Case Report

Home environmental fungus: the hidden killer

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ABSTRACT

Hypersensitivity pneumonitis is generally attributed to inhalational organic dust, commonly due to exposure to dust at occupation or hobbies. Hypersensitivity pneumonitis or extrinsic allergic alveolitis is an inflammatory syndrome of lungs resulting from immunologically induced inflammation secondary to various airborne allergens. It is relatively rare in childhood. Knowledge of classical HRCT finding of lungs and use of antigen specific IgG and IgM antibodies (despite the false positive and false negative) analysis can act as supportive evidence for making diagnosis of Hypersensitivity pneumonitis. We report a case of 4 yrs old girl child who despite being symptomatic, remained undiagnosed for 18-24 months, was on bronchodilator therapy suspecting pathophysiology being Hyperreactive airway disease but had recurrent episodes. When her hypoxic condition worsened later on, she was further investigated to rule out other differential, HRCT done was suggestive of childhood interstitial lung disease. The allergens test done for aspergillus species was positive in home environment and in her blood. She responded to steroids therapy and removal of offending agents from home environment. This case shows the importance of home environment in causing life threatening respiratory disease in children. She was diagnosed with Hypersensitivity pneumonitis, rare in childhood.

Keywords: Aspergillus fumigatus, Extrinsic allergic alveolitis, Hypersensitivity pneumonitis, Indoor pollutants

INTRODUCTION

Hypersensitivity pneumonitis (HP) is an immune-mediated syndrome affecting pulmonary alveoli and interstitium, associated with repeated exposure to varied environmental antigens. Hypersensitivity pneumonitis (HP) is rare in children; primary allergic sources being exposure to pet birds (or feather in bedding) or homes contaminated with pigeon’s antigens. Molds from prior flooding or damp floors, humidifiers and hot tubs are other sources. Symptoms includes cough, dyspnea, chest tightness, chills, malaise, fatigue; these can occur within 4-6 hours or over several years after exposure to antigen. Avoidance of the inciting antigens is the mainstay of treatment. Exposure to specific domestic indoor fungal spores is considered unlikely cause of Hypersensitivity pneumonitis hence we are presenting our case.2

CASE REPORT

4-year-old female child initially was brought with complaints of repeated episodes of cough and cold for 6 months. At that time her investigation showed increased Ig E levels and she was treated with anti-allergic (Montelukast) for 6 weeks following which she showed some improvement.1 year later she came with acute breathlessness at rest and productive cough on exertion. On examination she was found to have dyspnoea at rest, Spo2 <85 % on room air, bilateral decreased air entry and crepitation. She was reinvestigated, complete blood cell
Aspergillus fumigatus induced pulmonary disease may or may not involve elevated serum IgE or fungal colonization; luckily though both were positive in this child.4,6

Incidence of Interstitial lung disease is 30/100000 individual of which that HP was less than 2%.7 Since 1960, 95 cases of HP in children have appeared in literature (8-10) of which mean age of onset was 10 years and youngest case reported had onset of symptoms at 8 months of age. 97% of children were treated with removal from exposure and 65% with corticosteroids therapy, which is known to induce long term remission; as is the case with our patient who is 8-year-old now.

A history of worsening of symptoms on exposure to a potential agents and improvement in symptoms once the affected individual is away from that environmental space is essential for diagnosis and treatment. The interval between exposure to antigens and clinical feature of lung disease is unknown.

Its known that if its low level of continuous exposure, then onset of symptoms of hypersensitivity pneumonitis can be insidious as in the present case. Our patient had repeated episodes of cold and cough for 13 months; followed by episodes of acute breathlessness for which she was hospitalized. Her HRCT confirmed diagnosis of chronic hypersensitivity interstitial lung disease and her specific IgE to Aspergillus fumigatus was positive which was also grown from her home environment. Her dramatic clinical improvement away from home had helped us in identifying the source.

CONCLUSION

The mainstay of treatment for HP is removal from the offending agent and treatment with glucocorticoids which fastens the pulmonary function normalization as in our patient.

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REFERENCES
