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Smoking-Related Interstitial Lung Disease with Nonspecific Interstitial Pneumonia Pattern: A Case Report

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Abstract

Smoking-related interstitial lung disease (SR-ILD) manifesting as nonspecific interstitial pneumonia (NSIP) is a rare entity within the spectrum of tobacco-induced pulmonary disorders. We describe a 61-year-old asymptomatic male with a 40-pack-year smoking history and significant occupational exposures, whose abnormal chest X-ray—detected during routine mining surveillance—led to further evaluation. High-resolution computed tomography (HRCT) revealed bilateral ground-glass opacities, reticular thickening, and early honeycombing, consistent with fibrosing NSIP. Bronchoscopic biopsy confirmed a cellular NSIP pattern, and histological findings included anthracosis and silica-like crystals, supporting the contribution of environmental dust exposures. Comprehensive investigations excluded connective tissue disease and other interstitial lung disease etiologies. This case underscores the diagnostic challenges of SR-ILD with NSIP and highlights the importance of multidisciplinary team (MDT) input, especially in patients with complex exposure histories. In asymptomatic individuals with preserved lung function, a conservative management approach emphasizing smoking cessation, exposure mitigation, and longitudinal monitoring is appropriate. This report reinforces the critical role of occupational health surveillance in detecting subclinical ILD and the need for further research into SR-ILD pathogenesis and treatment strategies.

Keywords: Smoking-related interstitial lung disease; Nonspecific interstitial pneumonia; Fibrosing NSIP; Multidisciplinary diagnosis; Occupational exposure; Mining

Introduction

Smoking-related interstitial lung disease (SR-ILD) comprises a spectrum of parenchymal disorders linked to chronic tobacco exposure, including respiratory bronchiolitis-associated ILD (RB-ILD), desquamative interstitial pneumonia (DIP), and pulmonary Langerhans cell histiocytosis (PLCH) [1,2]. Nonspecific interstitial pneumonia (NSIP), although classically associated with connective tissue disease (CTD) and idiopathic presentations, is an uncommon manifestation of SR-ILD [3,4]. The true prevalence of SR-ILD with NSIP remains undefined, as diagnosis is often confounded by overlapping features with emphysema and chronic obstructive pulmonary disease (COPD), especially in asymptomatic individuals [3,5]. NSIP typically affects individuals aged 40–60, with a slight male predominance reflecting global smoking patterns [4,6]. Clinically, SR-ILD with NSIP presents with exertional dyspnoea, dry cough, and fine bibasilar crackles [5]. Radiologically, NSIP manifests as bilateral ground-glass opacities, reticular thickening, and basal-predominant fibrosis without honeycombing or traction bronchiectasis characteristic of usual interstitial pneumonia (UIP) [5,7]. Histologically, it demonstrates uniform interstitial thickening and chronic inflammatory infiltrates, lacking fibroblastic foci or granulomas [5,8]. Tobacco smoke induces repeated alveolar epithelial injury and macrophage activation, releasing pro-inflammatory cytokines such as TNF-α, IL-1β, and TGF-β, which promote fibrosis [9,10]. Occupational exposures to inhaled dusts—particularly silica or asbestos—may act

synergistically, further complicating the attribution of causality in patients with mixed exposure histories [11,12]. Diagnosis relies on multidisciplinary team (MDT) evaluation, integrating clinical presentation, HRCT, and histopathology. Discordance between imaging and biopsy findings is common and requires comprehensive synthesis of exposure history and clinical data [7,13]. This case report illustrates the diagnostic and management approach to a patient with asymptomatic SR-ILD manifesting as fibrosing NSIP.

Case Presentation

A 61-year-old male with a 40-pack-year smoking history (ceased 5 years ago) was referred to the respiratory clinic after an abnormal chest X-ray was noted during routine occupational screening for mining workers. He was entirely asymptomatic—denying dyspnoea, cough, chest pain, wheeze, or haemoptysis. Medical history included type 2 diabetes mellitus (treated with sitagliptin/metformin), hyperlipidaemia (rosuvastatin), and hypertension (perindopril). There was no history of previous respiratory conditions, autoimmune disease, or relevant family history. His occupational exposure history included 35 years in above-ground coal and mineral mining, 15 years in the construction industry (with potential asbestos and iron dust exposure), and 2 years in underground gold mining with tunneling and blasting activity, likely involving silica dust exposure. Use of personal respiratory protection was inconsistent. On examination, he was afebrile with a respiratory rate of 18 breaths/min, BP 142/86 mmHg, heart rate 64 bpm, and SpO_2 98% on room air. Chest auscultation revealed fine bibasilar crackles. There were no signs of digital clubbing or cutaneous features of CTD.

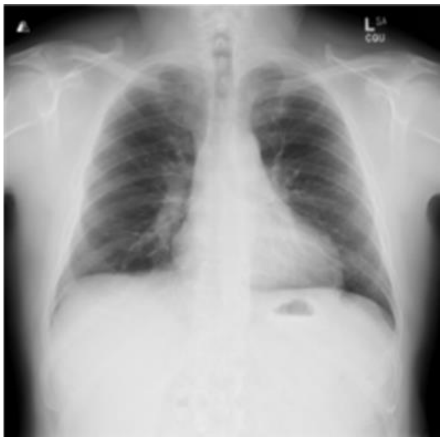

Figure 1: *Abnormal mining chest x-ray showing a prominent pulmonary trunk, prompting respiratory referral.*

Chest HRCT revealed bilateral ground-glass opacities, subpleural and perifissural reticular thickening, early honeycombing, and paraseptal emphysema (predominantly upper lobes), suggestive of fibrosing NSIP [3,7]. No mosaic attenuation, air trapping, or tree-in-bud patterns were identified. Pulmonary function tests (PFTs) were within normal limits: FEV_1/FVC 82% (104.3%), FEV_1 3.19 L (101%), FVC 3.92 L (96.8%), TLC 5.56 L (87.6%), RV/TLC 25.3% (83.9%), and KCO 3.75 mmol/min/kPa (96.4%). A six-minute walk test showed no desaturation (SpO_2 post-walk 97%), and distance walked was 580 m (92% predicted). Bronchoscopy showed no endobronchial lesions.

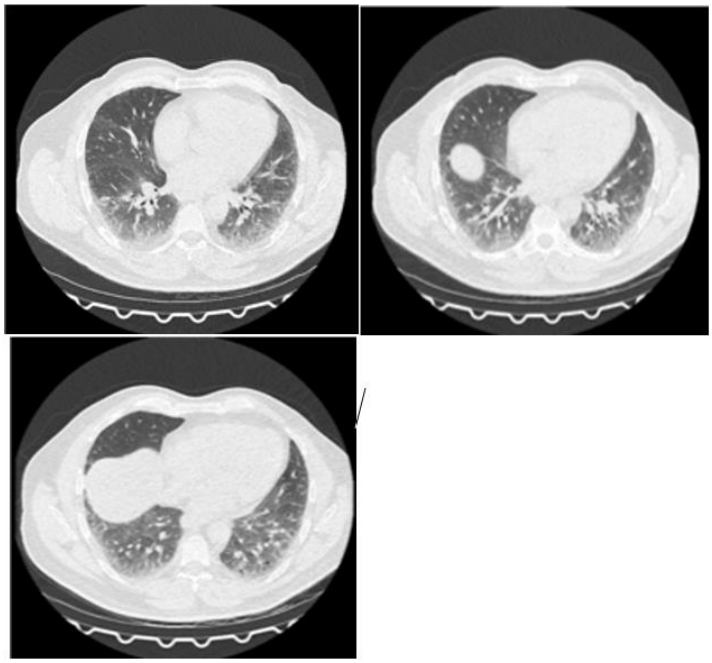

Figure 2: *HRCT showing bilateral ground-glass opacities with subpleural and perifissural reticular thickening, along with early honeycombing.*

BAL cytology revealed 60% macrophages, 30% lymphocytes, 8% neutrophils, and 2% eosinophils. Cultures for bacteria, mycobacteria, fungi, and viruses were negative. Transbronchial biopsies from the left lower lobe revealed a cellular NSIP pattern: interstitial lymphocytic infiltrates, mild fibrosis, patchy type II pneumocyte hyperplasia, and scattered alveolar macrophages. Polarized light microscopy revealed mild anthracosis and silica-like crystals [4,12]. Serologic testing was negative for ANA, RF, anti-CCP, ENA, anti-Scl-70, anti-Jo-1, and the myositis panel. ESR and CRP were normal. Echocardiogram showed normal biventricular function with no evidence of pulmonary hypertension. The MDT (including respiratory physicians, radiologists, and pathologists) concluded that the findings were consistent with SR-ILD with fibrosing NSIP pattern, given the radiologic, histologic, and exposure history. CTD-ILD, idiopathic NSIP, and hypersensitivity pneumonitis were excluded. Management involved smoking cessation reinforcement, avoidance of further occupational exposure, and conservative follow-up with annual HRCT and 6–12 monthly PFTs. No pharmacologic therapy was initiated.

Discussion

NSIP is a rare form of SR-ILD, with more common patterns including RB-ILD and DIP [1,3]. This case demonstrates an incidental diagnosis in an asymptomatic individual through mining surveillance, highlighting the utility of occupational health programs [9,11]. The patient's radiological features aligned with NSIP, but upper-lobe paraseptal emphysema suggested overlapping smoking-related pathology. Histology confirmed cellular NSIP, ruling out UIP (absence of fibroblastic foci), HP (no granulomas), and malignancy [5,8,15]. The presence of anthracosis and silica-like crystals suggested cumulative dust exposure but lacked the classical features of pneumoconiosis (e.g., silicotic nodules, pleural plaques) [12,16]. Differential diagnoses included CTD-ILD, idiopathic NSIP, HP, silicosis, and asbestosis. These were excluded by serologic, radiologic, and occupational history. Management strategies differ across these subtypes; thus, accurate classification is essential. Therapeutically, SR-ILD with NSIP lacks clear guidelines. Unlike CTD-NSIP, which often responds to corticosteroids, conservative management may suffice in asymptomatic, functionally preserved patients [5,10,13]. The evolving role of antifibrotic agents in progressive fibrosing ILD (e.g., nintedanib, pirfenidone) may apply in selected cases [20]. This case also raises important questions regarding the interplay between smoking and occupational exposures in ILD development and progression [11,12]. Subclinical disease in high-risk populations may be under-recognized, further emphasizing the importance of structured screening programs [17-21].

Conclusion

This case report describes a rare presentation of SR-ILD with a fibrosing NSIP pattern in an asymptomatic ex-smoker detected through occupational screening. It illustrates the complexities of diagnosis in ILD, particularly in patients with mixed environmental exposures, and reinforces the importance of multidisciplinary evaluation. In asymptomatic individuals with stable physiology, conservative management, exposure mitigation, and longitudinal surveillance represent appropriate strategies. Future studies are needed to better define the natural history and treatment thresholds in SR-ILD with NSIP.

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