Case Report

Symptomatic Pulmonary Siderosis – A case report

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Abstract

Pulmonary siderosis or Welder's lung is a rare occupational lung disease which occurs due to long term inhalation of iron compounds. It is most commonly seen in arc-welders. Most of the patients are asymptomatic with mild or no functional impairment. We report a case of pulmonary siderosis with symptomatic respiratory disease and restrictive pattern in pulmonary function test.

Key words

Pulmonary Siderosis, Welder's lung.

Introduction

Pulmonary siderosis otherwise known as Welder's lung is a rare occupational lung disease which occurs due to chronic inhalation of iron compounds. It is most commonly seen in arcwelders, oxyacetylene cutters, iron ore miners and silver polishers. It is considered to be a benign form of pneumoconiosis, because of the absence of associated respiratory symptoms and functional impairment [1]. However pulmonary siderosis associated with symptomatic disease has been reported among welders [6-8]. Here we report a case of pulmonary siderosis presenting with symptomatic respiratory disease.

Case report

A 30 year old male presented to the Department of Respiratory Medicine with complaints of exertional breathlessness of Grade 2 MMRC scale for a period of 1 year. He had no history of orthopnea or PND. He had no other respiratory complaints. There was no history of fever, loss of appetite or weight. He had no history of previous anti- tubercular treatment. There was no history of atopy or allergy and no history of exposure to

birds and pets. He was working as a welder for the past 14 years. He was a non-smoker and was not on any medications. On general examination, there was no pallor, icterus, clubbing, cyanosis, lymphadenopathy or pedal edema. His respiratory rate was 24/minute and his saturation was 97% on room air. His respiratory system examination was normal and other systemic examinations were normal.

His blood investigations were within normal limits. His spirometric evaluation showed a restrictive pattern with low FVC of 62% predicted. DLCO was decreased to 60 % predicted. His chest radiograph revealed bilateral diffuse nodular opacities (Photo - 1). A computed tomographic scan (CT) of the chest showed bilateral diffuse centrilobular nodules seen throughout the lung fields (Photo - 2). Based on clinical and radiological findings diagnoses differential of pneumoconiosis, sarcoidosis and hypersensitive pneumonitis were considered. Bronchoscopy was done which showed normal bronchial tree. BAL and TBLB were obtained. The bacterial, fungal and AFB cultures from the BAL fluid were negative. BAL cytology showed black and brown pigmented macrophages. The biopsy specimens showed type 2 pneumocyte hyperplasia and interstitial fibrosis (Photo - 3b) containing sheets of macrophages containing iron pigment which was confirmed with Perl's Prussian blue staining (Photo - 3a). The diagnosis of pulmonary siderosis was confirmed by histopathology. The patient was given counselling to change his job and was treated symptomatically with inhaled bronchodilators.

Discussion

Siderosis is most commonly observed in workers who are exposed to metal fumes containing iron during welding, mining, steel polishing and silver polishing. Siderosis was first described by Doig and McLaughlin in 1936 who conducted a prospective study in 16 arc-welders, 15 of them were followed up for a period of 9 years irrespective of their radiological progression or regression. There was no evidence of any respiratory symptoms or significant functional impairment among the subjects [2]. In another study pathological examination of 4 subjects who are exposed to iron oxide fumes did not demonstrate any evidence of pulmonary fibrosis [3]. Hence it was concluded that siderosis is a 'benign pneumoconiosis'.

<u>Photo - 1</u>: Chest X-ray showing bilateral nodular opacities.



Meyer et al did a spectrographic analysis of lung tissue in a symptomatic arc welder presenting with interstitial fibrosis and attributed the symptoms to the simultaneous inhalation of silica or asbestos which could be seen in the fumes arising from electrode coating [4]. However, Funahashi, et al. conducted a study on lung tissues collected from 10 symptomatic welders and demonstrated interstitial fibrosis and subsequently conducted energy dispersive X-ray analysis (EDXA) for elemental components and compared with controls. He demonstrated significant amount of iron content in the tissues and the silicon content did not vary from control subjects and hence concluded that interstitial pulmonary fibrosis can be seen solely due to the iron content present in the welding fumes [5]. Similarly cases of symptomatic respiratory disease with interstitial fibrosis secondary to chronic iron exposure have been reported [6-8]. Our patient had diffuse centrilobular nodules without any obvious areas of fibrosis on CT chest, however histopathological examination revealed significant interstitial fibrosis along with iron laden macrophages. He had exertional breathlessness of MMRC grade 2 at presentation. J. Szram, et al. conducted a systematic review of

all available published longitudinal studies on lung function decline in welders which found obstructive pattern and rapid decline in FEV1 in smokers compared to non-smokers [9]. Our patient was a non - smoker and had low FVC suggestive of restrictive pattern and decreased diffusion capacity.





<u>**Photo - 3A</u>**: Perl's Prussian blue staining demonstrating iron pigments.</u>



Conclusion

We conclude that patients with pulmonary siderosis can develop lung fibrosis and present with respiratory symptoms after significant years of exposure to iron fumes. We also insist the importance of obtaining the occupational history in all patients with respiratory diseases. Refraining from iron dust exposure and enacting strict preventive measures such as using respiratory protective masks and adequate ventilation of work places remains the mainstay of treatment. Our patient had significant improvement after the avoidance of exposure to iron dust.

<u>Photo - 3B</u>: Demonstrating interstitial fibrosis and pneumocytes hyperplasia.



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