OXFORD

*QJM: An International Journal of Medicine*, 2017, 233–234

**doi: 10.1093/qjmed/hcx010**
Advance Access Publication Date: 6 January 2017
Case report

CASE REPORT

# Feather bedding as a cause of hypersensitivity pneumonitis

J. Shaw, C. Leonard and N. Chaudhuri

From the Department of Respiratory Medicine, University Hospital of South Manchester, Wythenshawe M23 9LT, UK

Address correspondence to Dr J. Shaw, Respiratory Medicine, University Hospital of South Manchester, Wythenshawe M23 9LT, UK.
email: jonathonshaw@doctors.org.uk

**Learning points for clinicians**

Most cases of hypersensitivity pneumonitis have no underlying cause identified as the precipitant. Feather bedding is increasingly used in homes and may represent a more common aetiological agent in hypersensitivity pneumonitis than previously thought. Identification of precipitants requires careful history and investigation.

## Background

There are several antigens linked with the development of hypersensitivity pneumonitis. Amongst these, exposure to feather bedding is a 'rare' antigen linked to the development of hypersensitivity pneumonitis, although generally unrecognized as a causative antigen.[1] The current literature has scarcely investigated the link between feather bedding, an increasingly prevalent product in people's homes, and hypersensitivity pneumonitis.[2–4]

## Case presentation

In August 2012, a 47-year-old female suddenly developed generalised aches and pains, chills and dry cough which lasted for 10 days. Pre-morbid function was excellent. Exercise tolerance declined significantly. Home pulse oximetry showed oxygen saturations dropped to 80% on walking. Treatment with antibiotics and inhaled salbutamol prescribed by her GP gave no benefit. The patient found a temporal link between exposure to feather bedding and the onset of symptoms. On one occasion, camping with a feather duvet was associated with a general worsening in symptoms. Another episode occurred when staying on a friend's sofa which was stuffed with duck feathers. The patient works as a business analyst. There was no significant exposure to birds except occasional contact with a siblings budgerigar as a child over a 2-year period. The patient lives in a ground floor flat with no visible mould. She is a non-smoker and had no significant foreign travel prior to presentation. Past medical history is of resolved exercise induced asthma as a teenager and allergic rhinitis. The patient was not taking any medications prior to diagnosis.

## Investigations

In October 2013, the patient was referred for further investigation. Initial lung function testing showed a restrictive pattern with forced expiratory volume in 1 second (FEV1) of 2.04 (87% predicted), forced vital capacity (FVC) 2.43 (90% predicted), FEV1/FVC 96.7% and transfer factor of 3.9 mmolCO·min$^{-1}$·kPa$^{-1}$ (62% predicted). Forced expiratory flow (FEF) 25–75% was 2.42 (75% predicted). High resolution computed tomography (HRCT) showed minor patchy air trapping and mild ground glass shadowing in the lower lobes bilaterally (Figure 1). Broncho-alveolar lavage (BAL) showed a 70% lymphocyte predominance with a normal CD4/CD8 ratio. No mast cells were present. Transbronchial biopsy showed intra-alveolar macrophage accumulation and chronic interstitial mononuclear cell infiltrate. No granulomata were seen (Figure 2). Rheumatoid factor and anti-nuclear antibodies were negative. Total IgE (270 kU l$^{-1}$) and IgE to Cat Dander (5.3 kU l$^{-1}$), Dog Dander (2.1 kU l$^{-1}$) and Hamster (3.7 kU l$^{-1}$) were raised. Avian precipitin showed a raised Budgerigar IgG (45 mg l$^{-1}$, reference range 0.0–8.0) and normal pigeon IgG.

## Treatment

A diagnosis of subacute hypersensitivity pneumonitis was made at multi-disciplinary team (MDT). The patient was

**Submitted:** 17 December 2016

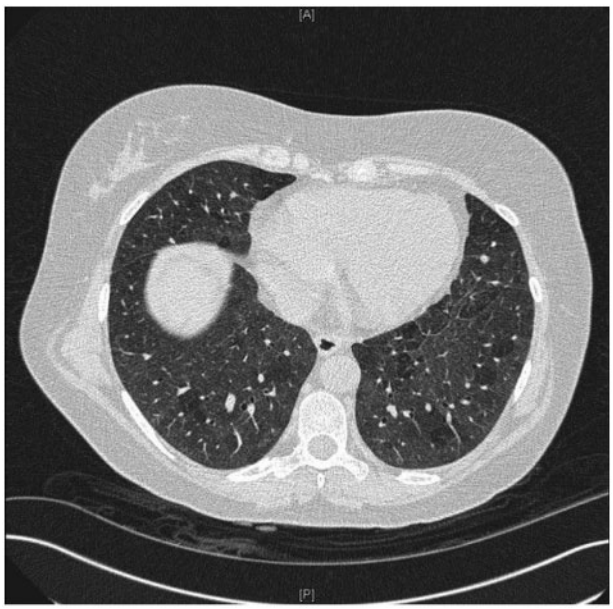


**Figure 1.** HRCT showing minor air trapping and mild ground glass change in the lower lobes bilaterally.

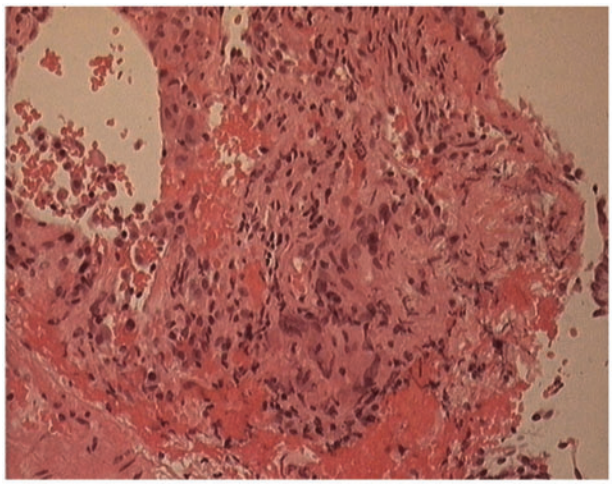

**Figure 2.** Transbronchial biopsy showing intra-alveolar macrophages and chronic interstitial mononuclear inflammatory infiltrate.

commenced on 30 mg prednisolone which gave excellent symptomatic benefit. Following cessation of a 6-week steroid course symptoms worsened. Following removal of her bedding, the patient found symptoms improved. Steroid therapy was restarted (30 mg prednisolone weaned by 2.5 mg every 2 weeks, then 1 mg per month wean from 10 to 0 mg) with good resolution of symptoms and improvement in lung function. Repeat lung function testing showed significant steroid responsiveness with a 32% improvement in FVC and 33% improvement in transfer factor [FEV1 2.62 (114% predicted), FVC 3.29 (122%), transfer factor 6.0 (95%)].

## Discussion

This case demonstrates an interesting temporal link between feather bedding exposure and respiratory symptoms. There were very high titres of budgerigar IgG despite no exposure since childhood. There is significant cross-reactivity between all avian antigens.[5] It is therefore possible that childhood exposure to a budgerigar acted as a primary sensitising event which led to immunological reaction to feather bedding. Exposure to occult avian antigens may represent a previously unrecognised aetiological factor in some cases of hypersensitivity pneumonitis. In many cases, no underlying antigen is identified. This suggests that in hypersensitivity pneumonitis, exposure to avian antigens including feather bedding should be screened for with careful history taking supplemented by immunological investigations if indicated.

*Conflict of interest*: None declared.

## References

1. Morell F, Villar A, Montero M, Muñoz X, Colby T, Pipvath S, *et al*. Chronic hypersensitivity pneumonitis in patients diagnosed with idiopathic pulmonary fibrosis: a prospective case-cohort study. *Lancet Respir Med* 2013; **1**:685–94.
2. Inase N, Ohtani Y, Sumi Y, Umino T, Usui Y, Miyake S, *et al*. A clinical study of hypersensitivity pneumonitis presumably caused by feather duvets. *Ann Allergy Asthma Immunol* 2006; **96**:98–104.
3. Koschel D, Wittstruck H, Renck T, Müller-Wening D, Höffken G. Presenting features of feather duvet lung. *Int Arch Allergy Immuol* 2010; **152**:264–70.
4. Haitjema T, van Velzen-Blad H, Van Den Bosch J. Extrinsic allergic alveolitis caused by goose feathers in a duvet. *Thorax* 1992; **47**:990.
5. Sennekamp J, Lange G, Nerger K, Berdel D, Meier-Sydow J. Human antibodies against antigens of the sparrow, blackbird, weaver finch, canary, budgerigar, pigeon and hen using the indirect immunofluorescent technique. *Clin Exp Allergy* 1981; **11**:375–84.