Section: Respiratory Medicine

Case Report

ERASMUS SYNDROME IN A FLOUR MILL WORKER: A RARE CASE OF SILICOSIS-INDUCED SYSTEMIC SCLEROSIS

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Received *: 04/05/2026*
Received in revised form *: 18/06/2026*
Accepted *: 03/07/2026*

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DOI: 10.70034/ijmedph.2026.3.39

Source of Support: Nil,
Conflict of Interest: None declared

Int J Med Pub Health
2026; 16 (3); 237-241

ABSTRACT

Silicosis is a progressive and irreversible occupational lung disease caused by inhalation of respirable crystalline silica dust. Apart from pulmonary complications such as mycobacterial infection, obstructive airway disease, pulmonary fibrosis and lung cancer, silica exposure has been associated with immune dysregulation and autoimmune diseases. Silica-exposed individuals may develop autoantibodies, including antinuclear antibodies and anti-Scl-70 antibodies, which are also associated with systemic sclerosis (SSc). Erasmus syndrome refers to the development of SSc in the background of silica exposure or silicosis. We report a 47-year-old male flour mill worker with long-term unprotected dust exposure who presented with progressive dyspnea, persistent dry cough, skin darkening and tightening, joint pain, Raynaud phenomenon, sclerodactyly, dysphagia and strong anti-Scl-70 positivity. High-resolution computed tomography (HRCT) thorax showed bilateral pulmonary nodules, interstitial septal thickening and calcified mediastinal lymphadenopathy, supporting chronic silicosis. The clinico-radiological and serological findings were consistent with Erasmus syndrome. This case highlights the importance of detailed occupational history in male patients presenting with systemic sclerosis-like illness.

Keywords: Erasmus syndrome; silicosis; systemic sclerosis; flour mill worker; occupational exposure.

INTRODUCTION

Silicosis is a progressive and irreversible lung disease caused by prolonged occupational exposure to inhaled crystalline silica dust. Respiratory illness related to dust exposure has been recognized since ancient times and the term “silicosis” was introduced by Visconti in 1870 from the Latin word “silex,” meaning flint.[1] The disease is characterized by inflammation and fibrotic nodules, classically involving the upper lobes. Industrial processes such as drilling, sandblasting, stone cutting and grinding increase exposure to respirable crystalline silica. Inhaled silica is phagocytosed by alveolar macrophages, leading to oxidative stress, inflammatory cytokine release and progressive pulmonary fibrosis.[2] Silica exposure is also implicated in immune dysregulation, autoantibody formation and autoimmune disease.[2,3]

Systemic sclerosis (SSc) is a multisystem autoimmune connective tissue disease affecting the skin, lungs, heart, kidneys, gastrointestinal tract and synovium, with a clear female predominance.[4] Therefore, when SSc occurs in men, an occupational or environmental trigger should be carefully considered. Occupational exposures such as silica, vinyl chloride, epoxy resins and solvents have been linked with SSc and silica exposure has been associated with more severe disease phenotypes.[3,5,6] The relationship between silica exposure and SSc was historically described by Bramwell and later by Erasmus, who reported systemic sclerosis among South African gold miners exposed to underground silica dust.[5,6] Subsequent reviews have also supported crystalline silica as an important environmental contributor to autoimmune disease.[7] Flour mill workers using traditional silica-containing

grinding stones are a recognized occupational group at risk for silicosis in India.[8] We report an uncommon case of Erasmus syndrome in a flour mill worker, where prolonged unprotected exposure to dust from traditional stone grinding was associated with chronic silicosis and systemic sclerosis.

CASE REPORT

A 47-year-old male flour mill worker presented to the outpatient department with dyspnea of insidious onset, gradually progressive for one year, associated with persistent dry cough without expectoration. He also complained of progressive darkening and tightening of skin for eight months, joint pain for two months, tingling and numbness of the fingers and toes for three to four months and painful bluish discoloration of the digits suggestive of Raynaud phenomenon. The skin tightening was gradual and involved the fingers, toes and face. He also had dysphagia. There was no history of fever, chest pain or relevant drug intake. There was no past history of diabetes mellitus, hypertension, tuberculosis, interstitial lung disease or asthma. He had a history of tobacco chewing for 10 years and occasional alcohol consumption every 15 days for 15 years. No relevant family history was reported.

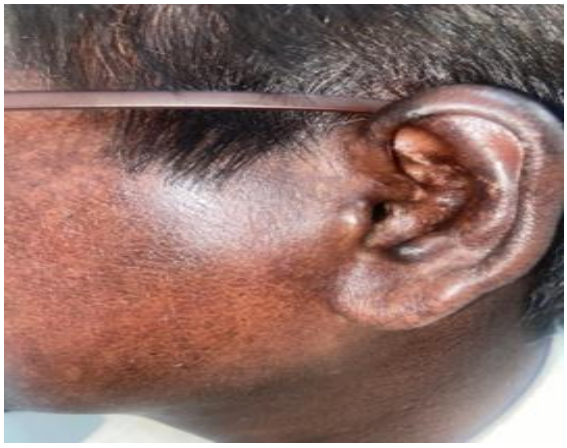

Figure 1: Facial hyperpigmentation with salt-and-pepper pigmentation.

On general examination, the patient was conscious, cooperative and well oriented to time, place and person. His height was 172 cm, weight 73 kg and body mass index was 24.7 kg/m2. He was afebrile, with pulse 80/min, respiratory rate 18/min, blood pressure 118/70 mm Hg and SpO2 97% on room air. Pallor, icterus, cyanosis, lymphadenopathy and pedal edema were absent. Grade II clubbing was present.

Respiratory system examination showed normal chest shape with bilaterally reduced chest movements and reduced chest expansion. Tactile vocal fremitus was reduced bilaterally. Percussion note was resonant throughout, with no area of significant dullness. Auscultation revealed bilateral end-inspiratory fine crepitations.

On cutaneous examination, there was reduced wrinkling over the forehead, salt-and-pepper pigmentation around the lips and diffuse facial hyperpigmentation. Mouth opening was restricted. The skin over the hands was taut and sclerodactyly was present. Clubbing was also noted.

Figure 2: Taut skin over the hands with sclerodactyly.

Relevant clinical, laboratory and pulmonary function findings from the case presentation are summarized in Table 1. HIV was non-reactive and hepatitis B surface antigen was negative. Sputum culture showed no organism isolated and GeneXpert for Mycobacterium tuberculosis was not detected. ANA blot was strongly positive for anti-Scl-70 antibodies, while rheumatoid factor was negative.

Table 1: Relevant clinical, laboratory and pulmonary function findings.

Domain	Findings
General examination	Afebrile; pulse 80/min; respiratory rate 18/min; BP 118/70 mm Hg; SpO2 97% on room air; BMI 24.7 kg/m2; no pallor, icterus, cyanosis, lymphadenopathy or edema; Grade II clubbing present.
Hematology and inflammation	Hb 11.9 g/dL; TLC 6010/mm3; platelet count 2.86 lakh/mm3; PT/INR 16.2/1.14; CRP 28.4.
Biochemistry	RBS 100 mg/dL; urea/creatinine 17/0.5 mg/dL; Na/K/Cl 140/4.2/109 mmol/L; total/direct bilirubin 0.8/0.4 mg/dL; AST/ALT/ALP 52/16/135.
Pulmonary function test	Restrictive ventilatory pattern; FVC 2.06 L (60% predicted), FEV1 1.61 L (58% predicted), FEV1/FVC 0.784; probable restriction on system interpretation.
Echocardiography	LVEF 60%; no diastolic dysfunction; pulmonary artery pressure 30 mm Hg.
Autoimmune workup	ANA blot strongly positive for anti-Scl-70 antibodies; rheumatoid factor negative.

Chest radiograph showed diffusely scattered small nodular opacities in both lungs (Figure 3). HRCT thorax revealed multiple small nodules with centrilobular/random distribution in bilateral lungs.

A few enlarged lymph nodes were noted in the pretracheal and prevascular regions; many showed peripheral or eggshell calcification, a characteristic radiological feature of silicosis. Interstitial septal thickening was seen in both lungs. Dilatation of the distal esophagus proximal to the gastroesophageal junction was also noted.

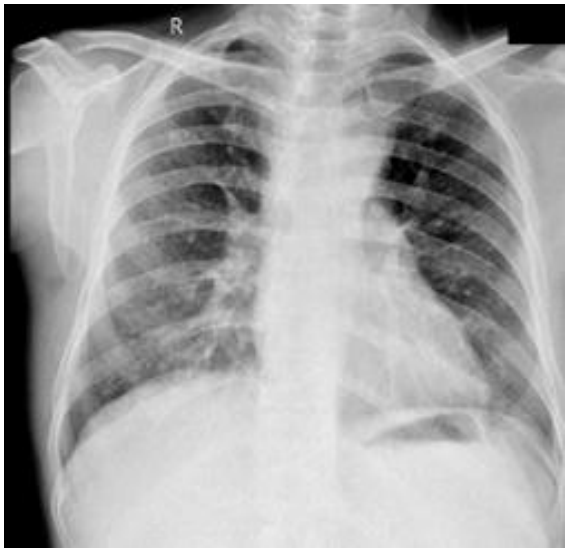

Figure 3: Chest radiograph showing bilateral diffuse small nodular opacities.

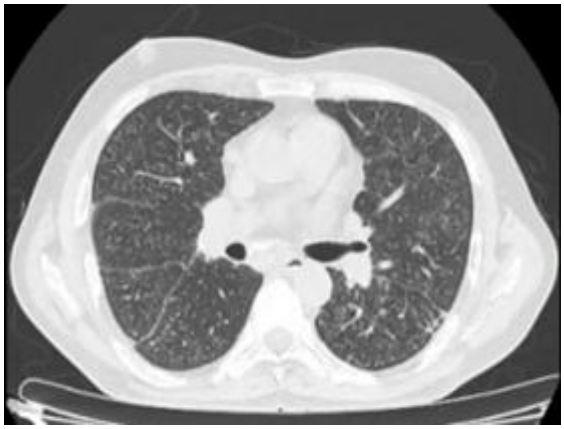

Figure 4: HRCT thorax showing bilateral small pulmonary nodules.

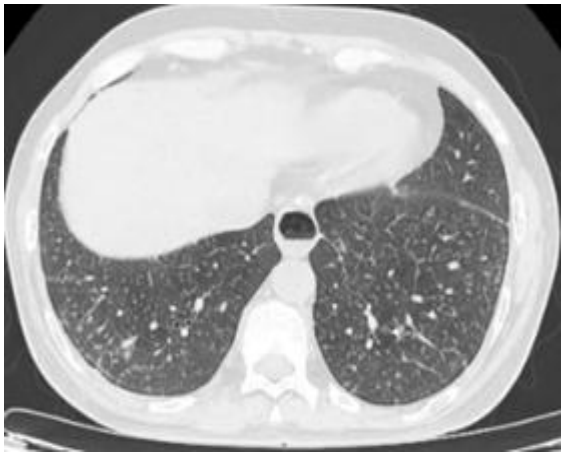

Figure 5: HRCT thorax showing interstitial septal thickening and bilateral nodularity.

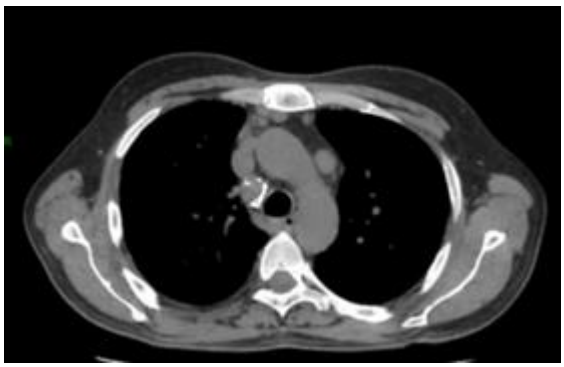

Figure 6: Mediastinal window showing calcified mediastinal lymph nodes.

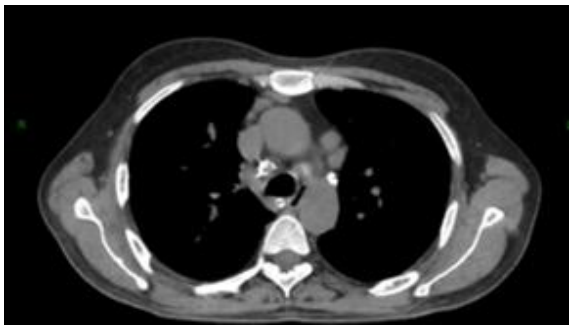

Figure 7: HRCT showing eggshell calcification of mediastinal lymph nodes.

These findings raised suspicion of an occupational lung disease. On further inquiry, the patient revealed a 17-year history of working in a flour mill for approximately 7–8 hours/day without any respiratory protective equipment. The approximate cumulative exposure index was 119–136 hour-years, reported in the case presentation as approximately 140 exposure-hours-years. Since workplace air monitoring was not available, silica exposure was inferred from prolonged traditional flour-mill stone-grinding exposure along with radiological evidence of silicosis.

The clinical-radiological differential diagnoses considered were sarcoidosis, pneumoconiosis, miliary tuberculosis, pulmonary metastases and disseminated fungal infections such as histoplasmosis and cryptococcosis. However, the long history of unprotected flour-mill dust exposure, bilateral nodular lung disease, calcified mediastinal lymphadenopathy, restrictive spirometry, negative infective workup and systemic sclerosis features supported Erasmus syndrome.

The diagnosis of systemic sclerosis was supported by Raynaud phenomenon, sclerodactyly, anti-Scl-70 positivity and esophageal involvement, consistent with ACR/EULAR clinical criteria. Chronic silicosis was diagnosed clinically and radiologically based on prolonged occupational dust exposure, bilateral nodular lung disease and calcified mediastinal lymphadenopathy. The final diagnosis was Erasmus syndrome. The patient was started on corticosteroids and immunosuppressive therapy and strict avoidance of further dust exposure with respiratory protection was advised.

DISCUSSION

Flour milling in India is commonly performed in the informal sector using traditional stone grinders. Stones such as "Agra" and buff stones have been reported to contain approximately 80–81% silica. Regular chiseling, grinding and dressing of these stones release respirable silica dust. In a study among flour mill workers, Athavale et al. reported silicosis in 30.4% of workers, highlighting the occupational risk in this group.[8]

Erasmus syndrome refers to the co-occurrence of systemic sclerosis and occupational silica exposure, with or without established silicosis. Crystalline silica triggers inflammation after inhalation by activating alveolar macrophages and promoting the release of cytokines such as interleukin-1, interleukin-2 and tumor necrosis factor-alpha. These mediators stimulate T-helper cells and fibroblasts, resulting in excess collagen deposition and fibrosis. Chronic exposure may also disrupt immune regulation and promote autoantibody formation, especially anti-topoisomerase I/anti-Scl-70 antibodies, which are associated with systemic sclerosis and interstitial lung disease.[2,3] The resulting immune response causes skin and internal organ fibrosis, along with small-vessel vasculopathy. Rustin et al. studied silica-associated systemic sclerosis and observed clinical, serological and immunological similarities with idiopathic systemic sclerosis, although pulmonary involvement and anti-Scl-70 positivity were more frequent among silica-associated cases.[9] Sanchez-Roman et al. also described multiple autoimmune manifestations in workers exposed to silica.[10] In a large Brazilian systemic sclerosis cohort, Rocha et al. identified Erasmus syndrome in 9 of 947 patients; all affected patients had interstitial lung disease, Raynaud phenomenon and esophageal dysmotility and the condition was associated with male predominance and higher mortality.[11] Similar cases have been reported in other silica-exposed occupations, including marble workers.[12]

The present patient had several features supporting Erasmus syndrome: male sex, prolonged occupational dust exposure in a flour mill, radiological evidence of silicosis, Raynaud phenomenon, sclerodactyly, dysphagia with distal esophageal dilatation, restrictive ventilatory defect and strong anti-Scl-70 positivity. The latency of 17 years between occupational exposure and systemic sclerosis-like manifestations is consistent with previous reports of long exposure-to-disease intervals in silica-associated systemic sclerosis. The absence of fever, negative sputum culture, GeneXpert negativity and lack of supportive features for malignancy or disseminated fungal infection made the major differentials less likely.

Management of Erasmus syndrome is directed toward both prevention of further silicosis progression and organ-based treatment of systemic sclerosis. The most important step is cessation of silica exposure, workplace modification, adequate ventilation, wet methods for dust suppression and use of appropriate respiratory protective equipment. Commonly used medications include corticosteroids or prednisolone for inflammatory manifestations, methotrexate or other immunosuppressive agents for skin and systemic involvement and endothelin receptor antagonists when pulmonary hypertension is present. Calcium channel blockers such as nifedipine or amlodipine are used for Raynaud phenomenon and antiplatelet therapy may be considered for vascular protection. Treatment should be individualized according to disease severity, duration and progression of interstitial lung disease.[12] Periodic monitoring for progression of interstitial lung disease, pulmonary hypertension, tuberculosis and other mycobacterial infections is essential.

CONCLUSION

Erasmus syndrome is a rare but clinically important overlap between occupational lung disease and systemic autoimmune pathology. This case emphasizes that chronic silica exposure should be considered in male patients presenting with systemic sclerosis, particularly when there is a history of long-term dust exposure and radiological features of silicosis. Flour mill workers using silica-containing grinding stones may represent an under-recognized high-risk occupational group. Early diagnosis, exposure cessation, respiratory protection and routine occupational health surveillance are essential to prevent progression and reduce complications such as interstitial lung disease and pulmonary hypertension.

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