CASE REPORT

Pulmonary oedema mimicking bronchiectasis

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SUMMARY

A previously completely fit and well 62-year-old man presented with a 4-week history of lethargy and increasing shortness of breath. This had been preceded by a flu-like illness that had been treated in the community with ciprofloxacin to no effect. There was no history of smoking, tuberculosis exposure or significant exposure to birds. Clinically the patient appeared well and was comfortable and conversant with no peripheral oedema. Chest auscultation revealed normal breath sounds and a loud pansystolic murmur over the cardiac apex. A chest radiograph and a subsequent CT scan showed widespread fibrotic and bronchiectatic changes, predominantly in the right upper lobe, with bilateral pleural effusions. Echocardiography revealed a posterior mitral valve prolapse. He was treated with loop diuretics and a mitral valve repair that resolved his symptoms and radiographic findings. This is the first English language case report of pulmonary oedema causing bronchiectatic lung appearances.

BACKGROUND

Congestive heart failure is increasingly common and represents a significant burden for the National Health Service (NHS). Early recognition and treatment of the condition improves prognosis considerably. Our patient had few physical signs suggestive of heart failure but had florid radiographic changes suggestive of bronchiectasis. Pulmonary oedema mimicking bronchiectasis is not a well-recognised phenomenon and there is little mention of it in the published literature. With the increasing use of CT scanning by hospital physicians, it is important to be aware of the ways in which heart failure can create misleading radiographic findings and thereby delay diagnosis and effective treatment.

CASE PRESENTATION

A 62-year-old Caucasian man was admitted in November 2011 with increasing shortness of breath. He had first started feeling unwell about 3–4 weeks prior to his admission with flu-like symptoms and shortness of breath. He was given ciprofloxacin for a chest infection with no relief. He was previously fit and well with no allergies and had never smoked. He worked as a welder and some weeks prior to his illness had started work at a biofuel processing plant dealing with waste products from a brewery but had never worked with asbestos. He denied ever having tuberculosis (TB) or being significantly exposed to birds. His brother required a 'triple heart bypass' in his early 30s but there was otherwise no family history of note.

On admission he looked well and was sat upright and speaking comfortably. Routine observations

revealed a heart rate of 90 bpm, blood pressure 136/86 mm Hg, respiratory rate of 22 breaths/min, a temperature of 37°C and oxygen saturations were 93% on air.

On examination there was no clubbing or signs of cyanosis and chest auscultation was clear. Jugular venous pulsations were not raised but cardiac auscultation revealed a pansystolic murmur loudest over the apex. There were no signs of peripheral oedema.

INVESTIGATIONS

Full blood count, urea and electrolytes were normal. C reactive protein was 21 mg/L. *Aspergillus* precipitins were weakly positive with normal serum IgE and negative radioallergosorbent test to *Aspergillus* but TB-QuantiFERON testing was also positive. ACE levels were normal.

A chest radiograph showed bilateral pleural effusions with bilateral upper lobe consolidation and volume loss (figure 1). High-resolution CT (HRCT) of the chest confirmed extensive upper lobe consolidation with traction bronchiectasis. There was also evidence of lower lobe consolidation bilaterally with large pleural effusions (figure 2). The size of the heart was normal with no evidence of asbestos exposure. Inpatient spirometry showed forced expiratory volume in 1 s (FEV₁) 2.2 (71% predicted), forced vital capacity (FVC) 3 (76% predicted) and FEV₁/FVC 0.73. Pleural fluid analysis showed a pleural/serum protein ratio of 0.42, pleural/serum lactate dehydrogenase (LDH) ratio of 0.51 with a pleural LDH less than 50% of the serum upper limit of normal, thereby fulfilling Light's criteria for a transudate.

Figure 1 Chest radiograph performed on admission showing bilateral upper lobe consolidation with pleural effusions.

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Figure 2 High-resolution CT scan showing consolidation, traction bronchiectasis, parenchymal bands with interlobular thickening.

Transthoracic echo showed posterior mitral valve (MV) prolapse with severe mitral regurgitation (MR) and preserved left ventricular (LV) function. His LV internal dimension in diastole was 5.7 cm with LV internal dimension in systole of 3.6 cm and a dilated left atrium at 5 cm. There was mild eccentric tricuspid regurgitation.

A transoesophageal echo confirmed the transthoracic echo findings and revealed a myxomatous posterior MV prolapse with torrential MR and pulmonary pressures of approximately 45 mm Hg (figure 3). A subsequent coronary angiogram showed a right dominant coronary system with only mild atheroma.

DIFFERENTIAL DIAGNOSIS

- ▶ Pulmonary oedema
- ▶ Bronchiectasis
- ▶ Pulmonary fibrosis
- ▶ Sarcoidosis

TREATMENT

His pulmonary oedema responded well to intravenous furosemide (figure 4) and he was referred for a MV repair at Liverpool

Figure 3 Transoesophageal echo images showing marked posterior mitral valve prolapse and severe eccentric mitral regurgitation.

Figure 4 A chest radiograph after treatment with diuretics. There is almost complete resolution of the consolidative changes with a resolution of the pleural effusions.

Heart and Chest Hospital. This was performed in December 2011 using a 32 mm Physio ring and was complicated by post-operative atrial fibrillation. He was cardioverted electrically and the patient took a 3-month course of warfarin following this. Subsequent reviews in clinic have found him to be in sinus rhythm and asymptomatic.

OUTCOME AND FOLLOW-UP

It is thought that his positive TB-QuantiFERON and *Aspergillus* tests were probably due to previous exposure to the microbes rather than active disease and a subsequent HRCT scan showed almost complete resolution of the bronchiectatic lung appearances with only a small left pleural effusion remaining (figure 5). A repeat echocardiogram performed in September 2012 showed completely normal valve and ventricular function. Pulmonary function tests following recovery showed FEV₁ 2.53 (79% predicted), FVC 3.99 (97% predicted), FEV₁/FVC 0.63, total lung capacity (TLC) 5.30 (76% predicted), transfer factor 9.08 (98% predicted) and transfer coefficient (Kco) 1.66 (123% predicted). The reduction in TLC and high Kco was thought to be due to his sternotomy.

Figure 5 High-resolution CT scan performed after mitral valve repair. The lungs now appear normal.

DISCUSSION

This case is a clear example of how radiographic findings can mislead the diagnostician. With the advent of HRCT scanning there is now an advanced system of categorising chest radiography patterns and, as recently reviewed by Oikonomou and Prassopoulos, pulmonary oedema is a well-recognised mimic of interstitial lung disease. The possibility of heart failure causing these appearances is raised by the coexisting presence of pleural effusions and the authors also describe how pulmonary oedema with a normal-sized heart may point towards MV prolapse as the cause.¹

MV prolapse has already been shown to cause atypical patterns of pulmonary oedema. Right upper lobe changes have been well associated with MV regurgitation and echocardiographic evidence of a regurgitant jet of blood preferentially entering the right pulmonary veins has been demonstrated as a possible explanation for this phenomenon. It is of significance therefore, that in our case the pulmonary changes were markedly worse in the right upper lobe.²

CT findings of traction bronchiectasis on the other hand, are not well known to be associated with hydrostatic pulmonary oedema. In 2000 Lewin *et al*³ from Massachusetts General

Hospital published their collation of radiological findings from chest CT scans performed on patients with advanced heart failure. Out of a cohort of 71 patients, 2 had radiological signs of bronchiectasis. However, it is quite possible that these two patients had underlying lung disease. Our patient had been completely well prior to his recent illness, had a complete resolution of his symptoms following MV repair and an almost complete resolution of the chest CT findings, so the possibility of underlying lung disease here seems unlikely. Our patient provides an instructive example of the protean radiological manifestations of pulmonary oedema and how the role of the clinical diagnostician in the age of high-tech investigation remains crucial.

Learning points

- ▶ Pulmonary oedema with radiographic appearances of bronchiectasis are a hitherto rarely recognised phenomenon and the clinician should be wary of the former diagnosis in the absence of a productive cough.
- ▶ Cardiogenic pulmonary oedema has been shown to cause focal lung consolidation. This should be considered as a possibility where there is no other evidence of infection as delayed treatment of cardiac failure in this circumstance has been linked to increased mortality.⁴

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Competing interests None.

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