A 67-year-old man was admitted due to fever and exertional dyspnea. He used to raise budgerigars and hill mynahs from 1980 to 1988. He was diagnosed as having chronic bird fancier’s lung (BFL) based on a positive peripheral lymphocyte proliferation to pigeon serum in 1994. His disease was stable until 2000. Three months before admission he became a daily user of a feather duvet. A chest CT showed newly-developed peribronchial ground-glass opacities and preexisting honeycombing. Inhalation provocation test was positive. Administration of steroids improved his condition. He has been well after refraining from the use of the feather duvet.

Key words: feather duvet lung, chronic hypersensitivity pneumonitis, BFL, pulmonary fibrosis

Introduction

Bird fancier’s lung (BFL) is a type of hypersensitivity pneumonitis (HP), which is induced by inhalation of bird-related antigens (1). Patients with BFL sometimes present with acute HP but more frequently as chronic HP, since exposure to birds tends to be chronic with small amounts of antigens. When patients with BFL are exposed to bird-related antigens for many years, they develop extensive pulmonary fibrosis, which might be indistinguishable from idiopathic pulmonary fibrosis (IPF). Early recognition of the disease and complete avoidance of the causative antigen are crucial. Bird feathers have rarely been reported as a causative antigen of this disease although breeding of birds is generally recognized to induce BFL (2–5). We report here a case of chronic BFL presenting with acute exacerbation induced by the regular use of a feather duvet for 3 months although 12 years had passed after discontinuation of raising birds.

Case Report

A 67-year-old man was admitted due to fever and exertional dyspnea in 2000. He is an ex-smoker until 1989 (100 cigarettes per day for 36 years). He used to raise about ten budgerigars and mynahs for 8 consecutive years (1980–1988). His chest X-ray taken on annual check-up showed interstitial densities and he was diagnosed as having IPF at another hospital in 1988. Since he was advised to have further evaluation although he was asymptomatic, he visited our hospital in 1994. A chest X-ray showed reticular infiltrates in the both lung fields (Fig. 1) and a chest CT showed honeycombing and reticular opacities predominantly in the upper lobes. A bronchoalveolar lavage (BAL) fluid showed a slight lymphocytosis (24.1% of total cells) with a CD4/CD8 ratio of 19.5, and transbronchial lung biopsy specimens demonstrated lymphocytic alveolitis, Masson bodies, and organizing pneumonia. He was diagnosed as having chronic BFL based on a positive peripheral lymphocyte proliferation to pigeon serum. His disease was stable until 2000 by complete avoidance of avian contact. Two years before admission, he started using a feather duvet only on weekends at home. On weekdays, he lived alone to work in another city without a feather duvet. Three months before admission he retired from his work and became a daily user of the feather duvet. He noticed low grade fever and exertional dyspnea for the last 5 days in April 2000.

Physical examination revealed bilateral fine crackles and clubbed fingers. A chest X-ray showed diffuse ground glass opacities as well as the pre-existing reticular infiltrates in both lung fields (Fig. 2) and a chest CT showed newly-developed peribronchial ground-glass opacities and preexisting honeycombing (Fig. 3). White blood cells (9,400/μl), erythrocyte sedimentation rate (23 mm/h), and C-reactive...
protein (1.3 mg/dl) were increased. Serum LDH (266 U/l), KL-6 (1,330 U/ml), and SP-D (136 U/ml) were also elevated. Blood beta-D-glucan was normal and cytomegalovirus antigenemia was not detected. No pathogenic organisms were cultured. PaO$_2$ was 58 mmHg and PaCO$_2$ was 36.6 mmHg while breathing room air. Pulmonary function tests disclosed a normal VC (3.46 l, 101.3% of predicted, but decreased from 111.2%) and a decreased DLco (12.6 ml/min/mmHg, 62.9% of predicted). We performed BAL on the 17th hospital day and recovered 47% of instilled fluid, showing a slight increase in lymphocytes (20.7% of total cells) with a CD4/CD8 ratio of 17.8, neutrophils (23.6%), and eosinophils (6.4%). Inhalation provocation test using pigeon dropping extracts (PDE) was positive (6). Video-assisted thoracoscopic biopsy specimens, obtained on the 27th hospital day, showed peribronchial and subpleural honeycombing with lymphoid follicles, intra-alveolar exudative organization, and lymphocytic alveolitis around the terminal and respiratory bronchioles (Fig. 4). Intraalveolar accumulation of alveolar macrophages was prominent. Scant neutrophils and eosinophils were seen in the alveoli.

After administration of steroids, his condition improved in 4 weeks. Ground glass opacities on his chest CT were clearing, arterial blood gas data improved (PaO$_2$ of 79 mmHg and PaCO$_2$ of 41.9 mmHg), and a decreased DLco has returned to normal (16.9 ml/min/mmHg, 88.3% of predicted). He has been well on a low dose of steroid (prednisolone, 5 mg/day) after refraining from using a feather duvet not only at home but also at hotels on his trips.

**Discussion**

We report here a case of chronic BFL presenting with acute exacerbation due to exposure to a feather duvet. Feather duvets are becoming popular worldwide because they are portable and warm. The patient formerly raised about ten birds for 8 years and developed chronic BFL. He
immunological tests employing the bird-related antigens sup-
chronic HP, including BFL. Positive results of both the 
sensitive and specific examinations for the diagnosis of 
proliferation test and inhalation provocation test are utilized 
itive rate is low in chronic HP (6). Therefore, lymphocyte 
antibody can be detected in most cases of acute HP, its posi-
tigen were not detected in this case. Though antigen-specific 
the summer time was observed, suggesting that Trichosporon 
steroid, no deterioration or progression of his disease during 
tended in four cases (2–5). Three cases were induced by the 
acause of death in BFL, acute exacerbation is crucial, which 
might be triggered by focal infection (10, 11) and re-
exposure to the bird (12). Pathological findings in acute ex-
cerabation of BFL include diffuse alveolar damage with 
organized exudates in the airspaces containing hyaline mem-
branes (10, 12), which are similar to those in acute exacerba-
tion of IPF.

The use of a feather duvet should be discouraged in cases 
of BFL, because it may induce deterioration and progression 
of disease even after discontinuation of raising birds (13).

Acknowledgements: We thank Dr. S. Saiki for his pathological evalu-
ation and Dr. Vernon L. Moore for his critical review of the manuscript.

References

1) Fink JN, Solson AJ, Barbioriak JJ, Schluter DP, Holmes RA. Pigeon 
breeders’ disease. A clinical study of a hypersensitivity pneumonitis. 
2) Burdon JGW, Stone C. Bird fancier’s lung after an unusual exposure to 
3) Haitjema T, van Velzen-Blad H, van den Bosch JM. Extrinsic allergic 
alveolitis caused by goose feathers in a duvet. Thorax 47: 990–991, 
4) Kim KT, Dalton JW, Klaustermeyer WB. Subacute hypersensitivity 
pneumonitis to feathers presenting with weight loss and dyspnea. Ann 
5) Meyer FJ, Bauer PC, Costabel U. Feather wing lung: chasing a dead 
6) Ohtani Y, Kojima K, Sumi Y, et al. Inhalation provocation tests in 
7) Yoshizawa Y, Ohtani Y, Hayakawa H, Sato A, Suga M, Ando M. 
Chronic hypersensitivity pneumonitis in Japan: A nationwide 
9) Greenberger PA, Pien LC, Patterson R, Robinson P, Roberts M. End-
stage lung and ultimately fatal disease in a bird fancier. Am J Med 
10) Tasaka S, Kanazawa M, Kawai C, et al. Fatal diffuse alveolar damage 
12) Ohtani Y, Inase N, Miyake S, Yoshizawa Y, Saiki S. Fatal outcome in 
13) Inase N, Ohtani Y, Endo J, Miyake S, Yoshizawa Y. Feather duvet 