai Ping[**], Tow Keang Lim[***]

Abstract

Introduction: Pulmonary edema is usually bilateral, but unilateral lobar pulmonary edema can also be encountered in clinical practice.

Clinical picture: We describe a case of unilateral lobar pulmonary edema in a patient without known cardiac history. It was first presentation of underlying cardiac disease in our patient and was difficult to differentiate from pneumonia.

Conclusion: Unilateral pulmonary edema can mimic as pneumonia. Clinician should be aware of differential diagnosis of pulmonary edema, otherwise it can lead to unnecessary investigation and delay in starting definitive treatment.

Introduction

Unilateral pulmonary edema is uncommon and usually involves the right upper lobe.[1] Atypical accumulation of fluid in the lung can produce unilateral lobar pulmonary edema.[2] Mechanism of such has been attributed to various causes. We present a case of unilateral right upper lobe pulmonary edema.

Case Report

A 74-year-old Chinese man with past medical history of chronic obstructive airway disease (COPD), asbestosis related pleural plaques, left lower limb deep vein thrombosis (DVT) and heavy alcohol consumption presented with shortness of breath associated with dry cough of one day duration. There was no history of heart disease. Patient was chronic smoker with 60 pack year's history.

On examination, his temperature was 37.6°C, pulse rate 117 / minute, blood pressure 134/71 mmHg and oxygen saturation 98% on 2 liter/min of nasal oxygen supplementation. Auscultation of the lungs revealed bilateral basal crepitations without any rhonchi. Results of the initial investigations are shown in Table 1.

Chest radiograph (Figure 1) showed right upper lobe infiltrates and sputum acid fast bacilli smear was negative.

Patient was started on intravenous Ceftriaxone 1 gm BD and PO Clarithromycin 500mg BD and given single dose of oral Furosemide 40mg in view of possibility of pulmonary edema. Chest radiograph done 3 days later (Figure 2) showed improvement in right upper lobe infiltrate. At the time of discharge 5 days after admission, the patient improved further and he was scheduled for outpatient echocardiography and cardiology follow-up.

Table 1 : Baseline blood tests

	Range	Patient's value
Hb (g/dL)	12.6 - 16.9	13.5
WBC (10^9/L)	3.26 - 9.28	13
Platelet (10^9/L)	160-398	262
Sodium (mmol/L)	135 - 150	138
Potassium (mmol/L)	3.5 - 5.0	3.4
Urea (mmol/L)	2.5 - 7.5	5.3
Creatinine (µmol/L)	65 - 125	102
NT Pro-BNP (pg/ml)	0-194	2279
Procalcitonin (mg/L)	<0.5	0.34

Hb: Hemoglobin, WBC : White Blood Cell, NTPro-BNP: N-terminal Pro- Brain Natriuretic Peptide

After fifteen days, patient was readmitted to cardiology with complains of shortness of breath associated with wheeze and exertional chest tightness. Chest radiograph showed bilateral infiltrates suggestive of pulmonary edema. 2D-echocardiography showed moderate pulmonary hypertension (estimated pulmonary artery systolic pressure 55mmHg), dilated left heart chambers, moderately severe mitral regurgitation and ejection fraction of 20%. Coronary angiogram showed normal coronary arteries with right dominant circulation. A diagnosis of non-ishaemic cardiomyopathy related to alcohol consumption was made. Patient was treated for heart failure and discharged well. Subsequently patient was found to have rectal tumor on sigmoidoscopy and biopsy (adenocarcinoma) and decided for conservative - hospice care in view of co-morbidities.

Discussion

Pulmonary edema is rarely unilateral, but may cause confusion and presents diagnostic challenges. The development of pulmonary edema is the result of complex mechanisms.[3] The force, which tends to push fluid out of the capillaries, is the capillary hydrostatic pressure less the hydrostatic pressure of the interstitial fluid. The force tending to keep fluid in the capillaries is the osmotic pressure of blood proteins less that of the proteins in the interstitial fluid (Starling forces). When the balance of these factors is altered, fluid passes from the pulmonary capillaries to the interstitial spaces – and, when the capacity of lymphatics in the lung interstitium is exceeded, to the alveolar spaces. The pressure of the gas in the alveoli and the presence of surfactant also influence this movement of fluid.

Unilateral pulmonary edema can occur when local forces predominate (Table 2) or when the pulmonary vasculature or pulmonary parenchyma is abnormal (Table 3).

In all conditions causing contralateral pulmonary edema, there is underperfusion of the abnormal lung, so pulmonary edema affects only the lung with normal vasculature.

Another important cause is neurogenic pulmonary edema. Possible mechanism includes increase intracranial pressure and subsequent vascular congestion due to alterations in the sympathetic pathways, resulting in alterations in intravascular hydrostatic pressures as well as myocardial rate and contractility. Unilateral pulmonary edema has been described also in patient with acute multiple sclerosis exacerbation [9] and end stage renal failure.[10]

[*]Medical officer, [**]Consultant, [***]Professor, Department of Medicine, National University Hospital, National University Health System, 5 Lower Kent Ridge Road, Main Building , Singapore 119074
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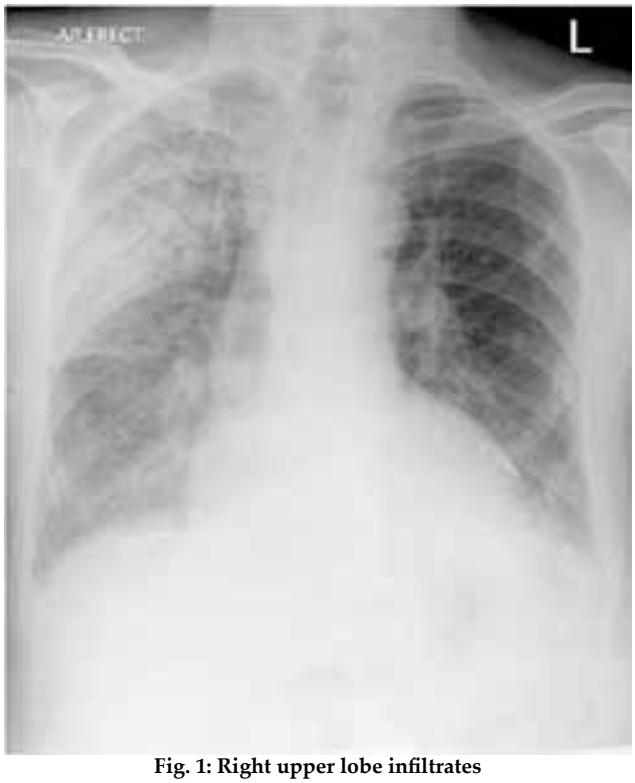

Fig. 1: Right upper lobe infiltrates

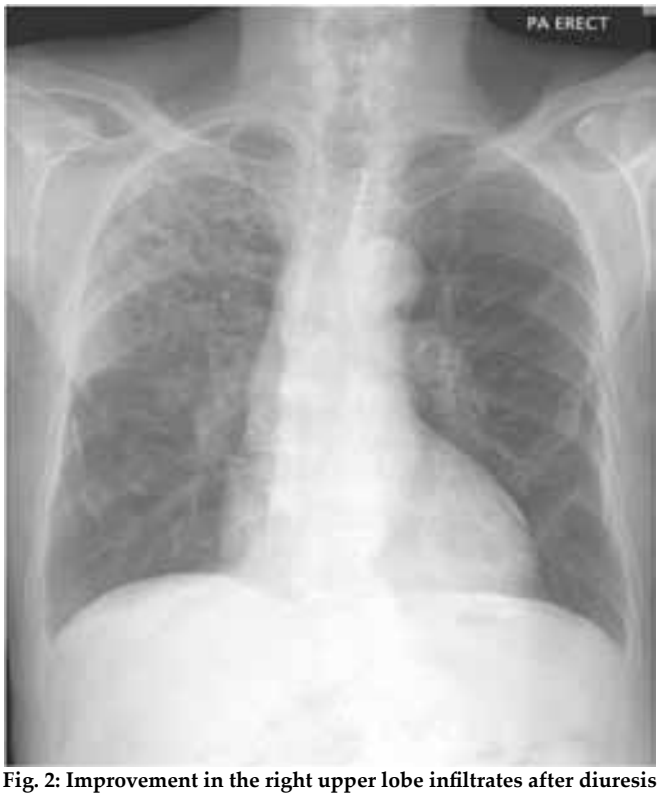

Fig. 2: Improvement in the right upper lobe infiltrates after diuresis

In our patient, right upper lobe involvement occurred initially before appearance of the bilateral pulmonary edema requiring further admission and reassessment. A retrograde flow of blood due to mitral regurgitation can be directed across the left atrium toward the orifices of the upper lobe pulmonary vasculature.[4,5] This backpressure into the right upper lobe pulmonary veins can cause a focal increase in pulmonary venous pressure and isolated pulmonary edema. Due to the unilateral infiltration in the chest radiograph and elevated WBC count, the possibility of a pulmonary infection was initially considered in our patient and was treated with antibiotics. In view of modestly raised N-terminal pro-brain natriuretic peptide (NT pro-BNP) and normal procalcitonin, single dose of diuretic was given. It is likely that the subsequent radiological improvement is due to the ensuing diuresis. It was patient's first presentation of underlying cardiac disease and although suspected, we were not aware of the full extent of his heart function. Therefore, failure to consider differential diagnosis of pulmonary edema lead to unnecessary investigations and significant delay in initiating definitive therapy.

Table 2 : Ipsilateral pulmonary edema

Condition	Mechanism
1. Papillary dysfunction, Mitral valve regurgitation [4,5]	A retrograde flow of blood directed across the left atrium toward the orifices of the right upper lobe pulmonary veins resulted in a focal increase in pulmonary venous pressure.
2. Dependent lung of unconscious or ventilated patient whose posture is not frequently altered	Contributed by gravity
3. Rapid removal of large amount of fluid or air by thoracocentesis [6,7]	Swift re-expansion of the decompressed lung causes a change in the hydrostatic pressure in the pulmonary capillaries. The production of surfactant may also have been inhibited while lung was collapsed
4. Aspiration of gastric content	Damage to the surfactant system and irritation of the pulmonary capillaries
5. Unilateral infusion of hypotonic saline through a catheter misplaced in pulmonary artery.	Local decrease in colloid osmotic pressure
6. Bronchial obstruction	Hypoxia damages those alveolar cells which produce surfactant

Table 3 : Contralateral pulmonary edema[8]

Condition	Mechanism
- Hypoplasia of the pulmonary arteries - Unilateral pulmonary thromboembolism - anastomosis of the left pulmonary artery to descending aorta(Potts procedure)	- Blood flow in the pulmonary capillaries may be reduced
- Primary emphysema - Compensatory emphysema after lobectomy	- Pulmonary capillaries may be reduced or damaged.

Summary

In our patient, the initial clinical and radiological diagnosis was pneumonia; however raised NT pro-BNP and rapid radiological improvement raised suspicion of pulmonary edema. Unilateral lobar pulmonary edema is rare but may present as first manifestation of underlying cardiac disease as in our case. The clinician should be aware that pulmonary edema need not necessarily affect both lungs, otherwise diagnosis may be mistaken and treatment delayed.

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