Budgerigar Breeders' Hypersensitivity Pneumonitis Presenting as Chronic Bronchitis with Purulent Sputum

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A 49-year-old woman complained of cough and a purulent sputum (about 40 ml/day). She kept budgerigars for a period of 30 years, she first noticed a productive cough 10 years previously. Her chest roentgenogram revealed a diffuse reticulonodular pattern. She was diagnosed as having a chronic type of budgerigar breeder's lung associated with chronic bronchitis. Peptostreptococcus anaerobius was cultured from her sputum, bronchoalveolar lavage fluid, as well as from droppings of her budgerigar. This is the second case of hypersensitivity pneumonitis presenting as chronic bronchitis with much sputum in Japan. (Internal Medicine 34: 676-678, 1995)

Key words: bird fancier’s lung, computed tomography, bronchoalveolar lavage, Peptostreptococcus anaerobius

Introduction

Bird fanciers’ hypersensitivity pneumonitis often involves chronic bronchitis (CB) and peripheral airway obstruction in addition to the typical restrictive pulmonary change (1, 2). Bourke and associates (1) reported that 8.4 percent of a total 287 pigeon fanciers had CB as their only manifestation of pigeon-related symptomatology. However, to our knowledge, there is only one report (3) of hypersensitivity pneumonitis presenting with copious sputum in Japan. We describe a case of budgerigar breeders’ hypersensitivity pneumonitis with a medium amount of purulent sputum, and discuss the bacteriological investigation of the patient and her budgerigar.

Case Report

The patient, a 49-year-old housewife, had kept budgerigars intermittently for a period of 30 years. She first noticed a productive cough in 1982. During the following nine years, she suffered from the cough and sputum almost every day, but never sought treatment. The color of the sputum changed to yellow in 1991. The symptoms began to worsen in June 1992, and she visited our hospital in August.

Her past medical history indicated no sinusitis. She was a lifelong nonsmoker. Her pulse rate was 76/min, and blood pressure was 116/80 mmHg. Auscultation of the chest revealed a fine crackle at bilateral back. A chest roentgenogram revealed a diffuse reticulonodular pattern (Fig. 1) and a high resolution CT scan showed poorly-defined nodular opacities and dense parenchymal consolidation (Fig. 2). A pulmonary function test revealed a reduction in carbon monoxide diffusing capacity to 42.0% of the predicted normal; PaO2 was 67.3 Torr. A 67-Gallium citrate radionuclide scan revealed no uptake in the lung. Serum precipitins against the serum and droppings of budgerigar and pigeon were all positive, however, the precipitins against P. anaerobius were negative. Transbronchial lung biopsy showed lymphocyte infiltration into the alveolar interstitium and the wall of bronchiolo, though no granuloma had formed. Bronchoalveolar lavage (BAL) recovered 425 cells per microliter in the following proportions: pulmonary alveolar macrophage, 41.4%; lymphocyte, 36.2%; neutrophil, 22.2%; and eosinophil 0.3%. The CD4/CD8 ratio of lymphocyte surface marker was 3.41.

The patient was hospitalized, after which the symptoms, the radiologic manifestations and the pulmonary function test showed gradual improvement. She returned home following 5 weeks of admission, re-exposure to her budgerigar for 7 days at home resulted in an increase of clinical signs: WBC increased from 4,500 to 10,000/cu mm, PA-aO2 gradient from 11.9 to 28.5, carbon monoxide diffusion capacity from 42.0 to 23.3%, one-hour erythrocyte sedimentation rate from 17 to 27mm and the opacities which had begun to fade from the CT were exacerbated again. Our tentative diagnosis of chronic form of budgerigar breeder’s lung was confirmed. She had also been...
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A patient was diagnosed with CB based upon the Medical Research Council definition: cough productive of sputum on most days of at least three months of two consecutive years (4). At the time of admission she had foul-smelling yellow sputa (40 ml daily), from which a large number of *P. anaerobius* was cultured. The same anaerobic organism was also cultured from the dropping of the patient's budgerigar and the BAL fluid, and both bacteria had the same antibiotic sensitivity. After 5 weeks admission and avoidance of the budgerigar, the cough disappeared and the sputa decreased to 10 ml daily. Two years later, the patient is doing well with little sputum.

**Discussion**

Bird breeders' lung was originally described by Pearsall et al (5) after examination of a breeder of parakeets, and has been shown to occur in persons handling various other birds. The major source of hypersensitivity pneumonitis is probably a bird serum that is secreted into the lumen of the gut, excreted, and inhaled in the form of droppings (6). CB occurs frequently in association with bird breeders' lung (1). CB occurred in 19.6–26.2% of nonsmoking pigeon fanciers with IgG antibody to pigeon gammaglobulin (1, 2), and in 23–26% of patients with farmer's lung (7, 8). However, in Japan there is only one case report (3) of hypersensitivity pneumonitis associated with CB having much sputum, and marked obstructive impairment of lung function being observed. In our case, there is no obstructive pulmonary change and asthmatic evidence.

The mechanism by which patients develop CB is unknown. The direct irritant effects of inhaled particle and immunological reactions of the host seem the most likely causative factors. Hargreave and colleagues (9) reported a case of pigeon breeders' disease with yellow sputa, as in our case; however, no bacteriological investigation was done. Bourke and associates (1) reported that the strong association between CB and the level of sensitization to pigeon gammaglobulin suggested immunologic rather than direct irritant effects as the most important in the pathogenesis of CB in pigeon breeders' disease. Hasani and colleagues (10) found that lung mucociliary clearance had been compromised in pigeon fanciers, thereby giving rise to an increased incidence of chest infections.

*P. anaerobius* was isolated in great number from both BAL of the patients and droppings of the budgerigar. Thus, this organism seemed to be the most probable cause of the infective exacerbation of CB in this case. *P. anaerobius*, a gram-positive streptococcus, was a part of the microflora in the bird's gut and the gums of the human oral cavity. In this case, the patient might have access to this organism by inhalation of dust of the budgerigar's droppings, because this organism was isolated from BAL fluid and the droppings and had the same antibiotic sensitivity. However, as this microorganism can be found in the oral cavity and the upper respiratory tract in humans, there is a possibility that *P. anaerobius* had been carried down from her oral cavity to the bronchial tree. We can not elucidate the origin of this organism. Nakajima et al (11) reported a case of bird-mediated *Pasteurella multocida* infection that exacerbated CB. The bacteria had been detected from that patient's sputum after feeding a macaw. The organism was not detected from the patient's sputum after treatment of the bird with antibiotics. It seems that a bacterial examination of the environment is also important in bird breeders' lung with CB. The precipitins to *P. anaerobius* were negative in this case and no case of hypersensitivity pneumonitis has been reported previously. We concluded that this case was diagnosed as having budgerigar.
breeders’ hypersensitivity pneumonitis.

Our case lacked acute symptoms of hypersensitivity pneumonitis such as sudden onset of chills, fever, malaise, or dyspnea; productive cough was the only manifestation. From the clinical course, this case had a chronic form of bird breeders’ lung. Pigeon breeders are normally exposed intermittently when cleaning out their pigeon lofts, while budgerigar fanciers, such as our case, are usually exposed more as their birds are kept indoors. For these reasons, pigeon fanciers generally present with acute symptoms relating to exposure, while budgerigar fanciers often present with a chronic disease (12).

References