An unusual cause of right upper- and mid-zone infiltrates on chest x-ray

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ABSTRACT

Chest x-ray remains a critical investigation in patients who present with shortness of breath. We report a 60-year-old man who presented with shortness of breath, haemoptysis and respiratory failure, a raised white cell count, and right upper-and mid-zone infiltrates on chest x-ray. He developed progressive multiple organ failure despite aggressive intensive care management with antibiotics, ventilation and inotropes.

As his haemodynamic instability continued to worsen, transthoracic, and subsequently transoesophageal, echocardiography revealed posterior mitral valve leaflet prolapse with severe mitral regurgitation. Mitral valve repair and annuloplasty led to eventual complete resolution of symptoms. The changes seen in the right upper and mid zones on initial chest x-ray were due to the mitral regurgitant jet being directed predominantly towards the right superior pulmonary vein. We report this case to highlight a rare but important cause of lobar oedema, and the usefulness of echocardiography in assessment of the patient with complex critical illness.

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Pulmonary oedema occurs in a bilateral distribution in the vast majority of cases. When it occurs unilaterally, or even solely in a lobar distribution, it is often confused with one of the more common causes of focal lung disease. There have been few reports of pulmonary oedema selectively located in the right upper lobe caused by acute mitral regurgitation.[1-4]

We report this case to highlight a rare, but important, cause of lobar oedema, and the pivotal role of echocardiography in the intensive care management of the complex critically ill patient.

Clinical record

A previously healthy 60-year-old bricklayer presented to the emergency department with a 12-hour history of acute shortness of breath, cough productive of blood-stained, purulent sputum, and pleuritic retrosternal chest pain. He described vague symptoms of generalised myalgia and increasing lethargy with decreased exercise tolerance over the preceding week, although he was still working. He had no relevant past medical history and was taking no regular medication.

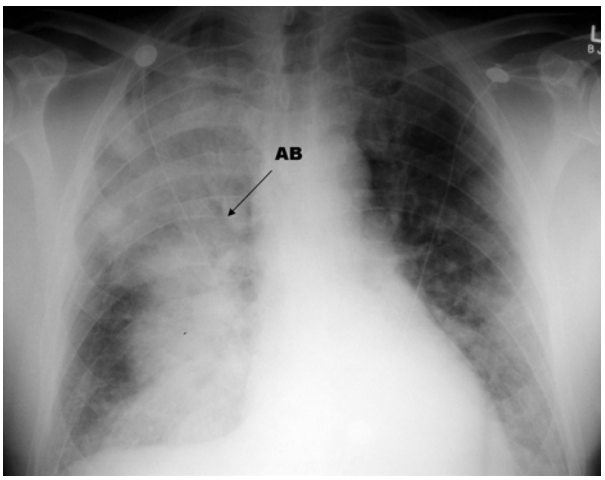
On presentation, the patient was conscious with a Glasgow Coma Score of 14 (opening eyes to voice), and was oriented to time, person and place, but looked unwell. He had tachypnoea (respiratory rate, 36 breaths per minute), with an oxygen saturation of 79% on room air, rising to 96% on 15 L/min oxygen via a non-rebreather facemask. Arterial blood gas measurements on 15 L/min oxygen were: pH, 7.43; Pa_{O_2}, 75 mmHg; Pa_{CO_2}, 32 mmHg; and base excess + 0.4. He had a heart rate of 120 beats per minute in sinus rhythm, blood pressure of 120/80 mmHg, and temperature of 37.7°C. The jugular venous pressure was not elevated, and there was no peripheral oedema. First and second heart sounds were present, and a soft systolic murmur was noted at the left sternal edge and apex. This murmur could not be further characterised because of the tachycardia and transmitted breath sounds. Air entry was reduced in the right lung, with coarse crackles in the right upper and mid zones. Physical examination was otherwise unremarkable.

An electrocardiogram showed sinus tachycardia. Blood tests revealed a raised white cell count (19.3×10^9/L; reference range [RR], 4–11×10^9/L) and neutrophil count (17.0×10^9/L; RR, 2–8×10^9/L). Troponin I was not detected. Chest radiography showed right-sided, predominantly upper- and mid-zone infiltrates, with air bronchograms (Figure 1).

The patient was admitted to the intensive care unit with a working diagnosis of severe community-acquired pneumonia. Fluid resuscitation, intravenous ceftriaxone and erythromycin, and non-invasive ventilation were commenced.

His condition deteriorated over the next 12 hours, with worsening haemodynamic status, requiring noradrenaline (0.18 μg/kg/min), and respiratory failure requiring intubation and ventilation. Arterial blood gas measurements while on 70% oxygen via a BIPAP facemask (inspiratory pressure, 15 cmH_2O; expiratory pressure, 10 cmH_2O) were: pH, 7.4; Pa_{O_2}, 64 mmHg; Pa_{CO_2}, 32 mmHg; base excess, –3.9. Bronchoalveolar lavage was performed after intubation. Copious bloodstained fluid was suctioned from the right upper lobe at bronchoscopy. Samples were taken for microscopy, culture and antibiotic sensitivity testing. At this time, the Pneumonia Severity Index was 140 (30-day mortality, 27%).

Figure 1. Chest x-ray on presentation to hospital with dyspnoea



Portable chest x-ray at presentation showing predominantly right-sided upper- and mid-zone infiltrates, air bronchograms (AB) in the right upper zone, and normal cardiothoracic ratio.

On ICU Day 3, the patient was febrile (temperature, 38.6° C) and developed fast atrial fibrillation with haemodynamic compromise. This was treated with direct-current cardioversion, magnesium and amiodarone. Given his fever, elevated white cell count and bronchoscopy findings, the diagnosis of severe sepsis with multiorgan dysfunction, secondary to community-acquired pneumonia, remained.

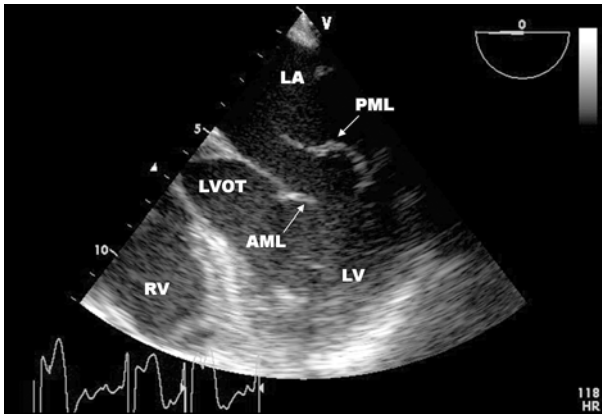
On ICU Days 4–7, cardiovascular instability increased, with increasing requirements for noradrenaline (up to 0.35 μg/kg/min) and ventilatory support to maintain oxygen saturation over 92%. The F_{IO_2} was 0.5, Pa_{O_2}, 77 mmHg, and positive end-expiratory pressure, 20 cmH_2O. His temperature peaked during this time at 42° C. Antibiotic cover was broadened empirically to ticarcillin–clavulanic acid, gentamicin, vancomycin and erythromycin, to cover potential nosocomial sources of sepsis.

Formal transthoracic, and subsequently transoesophageal, echocardiography was performed, which revealed a prolapsed posterior mitral valve leaflet (Figure 2A) and severe mitral regurgitation (Figure 2B). Systolic left ventricular function was preserved. The patient's diagnosis was revised to an acute chordal rupture with ensuing severe acute mitral regurgitation and pulmonary oedema with hospital-acquired sepsis. Dobutamine was commenced at a dose of 5 μg/kg/min, and noradrenaline was weaned over the next 12 hours. A balloon pump was inserted to improve ventricular loading and decrease myocardial work.

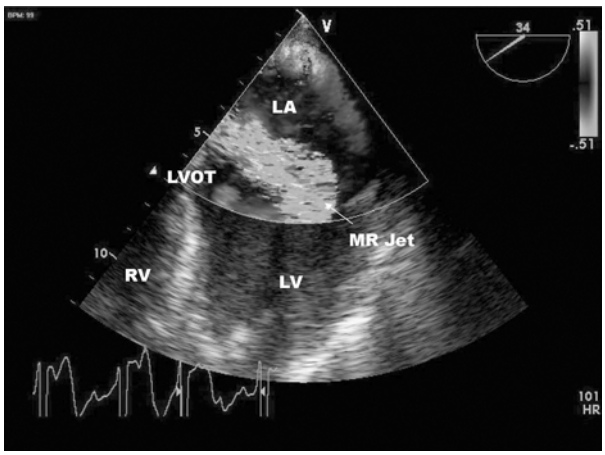
The patient underwent mitral valve repair and annuloplasty on Day 9. At operation, ruptured chordae to the middle scallop of the posterior mitral valve leaflet were noted. The valve was otherwise normal. The postoperative period was complicated by culture-negative nosocomial pneumonia, which was treated with a 5-day course of empirical ticarcillin–clavulanic acid. Follow-up echocardiography on Day 25 revealed an intact mitral valve repair, with no mitral regurgitation and normal left ventricular function. The patient was discharged home on Day 37.

Cultures of all specimens (blood, urine, sputum and bronchoalveolar lavage) taken during the patient's hospital admission were negative. Serological tests for chlamydia, mycoplasma and legionella were also negative. No respiratory viruses were detected in bronchoalveolar lavage specimens.

Figure 2. Transoesophageal echocardiograms



A. *Mid-oesophageal view in ventricular systole, showing prolapsed posterior mitral valve leaflet (PML).*
(***Key:*** *LA = left atrium. LVOT = left ventricular outflow tract. LV = left ventricle. RV = right ventricle. AML = anterior mitral valve leaflet.)*



B. *Mid-oesophageal view in ventricular systole with colour Doppler, showing large mitral regurgitant jet (MR Jet) directed into left atrium.*

Discussion

Pulmonary oedema is caused by extravasation of fluid from the pulmonary vasculature into the interstitium and alveoli of the lung. There are four main mechanisms: increased capillary hydrostatic pressure, increased capillary permeability, decreased plasma oncotic pressure, and lymphatic obstruction.[5] This may give the classic "butterfly" pattern on plain chest radiographs. Asymmetric distribution of oedema is not uncommon.[6,7] In case reports of unilateral pulmonary oedema, causes have included the patient lying in a lateral decubitus position,[8] re-expansion after thoracentesis or pneumothorax,[9,10] pulmonary arterial compression,[11] creation of systemic-to-pulmonary artery shunts,[12] and misplacement of central venous pressure catheters.[13]

There have been reports of pulmonary oedema localised to the right upper lobe in association with mitral regurgitation.[1-4] By 1993, 12 cases of unilateral pulmonary oedema associated with left ventricular failure had been reported in the English literature, eight with documented mitral regurgitation.[14] In the same year, Schnyder et al identified 131 patients with severe mitral regurgitation, and reviewed their chest radiographs for evidence of oedema.[15] Radiographic signs of vascular congestion and oedema were present in 117 (89%) of the patients, and, in 12 (9%), these findings were localised or predominant in the right upper zone. None of the patients had predominantly left-sided involvement. Despite these results, there continue to be few published cases of localised pulmonary oedema secondary to mitral regurgitation.

The pathogenic mechanism for the localised oedema has been attributed to a regurgitant jet through the mitral valve directed into the right superior pulmonary vein. This is best visualised by transoesophageal echocardiography.[16,17] The regurgitant jet causes flow reversal in the right pulmonary veins during ventricular systole, creating a relative obstruction to right-sided pulmonary venous return by increasing hydrostatic pressure.[2,3,17] There may be associated middle-lobe oedema, as the middle pulmonary vein frequently empties into the left atrium in a joint venous confluence with the superior pulmonary vein.[16] Other factors affecting the distribution of oedema include the volume of the regurgitant jet, the size of the left atrium, and the position of the pulmonary veins along the left atrial wall.[3] In cases of severe oedema, any propensity for it to be localised in the right upper lobe may be obscured.[3]

We revisit this rare cause of lobar pulmonary oedema, as misdiagnosis results in delay of appropriate treatment with potentially devastating consequences. Consideration should be given to cardiogenic causes of unilateral infiltrates on chest x-ray, particularly when the patient's clinical condition is worsening despite what is considered aggressive appropriate treatment. Echocardiography is the key investigation and should be considered early. Timely mitral valve repair or replacement should result in resolution of the oedema.[17]

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References

1 Bahl OP, Oliver GC, Rockoff SD, Parker BM. Localized unilateral pulmonary edema: an unusual presentation of left heart failure. *Chest* 1971; 60: 277-80.
2 Gamsu G, Peters DR, Hess D, et al. Isolated right upper lobe pulmonary edema. *West J Med* 1981; 135: 151-4.
3 Gurney JW, Goodman LR. Pulmonary edema localized in the right upper lobe accompanying mitral regurgitation. *Radiology* 1989; 171: 397-9.
4 Kusumi RK, Walker SS, Fulkerson PK, et al. Unilateral pulmonary edema associated with left ventricular failure. *Heart Lung* 1984; 13: 263-6.
5 Givertz MM, Colucci WS, Braunwald E. Clinical aspects of heart failure; pulmonary edema, high output failure. In: Braunwald E, Zipes DP, editors. Braunwald's heart disease: a textbook of cardiovascular medicine. 7th ed. Philadelphia; London: Saunders, 2005: 539-68.
6 Hublitz UF, Shapiro JH. Atypical pulmonary patterns of congestive failure in chronic lung disease. The influence of pre-existing disease on the appearance and distribution of pulmonary edema. *Radiology* 1969; 93: 995-1006.
7 Calenoff L, Kruglik GD, Woodruff A. Unilateral pulmonary edema. *Radiology* 1978; 126: 19-24.
8 Baraka A, Moghrabi R, Yazigi A. Unilateral pulmonary oedema/atelectasis in the lateral decubitus position. *Anaesthesia* 1987; 42: 171-4.
9 Humphreys RL, Berne AS. Rapid re-expansion of pneumothorax. A cause of unilateral pulmonary edema. *Radiology* 1970; 96: 509-12.
10 Mahfood S, Hix WR, Aaron BL, et al. Reexpansion pulmonary edema. *Ann Thorac Surg* 1988; 45: 340-5.
11 McTigue C, Scurry JP, Silberstein M. Unilateral pulmonary edema associated with pulmonary arterial compression. *Australas Radiol* 1988; 32: 390-3.
12 Salem MR, Masud KZ, Tatooles CJ, Yanes HO. Unilateral pulmonary oedema following aorta to right pulmonary artery anastomosis (Waterston's operation). Case report. *Br J Anaesth* 1971; 43: 701-4.
13 Royal HD, Shields JB, Donati RM. Misplacement of central venous pressure catheters and unilateral pulmonary edema. *Arch Intern Med* 1975; 135: 1502-5.
14 Roach JM, Stajduhar KC, Torrington KG. Right upper lobe pulmonary edema caused by acute mitral regurgitation. Diagnosis by transesophageal echocardiography. *Chest* 1993; 103: 1286-8.
15 Schnyder PA, Sarraj AM, Duvoisin BE, et al. Pulmonary edema associated with mitral regurgitation: prevalence of predominant involvement of the right upper lobe. *AJR Am J Roentgenol* 1993; 161: 33-6.
16 Chuang YS, Hsiao PN, Lin TY, et al. Right upper lobe pulmonary edema after mitral valve replacement caused by paravalvular leakage recognized by bedside transesophageal echocardiography. *Crit Care Med* 2002; 30: 695-6.
17 Dexter EU, Snider JM, Gordon EE, Richenbacher WE. Lobar pulmonary edema due to mitral regurgitation: diagnosis by echocardiography. *J Card Surg* 1997; 12: 412-5. ❑