Original Article

HYPERSENSITIVITY PNEUMONITIS PRESENTING WITH PNEUMOMEDIASTINUM

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Abstract
A 24-year-old woman presented to her general practitioner with exertional dyspnoea. There was a delay in diagnosis of the underlying respiratory condition, due to initial investigations being suggestive of cardiac disease. Subsequently, the patient developed clinical symptoms and signs of pneumomediastinum, which was discovered on radiological imaging. Detailed history-taking and further clinical testing confirmed the cause of this to be hypersensitivity pneumonitis due to sensitisation to pet birds. The patient was treated with high dose steroids and went on to make a good recovery. The birds were rehomed.

Keywords: pneumomediastinum, hypersensitvity, pneumonitis, sensitisation

Background

Although pneumomediastinum is a recognised complication of severe lung disease, it is only rarely reported in hypersensitivity pneumonitis.[1–3] In addition, this case highlights the vital importance of obtaining a full social history and making a full clinical examination in the dyspnoeic patient. Furthermore, the case illustrates how diagnoses can be delayed by misleading initial investigations.
A 24-year-old woman was referred to the cardiology outpatient clinic by her general practitioner, due to exertional dyspnoea. An ECG had shown a right bundle branch block and left axis deviation. A chest radiograph had revealed broadening of the upper mediastinum (thought to be secondary to unfolding of the thoracic aorta) and a prominent superior vena cava shadow.
In clinic, the patient reported having been short of breath for 4 months. The patient, a housewife, had never smoked, drank little alcohol, had no medical history of note and took no regular medications. She did not have any symptoms of heart failure and had no evidence of heart failure on clinical examination. However, transthoracic echocardiography demonstrated moderate left

ventricular systolic dysfunction and trabeculation. There was no valvular pathology and the right heart was poorly visualised. Ramipril and furosemide were started, and MRI of the heart was arranged to investigate for any underlying cardiomyopathy and to further assess the broadened mediastinum. The MRI, at variance with the echocardiogram findings, demonstrated normal left ventricular systolic function, an ejection fraction of 57% and no regional wall motion abnormality.
At follow-up, the patient reported feeling even more breathless and 'squeaks' could be heard on auscultation of her chest. She did not report any pain. Her respiratory rate was 30, heart rate 65, blood pressure 108/69, temperature 37.2°C and oxygen saturation was 95% on room air. A further chest radiograph showed increased lung markings with loss of definition of the heart borders and no overt pulmonary oedema, raising the clinical concern of interstitial lung disease. It also showed pneumomediastinum . An urgent high-resolution CT was arranged , following which the patient was immediately admitted to hospital and referred to the respiratory physicians.

Investigations

CT confirmed pneumomediastinum with pneumopericardium, a tiny left pneumothorax and mosaic attenuation of the lungs with areas of lucency, some areas of normal lung density and some areas of ground glass change. The overall picture suggested a combination of air trapping consistent with small airways pathology together with minor patchy ground glass change.

Differential diagnosis

These non-specific CT appearances gave a differential diagnosis including hypersensitivity pneumonitis, bronchiolitis obliterans, drug-related pneumonitis and possibly chronic venous thromboembolism (VTE). A CT pulmonary angiogram was performed and showed no evidence of acute or chronic VTE.

Treatment

On further questioning by the respiratory team, it transpired that the patient had pets, including a cockatiel and a parrot. Full blood count (including eosinophils), urea and electrolytes, liver function tests and thyroid function tests were all within normal limits. Arterial blood gas results were pH 7.43 (7.36–7.44), pCO2 5.2 (4.4–6.0), pO2 8.8 (12.0–15.0), HCO3 25.5 (21.0–27.0), BE+1.7 (−4.0 to +4.0) and oxygen saturation was 93.5%. Serum autoimmune screen, avian specific IgG antibodies and total IgE were taken, and pulmonary function tests were requested. A presumptive diagnosis of acute hypersensitivity pneumonitis was performed. Three days of methylprednisolone 1 g intravenously once daily was started, followed by oral prednisolone 40 mg once daily thereafter.

Outcome and follow-up

The patient made slow initial progress, however, her oxygen demands reduced over the following days and her breathlessness eased. The inspiratory squeaks on auscultation of her chest were less marked but were persistent. After 5 days she was deemed fit for discharge with early clinic review booked.
High-resolution CT repeated at her clinic review showed resolution of the pneumomediastinum and improvement in interstitial changes. The avian specific IgG antibodies confirmed the working diagnosis and the birds were rehomed with instructions to deep clean the house. Total IgE was 94.8

(0–250), budgerigar specific IgG antibodies 179 (0–16), pigeon avian specific IgG antibodies 131 (0–21) and parrot avian specific IgG antibodies 163 (0–26). Antinuclear antibody, anti-dsDNA, extractable nuclear antigens and anti-CCP were negative or within normal limits. Pulmonary function tests showed forced expiratory volume in 1 s (FEV1) 1.06 (42% predicted), forced vital capacity (FVC) 1.14 (39% predicted), FEV1/FVC 0.93, KCO 86% predicted, residual volume 1.95 (117% predicted), vital capacity 1.03 (34% predicted) and total lung capacity 2.98 (62% predicted).
At recent clinic review, clinical and radiological improvement had continued while steroid requirements were reduced.

Discussion

Pneumomediastinum is a rarely reported manifestation of hypersensitivity pneumonitis: the authors found only three such cases in the literature.[1–3] More common causes of pneumomediastinum include the Valsalva manoeuvre, violent cough, emesis, acute bronchial asthma and mechanical ventilation.[2]
The mechanism behind pneumomediastinum is the rupture of marginally situated alveoli due to increased alveolar pressure.[2] In hypersensitivity pneumonitis, inflammation may lead to narrowing of the bronchioles, in turn increasing alveolar pressure due to obstruction of air flow.[3] Following rupture of the alveoli, air dissects along the bronchovascular sheaths to the lung hilum and mediastinal soft tissues, where it collects.[2]
In terms of explaining the clinical examination findings, in retrospect, the ‘squeaks’ heard on examination are likely to have been Hamman's sign. This is crepitus heard in time with the heartbeat (rather than respiration), due to movement of air in the mediastinum.[2]

Conclusion

this case highlights how initial investigations may mislead the clinician, and must be correlated with a thorough history and clinical examination in order to make a clear and timely diagnosis.

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