Pulmonary siderosis presenting with a miliary nodular pattern: A case report

Sonal P. Karpe[1], Ganesh B. Shiral[1], Kunal R. Mahajan[1], Shivraman Natarajan[1], Jairaj P. Nair[1]

ABSTRACT

Pulmonary siderosis is an uncommon occupational lung disease resulting from chronic inhalation of iron oxide particles, typically affecting welders, grinders, and iron industry workers. Although classically regarded as a benign pneumoconiosis, radiological and histopathological overlap with other diffuse lung diseases can pose diagnostic challenges, particularly in tuberculosis-endemic regions. We report a case of a middle-aged male, a former iron-factory worker, who presented with cough and exertional dyspnea and was found to have a diffuse miliary nodular pattern on high-resolution computed tomography (HRCT). Bronchoalveolar lavage (BAL) revealed hemosiderin-laden macrophages positive for Prussian blue stain, confirming pulmonary siderosis. Pulmonary siderosis can closely mimic miliary tuberculosis, particularly in TB-endemic regions. Careful occupational history and bronchoalveolar lavage with Prussian blue staining are essential to establish the correct diagnosis and avoid unnecessary antitubercular therapy.

AFFILIATION

1 Department of Pulmonary Medicine, Lokmanya Tilak Municipal General Hospital and Medical college, Mumbai, India

CORRESPONDENCE TO

Sonal P. Karpe. Department of Pulmonary Medicine, Lokmanya Tilak Municipal General Hospital and Medical College, Sion, Mumbai, India
E-mail: sonimoni85@gmail.com
ORCID iD: https://orcid.org/0000-0002-3007-1155

KEYWORDS

miliary nodules, pulmonary siderosis, Prussian blue stain

Received: 8 October 2025
Revised: 22 December 2025
Accepted: 10 June 2026

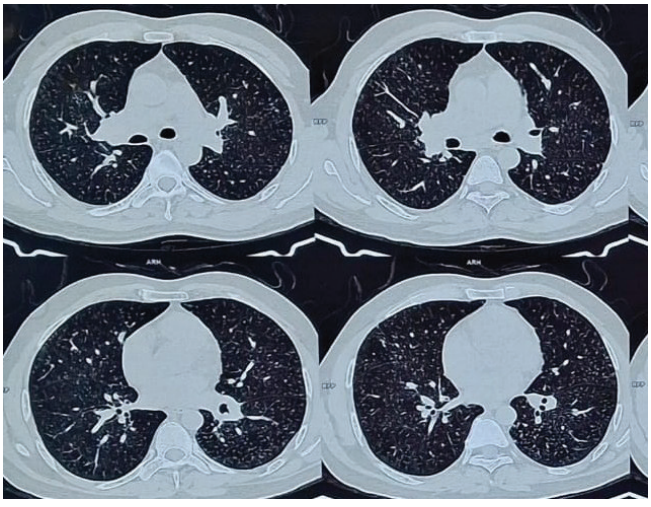
INTRODUCTION

Pulmonary siderosis, also known as welder's lung, is an occupational lung disease caused by chronic inhalation of iron oxide dust or fumes[1,2]. Traditionally considered a benign pneumoconiosis without fibrosis, recent evidence suggests that prolonged or mixed-dust exposure may result in interstitial lung disease[3-5]. Radiologically, pulmonary siderosis may present as diffuse micronodules, occasionally mimicking miliary tuberculosis, particularly in TB-endemic countries[6,7]. Bronchoalveolar lavage (BAL) with Prussian blue staining offers a simple, cost-effective, and confirmatory diagnostic approach[8]. This case is unique due to its striking miliary radiological pattern in a patient with prior extrapulmonary tuberculosis, creating a significant diagnostic dilemma.

CASE REPORT

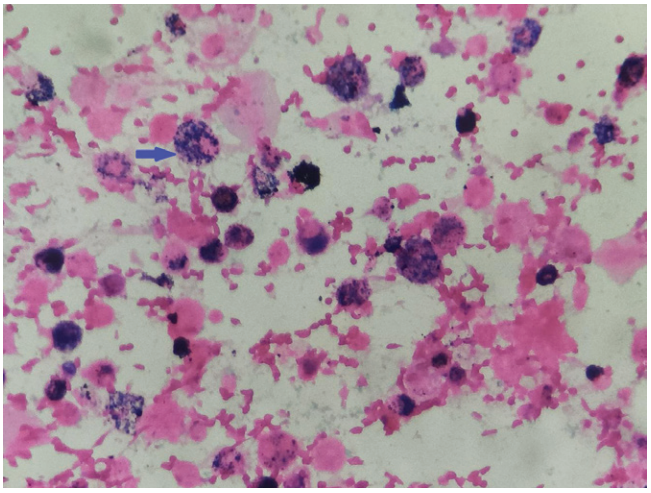
A 51-year-old man presented to the respiratory outpatient department with cough producing mucoid expectoration for 10 days, associated with low-grade evening fever for 5–6 days and mild exertional dyspnea (Modified Medical Research Council Grade I, static for one month). He denied hemoptysis, chest pain, palpitations or orthopnea. He had worked in a stainless-steel factory for several years but had ceased exposure two years prior and reported no ongoing or alternative iron exposure. He was currently employed as a security guard. He had been treated for extrapulmonary tuberculosis (cervical lymphadenopathy) eight months earlier, with no documented pulmonary involvement at that time for which he completed a full six-month course of antitubercular therapy. There was no history of diabetes, hypertension, ischemic heart disease, or occupational exposure to silica, asbestos, or organic dusts. He denied any history of TB contact or smoking. On examination, he was afebrile and hemodynamically stable. Oxygen saturation was 97% on room air. Bilateral diffuse fine inspiratory crackles were heard over the chest. There was no cyanosis, clubbing,

Figure 1. High resolution chest CT scan showing randomly distributed miliary nodules



Pneumon 2026;39(2):8
https://doi.org/10.18332/pne/225707

Figure 2. Prussian blue-stained bronchoalveolar lavage specimen from a 51-year-old Indian male, former iron-factory worker, demonstrating hemosiderin-laden macrophages (blue arrow) consistent with pulmonary siderosis



or pedal edema. Routine hematological and biochemical investigations were within normal limits.

Chest radiograph showed multiple tiny diffuse nodular opacities in both lung fields. HRCT thorax showed diffuse miliary nodules involving all lobes without fibrosis or cavitation (Figure 1). BAL analysis was negative for AFB smear, Lowenstein–Jensen culture, MGIT culture, bacterial and fungal cultures, and malignant cells. Prussian blue staining demonstrated numerous hemosiderin-laden macrophages (Figure 2). The patient was counseled to avoid any further exposure to iron dust and was advised to use protective respiratory equipment at work. The patient received inhaled bronchodilators, formoterol (4.8 µg) with glycopyronium (9 µg) twice a day for 1 month and supportive care. At follow-up at 3 months, symptoms improved and imaging showed no progression.

DISCUSSION

Pulmonary siderosis results from occupational inhalation of iron oxide particles[1-4]. Although historically considered benign, persistent radiological abnormalities may remain long after exposure cessation[5]. However, modern HRCT and histopathological studies indicate that some patients may develop interstitial fibrosis, particularly with concurrent exposure to silica or other irritants[2,5]. Inhaled iron particles are engulfed by alveolar macrophages and deposited as hemosiderin within alveoli and interstitium. These iron-laden macrophages can be demonstrated by the Prussian blue reaction, which stains ferric iron as deep blue granules[3]. Persistent exposure triggers macrophage activation and release of pro-fibrotic cytokines, contributing to interstitial changes[5].

Most cases are asymptomatic and detected incidentally on imaging. Symptomatic patients may present with chronic cough or exertional dyspnea. In our patient, respiratory symptoms appeared after cessation of exposure, suggesting a delayed inflammatory response possibly potentiated by recent infection. Chest radiographs typically show fine nodular opacities predominantly in the upper lobes. HRCT provides greater sensitivity, demonstrating micronodules, ground-glass opacities, or reticulation[6]. Pulmonary siderosis may mimic miliary tuberculosis, emphasizing the need for tissue or cytological confirmation[7]. Cases with diffuse deposition of iron particles and miliary patterns have been rarely described, especially in workers with prolonged exposure and fine-particle inhalation[1,8]. Importantly, in our patient, co-existent miliary tuberculosis was carefully excluded through repeated microbiological evaluation and radiological stability on follow-up. Although coexistence has been reported, negative cultures and absence of progression argued against active TB[6,7].

Bronchoalveolar lavage is a minimally invasive diagnostic method when transbronchial or surgical biopsy is not indicated. Demonstration of hemosiderin-laden macrophages positive for Prussian blue stain confirms the presence of intra-alveolar iron[3]. Differentials include idiopathic pulmonary hemosiderosis, chronic alveolar hemorrhage, and mixed-dust pneumoconiosis; clinical and occupational history aids differentiation[4]. There is no specific pharmacological therapy for pulmonary siderosis. The mainstay is elimination of exposure to iron oxide dust. Symptomatic management includes bronchodilators and, occasionally, corticosteroids if airway inflammation predominates. Prognosis can be good in pure siderosis and sometimes regression of radiological findings after cessation of exposure have been documented[5,9].

Strengths and limitations

Strengths of this report include detailed occupational history, comprehensive microbiological exclusion of tuberculosis, and confirmatory cytological diagnosis. Limitations include absence of histopathology and relatively short imaging follow-up.

CONCLUSION

Pulmonary siderosis can closely mimic miliary tuberculosis, particularly in TB-endemic settings. This case highlights the critical importance of occupational history and targeted diagnostic evaluation. BAL with Prussian blue staining can prevent misdiagnosis and unnecessary antitubercular therapy.

CONFLICTS OF INTEREST

The authors have completed and submitted the ICMJE Form for disclosure of Potential Conflicts of Interest and none was reported.

FUNDING

There was no source of funding for this research.

ETHICAL APPROVAL AND INFORMED CONSENT

Ethical approval was not required for this study. The patient provided informed consent for the publication of his clinical data. A CARE checklist is provided in the Supplementary file.

DATA AVAILABILITY

The supporting data are available from the authors upon reasonable request.

AUTHORS' CONTRIBUTIONS

SPK: conception and study design, literature search, clinical data acquisition, data analysis, experimental studies, statistical analysis, manuscript drafting and editing. GBS, KRM, SN and JPN: study design, literature search, clinical data acquisition, and data analysis. All authors reviewed and approved the final version of the manuscript.

PROVENANCE AND PEER REVIEW

Not commissioned; externally peer reviewed.

SUPPLEMENTARY FILE DISCLAIMER

The content has been provided by the author(s) and has not been reviewed, verified, or endorsed by European Publishing. It may not have undergone peer review. The views, opinions, and recommendations expressed are solely those of the author(s) and do not necessarily reflect the position of European Publishing. European Publishing accepts no responsibility or liability for any consequences arising from the use of, or reliance on, this content.

REFERENCES

1. Oh SJ, Hwang KE, Jeong ET, Kim HR. A case of pulmonary siderosis misdiagnosed as pneumonia. Respir Med Case Rep. 2018;25:58-60. doi:10.1016/j.rmcr.2018.06.006
2. McCormick LM, Goddard M, Mahadeva R. Pulmonary fibrosis secondary to siderosis causing symptomatic respiratory disease: A case report. J Med Case Rep. 2008;2:257. doi:10.1186/1752-1947-2-257
3. Meyer KC, Raghu G, Baughman RP, et al. An official American Thoracic Society clinical practice guideline: The clinical utility of bronchoalveolar lavage cellular analysis in interstitial lung disease. Am J Respir Crit Care Med. 2012;185(9):1004-14. doi:10.1164/rccm.201202-0320ST
4. Banks DE, Parker JE. Occupational lung disease. In: Broaddus VC, Mason RJ, Ernst JD, King TE Jr, Lazarus SC, Murray JF, eds. Murray & Nadel's Textbook of Respiratory Medicine. 7th ed. Elsevier; 2022.
5. Masanori A. Imaging diagnosis of classical and new pneumoconiosis: Predominant reticular HRCT pattern. Insights Imaging. 2021;12(1):33. doi:10.1186/s13244-021-00966-y
6. Chong S, Lee KS, Chung MJ, Han J, Kwon OJ, Kim TS. Pneumoconiosis: Comparison of imaging and pathologic findings. Radiographics. 2006;26(1):59-77. doi:10.1148/rg.261055070
7. Zhou Y, Wang Y, Liu W, et al. Case report: Application of morphology in the diagnosis of siderosis in a patient with tuberculosis infection. Front Oncol. 2023;13:1001802. doi:10.3389/fonc.2023.1001802
8. Spalgais S, Kumar R, Mrgipuri P. Symptomatic pulmonary siderosis in scissors/knife sharpening worker: A case report. Indian J Occup Environ Med. 2020;24(1):42-44. doi:10.4103/ijoem.IJOEM_77_19
9. International Labour Organization. Guidelines for the Use of the ILO International Classification of Radiographs of Pneumoconioses. ILO; 2011. Accessed June 10, 2026. https://www.ilo.org/publications/guidelines-use-ilo-international-classification-radiographs-pneumoconioses-0