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Review Article

Silicotuberculosis – An Updated Overview

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Abstract

Purpose of Review: This review focusses on the burden of the problem of silicotuberculosis, clinical scenario and challenges encountered in its management.

Recent Findings: In accordance to the Indian Council of Medical Research (ICMR), approximately 3.0 million workers are at high risk of silica exposure; of these, 1.7 million work in mining or quarrying activities, 0.6 million in non-metallic product manufacturing (such as refractory products, structural clay, glass, and mica), and 0.7 million in the metals industry. In addition, over 5.3 million construction workers are in danger of silica exposure. Because of differences in silica concentrations and duration of exposure in the workplace, the reported prevalence of silicosis and silico-TB in India varies greatly.

Summary: Silicotuberculosis, a condition where tuberculosis develops as a complication of silicosis, underscores a critical public health concern, particularly prevalent in low- and middle-income countries where prolonged exposure to crystalline silica particles is widespread. This exposure heightens the vulnerability of workers to tuberculosis infection, exacerbating the already substantial burden of both diseases. Management of silicotuberculosis involves a multifaceted approach. These strategies include controlling silica dust exposure, ensuring continuous TB treatment, implementing occupational health management by professionals and policy making by the government.

Keywords: Silicotuberculosis, Public health concern, Silica particles.

Introduction

Silicotuberculosis, a condition where tuberculosis develops as a complication of silicosis, and a major public health problem, especially prevalent in developing countries, where prolonged exposure to crystalline silica particles is common.[1] This exposure heightens the vulnerability of workers to tuberculosis infection, exacerbating the already substantial burden of both diseases.[2] The chronic exposure of silica dust by the respiratory route not only enhances the contracting tuberculosis but also exacerbates existing cases of pulmonary tuberculosis in individuals suffering from silicosis.[3] This dual impact amplifies the complexity of managing these conditions, posing significant challenges for healthcare systems.[4]

History

Achille Visconti (1836–1911), in Milan, coined the term silicosis in 1870, but concerns about the harmful effects of dust on workers' lungs date back much earlier.[5,6] In the mid-16th century, Agricola observed respiratory issues among miners due to the inhalation of mining dust. [7,8] Later, in 1713, Bernardino Ramazzini noted similar complications—such as asthma and the presence of sand-like material—in stone cutters. By 1720, British workers in the ceramics industry were found to suffer severe lung damage from inhaling milled calcined flint, often developing symptoms in less than a decade of exposure.[9] With the rise of industrialization, dust generation increased significantly, leading to a higher incidence of respiratory diseases. Technological advancements such as the innovation of the pneumatic hammer drill in 1897 and sandblasting in 1904 significantly contributed to the rising incidence of silicosis.[10]

By the 1930s, silicosis had become relatively uncommon in high-income countries; however, a pivotal milestone

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occurred in 1930 when its pathology was formally described at the 'International Labour Office Conference' at Johannesburg. Previously referred to as 'miner's phthisis,' silicosis was clinically and radiologically differentiated from tuberculosis.[11] The United States Department of Labor launched the film *Stop Silicosis* to highlight the increasing prevalence of the disease and to promote public awareness.[12] Decades later, in 1995, the International Labour Organization (ILO) and the World Health Organization (WHO) launched together a global initiative aimed at the elimination of silicosis, based on the premise that sufficient knowledge and resources were already available to achieve this goal.[13]

Epidemiology

As silicosis predominantly afflicts workers in sectors such as mining and construction, the burden of silicotuberculosis looms large, particularly in middle-income countries where factors like population growth and aging trends exacerbate the risk. Despite a global decline in the prevalence of silicosis, its burden remains substantial, with millions of 'disability-adjusted life years' (DALY) attributed to the disease and its complications.[14] Men are at higher risk of developing silicosis as compared to women. Additionally, miners living with HIV exhibit a high risk of silicosis.[15] Yi *et al.*[16] reported a decrease in Global Age-Standardized Rates (ASRs) for silicosis from 1990 to 2019, revealing notable epidemiological trends. The most affected age group was individuals aged 45-59, with higher mortality observed in those aged 80 and above. A study done by Ehrlich *et al.*[17] it was observed that over the period of 7 years, one in four men with silicosis developed tuberculosis by the age of 60. According to ICMR Annual Report 2022-2023[18], the mean age of mine/quarry workers with TB working in the sandstone belt of Jodhpur, Rajasthan in Western India was 36.44 ± 11.23 years and an observed male: female ratio of 332:3.

Silicosis workers are 2.8 times and 3.7 times more likely to develop pulmonary and extra-pulmonary tuberculosis, respectively, as compared to those without silicosis. A meta-analysis by Jamshidi *et al.*[19] showed a Mantel-Haenszel pooled risk ratio of 1.35, revealing an increased risk of tuberculosis patients with exposer to silica.

Tyagi *et al.*[20] directed a study in the Jodhpur district involving 221 mine workers and reported a mean age of the workers of 40.7 years within a range of 15 years to 84 years. About 28.6% workers were under the age of 30 years, while 24% were over 50 years old. A high prevalence of substance use was observed among mine workers, about 80% workers had tobacco addiction and 52% workers had alcohol addiction. These addictions are associated with reduced immunity and poor nutritional status, thereby increasing the risk of developing silicosis and tuberculosis. Furthermore, 26% of workers exhibited a respiratory rate exceeding 20 breaths per minute, suggesting compromised respiratory function or underlying conditions such as anemia, malnutrition. About 57% workers had a body mass index (BMI) below 20, indicating a high prevalence of malnutrition among the mine workers. In a study from Rajasthan, Rajavel *et al.*[21] analysed 174 mine workers with a mean age of 39.13 ± 11.09 years among them. Notably, about 75.3% workers had been employed in mining for over ten years, and out of them, about 128 (73.6%) were males and 46 (26.4%) were females. Around 30.0% had a history of tuberculosis. The same study observed a prevalence of silicosis of 37.3% and that of silicotuberculosis of 7.4% among the mine workers. Additionally, tuberculosis alone was present in 10% of the cohort, and 4.3% were diagnosed with other respiratory conditions, including emphysema and pleural effusion. Rupani *et al.*[22] also report the median age of 42 years within a range of 30 years to 55 years among 2748 TB patients with silicosis in Khambhat block in western India and about 66% TB patients with silicosis were males and 34% were females. Few states in India are known for significant mining activities that include Rajasthan, Chhattisgarh, Jharkhand and Odisha, apart from West Bengal. As per the previous report of ICMR, there were approximately 3 million workers in high-risk occupational settings with a high risk of silica exposure. Of these, approximately 1.7 million were employed in mining and/or quarrying and 0.6 million in 'non-metallic' products manufacturing, such as glass, mica, structural clay, and refractory materials, etc, and 0.7 million in the 'metallic' industry. Furthermore, over 5.3 million construction workers are potentially exposed, owing to variable silica concentrations and diverse exposure durations across job sites. Hence, there is considerable variability in the prevalence of silicosis and silico-tuberculosis across different areas and occupational groups in India.[23]

Pathophysiology

Silicosis is a progressive and incurable occupational lung disease caused by exposure to crystalline silica dust, which triggers inflammatory and fibrotic processes in the lungs. Jamshidi *et al.*[19] describe that increased silica exposure disrupts the function of alveolar macrophages, inducing programmed cell death and increased susceptibility to mycobacterium infections. Upon deposition in the alveolar space, inhaled silica particles interact with macrophages and are internalized through phagocytosis into a membrane-bound phagosome. When inhaled, silica disrupts these macrophages' normal function, leading to the secretion of pro-inflammatory substances like interleukin-1 (IL-1) that activate lymphocytes, which further stimulate macrophages, creating a cycle that perpetuates inflammation and fibrosis in the lungs. 'Macrophage receptor with collagenous structure' (MARCO), a class A scavenger protein, plays a crucial role in recognizing and internalizing unopsonized environmental particles like silica. In the absence of this receptor, particle uptake and subsequent cell death do not occur.[24] Downregulation of Toll-like Receptor (TLR-2) in response to chronic exposure to silica contributes to increased susceptibility to tuberculosis (TB) in such patients. In macrophages, genetic polymorphisms in molecules such as tumor necrosis factor-alpha (TNF-

α), natural resistance-associated macrophage protein 1(NRAMP1), and inducible nitric oxide synthase (iNOS) have been demonstrated to modulate the macrophage response to silica exposure. T-lymphocytes play a crucial role by producing interferon-gamma (IFN-γ), a cytokine that prompts macrophages to release growth factors contributing to fibrotic tissue formation. Additionally, neutrophils and macrophages can cause direct lung tissue damage, exacerbating the disease.[25] Another theory proposes that silica particles may serve as a reservoir of iron, which is an essential micronutrient for the growth of mycobacteria. The iron released from silica-iron complexes could potentially activate dormant bacilli, leading to the reactivation of tuberculosis. Additionally, tubercle bacilli may remain encapsulated within silicotic nodules, contributing to disease reactivation.

Clinical Picture of Silicotuberculosis

Initially asymptomatic, the affected worker typically presents with exertional dyspnea, which may progress to dyspnea at rest over time. There is parenchymal damage due to silicosis, which is evident in abnormal chest x-ray findings. The onset or worsening of dyspnea may signal the emergence of complications, such as tuberculosis. A productive cough is frequently observed, often resulting from chronic bronchitis due to occupational dust exposure, smoking, or both. In some cases, coughing may also result from compression of the trachea or mainstem bronchi by enlarged silicotic lymph nodes. Although hemoptysis is uncommon, its presence should prompt evaluation for possible mycobacterial disease. Wheezing and chest discomfort can occur in silicosis, but are more commonly linked to bronchitis and airflow obstruction. Chest pain and clubbing are not seen in silicosis, though they have been reported in certain studies. Systemic symptoms like fever and weight loss raise suspicion for coexisting tuberculosis. In advanced silicotuberculosis, patients may develop progressive decline in pulmonary function, with or without cor pulmonale, and imaging may reveal progressive massive fibrosis. Chopra *et al*.[26] reported that cough with expectoration comprised the predominant symptomatology (80%), followed by dyspnea (70%), chest pain (52.6%), hemoptysis (43%), fever (40%) and hoarseness of voice (1%). 35% were anemic, 30% had clubbing, 10% had cyanosis, 9% had pedal edema and 2% had lymphadenopathy among the mine workers with TB. Tyagi *et al*.[20] reported that among 221 mine workers in Jodhpur district, the majority of workers (58.8%) reported respiratory symptoms. The most common symptom was breathlessness (32.1%), followed by cough (31.7%) and chest pain (24.4%), indicating predominant respiratory involvement. Additionally, headache (14%) and fever (13.6%) were also commonly reported systemic symptoms.

Diagnosis and Challenges Encountered in Silicotuberculosis

Diagnosing active tuberculosis in patients with silicosis requires a strong suspicion and is primarily based on history of exposure, clinical presentation, and characteristic chest radiographic findings. It is difficult to differentiate between silicosis with silicotuberculosis on the basis of abnormalities in the chest radiograph. In patients with silicosis, radiographic findings suggestive of tuberculosis mostly appear in the apical regions of the lungs. These typically present as poorly defined infiltrates of varying sizes that respect fissural boundaries and may encircle existing silicotic nodules. Cavitation within a nodule strongly suggests tuberculosis, though fluid-filled cavities may also occur in silicosis alone.[27] With extensive fibrosis, hilar structures are usually pulled up, leading to hyper-translucent zones of lung in the periphery area of the lung and lower zones, also associated with multiple bullae. Lymph node involvement in chronic silicosis is a characteristic finding, often demonstrating classic "eggshell" appearance. The hilar and mediastinal lymph nodes are commonly affected, though other intrathoracic and extrathoracic nodes may also be involved. When the visceral pleura is affected, it typically shows diffuse fibrotic thickening, occasionally accompanied by focal calcifications.[28,29] Distortion of lung parenchyma due to fibrosis may lead to bronchial stenosis, which may be irregular, especially of the right middle lobe. Additional indicative findings may include rapid progression of radiographic changes and the presence of pericardial or pleural effusions. Computed tomography [CT] of the chest is useful in some patients to distinguish between silicosis and silicotuberculosis by the appearance and distribution of nodules, fibrosis and cavitation.[27]

Establishing the diagnosis by bacteriological methods is often difficult as due to fibrotic changes within the silicotic nodule because mycobacterium tubercular bacilli may not expectorate out in the sputum of silico-tuberculosis patients.[30,31]These fibrotic alterations in the lung tissue hinder the effective release of tubercle bacilli into the sputum, complicating the diagnostic process and potentially resulting in false-negative results in tuberculosis sputum tests for these individuals and may raise concern about potential bacterial reactivation within these individuals. Consequently, conventional sputum tests for tuberculosis may fail to detect the presence of Mycobacterium tuberculosis in these cases, leading to inaccurate diagnoses and delays in initiating appropriate treatment.[32] Therefore, frequent sputum smear examinations for acid-fast bacilli (AFB) are recommended. Mycobacterial culture is of paramount importance in regions facing a significant load of non-tuberculous mycobacterial (NTM) infections.[33,34] Regular chest radiographic screening is considered more efficient than sputum examination for the early detection of tuberculosis in patients with silicosis, emphasizing the significance of proactive radiographic monitoring in this population for timely intervention and treatment.[35] Serial radiographic evaluations are recommended, with close monitoring for asymmetric nodules, areas of consolidation, pleural effusions, cavitary lesions, and any focal or rapidly progressive changes.[36] Although cavitation is

a key radiological marker suggestive of silico-tuberculosis, it may also arise secondary to ischemic necrosis within fibrotic silicosis masses.[37]

Sureka *et al*.[38] reported that a case of silicotuberculosis in consecutive chest radiographs over a six-year period demonstrated progressive pulmonary volume loss, superior displacement of the hilum, and increasing density of conglomerate fibrotic mass lesions in the upper and mid lung zones bilaterally, accompanied by bilaterally lower lobes compensatory hyperinflation. The imaging also revealed calcified mediastinal lymph nodes, calcification in the bilateral lung parenchyma, and areas of cavitation within the conglomerate masses. A lung biopsy from the lesion demonstrated silicotic nodules and the presence of acid-fast bacilli, confirming the diagnosis of silicotuberculosis. Shitrit *et al*.[39] reported that among 28 patients with suspected silicosis, 11 patients (39%) with mediastinal lymphadenopathy underwent evaluation using 'endobronchial ultrasound-guided transbronchial needle aspiration' (EBUS-TBNA), which identified silica particles under polarized light. In the remaining 17 patients, transbronchial biopsy (TBB) was diagnostic in only 76% of cases, necessitating video-assisted thoracoscopic surgery (VATS) for further evaluation. Sarkar *et al*.[40] highlighted the potential of 'Clara cell secretory protein' (CC16) as a biomarker for early detection of silica-induced lung damage, validated through chest radiography. Early detection of silicosis is essential as it significantly increases the risk of pulmonary tuberculosis. The Indian Council of Medical Research has authorized the use of the CC16 serum detection kit for the periodic screening of workers exposed to silica. Addressing silicosis is crucial for India's TB elimination efforts due to its high prevalence among workers.

Functional Assessment in Silicotuberculosis

Silico-tuberculosis results in a significant decline in lung function, especially in airway obstruction. Spirometry, conducted during initial evaluation and during follow-up to assess potential functional deterioration, helps to evaluate the extent of pulmonary dysfunction following effective tuberculosis treatment.[41] Radiographic abnormalities have a limited correlation with ventilatory impairment, with lung function deterioration in workers primarily reflecting the exposure burden characterized by duration and concentration of silica dust.[42] A reduced six-minute walk test (6MWT) has been identified as a predictive marker for both morbidity and mortality in silicosis patients, suggesting its use in continuous health surveillance.[43]

Hnizdo *et al*.[44] conducted a study on South African gold miners, revealing that pulmonary tuberculosis can result in chronic lung function impairment, with functional loss stabilizing approximately 12 months post-diagnosis. The study found that the average chronic deficit in lung function, measured by FEV1 and FVC, worsened with the number of tuberculosis episodes, irrespective of HIV status. Chronic airflow impairment was present in 18.4 to 35.2% of miners with tuberculosis episodes.

Management of Silicotuberculosis

Management of silicotuberculosis involves a multifaceted approach. These strategies include controlling silica dust exposure, ensuring continuous TB treatment, and implementing occupational health management by professionals. Early detection through routine health check-ups and TB screening, along with policy-making for the high-risk population to prevent dust inhalation.[45] Exposure to crystalline silica dust elicits oxidative stress and nitrosative stress in the lungs that impairs alveolar macrophage function, disrupts innate immune responses, and creates barriers to effective drug delivery and distribution within the tissues, which facilitates the persistence of Mycobacterium tuberculosis within alveolar spaces, thereby increasing the risk of disease relapse.[46] Individuals with silico-tuberculosis exhibit an elevated risk of disease relapse and are more prone to treatment interruptions.[47,48] The WHO End-TB Strategy targets a treatment success rate of at least 90% among tuberculosis patients by 2025.[49] However, under the TB programme, the exact risk of treatment non-completion in patients with silico-tuberculosis remains unclear. Silicosis and tuberculosis have both been prioritized for global elimination by 2030, in line with international health and occupational safety goals.[50,51,52]

A clinical trial in Hong Kong[53] randomized patients with silico-tuberculosis patients receive either a 6 or 8-month regimen of thrice-weekly therapy, consisting of isoniazid, rifampicin, pyrazinamide, and streptomycin. Those previously treated tuberculosis patients also received ethambutol for the first three months. In 80% of patients, sputum conversion was done within two months. However, relapse rates during a three-year follow-up were higher in the six-month treatment group (22%) compared to the eight-month group (7%). About 22% patients experienced significant adverse drug reactions. Lanzafame *et al*.[54] also suggested that an extended treatment duration for at least eight months of antituberculosis therapy under 'Directly Observed Treatment, Short-Course' (DOTS) is recommended for patients with silicosis-tuberculosis to reduce the risk of relapse, as there is impaired macrophage function due to free silica exposure and limited drug penetration into fibrotic nodules. In a study, Singh D *et al*.[55] investigated the association between silica dust exposure, silicosis, and tuberculosis (TB) drug resistance patterns and demonstration that both the incidence of tuberculosis(TB) and the prevalence of mono-drug resistance are directly related to the intensity and time period of silica exposure. Among workers with radiologically confirmed silicosis, 16.4% were found to have TB resistant to both isoniazid and rifampicin. Additionally, the occurrence of multidrug resistance showed a statistically significant linear trend. In a study done by Rupani *et al*.[56] in Khambhat block, India. Over

138 patients with silico-tuberculosis were compared to 2,610 tuberculosis (TB) patients without silicosis. The study reveals that individuals with silico-tuberculosis had 2.3 times higher risk of experiencing unfavorable treatment outcomes. Sputum positivity rates were almost similar between the silicosis with tuberculosis group (57%) and the silicosis without tuberculosis group (55%). However, mortality during treatment was significantly higher in silico-tuberculosis patients (23%) as compared to TB patients without silicosis (11%).

Complications in Silicotuberculosis

Maintaining continuity of care is vital, as silicotuberculosis can lead to complications like chronic airways obstruction and non-tuberculous mycobacterial disease and other infectious diseases. The presence of fever, loss of weight, progressive deterioration of respiratory symptoms, or new radiographic abnormalities in silica-exposed individuals should prompt thorough evaluation to exclude treatable conditions such as tuberculosis or malignancy. In 1997, the 'International Agency for Research on Cancer' (IARC) categories crystalline silica (quartz and cristobalite forms) as a Group I carcinogen, based on enough evidence of its carcinogenicity in humans. Occupational silica exposure has been firmly established as a causal factor in the development of lung cancer.[57] Since the early 20th century, silica exposure has been linked to renal complications. A study in 2002 identified a 5.1% increased chance of end-stage renal disease (ESRD) and a 1.8% rise in mortality from kidney disease among exposed individuals. Additionally, silica exposure also leads to conditions like glomerulonephritis and nephrotic syndrome.[58-61] According to a review by the National Institute for Occupational Safety and Health (NIOSH), silica exposure may have a synergistic effect in triggering autoimmune responses. Scleroderma, systemic lupus erythematosus (SLE), rheumatoid arthritis, autoimmune hemolytic anemia, and dermatomyositis or polymyositis are the autoimmune diseases commonly associated with silicotuberculosis.[62] Other complications that may occur in silicotuberculosis patients are spontaneous pneumothorax, broncholithiasis, cor pulmonale and end-stage hypoxemic ventilatory failure.

Recommendations for Prevention

Prompt detection of silicosis and tuberculosis among occupationally exposed silica workers is crucial for preventing progressive lung damage and reducing TB transmission within the community. Effective control or elimination of respirable crystalline silica exposure would substantially decrease the incidence of both diseases. As a part of a comprehensive strategy to eliminate silicosis, primary prevention measures—such as health awareness programs, workforce training, active TB case detection in high-risk areas, targeted surveillance, and worker rehabilitation— play a critical role in minimizing the overall health burden. After exposure to silica for longer than 10 years, irrespective of silicosis disease, individuals should have periodic chest radiographs and tuberculin skin test [TST] in the initial evaluation. For latent tuberculosis infection [LTBI] in people with silicosis, short course prophylactic regimens should be considered under the Tuberculosis preventive treatment (TPT) guidelines.[63,64] Active surveillance of workers should be implemented during both pre-employment and post-employment periods to enable early detection of occupational illnesses. Given the irreversible nature of diseases caused by silica dust exposure, state and national governments must establish policies for appropriate compensation to affected individuals who develop occupational diseases. Strict regulations must be there for the industries to improve the working conditions, maybe by improved engineering controls or compulsory use of advanced personal protective equipment for the workers, resulting in limited silica dust exposures.

Conclusion

A comprehensive, multifaceted approach is needed to better manage TB in the context of occupational silica exposure, that includes active surveillance for early TB detection in individuals with silicosis, development of enhanced diagnostic methods like molecular diagnostics, optimization of TB treatment regimens specific to this population, and preventive strategies such as effective dust control measures in the working area to decrease the incidence of silicosis and associated TB. A stronger emphasis on occupational health and safety measures to minimize silica dust exposure is essential for the primary prevention of both TB and silicosis. Moreover, silicosis patients who develop tuberculosis require not only frequent monitoring during treatment but also close follow-up even after treatment so as to detect complications, recurrence and need for pulmonary rehabilitation in these patients.

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