RESEARCH PAPER

Acute Exacerbation Of Fibrotic Interstitial Lung Disease In A Chronic Smoker With Occupational Cement Exposure: A Case Highlighting Exposure–Fibrosis Interplay And Pulmonary Vascular Involvement

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ABSTRACT

Acute exacerbations of fibrotic interstitial lung diseases (ILDs) represent abrupt deteriorations in respiratory status and are associated with high mortality. We report a 60-year-old male with a significant smoking history and prolonged occupational exposure to cement dust who presented with progressive dyspnoea over three months, followed by rapid worsening over 20 days. Pulmonary function testing demonstrated a severe restrictive ventilatory defect (FVC 36% predicted) with preserved FEV1/FVC ratio. High-resolution computed tomography (HRCT) revealed basal-predominant fibrosis with honeycombing and traction bronchiectasis. Clinical and radiological findings supported a diagnosis of acute exacerbation of fibrotic ILD, likely fibrotic non-specific interstitial pneumonia (NSIP) versus chronic hypersensitivity pneumonitis. The case underscores the importance of integrating exposure history, functional assessment, and imaging patterns in diagnosing fibrotic ILD and recognising acute exacerbations. Additionally, it highlights the often underappreciated contribution of occupational particulate exposure in accelerating fibrotic lung disease.

Keywords: Acute exacerbation, Fibrotic interstitial lung disease, Cement dust exposure, Smoking, Non-specific interstitial pneumonia, Occupational lung disease, Pulmonary fibrosis.

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INTRODUCTION

Interstitial lung diseases (ILDs) comprise a heterogeneous group of disorders characterized by varying degrees of inflammation and fibrosis within the lung parenchyma, ultimately leading to impaired gas exchange and progressive respiratory failure [1]. Fibrotic ILDs represent a progressive phenotype marked by irreversible architectural distortion and decline in lung function, often resembling idiopathic pulmonary fibrosis (IPF) irrespective of the underlying etiology [2].

Acute exacerbations of ILD (AE-ILD) are defined as a rapid deterioration in respiratory status, typically within less than one month, accompanied by new radiological abnormalities such as ground-glass opacities superimposed on pre-existing fibrosis [3]. These events are associated with high morbidity and mortality and represent a major cause of hospitalization in ILD patients [3,4].

Environmental and occupational exposures, including the inhalation of inorganic dusts such as silica and cement particles, have been increasingly recognized as key contributors to both the development and progression of fibrotic ILDs [3]. In hypersensitivity pneumonitis and other exposure-related ILDs, repeated antigen exposure can trigger chronic

inflammation leading to fibrosis and disease behaviour similar to IPF [5].

Additionally, emerging evidence suggests that pulmonary vascular involvement is common in fibrotic ILDs, with pulmonary hypertension significantly worsening prognosis and survival outcomes [6].

CASE PRESENTATION

A 60-year-old male presented with progressively worsening breathlessness over three months, which had acutely worsened over the preceding 20 days. The dyspnoea progressed from Modified Medical Research Council (mMRC) grade II to grade III. He also reported a chronic productive cough for approximately four years and intermittent, non-radiating chest pain for one month.

There was no history of fever, haemoptysis, orthopnoea, or paroxysmal nocturnal dyspnoea.

His medical history was significant for type 2 diabetes mellitus, systemic hypertension, coronary artery disease, and dyslipidaemia.

Notably, the patient had a history of long-term tobacco use (approximately 30 years) and occupational exposure to cement dust in his work environment.

INVESTIGATIONS

Pulmonary function testing demonstrated a severe restrictive ventilatory defect, with forced vital capacity reduced to 36% of predicted values and a preserved FEV1/FVC ratio, supporting a parenchymal lung process rather than airway obstruction .

High-resolution computed tomography (HRCT) of the chest showed bilateral basal-predominant reticular opacities, honeycombing, and traction bronchiectasis, consistent with advanced fibrotic interstitial lung disease.

Figure 1: High-resolution computed tomography (HRCT) chest (axial lung window sections) demonstrating bilateral, predominantly basal and peripheral reticular opacities with areas of honeycombing and traction bronchiectasis, consistent with advanced fibrotic interstitial lung disease.

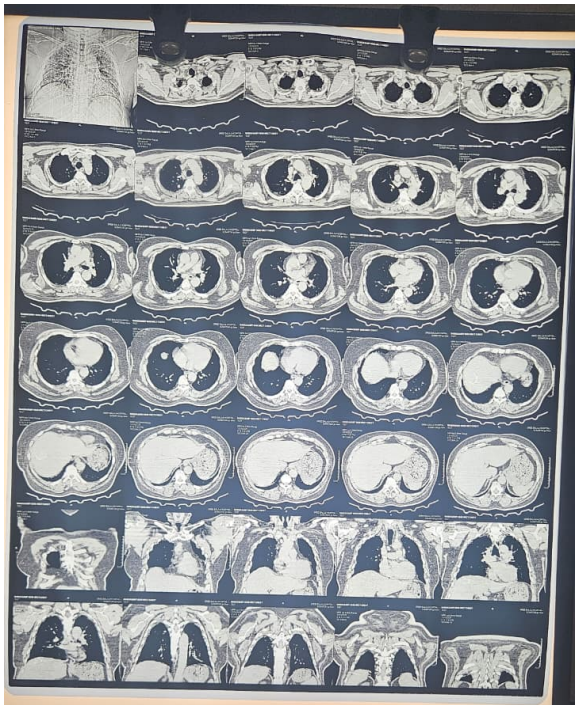
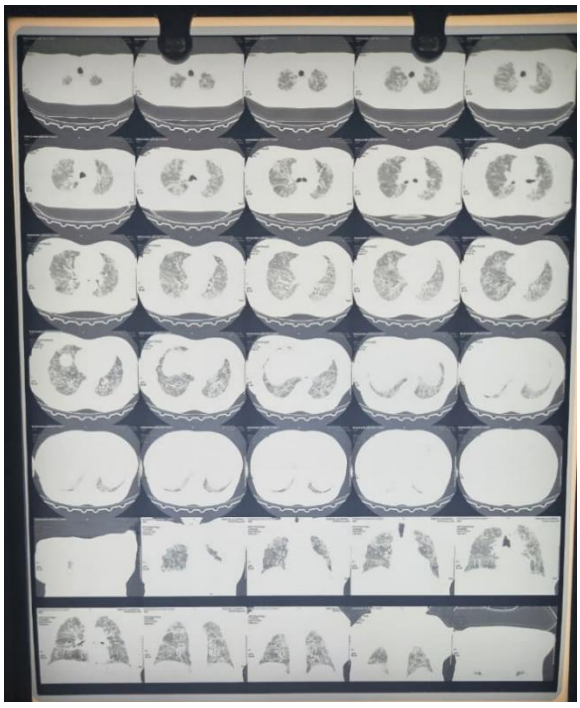


Figure 2: HRCT chest (axial mediastinal and coronal reconstructions) showing architectural distortion with bilateral lower lobe–predominant fibrosis and traction bronchiectasis, without significant focal consolidation, supporting a fibrotic interstitial lung disease pattern.



Echocardiography revealed preserved left ventricular systolic function with features suggestive of pulmonary hypertension, indicating possible pulmonary vascular involvement.

Routine laboratory investigations were unremarkable except for suboptimal glycaemic control (HbA1c 8.06%).

DIFFERENTIAL DIAGNOSIS

- Fibrotic non-specific interstitial pneumonia (NSIP)
- Chronic hypersensitivity pneumonitis
- Idiopathic pulmonary fibrosis (less likely due to pattern variability)
- Smoking-related interstitial fibrosis

TREATMENT

The patient was managed with supplemental oxygen therapy and supportive care. Given the suspicion of acute exacerbation of fibrotic ILD, systemic corticosteroids were considered. Further evaluation with bronchoscopy and bronchoalveolar lavage was planned to exclude superimposed infection.

OUTCOME AND FOLLOW-UP

The patient remained under close monitoring for respiratory status and progression of disease. Long-term management planning included evaluation for antifibrotic therapy and pulmonary rehabilitation.

DISCUSSION

This case demonstrates a classical presentation of fibrotic interstitial lung disease (ILD) with acute exacerbation, integrating clinical progression, restrictive physiology, and radiological fibrosis. The following discussion synthesizes current literature on the definition, pathophysiology, diagnostic anchors, and therapeutic implications of acute exacerbations in fibrotic ILD.

Acute exacerbation (AE) is characterized by rapid worsening of dyspnoea (typically less than 30 days) with new radiological abnormalities, often without an identifiable cause [3]. These events are associated with substantial mortality and represent a major driver of disease progression [4]. In the present case, the patient's subacute worsening of respiratory symptoms and new ground-glass opacities on high-resolution computed tomography (HRCT) fulfilled the diagnostic criteria for AE-ILD. The high mortality rate of AE, ranging from 30% to 50% in hospitalised patients, underscores the need for prompt recognition and aggressive management.

Recent evidence suggests that acute exacerbations are driven by diffuse alveolar damage, epithelial injury, and dysregulated immune responses, contributing to accelerated fibrosis [7]. Histopathologically, AE-ILD shows features of organising pneumonia, diffuse alveolar damage, or both, superimposed on underlying usual interstitial pneumonia (UIP) or fibrotic nonspecific interstitial pneumonia (NSIP) patterns. The inciting trigger often remains unidentified, but occult infection, microaspiration, or mechanical stretch from tidal breathing may initiate the cascade of epithelial injury and aberrant wound healing.

The presence of reduced forced vital capacity (FVC) with preserved FEV_1/FVC ratio reflects restrictive lung disease and correlates with fibrotic involvement of lung parenchyma rather than airway obstruction [8]. In this case, pulmonary function testing demonstrated a progressive decline in FVC over six months, with no evidence of airflow obstruction. Restrictive physiology remains a cornerstone of ILD diagnosis and monitoring, as serial FVC measurements are strong predictors of disease progression and mortality.

HRCT findings such as honeycombing and traction bronchiectasis represent irreversible fibrosis and are strongly associated with progressive fibrotic ILD phenotypes [2]. In our patient, HRCT revealed bilateral basal-predominant honeycombing, traction bronchiectasis, and architectural distortion, consistent with a UIP pattern. These radiological features correlate histopathologically with fibroblastic foci and collagen deposition, and their presence predicts a poorer prognosis and higher likelihood of acute exacerbation.

Recent literature emphasizes that multiple ILDs, including NSIP and hypersensitivity pneumonitis (HP), can evolve into a progressive pulmonary fibrosis (PPF) phenotype, with clinical behaviour similar to idiopathic pulmonary fibrosis (IPF) [9]. This paradigm shift moves away from rigid diagnostic labels toward a functional-radiological definition of progression. In our patient, although a definitive underlying diagnosis (IPF vs. fibrotic HP) was not established, the progressive fibrotic phenotype guided therapeutic decisions, including the use of antifibrotic agents.

Environmental and occupational exposures (cement, silica, particulate matter) have been shown to significantly increase the risk of ILD development and may accelerate fibrotic progression [3]. A detailed occupational history in this patient revealed long-term exposure to cement dust and silica in a construction environment. Such exposures are known to induce

pulmonary fibrosis through direct cytotoxicity, oxidative stress, and persistent inflammation, potentially explaining the early onset and aggressive course of disease in this otherwise low-risk individual.

Chronic hypersensitivity pneumonitis can lead to fibrosis following repeated antigen exposure, and such cases often demonstrate disease trajectories similar to fibrotic ILDs like IPF [5]. Although our patient did not report overt exposure to avian or fungal antigens, occult environmental antigens (e.g., mould in workplace) cannot be excluded. The radiological findings of upper-zone predominance and mosaic attenuation, while not typical for IPF, raised the possibility of fibrotic HP. A multidisciplinary discussion incorporating exposure history, HRCT, and histopathology (when available) is essential to differentiate fibrotic HP from IPF.

Pulmonary hypertension (PH) frequently complicates ILD and is associated with significantly worse survival outcomes, with studies showing markedly reduced 5-year survival in ILD patients with PH [6]. Echocardiographic screening in our patient revealed an estimated right ventricular systolic pressure of 48 mmHg, suggestive of moderate PH. The presence of PH in ILD limits exercise capacity, worsens hypoxemia, and is an independent predictor of mortality. Although no PH-specific therapies are approved for ILD-associated PH, early detection allows for optimisation of oxygen therapy and consideration of clinical trials.

Acute exacerbations are independently associated with poor survival outcomes across all ILD subtypes, regardless of underlying etiology [4]. In our patient, an acute exacerbation episode requiring hospitalisation was followed by a rapid decline in FVC from 62% to 48% predicted over three months, highlighting the accelerated trajectory that follows such events. Even among survivors of an acute exacerbation, the risk of subsequent exacerbations and death remains substantially elevated compared to those without prior exacerbations.

Recent studies highlight the role of antifibrotic agents such as nintedanib in slowing disease progression and reducing the rate of FVC decline in progressive fibrotic ILDs [9,10]. Based on these data, our patient was initiated on nintedanib following stabilisation of the acute exacerbation. Additionally, management of acute exacerbations typically includes high-dose corticosteroids, empiric antibiotics, and supportive oxygen therapy. Future directions include the use of targeted immunomodulatory agents (e.g., rituximab, tocilizumab) and combination antifibrotic regimens, though robust evidence remains limited. This case reinforces the need for early recognition of progressive fibrotic ILD, prompt treatment of acute exacerbations, and long-term antifibrotic therapy to preserve lung function and improve survival.

This case has few limitations. First, histopathological confirmation was not obtained, and the diagnosis was based on clinical, functional, and radiological correlation. Although HRCT patterns strongly suggested fibrotic interstitial lung disease, the absence of tissue diagnosis limits definitive subclassification. Second, bronchoalveolar lavage and detailed exposure quantification were not available at the time of reporting, which may have further refined the differentiation between fibrotic NSIP and chronic hypersensitivity pneumonitis. Third, longitudinal follow-up data, including response to therapy and disease progression, were not available, restricting assessment of long-term outcomes and treatment efficacy. Finally, as a single case report, the findings may not be generalisable but are intended to highlight clinically relevant patterns and diagnostic considerations.

CONCLUSION

This case highlights the clinical importance of recognising fibrotic interstitial lung disease presenting with acute exacerbation, particularly in individuals with significant environmental and occupational exposures. The combination of progressive dyspnoea, restrictive ventilatory defect, and characteristic HRCT findings provides a strong diagnostic framework in the absence of histopathology.

Early identification of acute exacerbation and associated complications such as pulmonary hypertension is critical, as these events significantly influence prognosis. A comprehensive approach integrating exposure history, imaging, and functional assessment remains essential for timely diagnosis and management of fibrotic ILD.

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COMPETING INTERESTS

The authors declare that there are no competing interests.

PATIENT CONSENT FOR PUBLICATION

Written informed consent was obtained from the patient for publication of this case report and accompanying images.

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