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Case Report

A Pigeon Fancier's lung; Subacute on Chronic Hypersensitivity Pneumonitis: A Rare Case Report

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ABSTRACT

Pigeon fancier's lung, a form of hypersensitivity pneumonitis (HP), is a rare but important occupational and recreational preventable cause of severe and debilitating breathlessness. We report the case of a 44-year-old Sri Lankan lady suffering with severe breathlessness due to pigeon fancier's lung. She has bred pigeons for 20 years. She presented with progressive dyspnea, non-productive cough, fatigue and had bilateral end inspiratory velcro type crackles with hypoxemia at rest. There were reticular nodular opacities on CXR and diffuse ground glass opacities, inter- and intralobular thickening with subpleural sparing associated with patchy traction bronchiectasis and lobular air trapping. She had marked improvement to glucocorticoid pulses followed by maintenance. We diagnosed this patient as subacute on chronic HP considering clinical and radiological evidence. Avoidance of allergen and steroids advised with regular follow up to monitor progression.

Keywords: Hypersensitivity pneumonitis (HP); Bird fancier lung (BFL); Pigeon fancier's lung

INTRODUCTION

Hypersensitivity pneumonia (HP), or extrinsic allergic alveolitis, is a spectrum of interstitial, alveolar and bronchiolar lung disease caused by non IgE mediated immunologic reaction occurring within lung parenchyma to sensitization and subsequent recurrent exposure to various inhaled antigens that may lead to irreversible damage. Pigeon fancier's lung, a form of hypersensitivity pneumonitis, is an unusual but important occupational and recreational preventable cause of severe and debilitating breathlessness. It is caused by exposure to air-borne avian antigen which provokes a hypersensitivity reaction in a susceptible host. We report the case of a 44-year-old Sri Lankan lady suffering with severe breathlessness due to pigeon fancier's lung. She has bred pigeons for 20 years.

CASE STUDY

A 44-year-old non-smoker, housewife from an urban area admitted to National Hospital for respiratory diseases emergency treatment unit due to progressive dyspnea at rest for six days. She had an exertional dyspnea of Modified Medical Research Council (mMRC) grade 2 severity, a dry cough and fatigue for past 3 months. Recently she has developed acute worsening of dypnoea up to mMRC grade 4 associated with fever and productive cough for six days. She had neither pleuritic type chest pain nor hemoptysis nor epistaxis. She had no childhood asthma. She denied any oral ulcers, photosensitivity rashes, arthralgia and myalgia. There were no known allergies. She had not been on any long-term medications. She had no self or family history of any rheumatological diseases. She has been exposed to pigeon dust since her marriage due to pigeon keeping and cleaning the lofts at home for twenty years (Figure 1).

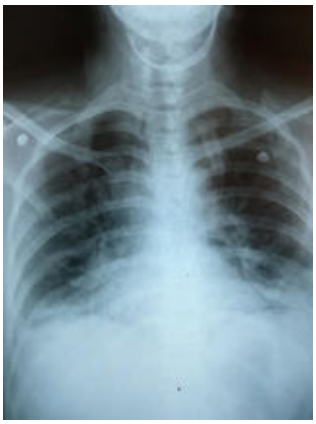

Figure 1: CXR AP of the patient on day 1 symmetrical reticular nodular shadow involving both lower zones.

On examination, she was breathless at rest but alert and rational. She was afebrile. She had no cervical lymphadenopathy, no finger clubbing and no peripheral oedema. She was admitted with

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tachypnea (32/min), tachycardia (108beats/min), blood pressure around 125/76 mmHg, and cyanosed with oxygen saturations 89% on 60% oxygen; jugular venous pressure was raised and pulsatile. On auscultation, there was bilateral vesicular breath sound with coarse bilateral late inspiratory crackles along with accentuated P2. Head, neck and abdominal examination was unremarkable. She had no features of rheumatological diseases.

Her sputum and blood gram stain were negative, and cultures had no bacterial growth. Sputum was negative for acid fast bacilli on both direct smears and TB PCR. Urine for Legionella antigen, serum for mycoplasma antibody, PCR for CMV, Sputum for pneumocystis on silver stain, sputum for fungal studies and cryptococcal antigen, nasopharyngeal swabs for H1N1 and retroviral studies were negative.

Her autoimmune profile was negative for ANA titer, dsDNA, cANCA, pANCA and rheumatoid factor.

Her chest Xray showed symmetrical reticular nodular shadows involving both lower zones. Her HRCT revealed patchy ground glass opacities and inter- and intra-lobular thickening with subpleural sparing involving both lungs in anterior segment of right upper lobe, apicoposterior segment of left upper lobe, right middle lobe, left lingular lobe and both lower lobes. Patchy areas of traction bronchiectasis were seen in right middle lobe, left lingular lobe and both lower lobes with no areas of honeycombing. Lobular air trapping was noted with no pleural effusions. The overall impression was subacute on chronic hypersensitivity pneumonitis.

Echocardiogram showed mild pulmonary hypertension. Pulmonary function tests and bronchoalveolar lavage could not be done during this acute clinical presentation.

She was given continuous positive airway pressure support (CPAP) in the intensive care unit (ICU). She was initially started on broad spectrum antibiotics – piperacillin tazobactam, clarithromycin, linezolid, clindamycin and cotrimoxazole in treatment doses. With very low procalcitonin levels and radiological evidence of subacute on chronic hypersensitivity pneumonitis she was started on intravenous methylprednisolone 750 mg daily up to three pulses followed by high dose oral prednisolone 1 mg/kg/day maintenance dose. Her oxygen requirements dramatically reduced within three days of glucocorticoids. The patient showed remarkable response to glucocorticoids and discharged from ICU on third day to continue care at high dependency unit.

During hospital stay, she improved gradually. Except for steroid induced hyperglycemia she did not complaint of gastrointestinal side effects of glucocorticoids. She was also started on calcium 1200 mg and vitamin D 800IU daily for bone protection.

We suspected that this case was pigeon dust-induced hypersensitivity pneumonitis due to long duration of direct exposure at home with pigeon keeping. She was diagnosed to be a case of chronic HP considering her prolonged exposure along with pulmonary hypertension, which was secondary to chronic HP. Her condition was explained to her and her family members. Thereafter, she was counseled to consider modification in her livelihood. She was discharged on oral prednisolone 30 mg daily and planned to readmit for further evaluation in two weeks.

RESULTS AND DISCUSSION

Hypersensitivity pneumonia (HP), or extrinsic allergic alveolitis, is a spectrum of interstitial, alveolar and bronchiolar lung disease caused by non IgE mediated immunologic reaction occurring within lung parenchyma to sensitization and subsequent recurrent exposure to various inhaled antigens, such as proteins, bacteria, and fungi that may lead to irreversible damage. HP can present in acute, subacute or chronic forms depending on the type, intensity and duration of exposure to the causative agent [1-5].The disease is histologically characterized by a triad of non-necrotizing granulomas, chronic inflammatory changes in the small airways, and diffuse interstitial infiltrates of the chronic inflammatory cells[6].

Pigeon fancier's lung, a form of hypersensitivity pneumonitis, is an unusual but important occupational and recreational cause of severe and debilitating breathlessness. It is caused by exposure to air-borne avian antigen which provokes a hypersensitivity reaction in a susceptible host. The prevalence depends on the intensity, duration of exposure and individual host factors [7]. Studies document 6000–21,000 cases per 100,000 persons per year for pigeon breeders in U.S but the incidence in Sri Lanka is unknown. 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 [8].

The diagnosis of bird fancier's lung (BFL) is of a high clinical suspicion with extensive history of potential hazards from occupational and animal exposure. 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 [9]. Our patient who met with three major and two minor criteria with the limited invasive diagnostic investigations at this acute setup to diagnose chronic HP. 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 from noninvasive testing as in [10]. Thus the requirement of BAL or lung Biopsy would be Unnecessary for the confirmation especially in the HRCT.
Also consistent with the diagnosis [11] as in our case. The prognosis is generally favorable with early identification, prompt removal of the offending antigen and symptomatic treatment with corticosteroids [1]. In our patient the disease has progressed to chronic state with complications. However, continued antigen exposure can lead to a permanent loss in lung function, irreversible pulmonary fibrosis and even premature death [1].Steroids are also recommended for patients with severe or progressive chronic HP though its therapeutic efficacy is variable. Long – term corticosteroid therapy for the treatment of chronic HP should be considered only if objective improvements in clinical signs, pulmonary function, or radiographic abnormalities are documented [6].

CONCLUSION

We emphasize the importance of professional history taking in patients presenting with dyspnooea in regard to potential occupational and recreational hazards because early detection and avoidance have proven good prognosis.

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