

Case Report

Interstitial Lung Disease due to Siderosis in a Lathe Machine Worker

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Abstract

Since its first description in 1936, siderosis of lung has been considered a benign pneumoconiosis due to absence of significant clinical symptoms or respiratory impairment. Subsequently, authors have questioned the non-fibrogenic property of iron. However, siderosis causing interstitial lung disease with usual interstitial pneumonia (UIP) pattern has not been described in the past. We report a case of UIP on high resolution computed tomography, proven to be siderosis on transbronchial lung biopsy in a lathe machine worker.

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Key words: Siderosis, Usual interstitial pneumonia.

Introduction

Siderosis is caused by the accumulation of iron oxide in macrophages within the lung. Though it has been described to cause interstitial lung disease (ILD) with severity linked to duration of exposure since 1946,¹ it had been believed to be a benign pneumoconiosis because of near absence of any significant signs or symptoms or associated fibrosis. Subsequently, functional impairment,²⁻⁴ symptomatic disease with interstitial fibrosis,⁵ and even progressive massive fibrosis⁶ has been described secondary to iron exposure. However, siderosis causing usual interstitial pneumonia (UIP) has not been reported in the past. We report a case of chronic iron exposure with radiological evidence of possible UIP, due to siderosis.

Case Report

A 62-year-old male, presented to the outpatient department with complaints of cough with minimum mucoid expectoration and a gradually progressive dyspnoea on exertion for a period of three years. He had worked as a lathe machinist, involved in making iron spare parts from casted iron, without the use of protective gear, exposing him to iron dust for 40 years. The patient was a chronic *bidi* smoker with smoking index of 900. He was hypertensive and under treatment with amlodipin and atenolol for the last two years. General physical examination revealed clubbing. Respiratory system examination revealed presence of bibasilar fine end inspiratory crepitations. Haematological, renal and hepatic investigations were within normal limits. The pre- and post-exercise (walk test) saturation and pulse were 95/88 and 89/98,

respectively. Two-dimensional echocardiography demonstrated a grade I diastolic dysfunction with left ventricular ejection fraction of 55% and pulmonary artery pressure of 25 mmHg. Spirometry was suggestive of a mixed defect. The forced vital capacity (FVC) was 2.36 (69% predicted), forced expiratory volume in the first second (FEV₁) was 1.63 (56% predicted) and FEV₁/FVC ratio was 68 with 11% and 180 mL improvement in FEV₁ following inhaled bronchodilator.

Chest radiograph (postero-anterior view) projection (Figure 1) demonstrated the presence of reticular opacities in bilateral lung fields. High resolution computed tomography (HRCT) demonstrated the presence of intra-lobular septal thickening, ground-glass opacities with evidence of honey-combing at

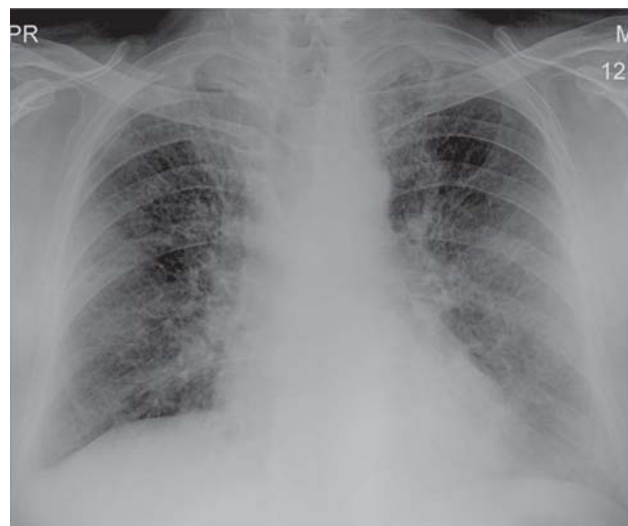


Figure 1. Chest radiograph (postero-anterior view) showing the presence of reticular opacities in bilateral lung fields.

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places (Figure 2). There was predominant upper lobe involvement with relative sparing of lower lobes.

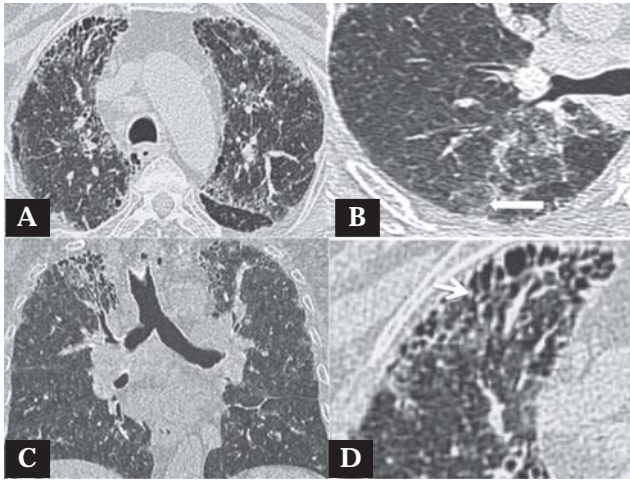


Figure 2. High resolution computed tomography of lung showing (A) axial images at the level of arch of aorta showing interstitial thickening and sub-pleural honey-combing, (B) axial images at the level of carina showing ground-glass opacities (arrow) and interstitial thickening, (C) (coronal image) showing predominant upper lobe involvement and relative sparing of lower lobes, and (D) magnified view of honey-comb cysts (arrow).

Transbronchial lung biopsy showed interstitial fibrosis with an inflammatory infiltrate, composed of chronic inflammatory cells. The macrophages among inflammatory cells were laden with pigment and stained positive with Perl's Prussian blue stain, suggestive of siderosis (Figure 3).

occupations at risk include mining and processing of iron ores, iron and steel rolling mills, foundry workers and silver polishers. Siderosis has not been reported in a lathe machine worker till date.

Doig and McLaughlin⁵ first described 'welders' siderosis in 1936. They carried out a prospective study examining the clinical and radiological features of lung in 16 electric-arc welders and followed 15 of them for nine years. Irrespective of radiological progression or resolution, all subjects demonstrated absence of respiratory symptoms or significant pulmonary functional impairment. The pathological examination of the lung did not demonstrate any evidence of pulmonary fibrosis.⁶ This led to the conclusion that siderosis is a 'benign pneumoconiosis'. Subsequently, Buckell *et al*¹ in 1945 reviewed siderosis and did suggest the possibility of fibrosis. Multiple case series with spirometric abnormalities have also been reported.²⁻⁴ Symptomatic disease with interstitial fibrosis has also been described secondary to iron exposure.^{7,8} Even, a case of progressive massive fibrosis has been reported recently.⁹

In a study by Akira in 1995,¹⁰ correlating computed tomography (CT) features of pneumoconiosis with histological findings, honey-combing was observed on CT in 3 out of 21 arc welders. The CT appearance resembled that of UIP; however a pathological basis of this finding was not established. In another study,¹¹ lung mineralogical analysis of patients with idiopathic pulmonary fibrosis (IPF) revealed iron content in the lung but this was not correlated with exposure.

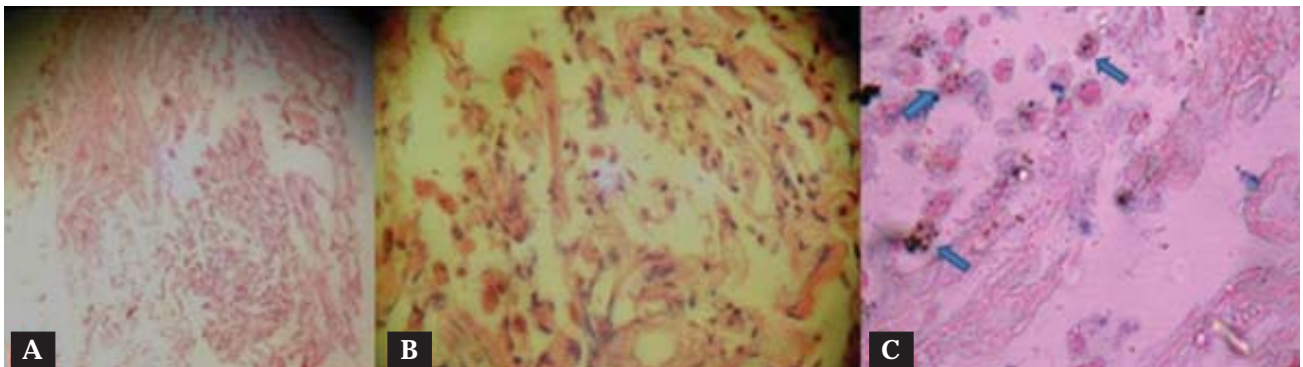


Figure 3. Transbronchial lung biopsy showing (A) alveoli with interstitial fibrosis, pigment laden macrophages in alveoli and interstitium with mild inflammation (Haematoxylin and Eosin \times 100), (B) alveolar and interstitial foamy macrophages with golden brown pigment (Haematoxylin and Eosin \times 400), and (C) Perl's Prussian blue stain positive for iron (arrows) X400 magnification.

The patient on follow-up after one year showed reduction in FEV₁ by 100 mL and FVC by 20 mL but there was no significant worsening on HRCT thorax.

Discussion

Siderosis is observed most commonly in workers exposed to metal fumes during welding, either electric-arc or oxyacetylene, and thus, is popularly called welders siderosis or arc welder pneumoconiosis. Other

In the present case, HRCT was suggestive of an ILD possibly with a UIP pattern. Absence of traction bronchiectasis ground-glass opacification and upper lobe predominance were features inconsistent with definite UIP seen in IPF. Histopathology confirmed the diagnosis of interstitial fibrosis due to siderosis showing the presence of iron in fibrotic alveolar septa. The patient was a chronic smoker but the clinico-radiological and pathological findings cannot be

attributed to smoking. This is because the classical finding of UIP associated with smoking², i.e., lower lobe bibasilar involvement was not seen in the present case. Instead there was predominant upper lobe involvement which is characteristically seen in siderosis.¹³ Moreover, the transbronchial lung biopsy showed interstitial fibrosis with iron laden macrophages confirming to interstitial fibrosis secondary to siderosis.⁷

Spirometry demonstrated a mixed abnormality. Iron exposure is known to cause obstructive airway disease even in non-smokers²⁻⁴ and smokers are at a greater risk of developing obstructive defect while exposed to iron dust.¹⁴ Thus, obstructive abnormality in this patient could be due to both smoking and iron exposure. Smoking likely reduces the lung's ability to remove dust by damaging the respiratory epithelium. Also, by inhaling more deeply during smoking in the work-place, smokers may inhale more dust than non-smokers. Thus, smoking may have compounded the effect of iron exposure in the present case. Absence of any protective gear probably increased lung deposition of iron dust.

The present case is supported by a review where Taskar and Coultas¹⁵ had suggested that IPF may be a misnomer and multiple environmental agents may cause pulmonary fibrosis in susceptible individuals. According to the authors, six exposures were significantly associated with IPF including smoking, agriculture/farming, livestock, wood dust, metal dust and stone/sand. However, the classical IPF has a lower lobe predominance in contrast to the upper lobe predominance seen in our case.

In conclusion, our report illustrates symptomatic siderosis with ILD and a possible UIP pattern. Siderosis may not be considered a benign pneumoconiosis any further since cases demonstrating fibrosis and functional impairment are being documented. It is imperative to advocate use of protective gear even for iron exposure. It should be realised that the clinical, radiological and pathologic manifestations of occupational ILD are similar to the non occupational

cause. The clinician must maintain a high degree of suspicion and obtain a complete occupational history to establish a potential exposure causing ILD.

References

1. Buckell M, Garrad J, Jupe MH, McLaughlin AIG, Perry KMA. The incidence of siderosis in iron turners and grinders. *Br J Industrial Med* 1946;3:78-82.
2. Skloot G, Goldman M, Fischler D, Goldman C, Schechter C, Levin S, et al. Respiratory symptoms and physiologic assessment of iron workers at the World Trade Center disaster site. *Chest* 2004;125:1248-55.
3. Johnson A, Moira CY, MacLean L, Atkins E, Dybuncio A, Cheng F, et al. Respiratory abnormalities among workers in an iron and steel foundry. *Br J Industrial Med* 1985;42:94-100.
4. Gomes J, Lloyd O, Norman N, Pahwa P. Dust exposure and impairment of lung function at a small iron foundry in a rapidly developing country. *Occup Environ Med* 2001; 58:656-62.
5. Doig AT, McLaughlin AIG. X-ray appearances of the lungs of electric arc welders. *Lancet* 1936;1:771-5.
6. Doig AT, McLaughlin AIG. Clearing of X-ray shadows in welders' siderosis. *Lancet* 1948;1:789-91.
7. McCormick LM, Goddard M, Mahadeva R. Pulmonary fibrosis secondary to siderosis causing symptomatic respiratory disease: a case report. *J Med Case Reports* 2008;5:257.
8. Nemery B. Metal toxicity and the respiratory tract. *Eur Respir J* 1990;3:202-19.
9. Cheng JI, Gang C, Hou-rong C, Fan-qin M, Yan-bin C, Ling-chuan G, et al. An unusual case of welder's siderosis with local massive fibrosis: a case report. *Chinese Med J* 2012;125:552-4.
10. Akira M. Uncommon pneumoconioses: CT and pathologic findings. *Radiology* 1995;197:403-9.
11. Siegesmund KA, Funahashi A, Pinter K. Identification of metals in lung from a patient with interstitial pneumonia. *Arch Environ Health* 1974;28:345-9.
12. Attili AK, Kazerooni EA, Gross BH, Flaherty KR, Myers JL, Martinez FJ. Smoking-related interstitial lung disease: radiologic-clinical-pathologic correlation. *Radiographics* 2008;28:1383-96.
13. Glazer CS, Newman LS. Occupational interstitial lung disease. *Clin Chest Med* 2004;25:467-78.
14. Kuo HW, Chang CL, Liang WM, Chung BC. Respiratory abnormalities among male foundry workers in central Taiwan. *Occup Med* 1999;49:499-505.
15. Taskar VS, Coultas DB. Is idiopathic pulmonary fibrosis an environmental disease? *Proc Am Thorac Soc* 2006;3:293-8.