BIRD-FANCIER'S LUNG: A CASE REPORT OF PROBABLE EXTRINSIC ALLERGIC ALVEOLITIS IN A PIGEON-BREEDER

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ABSTRACT

Pigeon-breeding in Singapore, as contrasted to bird-fancying in general, is usually on the scale of small holdings. This report of a case of pigeon-breeder's lung is made so that, among occupations in highly urbanised Singapore, those in the agronomics sector should not be forgotten.

Keywords: Avian proteins, hypersensitivity pneumonitis, removal from antigenic environments

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INTRODUCTION

As a cause of bird-fancier's lung, the pigeon is in league with other avians like chicken, canary and budgerigar. Wild birds, for reasons of scarcity in Singapore, much less a fancy for them, are seldom of importance.

Intermittent doses of inhaled avian protein and enzymes in pigeons' droppings and feathers cause pigeon-breeder's lung, which is a form of extrinsic allergic alveolitis (EAA), manifested in the late stage as interstitial fibrosis histopathologically (1). Hypersensitivity pneumonitis predates this change (2).

The inhaled avian antigen is usually two to five microns in aerodynamic diameter and can therefore reach the alveoli. There is evidence that the disease is more common in non-smokers. Perhaps the excess mucus secretion of smokers acts as an antigen trap and protects the acini (2). There is no relationship to atopy, unlike in the case of allergic broncho-pulmonary asper-gillosis (3).

Pigeon-breeder's lung is uncommon in Singapore. Understandably, extreme diagnostic vigilance is the basis for further relatively simple diagnostic elucidation. Presentation with cough is non-specific. However, after repeated exposures, breathlessness on exertion becomes a prominent symptom. It may become continuous and is not usually associated with wheezing (2). In the chronic stage, auscultatory signs are slight (4). Immunologically, IgA, IgG and IgM antibodies to avian antigen in sera can be detected by the ELISA technique (2).

However, there is no definite relationship between the antibodies titre and the occurrence of extrinsic allergic alveolitis (4).

CASE REPORT

A 38-year old commercial pigeon-breeder works out of several farms in Thailand. He has no such farm in Singapore. He visits and inspects his pigeon lofts once every two months or so, and stays at an out-house for up to a fortnight per visit. Other than making marketing decisions, he has to enter and check on the breeding areas daily, including lending a hand with the cleaning of the lofts whenever shorthanded. This takes a couple of hours of exertion at a time.

On 14 October 1987, a day after returning from one such trip, he presented with the complaint of nocturnal cough with sputum production, typically about six hours after work which ends at about 6 pm. There was breathlessness on exertion but no wheezing. He has no history of atopy or asthma. His symptoms usually started as "prodroma" at the commencement of his farm visits and "dragged on and worsened" when he was back in Singapore. Although he smokes more than twenty cigarettes a day, he claims to be hardly ever troubled by excessive mucus secretion.

Clinically, he was not cyanosed nor was there any finger clubbing. There were no adventitious sounds in the lungs. Peak expiratory flow rate as measured on a validated Wright mini peak flow meter were 410, 390, 420,380 and 400 L/min (5,6). He had no past PEFR readings in the absence of a nomogram for PEFR in the healthy Singapore population, PEFR nomogram for the British population by Ian Gregg and A J Nunn was adopted as a guide (7). The read-off for his height and age was 630 L/min.

Additional tests like antibody assay, which can be found in asymptomatic exposed individuals, were not done (4).

Prednisolone,30 mg per day plus orciprenaline 15 mg per day, were given as treatment for a week. At the eighth day, effort dyspnoea and nocturnal cough had abated. PEFR monitor readings were 550, 530, 500, 570 and 560 L/min. This reflects dramatic improvement.

DISCUSSION

The diagnosis of pigeon-breeder's lung was made on heightened index of suspicion and simple assessment. The obvious correlation of symptoms with "return to the antigenic environment" was sufficient from the practical point of view. Fulfilment of three of the five main known criteria of EAA diagnosis makes the case more than probable, viz. there was proof of the source of avian antigen, there were respiratory symptoms in the classical 4 to 10 hours after exposure and there was disturbance of lung function (4). The clear reversal

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of the lung function after appropriate treatment 'precludes' the need for chest radiograph, antibodies assay and broncho-alveolar lavage for IgA and IgG. Radiographic changes are not specific to extrinsic allergic alveolitis (2). They only confirm lung changes and also indicate severity of the disorder at that point of time for employees who seek compensation under occupational disease notification provisions. In the context of this patient's presentation, he being self-employed, what is of paramount importance is awareness of the source of antigen and its minimisation, amelioration of his symptoms and monitoring of his lung function. When the latter deteriorates, chest radiograph then becomes an obligatory adjunct to support the diagnosis.

The patient most likely suffers from the subacute form of EAA. He did not have constitutional symptoms like loss of appetite, weight loss, fever, chills, muscle aches etc which is the case in the acute form of EAA (4). He also probably has not reached the chronic stage because his impaired lung function was reversed after treatment. It is known that the chronic form may progress long after cessation of antigenic exposure (2). He may or may not have lung fibrosis in the future. Even if mild fibrosis should be detected on any future chest radiograph, there is no evidence that steroids can prevent progression of the fibrosis (2). The established place of steroids in therapy is at present one of amelioration of the acute symptoms. Monitoring of lung function cannot be over-emphasized. Detection of any decrease in carbon monoxide diffusion in future is evidence of failure in prevention.

A certain degree of air way obstruction is known to occur before it reaches chronicity (4).

CONCLUSION

The five main criteria of EAA diagnosis are history, immunology, bronchopulmonary lavage, lung function tests and chest radiograph interpretation (4). Not all are necessary as illustrated by this case. A well taken history with heightened suspicion of the condition enables the correct diagnosis to be made. Occupational history must never be neglected.

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