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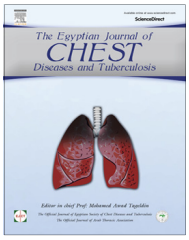

CASE REPORT

Pigeon fancier's lung – An under-diagnosed cause of severely debilitating and chronic breathlessness

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KEYWORDS

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Abstract Pigeon fanciers lung or Bird fanciers lung (BFL) is one of the common and preventable causes of hypersensitivity pneumonitis. It is an under diagnosed cause of severe incapacitating breathlessness and can be acute, sub-acute or chronic. We report a case of 53 year old female who presented with severe chronic breathlessness due to regular exposure to pigeons for last 35 years. Clinicians should take a detailed history of exposure in patients with unexplained breathlessness as the avoidance of exposure to the antigens can reverse the disease preventing the morbidity and mortality of the patient.

Introduction

Hypersensitivity pneumonitis (HP) also known as extrinsic allergic alveolitis is a non IgE mediated immune lung disease. Pigeon fanciers lung or Bird fanciers lung (BFL) is the most common form of hypersensitivity pneumonitis. It is caused by exposure to air-borne avian antigen which provokes a hypersensitivity reaction in a susceptible host. It is a rare and under diagnosed cause of severe debilitating breathlessness and needs a high clinical suspicion. Only a small proportion of exposed individuals develop clinically significant HP and genetic factors have been postulated to play a major role in determining an individual's risk of disease. Incidence also varies considerably and however incidence in India is not known.

Case report

A 53 year female married since last thirty five years, presented with history of progressive shortness of breath and dry cough for the past 4 years. Initially she complained of dyspnoea on mild to moderate exertion which progressively worsened since last 2 years and but now had dyspnea even at rest since last two months. She did not give any history of chest pain, orthopnea, paroxysmal nocturnal dyspnea (PND) or hypertension. There was no history of joint pains. She was a diabetic and was on treatment with metformin. She denied any history of active or passive smoking or any exposure to asbestos. She gave history of weight loss of about 7 kg in last 8 months and also having been exposed to pigeons since her marriage for last 35 years. The family had been feeding pigeons staying and breeding in the compound of their house since last two generations. There was no history of any of the other family members suffering from any pulmonary symptoms or disease.

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Patient was afebrile with a pulse rate of 100 beats/minute, a blood pressure 140/76 mmHg and a respiratory rate of 25/min. General physical examination did not reveal any digital clubbing, peripheral edema or cyanosis. Oxygen saturation was 85% by pulse oximetry at room air. Arterial blood gases (ABG) revealed a normal pH and $PaCO_2$ but hypoxemia with a PaO_2 of 65 mm Hg. Full blood count, serum electrolytes, serum IgE, serum ACE levels, renal, hepatic and thyroid function were within normal limits with hemoglobin of 12 g m% and a raised erythrocyte sedimentation rate (ESR). Rheumatoid arthritis (RA) factor and anti-neutrophilic cytoplasmic antibody (ANCA) were negative. Electrocardiogram (ECG) showed a normal sinus rhythm. Cardiac examination did not reveal any abnormality. Examination of the chest revealed bilateral basal and mid zone velcro type late inspiratory crackles with no wheeze. On further assessment she was found to be positive for anti-avian antibodies confirming a diagnosis of pigeon fanciers lung.

Chest radiograph showed bilateral interstitial reticular shadowing (Fig. 1). Computed tomography of the chest showed bilateral ground glass opacities with extensive honeycombing in both lungs (Fig. 2).

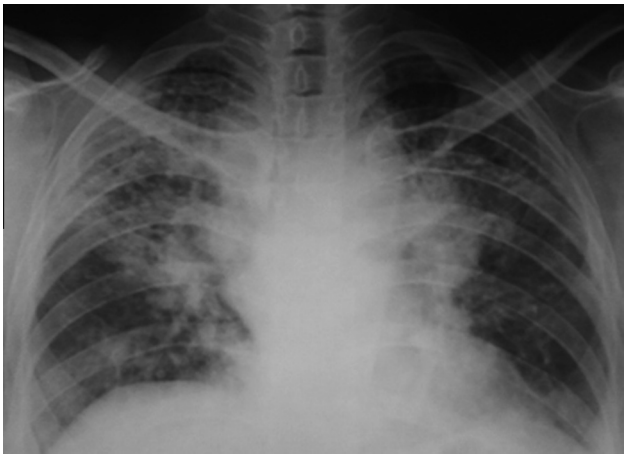

Figure 1 Xray Chest showing bilateral interstitial reticular shadowing.

Bronchoalveolar lavage (BAL) showed increased cellularity with elevated lymphocytosis. History along with clinical and radiological findings suggested the diagnosis of hypersensitivity pneumonitis (extrinsic allergic alveolitis).

Discussion

HP is an immunologic-induced, non-IgE mediated inflammatory lung disease resulting from the sensitization and subsequent recurrent exposure to any of a wide variety of inhaled organic dusts [1]. These diseases have been described in different occupational groups after exposure to aerosolized antigenic stimulus. Pigeon fanciers lung also known as Bird fanciers lung (BFL) is a very common type of HP [2] caused by airborne exposure to avian antigens which is known to be a particularly strong stimulus [3]. Susceptibility to the disease following exposure varies and differs in each individual as only a small proportion of the individuals exposed develop the disease. It is an uncommon and an under-diagnosed entity. Studies document 6000–21,000 cases per 100,000 persons per year for pigeon breeders in U.S [1] but the incidence in India is unknown.

HP presents as acute, sub acute or chronic forms based on the exposure to antigens and host response. Acute form presents after high level of exposure and the symptoms develop within 4 to 8 hours in the form of high grade fever with chills, muscle pains, fatigue and a non productive cough. Sub acute form is due to a relatively low level of exposure and the symptoms are more insidious. Chronic HP results from prolonged low level exposure to the antigens which lead to irreversible pulmonary damage without acute attacks [4]. It can be categorized as either chronic insidious or chronic recurrent HP [5]. Chronic HP presents as cough, malaise, weight loss and progressive dyspnea as in this case. Fever is mostly absent in the patients of chronic HP. Acute & sub acute form of disease may resolve by the avoidance of exposure. Chronic HP is a potentially severe disease which may be progressive, irreversible, and result in debilitating fibrotic lung disease [6]. It

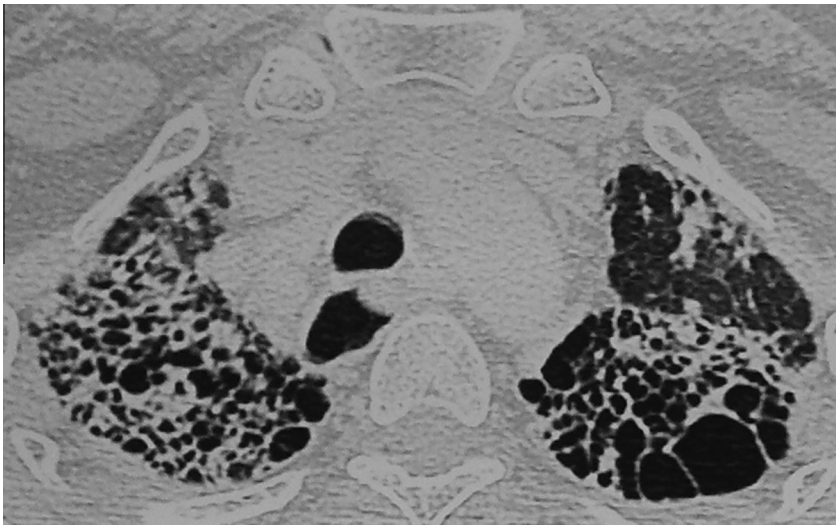

Figure 2 CT Chest showed bilateral ground glass opacities with extensive honeycombing in both lungs.

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Table 1 Diagnostic criteria for hypersensitivity pneumonitis.

Major criteria (four major criteria need to be present)
1. History of symptoms compatible with HP
2. Evidence of exposure to the offending antigen by history or through detection in serum or BAL fluid antibody
3. Changes of characteristic HP on chest radiograph (reticulonodular infiltrates, linear opacities) or HRCT of the chest (ground-glass opacities, micronodules, honeycombing, linear opacities, air trapping)
4. Demonstration of BAL fluid lymphocytosis, if BAL is performed
5. Demonstration of histologic changes consistent with HP, if lung biopsy is performed, such as alveolitis, noncaseating granulomas, giant cells, foamy alveolar macrophages, or fibrosis
6. Positive 'natural challenge' that produces symptoms and objective abnormalities either through controlled inhalational challenge or after re-exposure to the offending environment

Minor criteria (two minor criteria need to be present)
1. Bibasilar rales
2. Decreased diffusion capacity
3. Arterial hypoxemia, either at rest or with exercise

Table 2 Clinical prediction rules.

(1) Exposure to a known offending antigen
(2) Positive precipitating antibodies to the offending antigen
(3) Recurrent episodes of symptoms
(4) Inspiratory crackles on physical examination
(5) Symptoms occurring 4 to 8 hours after exposure
(6) Weight loss

may lead to respiratory failure. Since chronic form is characterized by fibrosing ILD the avoidance of the offending antigen may not result in complete resolution of symptoms though the continued exposure portends a poor prognosis [4].

The diagnosis of BFL is of a high clinical suspicion and requires an extensive environmental history. Lifestyle questions are integral part of the history and must include an assessment of potential hazards from occupational and animal exposure. Clinicians must take a detailed occupational and recreational history in any patient presenting with unexplained breathlessness. Early diagnosis of this entity is important as it may reverse the disease and if unchecked can lead to irreversible lung damage, respiratory insufficiency and even death [7]. Many criteria have been put forward for the diagnosis of HP of which BFL can be considered as HP prototype for sub-acute and chronic forms but none has been validated [2]. A diagnostic criteria of six major and three minor criteria out of which four major and two minor may be present has been suggested as in Table 1 [8]. This case fulfilled the four major and two minor criteria as required. A clinical prediction rule as an aid to accurate diagnosis of active HP has been developed and has been demonstrated to have a maximum probability of 98% of having HP when these predictors are present though it may guide the clinical practice by providing estimates of the probability of acute, subacute, or chronic progressive HP from noninvasive testing as in Table 2 [9]. Thus the requirement of BAL or lung biopsy would be unnecessary for the confirmation especially if HRCT is also consistent with the diagnosis [2] as in this case.

Treatment strategies include environmental control and medical therapy. Antigen avoidance and removal remains the single most important facet in the treatment of BFL and is crucial in it's management [2]. Continued exposure leads to persistent symptoms and progressive lung damage. Acute & sub acute form of disease may resolve by the avoidance of exposure. Medical therapy in the form of systemic corticosteroids may be useful. It will usually ameliorate symptoms of acute and subacute cases. Chronic form is characterized by fibrosing ILD. The avoidance of the offending antigen may not result in complete resolution of symptoms though the continued exposure portends a poor prognosis [4]. Steroids are also recommended for patients with severe or progressive chronic HP though its therapeutic efficacy is variable.

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