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The covert struggle: Tuberculosis amidst silicosis cases

Maria Jose[1], Nagarjun S[2], Abi G[3], Sowmya P[4], Nalini Jayanthi Nagesh[5]

[1,3]Postgraduate Resident, [4]Senior Resident, [2]Assistant Professor, [5]Professor and Head, Department of Respiratory Medicine, SRM Medical College Hospital and Research Centre, Chennai, Tamil Nadu, India

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ABSTRACT

Silicosis is a fibrotic lung disease that occurs due to the inhalation of respirable crystalline silicon dioxide, or silica, typically encountered in occupational settings. In countries where tuberculosis (TB) is prevalent, TB often coexists with silicosis and significantly increases the mortality rate among affected individuals. Educating workers in industries such as quarrying and sandblasting about the risks of occupational lung diseases is essential. This case series involves patients who presented to a tertiary care hospital with a history of occupational silica dust exposure. The study assessed their symptoms, medical history, and diagnostic workup. Patients who reported shortness of breath and cough, along with a history of prolonged exposure to silica, were included. Three of the silicotuberculosis cases had occupational risk factors such as stone cutting, work at construction sites, and borewell drilling. The final case developed silicosis due to chronic exposure to inorganic dust during activities such as tilling and plowing. Detailed histories and clinical examinations were recorded for all patients, who exhibited radiological signs consistent with silicotuberculosis. Although their sputum acid-fast bacillus smears were negative, sputum and bronchoalveolar lavage (BAL) GeneXpert tests confirmed the presence of TB. Anti-TB treatment was initiated for all four patients following microbiological confirmation. Active surveillance of workers exposed to silica, including spirometry, chest X-rays, and respiratory questionnaires, is vital both before and during employment. Workers involved in stone grinding and construction are at particular risk, as they are exposed to fine silica particles that commonly affect the distal airways and alveoli, leading to progressive massive fibrosis. This approach can substantially reduce the morbidity and mortality associated with silicotuberculosis. The use of respiratory masks and ensuring proper ventilation in the workplace are key preventive measures against the onset of silicosis.

Key words: Silicosis; Tuberculosis; Occupational lung disease; Progressive massive fibrosis; Silicotuberculosis; Pneumoconiosis

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INTRODUCTION

Silicosis is a form of pneumoconiosis caused by inhaling and accumulating free silica particles in the lungs, resulting in granulomatous inflammation and pulmonary fibrosis. This disease is primarily associated with occupational exposure and can arise in various industrial settings, such as coal and gold mining, quarrying, tunnel construction for railways and highways, foundries, as well as in the production of cement, glass, ceramics, porcelain, and marble, along with sand extraction. It is estimated that around 227 million workers are at risk, many of whom are informal and often migrant laborers.[1,2] Within this group, approximately 40.5 million are artisanal small-scale miners active in over 80 countries worldwide. While there are rare instances of non-occupational exposure to silica, these cases are infrequent. The widespread nature of silicosis highlights the urgent need for enhanced safety measures and health protections in high-risk industries.[3] In 1995, the World Health Organization and the International Labour

Address for Correspondence:
Dr. Nagarjun S, Assistant Professor, Department of Respiratory Medicine, SRM Medical College Hospital and Research Centre, Chennai, Tamil Nadu, India. **Mobile:** +91-9884722003. **E-mail:** nagarjus@srmist.edu.in

Organization launched a worldwide initiative to enhance awareness and prevent silicosis, aiming for the disease's eradication by 2030. Despite efforts by several low- and middle-income countries to implement national strategies to address silicosis, it remains a serious occupational health concern, making the goal of elimination by 2030 seem unlikely.[4] Among the various health complications linked to silicosis, tuberculosis (TB) stands out as the most significant, especially in low- and middle-income regions.[5] Silicosis is recognized as the second-leading risk factor for developing TB, just after HIV infection.[6] Here, we present a case series of 4 silicotuberculosis patients.

CASE PRESENTATION

Case 1

55-year-old male, a known case of chronic kidney disease stage 4 on hemodialysis, twice weekly, who came for his regular dialysis was referred to respiratory medicine outpatient department with complaints of cough with mucoid expectoration for 2 weeks, breathlessness grade 2mMRC and low-grade fever for 1 week. The patient was not aware of any close contact with anyone diagnosed with TB, nor had he ever taken anti-tuberculous medications, based on his medical history. He reported no symptoms of hemoptysis, rashes, or lymphadenopathy. In addition, he stated that he had no prior history of allergies or any other significant medical conditions. He worked as a stone grinding worker for more than 12 years and quit 10 years back. He is an ex-smoker, quit 2 years back, with exposure of 30 years. Smoking index: 240.

On physical examination, the patient presented with, a pulse rate of 112 beats/min, a respiratory rate of 24 breaths/min, a blood pressure reading of 140/88 mmHg, and a temperature of 98.7°F with unremarkable neurological, cardiovascular, and abdominal findings. Computed tomography (CT) Chest shows bilateral calcified lymphadenopathy and progressive massive fibrosis (PMF). Sputum samples were sent for analysis. Sputum acid-fast bacillus (AFB) was negative. Sputum GeneXpert showed *Mycobacterium tuberculosis* (MTB) detected with rifampicin sensitivity. The patient was started on antitubercular drugs (Figures 1 and 2).

Case 2

48-year-old male, sandblaster for 20 years, presented with complaints of cough with expectoration; increased in 10 days, dyspnea for 1 year, increased for 1 week, insidious in onset, gradually progressive from MMRC 1 -1V, associated with wheeze, history of seasonal variation present (increases in winter seasons), chest pain for 7 days more on left side, pricking type of pain, associated with chest palpitations, not associated with sweating, relieved by taking medications, fever for 1 day; low grade, intermittent not associated with chills and rigors, relieved by medication.

Patient had a history of pulmonary TB-MDR-TB 5 years ago, took ATT with injectables for 9 months and was declared cured, systemic hypertension for 5 years on regular treatment and post pulmonary TB- obstructive airway disease for 1 year on treatment. He had no additional exposure to dust or other harmful substances. During follow-up, his condition remained stable. The patient had never smoked and did not consume alcohol.

On examination, his vitals were normal, with respiratory system auscultation showing blateral air entry decreased, left infra clavicular and mammary area squeaks, and other areas normal vesicular breath sounds. Other systems were within normal limits high-resolution CT chest showed multiple thick-walled cavitatory changes with

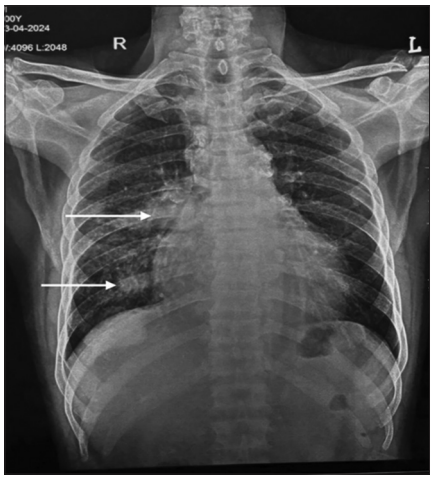

Figure 1: Chest X-ray showing right mid zone and lower zone nodular opacity noted

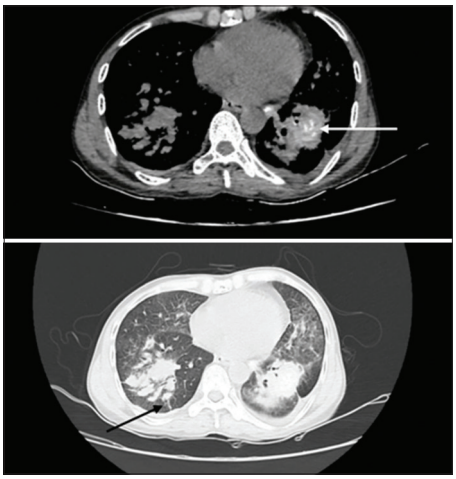

Figure 2: Contrast computed tomography chest showing bilateral calcified lymphadenopathy with progressive massive fibrosis

areas of consolidation noted in bilateral upper lobes with associated bronchiectatic changes and partial volume loss, predominantly on the right upper lobe with the focal tracheal shift to right, right mild pleural effusion noted, left trace pleural effusion noted (Figure 3).

Fiberoptic bronchoscopy with bronchoalveolar lavage (BAL) tested positive for Mycobacterium tuberculosis (MTB) using GeneXpert, indicating rifampicin resistance. Mycobacterial Growth Indicator Tube (MGIT) culture also confirmed the presence of MTB with rifampicin resistance and high-level isoniazid resistance. The patient was started on a multidrug-resistant tuberculosis (MDR-TB) regimen consisting of bedaquiline (Bdq), levofloxacin (Lfx), linezolid (Lzd), clofazimine (Cfz), and cycloserine (Cs). Patient was non-compliant and died within 2 weeks after the initiation of treatment.

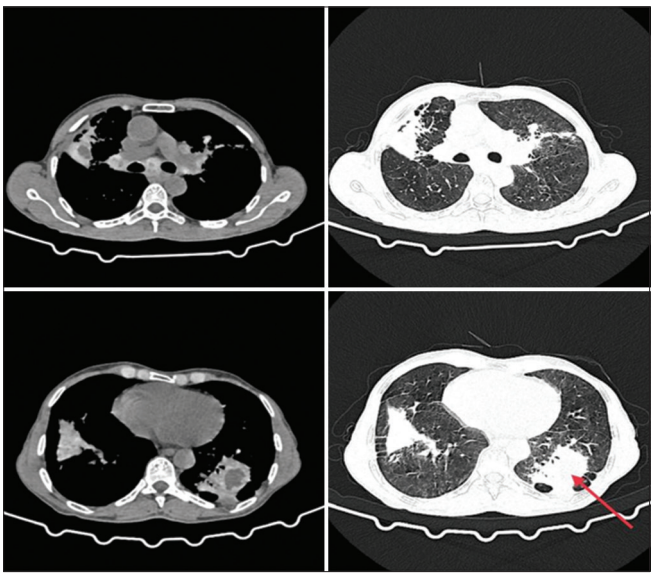

Figure 3: Computed tomography chest showing multiple thick walled cavitatory changes with areas of consolidation in bilateral upper lobes with bronchial wall thickening

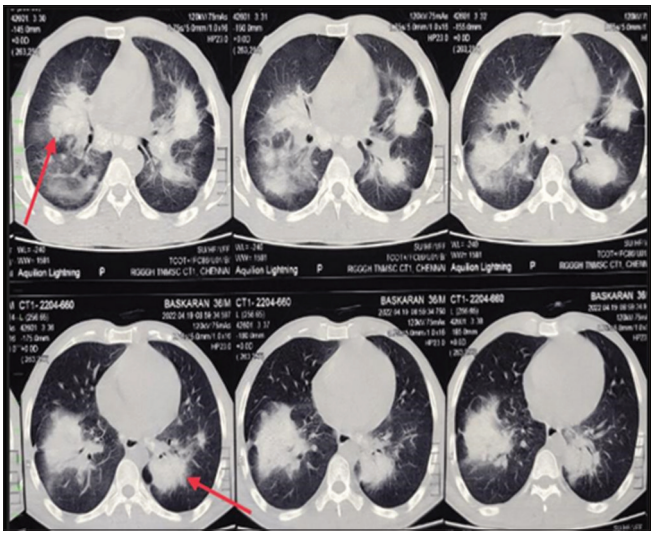

Figure 4: Computed tomography chest showing bilateral conglomerate mass with progressive massive fibrosis

Case 3

A 57-year-old male, who is a case of cholangiocarcinoma on regular follow-up came with incidental CT findings. He worked in a Quarry for 15 years, 10 years back. The patient also had complaints of loss of weight for 1 month and loss of appetite for 2 weeks. He is an ex smoker, stopped 1 year back with 41 years of exposure and a smoking index of 205.

Upon this hospital admission, the patient appeared to be in good health during the physical examination, with no detectable peripheral lymph nodes. Blood tests revealed a hemoglobin level of 133 g/L, a white cell count of 4.64 × 10^9/L, and a platelet count of 164 × 10^9/L. Coagulation, renal, and liver function tests were all within normal ranges, and tumor marker screenings returned negative results.

His CT chest showed bilateral conglomerate mass with PMF. Sputum AFB was Negative. Sputum Gene Xpert turned out to be MTB detected, Rifampicin sensitive. He was initiated on antitubercular drugs (Figure 4).

Case 4

A 63-year-old male presented with chest pain, grade 2 mMRC shortness of breath, and a 20-day history of cough with mucopurulent expectoration. He is a farmer for 40 years. On further questioning, he disclosed a history of exposure to burnt rice husk over the past 11 years in a nearby rice mill.[7] He used to work there for an additional living.

On clinical examination, he was afebrile and tachypneic with a respiratory rate of 30 per min. His oxygen saturation was 88% on room air at rest. There was a presence of pallor. A respiratory examination showed bilateral decreased breath sound and fine crepitations. Other systemic examinations were normal. Routine lab tests, including a complete blood count, liver and kidney function panels, and serum electrolytes, returned normal results. The fasting blood glucose was measured at 210 mg/dL, and the rheumatoid factor test came back negative.

His chest CT scan revealed bilateral multiple nodular opacity coalescing to form PMF with surrounding emphysematous changes, giving the appearance of angle wing sign (Figure 5).

Sputum testing for AFB and Gene Xpert was negative. Bronchoscopy and transbronchial lymph node biopsy were done, BAL turned out to be Gene Xpert- MTB detected, Rifampicin sensitive (Figure 6).

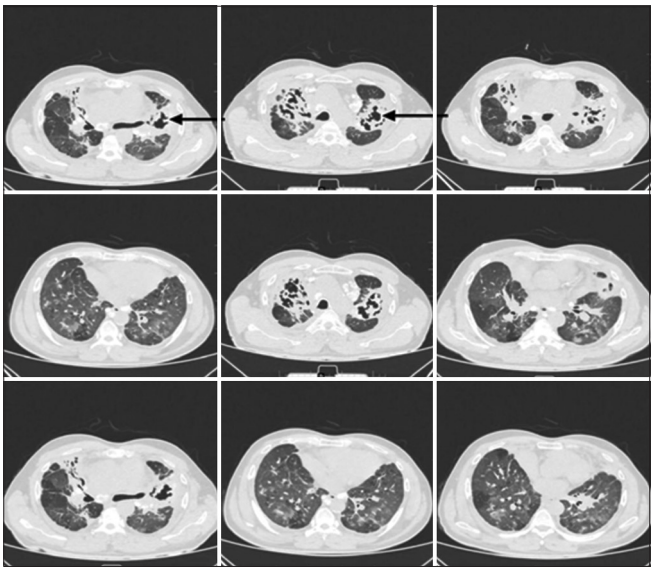

Figure 5: Contrast computed tomography chest showing bilateral multiple nodular opacity coalesce to form progressive massive fibrosis with surrounding emphysematous changes giving appearance of angel wing appearance

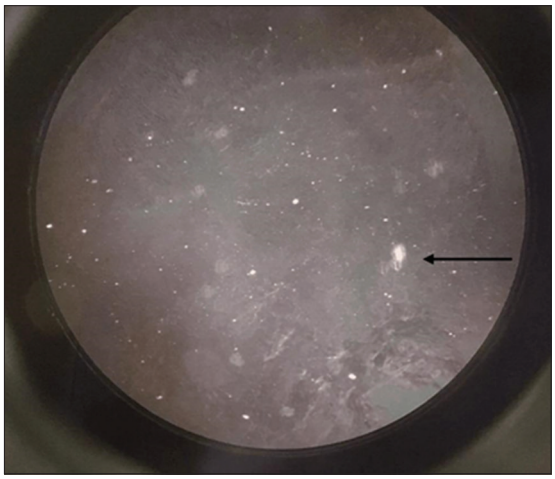

Figure 6: Under polarized microscope, birefringent polyhedral silica particles seen

DISCUSSION

Air pollution refers to the buildup of unwanted organic, inorganic, and biological substances, along with or without toxic gases like ozone (O_3), sulfur dioxide (SO_2), and nitrogen dioxide (NO_2), in the ambient air at elevated concentrations, which can potentially diminish the quality of life for populations.[8,9] It is estimated that 91% of the global population resides in cities and towns where air quality falls short of the minimum guidelines set by the World Health Organization (WHO), resulting in over 4.2 million premature deaths annually worldwide.[8,10] Silicosis is a permanent, advancing, and untreatable lung disease resulting from the inhalation of silica dust, and it has been recognized for many years as a significant occupational risk for workers in various industries.[11] By the end of 2025, it is estimated that India will have around 52 million workers who will continue to be exposed to silica dust in their occupations.[12] The recent rise in silicosis cases among construction and mining workers, especially among young individuals engaged in sandblasting, bolting, cutting, shaping, and installing quartz conglomerate (engineered stone) kitchen countertops, is a significant concern.[12] Silica exists in seven polymorphic forms based on the elemental arrangement of atoms, with quartz (α-quartz) being the most prevalent. Exposure to respirable quartz has been linked to the development of various interstitial lung diseases, including silicosis and pulmonary sarcoidosis.[13,14] Air-burning rice husk generates rice husk ash (RHA), composed of 85–98% silica. This process contributes to environmental pollution and poses health dangers. Although RHA is abundant in silica, there have been no attempts to extract silica from it.[7] Silicosis arises after the inhalation and deposition of respirable crystalline silica (RCS) in the smaller airways and lung parenchyma. The severity and progression of the disease are influenced by the amount of dust inhaled and the duration of exposure. Although silicosis has been recognized for many decades, the precise mechanisms behind its development remain unclear. It is believed that alveolar macrophages engulf the inhaled crystalline silica (quartz) particles within the lungs, leading to lysosomal damage and subsequent activation of the NLR family pyrin domain containing 3 inflammasome, a process also observed in severe asthma. This activation triggers a series of reactions that include the release of inflammatory and pro-inflammatory cytokines, such as interleukin (IL)-1β and tumor necrosis factor-alpha (TNF-α), as well as the production of reactive oxygen species (ROS) and reactive nitrogen species intermediates. These processes contribute to the cycle of apoptotic cell death and the development of lung fibrosis.[15-17]

Patients with simple silicosis typically do not exhibit symptoms and generally maintain normal pulmonary function, with only a minor reduction in vital lung capacity. However, they may experience a dry cough due to tracheal irritation. Histological analysis of lung biopsies, along with chest X-rays and HRCT scans, often reveals the presence of silica nodules, up to 10 mm in diameter, in the upper lobes. These nodules are commonly associated with enlarged hilar peribronchial lymph nodes, which are often calcified.[8,13]

Complicated silicosis, also known as PMF, is characterized by the merging of nodules that extend into the lower lobes, each larger than 1 cm in diameter. The color of the nodules varies based on the chemical composition of the dust exposure: they can be grey to blue from pure silica, black in coal miners, or red in haematite miners. These nodules are frequently accompanied by dust-laden macrophages and

increased collagen deposition at their centers. Ongoing dust exposure can lead to the recruitment of additional immune cells and the progression of the disease toward idiopathic pulmonary fibrosis. PMF mainly affects the upper lobes, resulting in a progressive reduction in lung volume and upward traction of mediastinal structures.[13,18]

Accelerated silicosis is distinguished from traditional silicosis by its rapid onset, occurring within <10 years due to high exposure to RCS particles. The radiological presentation can vary from simple to complicated silicosis, but lung function typically deteriorates significantly over a relatively short period. The resurgence of accelerated silicosis is often linked to an inadequate understanding of risk or non-compliance with workplace safety regulations. For instance, Hoy and Chambers[19] documented six Australian workers who developed radiological signs of PMF after 7 years of working with artificial stone. In Australia, 6.6% of workers are exposed to crystalline silica dust. Traditional diagnostic methods, such as plain chest X-rays, may have limited sensitivity for early detection, making it challenging to prevent disease progression before irreversible damage occurs. Utilizing established biomarkers for early detection in at-risk populations could be highly beneficial. Potential biomarkers include elevated levels of TNF-α, IL-8, ROS, glutathione, glutathione peroxidase, glutathione S-transferase, club cell secretory protein (CC16), 8-isoprostane, and platelet-derived growth factor.[20,21] A cross-sectional study of workers with silicosis found increased plasma levels of CXC motif chemokine ligand 16, a chemokine released by respiratory epithelial cells, as an early disease biomarker.[22] Additionally, patients with silicosis are more prone to infections with MTB, which can lead to the progression from latent to active TB and fungal infections that may become life-threatening.[23] Silica exposure reduces the activation of dendritic cells, leading to a general impairment in the body's inflammatory response.[24] This weakens antibacterial defenses and heightens vulnerability to bacterial infections, particularly to MTB and other mycobacterial species. The suppression of Toll-like receptor 2 due to silica exposure may contribute to an increased risk of TB. Several pathways may facilitate disease development in the presence of both silica and MTB. Additionally, genetic variations in factors such as TNF-α, the natural resistance-associated macrophage protein 1, and inducible nitric oxide synthase within macrophages can affect how the body responds to both silica exposure and TB. Silica particles enhance the intracellular replication of MTB and promote its release from macrophages. The exact pathophysiological mechanisms are yet to be completely identified.[24]

Newly inhaled silica particles can generate reactive free radicals on their surfaces. These radicals can lead to DNA damage, mutations, and ultimately result in cell death.[23] As silicosis advances, it can be complicated by severe mycobacterial or fungal infections, similar to those observed in our patients. TB is a common infection that arises when macrophages, overwhelmed by silica dust, are unable to eliminate the pathogen. The link between silicosis and TB is well-documented, with pulmonary TB potentially serving as either a differential diagnosis or a complication of silicosis. In fact, about one-quarter of patients with silicosis, including those with coal workers' pneumoconiosis who often exhibit peripheral lymph node calcification known as "eggshell calcification," also have silico-TB.[25,26] Chest X-rays and sputum smear tests play a critical role in diagnosing pulmonary TB, particularly in patients with silicosis.[27] Chest X-rays frequently detect TB signs in silicosis patients before clinical symptoms emerge, making regular radiographic screening potentially more effective than sputum tests for early TB detection.[27,28] Comparing sequential X-ray images is crucial, with careful attention to asymmetric nodules, consolidation, effusions, cavities, and any rapid changes.[18] While cavitation is a strong indicator of possible silico-TB, it can also result from ischemic alterations within fibrotic masses due to silicosis.[29] If sputum smear microscopy and chest X-rays do not provide clear results, a chest CT scan can offer additional insights. However, identifying silico-TB, particularly in the context of PMF, can be challenging.[26] In such cases, bronchoscopy with bronchoalveolar lavage and, where feasible, a transbronchial biopsy should be considered.[30] Silico-TB is often associated with significant lung function impairment, especially airway obstruction.[31] Spirometry, performed at diagnosis and during follow-up, can assist in assessing the extent of lung function decline and the effectiveness of TB treatment. Treating TB in patients with silicosis presents significant challenges, likely due to impaired macrophage function caused by exposure to free silica and/or reduced drug penetration into fibrotic nodules.[32-36] Standard anti-TB therapy with directly observed treatment is recommended, but extending the treatment duration to at least 8 months may be necessary to reduce the risk of relapse. In a well-controlled clinical trial conducted in Hong Kong, patients with silico-TB were randomly assigned to receive either 6 months or 8 months of thrice-weekly therapy with isoniazid, rifampicin, pyrazinamide, and streptomycin.[35] Patients with a history of previous anti-TB treatment also received ethambutol for the first 3 months. The study found that 80% of patients achieved a negative sputum culture after 2 months. Relapse rates during a 3-year follow-up were 22% in the 6-month treatment group compared to 7% in the 8-month group, and 22% of patients experienced significant adverse drug reactions.[35] In a subsequent study conducted in South Africa, gold miners diagnosed with pulmonary TB were

treated with isoniazid, rifampicin, pyrazinamide, and streptomycin on weekdays for 5 months. Chest X-rays were examined for signs of silicosis at the time of diagnosis, and all participants were monitored for at least 5 years after completing treatment.[35] The study found that the risk of TB relapse was 1.55 times higher in patients with silicosis compared to those without the condition. Additional research is necessary to determine the optimal treatment duration and assess the tolerability of anti-TB therapy in patients with silicosis.[24]

CONCLUSIONS

TB continues to be a significant issue for those suffering from silicosis. Enhanced surveillance and reporting of both silicosis and TB among former miners are urgently needed to gauge the effectiveness of current control strategies. It is crucial to enforce strict dust control measures and bolster them with comprehensive TB prevention initiatives for active miners. However, implementing these measures can be particularly difficult in certain environments, such as deep-level mining operations in low- and middle-income countries, where conditions are often unstable. Surprisingly, even in wealthy nations like Australia, these measures are sometimes inadequate. Furthermore, some countries have mining regulations that fail to specify exposure limits for crystalline silica, an issue that requires immediate attention. The challenge of managing TB among miners is complex. Workers who are barred from mining due to TB may return to poor communities, where completing treatment is challenging, and the risk of TB transmission is high. On the other hand, permitting these workers to continue working in the mines could exacerbate the spread of the disease. Expanding chemoprophylaxis on a global scale is vital to reducing the incidence of both pulmonary and extrapulmonary TB among miners. Awareness among physicians is crucial, as silicosis can affect those who are exposed to burning rice husk.

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Authors' Contributions:
MJ- Definition of intellectual content, prepared first draft of manuscript, data collection, manuscript preparation and submission of article; **NS-** Concept, design, data collection clinical protocol; **AG-** Literature survey, manuscript preparation and editing; **SP-** Design of study and manuscript revision; **NNJ-** Review manuscript

Work attributed to:
SRM Medical College Hospital and Research Institute, Chennai, Tamil Nadu, India

Orcid ID:
Maria Jose- https://orcid.org/0009-0004-2285-1575
Nagarjun S- https://orcid.org/0009-0008-4462-5359
Abi G- https://orcid.org/0009-0000-4136-3009
Sowmya P- https://orcid.org/0009-0006-1278-9960
Nalini Jayanthi Nagesh- https://orcid.org/0000-0002-4672-0578

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