Radiology Forum

Hypersensitivity Pneumonitis Following Exposure to Lovebirds

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Clinical Summary

A 54-year-old man was referred for evaluation of respiratory symptoms of cough and breathlessness of 4 years. The symptoms had a temporal relationship with keeping lovebirds (*Agapornis roseicollis)* as pets. His vital parameters were within normal limits. Physical examination revealed clubbing, exercise desaturation and bilateral bibasilar crackles on chest auscultation.

Investigations

Haemogram and serum biochemistry were within normal limits. Chest radiograph (postero-anterior view) showed bilateral mid and lower zone reticulonodular opacities (Figure 1). High resolution computed tomography (HRCT) of thorax showed bilateral ill-defined centrilobular nodules, coalescing into areas of ground-glass attenuation, along with the presence of small apical para-septal bullae (Figure 2).

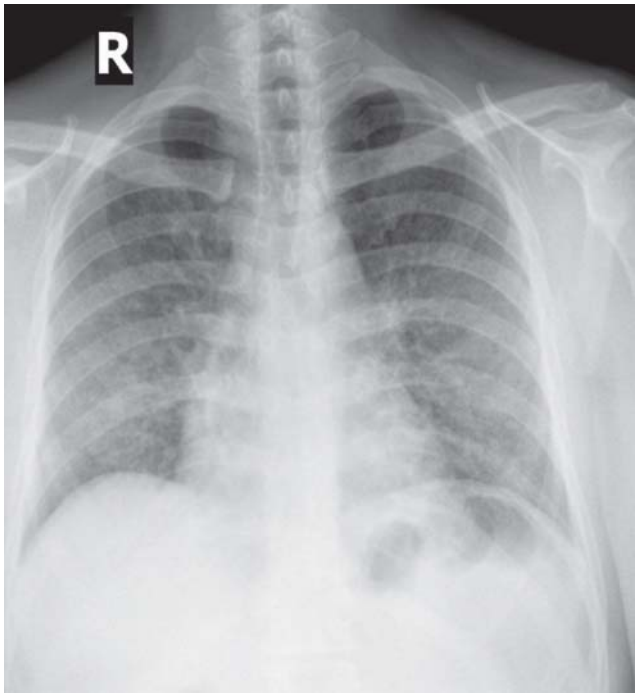

Figure 1. Chest radiograph (postero-anterior view) showing bilateral reticulonodular opacities.

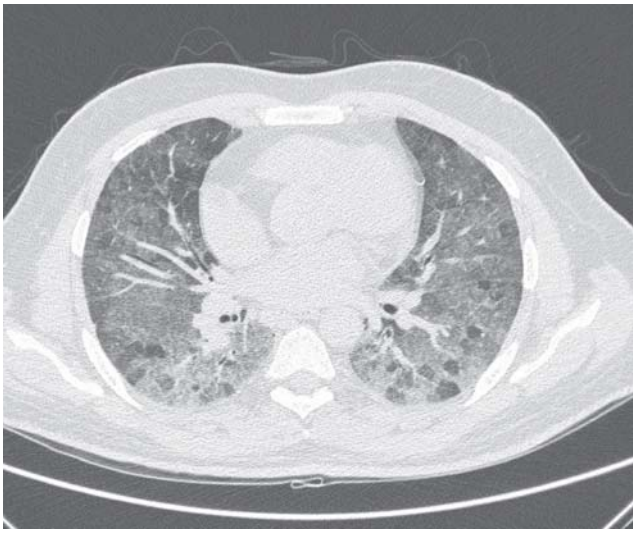

Figure 2: High resolution computed tomography (HRCT) thorax showing an ill-defined, centrilobular nodules, coalescing into areas of ground-glass attenuation in bilateral lung parenchyma.

Spirometry showed a restrictive abnormality with a forced vital capacity (FVC) of 1.70 litres (44% predicted), forced expiratory volume in first second (FEV_1) of 1.40 litres (45%), and FEV_1/FVC of 82%. Arterial blood gas analysis showed an increased $(A–a)O_2$ gradient of 54.35. A thoracoscopic lung biopsy was performed, which on histopathological examination showed inflammatory infiltrate around the bronchovasculature and ill-defined granulomas with giant cells around airways (Figure 3 A and B).

Diagnosis: *Hypersensitivity pneumonitis due to exposure to lovebirds.*

Discussion

Hypersensitivity pneumonitis (HP) is also known as extrinsic allergic alveolitis. It is an inflammation in and around terminal airways and the alveoli of the lung caused by hypersensitivity to inhaled organic dusts. The first account of this disease was presented in 1713 by Italian medical professor, Bernardo Ramazzini, who observed that grain workers are often short of breath and cachexic and are liable to develop orthopnoea.[1] The first detailed clinical description of HP was in 1932 in which reports mentioned the outbreak of

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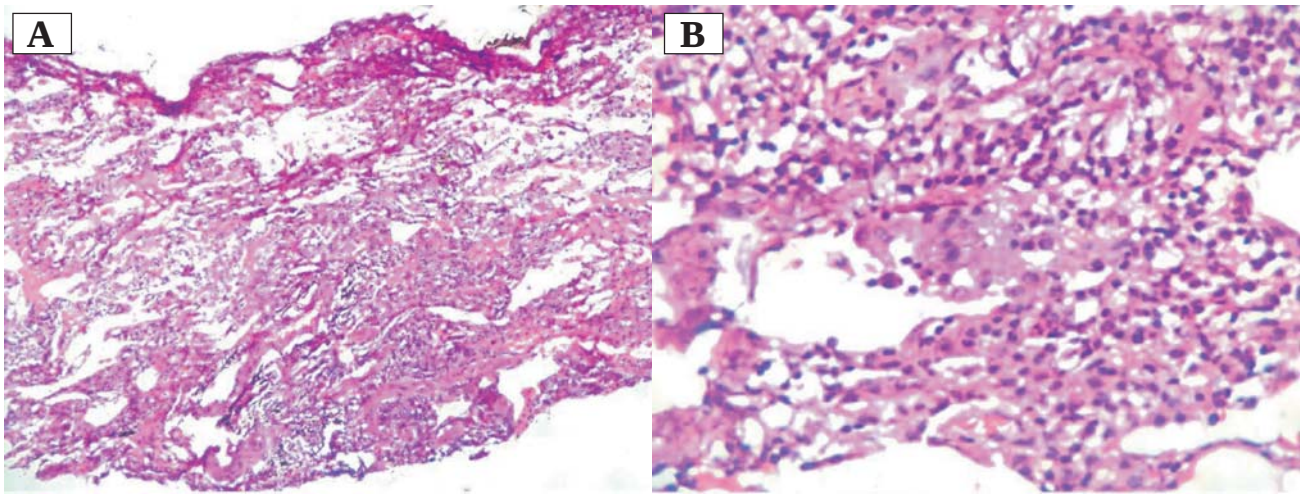

Figure 3. (A) Histopathology (Hematoxylin & Eosin × 10) showing inflammatory infiltrate around the bronchovasculature and (B) (Haematoxylin & Eosin × 40) showing mixed inflammatory infiltrate with occasional giant cell and ill-formed granuloma.

respiratory symptoms in employees of a rail road company in Michigan, USA.[2] Innumerable aetiological agents — bacterial, fungal, animal proteins, and chemical agents — have since been identified. Development and progression of the disease requires repeated exposure to the antigen, sensitisation of the host to the antigen, and immune mediated lung damage.

The clinical features of HP are also variable and start with a temporal relation with symptoms and certain activities/environmental or occupational exposure. Patients may present with rhinitis, cough, breathlessness, chest tightness, haemoptysis, night-sweats, palpitations, fatigue or weight loss of varying duration depending on the antigen. On chest radiograph, acute HP has lower lobe predominance, whereas chronic forms have upper lobe predominance. These may present as ground-glass opacities, ill-defined centrilobular nodules, coin lesions, diffuse nodular opacities, mediastinal lymphadenopathy, mosaic perfusion, honey-combing, consolidation and rarely pleural involvement in the form of pleural effusion or thickening.[3] Details of exposure to an offending antigen predicts HP with a very high odds ratio.[4] In addition, identification of the offending antigen is possible by specific precipitins using extracts from the patient's environment.[4] Histopathology shows characteristic airway centered inflammation, diffuse chronic interstitial inflammation, poorly-circumscribed, non-caseating granulomas with or without giant cells.[5]

The diagnosis of HP requires a strong clinical suspicion, temporal relation between symptoms and exposure, demonstration of precipitins against antigens directly extracted from the patient's environment and histopathological findings. Till date there are no formal guidelines or criteria to diagnose this condition. Our patient developed respiratory symptoms ever since acquiring lovebirds as pets. Clinical picture of the patient resembled an interstitial lung disease. HRCT thorax findings were consistent with chronic HP and it was confirmed on lung biopsy. Patient was started on oral corticosterioids (1mg/Kg/day) with an advise to avoid exposure to lovebirds. Follow-up after 3 months showed an improvement in his clinical condition and an increase in FVC by 500 mL and in FEV_1 by 440 mL. HP due to exposure to lovebirds has been reported previously[6] and is a recent addition to the long list of offending agents.

References

1. Ramazzini B. "*Diseases of Workers*" Wright WC, trans. New York: Hafner;1964:p.243.
2. Towey JW, Sweany HC, Huron WH. Severe bronchial asthma apparently due to fungus spores found in maple bark. *JAMA* 1932;99:453–9.
3. Hirschmann JV, Pipavath SNJ, Godwin JD. Hypersensitivity pneumonitis: a historical, clinical and radiological review. *Radiographics* 2009;29:1921–38.
4. Lacasse Y, Selman M, Costabel U, Dalphin JC, Ando M, Morell F, *et al*. Clinical diagnosis of hypersensitivity pneumonitis. *Am J Respir Crit Care Med* 2003;168:952–8.
5. Barrios RJ. Hypersensitivity pneumonitis: histopathology. *Arch Pathol Lab Med* 2008;132:199–203.
6. Funke M, Fellrath JM. Hypersensitivity pneumonitis secondary to lovebirds: a new cause of bird fancier's disease. *Eur Respir J* 2008;32:517–21.