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Case Report

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COMPLICATED CHRONIC SILICOSIS: A THERAPEUTIC CHALLENGE – A CASE REPORT

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ABSTRACT

Silicosis is an occupational lung disease caused by inhalation of respirable crystalline silica dust, typically encountered in industrial activities. We report a case of a 40-year-old dental technician with 18 years of silica exposure and occasional hookah use. The disease presented with worsening dyspnea over three months, progressing to mMRC stage III. Chest CT showed bilateral reticulonodular infiltrates, traction bronchiectasis, and calcified "egg-shell" mediastinal lymphadenopathies. Bronchoscopy and transbronchial biopsies were non-specific. The sarcoidosis work-up was negative. Multidisciplinary discussion concluded a diagnosis of silicosis. The patient was started on long-term oral corticosteroids. The disease progressed with worsening fibrosis and declining pulmonary function. Nintedanib was initiated following multidisciplinary review. After two months, the patient showed clinical improvement..

INTRODUCTION

Silicosis is a devastating, incurable, and sometimes fatal disease, and its control relies entirely on prevention. It remains a major occupational health risk, particularly among workers in construction, mining, and stone manufacturing. Silicosis significantly contributes to respiratory morbidity and impaired lung function. Comorbidities such as silico-tuberculosis, lung cancer, and COPD often complicate its course.[1]

Case Presentation

A 40-year-old male, occasional alcohol and hookah user, working as a dental technician with 18 years of silica exposure, presented with chronic dyspnea over one year. In the past 3 months, his symptoms worsened to mMRC stage III with mucoid bronchial syndrome.

Clinical examination revealed a patient in fair general condition (PS 1), without digital clubbing or respiratory distress. Oxygen saturation was 98% in ambient air. Pulmonary auscultation revealed bilateral ronchi.

Chest X-ray showed scattered bilateral nodular and micronodular infiltrates. Chest CT scan revealed bilateral reticulonodular infiltrates (predominantly upper and middle lobes), traction bronchiectasis, and "egg-shell" calcified mediastinal lymphadenopathies.

Bronchoscopy revealed diffuse grade 2 inflammation with mucous secretions. Biopsies showed non-specific chronic fibro-inflammatory changes. BAL was non-diagnostic; mineralogical analysis was unavailable. Bronchial aspirations were sterile with no evidence of Mycobacterium tuberculosis (Ziehl-Neelsen staining and GeneXpert). The sarcoidosis work-up (CBC, ACE, calcium-phosphate balance, ENT and ophthalmology consults) and QuantiFERON (IGRA) test were negative.

Given the occupational history and CT findings, and exclusion of other causes, a diagnosis of fibrosing pneumoconiosis – silicosis – was made.

Functional Assessment

The 6-minute walk test showed slight desaturation (92%) after 400 meters. Arterial blood gases were unremarkable (pH 7.48; PaO_2 129 mmHg; $PaCO_2$ 32 mmHg; HCO_3^- 23 mmol/L). Plethysmography revealed restrictive ventilatory defect (TLC at 44%). Echocardiography showed preserved ejection fraction without pulmonary hypertension.

Management and Evolution

Multidisciplinary team (MDT) concluded on complicated chronic silicosis. The patient received oral corticosteroids (1 mg/kg/day over 8 weeks, tapered), inhaled steroids, long-acting bronchodilators, and supportive care including work cessation.

Initially, clinical improvement was noted (dyspnea from stage III to II mMRC). After 4 months, deterioration occurred with radiological progression and worsened lung function (TLC 43% vs. 51%).

At a second MDT meeting, antifibrotic therapy was initiated with nintedanib due to worsening fibrosis. After two months of therapy, clinical status stabilized. Thoracic imaging and plethysmography are planned after one year.

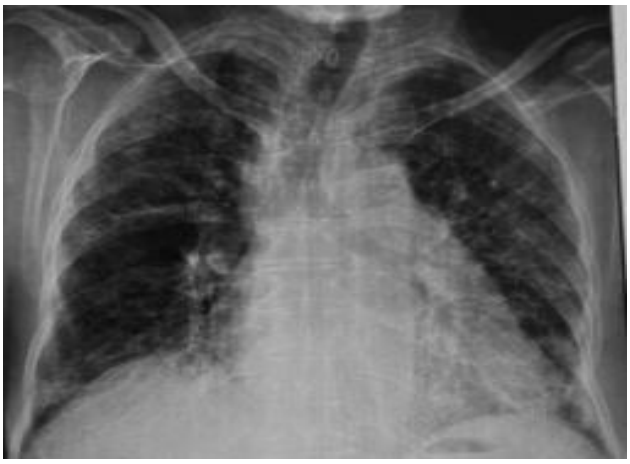

Figure 1: Frontal chest X-ray showing diffuse bilateral nodular and micronodular infiltrates.

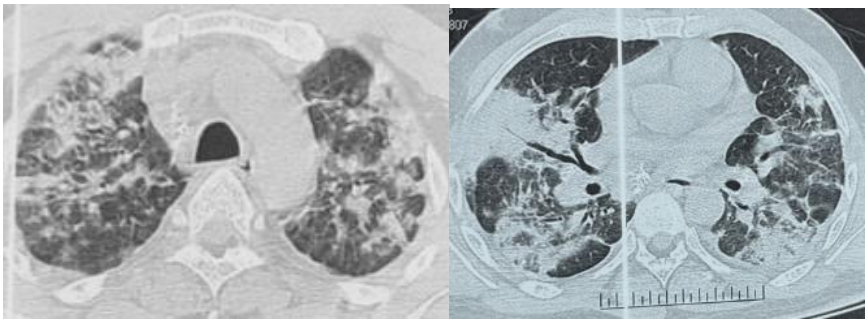

Figure 2: Chest CT scan revealing bilateral reticulonodular infiltrates predominantly in the upper and middle lobes, associated with traction bronchiectasis.

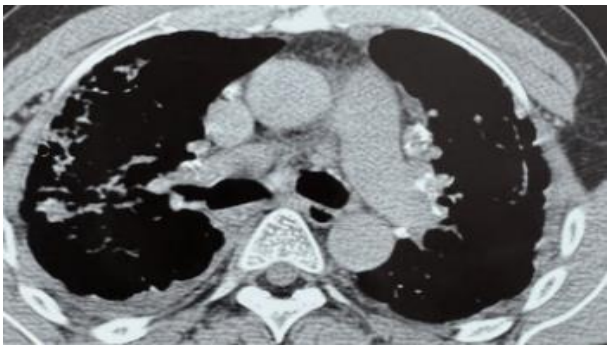

Figure 3: TDM Chest CT (mediastinal window) showing mediastinal lymph nodes with egg-shell calcifications.

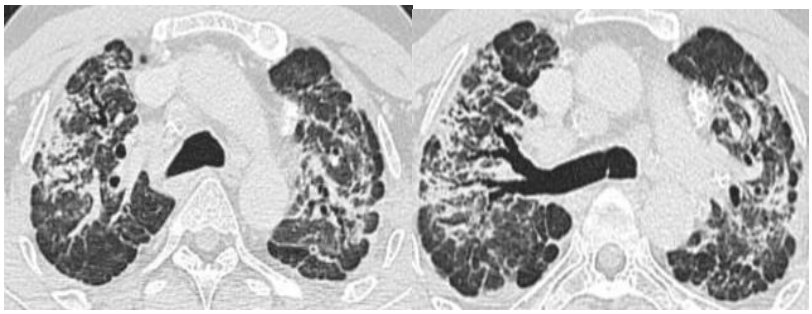

Figure 4: Follow-up chest CT showing progression of fibrotic lesions.

DISCUSSION

Silica is one of the most abundant minerals in Earth's crust. Silicosis is caused by inhalation of respirable crystalline silica particles.[2] High-risk occupations include mining, quarrying, stone cutting, construction, sandblasting, pottery, glass and ceramic work, and dental prosthetics. The severity of disease is influenced by exposure duration, intensity, and particle properties.

This patient had 18 years of unprotected exposure as a dental technician. Silicosis is common in low- and middle-income countries with underreported burden.[3]

Despite research efforts, silicosis pathogenesis remains partially understood, involving parenchymal injury, inflammation, and fibrosis (Figure 1). These stages are interactive rather than sequential.[3,4,5]

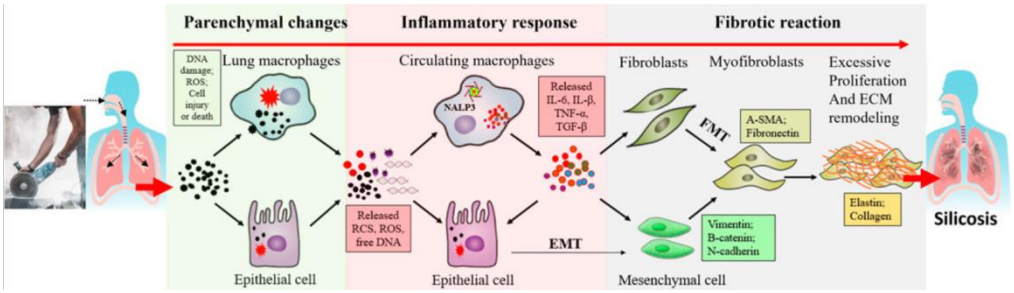

Diagram 1: General diagram describing the different stages of silicosis.

Diagnosis relies on occupational history and imaging. CT is superior to X-ray for early detection and correlates inversely with pulmonary function. Common CT findings include centrilobular nodules, bilateral consolidation, calcified adenopathies, and pleural thickening.[8,9]

Radiologic classification differentiates simple (<1 cm nodules) from complicated silicosis (massive progressive fibrosis >1 cm).[10]
Currently, therapeutic options are limited. Lung transplantation offers partial survival benefit. Pirfenidone and nintedanib, FDA-approved for idiopathic pulmonary fibrosis (IPF), show promise in reducing inflammation, granuloma formation, and fibrosis.[9]

In our case, nintedanib (approved in Morocco) was initiated. It has shown efficacy in silica-induced fibrosis in murine models.[5,11]

Pirfenidone's anti-fibrotic mechanism remains unclear, though it inhibits fibroblast proliferation, myofibroblast differentiation, collagen synthesis, and oxidative stress markers.[5,12]

Both antifibrotics are promising in chronic inflammatory lung disease. Further prospective studies are needed to clarify efficacy in silicosis.[5]

Preventive measures include smoking cessation and vaccination (pneumococcal, influenza, COVID-19). Clinicians should be vigilant for tuberculosis and non-tuberculous mycobacteria in exposed individuals, with annual tuberculin or IGRA screening recommended.[2]

CONCLUSION

Silicosis is a fibrosing pneumoconiosis caused by chronic inhalation of crystalline silica. Often diagnosed late, it is irreversible and therapeutically challenging, with a poor prognosis. Some studies suggest stabilization under antifibrotics, but this remains experimental and warrants continued clinical resear.

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