Guidelines for Management of Silicosis in Primary Care Settings[1]

- Dr Ramani Atkuri, MD

What is silicosis?

Silicosis is a lung disorder caused by inhalation of tiny particles of silicon dioxide (SiO2) in the form of unbound (free) crystalline silica (quartz) or less commonly, by inhaling silicates where silica is bound to other materials like talc. It is the oldest known occupational disease of the lungs and even today is responsible for thousands of deaths.

Who is at risk of silicosis?

Anyone who is exposed to silica for any length of time is at risk of developing silicosis. The following industries are the ones where the worker is at greatest risk:

a. Mining – minerals
b. Stone quarrying
c. Natural and artificial stone cutting
d. Sandblasting
e. Construction workers
f. Stone masons
g. Foundry workers
h. Glass workers
i. Mica mining
j. Slate pencil workers

Artificial or engineered stone is used in construction, for counter-tops, flooring, etc. It has more than 90% silica and exposure while cutting, grinding and polishing engineered stone exposes workers to high levels of silica, leading to severe forms of silicosis.

Development of silicosis depends on various factors –

a. Duration and intensity of exposure
b. Form of silica (exposure to crystalline silica is more dangerous)
c. Rapidity of inhalation after the dust is fractured and becomes airborne (exposure immediately after fracturing has greater risk than delayed exposure).

Silicosis in India

India has a large mining industry, with the states of Chhattisgarh, Jharkhand, West Bengal and Odisha being particularly mineral-rich. Workers in these mines are at risk of developing silicosis. An ICMR report of 1999[i] puts the number of workers at high risk of silicosis at 3 million. Of these, 1.7 million are in mining or quarrying industry, 0.6 million in

[1] Cite as: Guidelines for Management of Silicosis in primary care settings. BHS working paper series. Ramani Atkuri, January 2023. Basic Health Care Services

manufacturing non-metallic products (like refractory clay, glass and mica), and 0.7 million workers in the metals industry. These numbers are likely to be under-estimates. Currently India has around 74 million informal workers in the construction industry[ii] alone, many of whom are at risk of developing silicosis. Apart from this, work like stone-carving, marble work, polishing of granite etc – all are high risk jobs for contracting siliciosis.

Stone quarrying and crushing happens all across India, and is mostly carried out by poorer people in informal work settings. Hence, getting accurate information on areas where silicosis is more prevalent, or the number of people working in jobs that expose them to silica dust, is very difficult. However, what is evident is that most of the people affected by silicosis are in the productive age group.

India is one of the largest manufacturers and exporters of engineered stone. The silica content of artificial stone is greater than 90%, and exposures are often intense leading to severe forms of the disease.

What happens when a person is exposed to silica dust?[iii, iv]

Respirable particles of silica (< 10microns in diameter) are deposited in the distal airways and alveoli after dust inhalation. Inhalation of crystalline silicon dioxide causes mineral deposits to form at the level of terminal bronchioles and alveoli. The presence of foreign material results in the activation of alveolar macrophages and also exerts direct toxic effects on the surrounding lung parenchyma. Cellular damage results in the release of inflammatory cytokines (such as IL-1 and TNF-alpha), the generation of free radicals, and augmentation of cell-signaling pathways. The different cytokines result in the promotion of fibrosis. There is also evidence that silica interferes with the ability of macrophages to inhibit the growth of mycobacteria and this effect explains the common association of silicosis with tuberculosis.

When the macrophages die, they release the silica into interstitial tissue around the small bronchioles, causing the characteristic silicotic nodule. These nodules initially contain macrophages, lymphocytes, mast cells, fibroblasts with patches of collagen, and some birefringent particles. As these nodules mature, their centres become dense balls of fibrotic scar with a classic onion-skin appearance, and are surrounded by an outer layer of inflammatory cells. Fibrotic changes are seen alongside silicotic nodules, leading to distortion of lung parenchyma and reduction in gas-exchange sufaces.

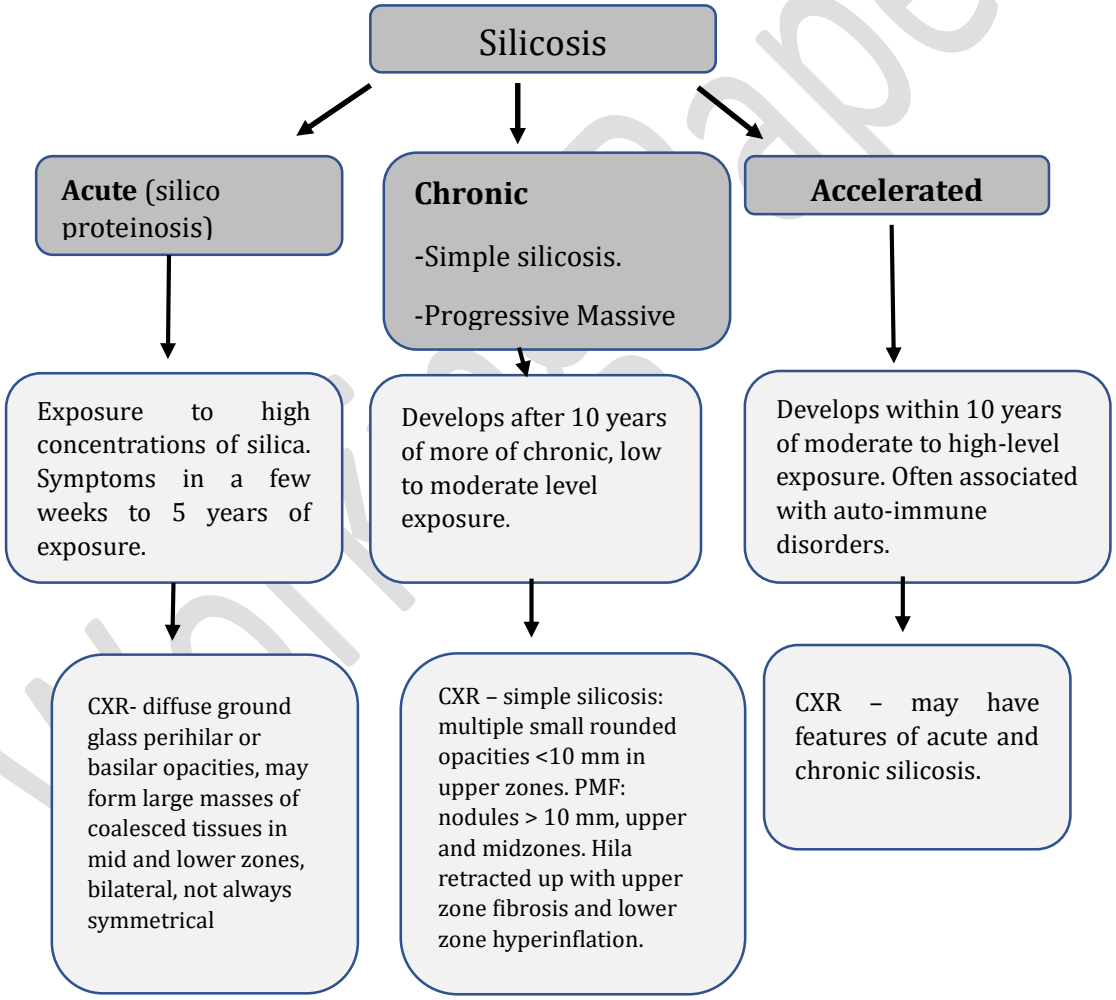
Damage caused by silica is dose-dependent. The current safe exposure limit is set at 0.05 mg/m3 of ambient air but even at this level, the risk of developing simple silicosis after a lifetime of working in this environment is 25-40%.

When exposure is less or of short duration, the nodules are discrete and do not affect lung function (simple chronic silicosis). But when exposure is of higher intensity or with more prolonged exposures (complicated chronic silicosis), the nodules join and cause progressive fibrosis with compromise of lung function. There is reduction of total lung capacity and vital capacity on lung function tests. Sometimes the nodules coalesce, leading to progressive massive fibrosis.

Clinical presentations of silicosis

Three types of silicosis are seen:

1. Acute silicosis (also called silicoproteinosis) occurs with high intensity, short duration exposure. Patients present within weeks to 5 years of the initial insult. Surface drilling and sandblasting can expose the worker to such high concentrations of silica dust (1-10mg/m3/year)
   Symptoms are dysnoea, fatigue, weight loss and pleurtic pain. The patient can progress rapidly to respiratory failure due to decline in gas exchange and pulmonary function.
2. Accelerated silicosis develops within 10 years of moderate to high-level exposure. The disease can progress even after the person is removed from silica exposure. This kind of silicosis is often associated with auto-immune disorders.
3. Chronic silicosis occurs after 10 years or more of low to moderate level exposure. Patients can present with simple (nodular) silicosis or progressive massive fibrosis (PMF). Patients with simple silicosis can be asymptomatic, while patients with PMF can present with chronic respiratory failure.



Complications of silicosis

Patients with silicosis are at higher risk of the following diseases –

- Tuberculosis (this is discussed separately)
- Lung cancer
- Progressive systemic sclerosis
- Chronic kidney disease
- Possibly rheumatoid arthritis

Patients with silicosis have a 30-fold higher risk of developing tuberculosis or non-tubercular mycobacterial disease; and have higher risk of both pulmonary and extrapulmonary symptoms. People with exposure to silica but with no silicosis have a three-fold increased risk of developing TB.

Spontaneous pneumothorax and emphysema may also occur.

What are the symptoms of a patient with silicosis?

1. Persistent cough
2. Cough with sputum
3. Shortness of breath (Dyspnoea) – progressive.
4. Weight loss
5. Fever
6. Fatigue and weakness

What do you find in a patient with silicosis?

- The patient may initially be asymptomatic, but will eventually develop dyspnoea during exertion that progresses to dyspnoea at rest.
- Productive cough that may be due to silicosis, or chronic occupational bronchitis or smoking.
- Breath sounds reduce as the disease progresses, and pulmonary consolidation, pulmonary hypertension and respiratory failure with or without right ventricular failure may develop.
- Clubbing is **not** a typical feature of silicosis and if present in a silicotic patient, must raise suspicion of lung cancer.

Diagnosing a patient with silicosis

a. Acute silicosis

This is also known as acute silico-proteinosis, and develops after exposure to high concentrations of respirable, crystalline silica. Symptoms develop in a few weeks or few years after exposure.

Patient presents with a rapid onset of dyspnoea, cough, weight loss, fatigue, pleuritic pain and fever. On examination, crepitations are usually present. In addition to a history of exposure to silica, other causes of similar symptoms like pneumonia, acute respiratory distress syndrome, heart failure etc are ruled out.

Assessment of oxygen saturation is important.

Lung function tests show spirometric reduction in FVC and FEV1.

Chest X-ray shows bilateral, diffuse, ground-glass opacites that are basilar or perihilar. These may progress to large masses of coalesced lung tissue in the mid and lower zones that are bilateral, though not always symmetrical.

Diagnosis is based on the history of acute, high dose silica exposure, X-ray findings of diffuse nodular and patchy opacities, (a milky and proteinaceous broncho-alveolar lavage effluent) and ruling out other causes of acute respiratory distress.

Treatment – acute silicosis is progressive with no specific therapy. Complete removal from exposure and supportive care is the treatment indicated.

Look for, and treat associated infections like tuberculosis.

b. Chronic silicosis

Chronic silicosis (which includes simple silicosis and progressive massive fibrosis) develops slowly, usually 10-30 years after initial exposure. The X-ray changes of silicosis may develop long after the person's exposure to silica has stopped. The progressive coalescence of silicotic nodules leads to the replacement of the upper lobe parenchyma with nodules and air trapping and emphysema in the lower lobes due to fibrotic retraction by the upper lobes. This results in respiratory impairment.

Clinical picture is variable. Patient with simple silicosis may be asymptomatic with only an abnormal chest-X-ray. Patient my develop chronic cough and dyspnoea on exertion which worsen with worsening radiographic findings.

PMF is associated with more severe symptoms of cough and dyspnoea.

Chest examination – may be normal in simple silicosis, or there may be a variety of sounds including coarse (end-inspiration) crepitations, fine crepitations, rhonchi and / or wheeze. PMF patients have inspiratory crackles. PMF is not associated with digital clubbing.

Evaluation – confirm degree of exposure, assess respiratory function, exclude other causes of symptoms. A sputum smear and culture for AFB is necessary to rule out tuberculosis as a complication of silicosis. Pulmonary function tests may be normal or have a range of abnormalities with PMF associated with the worst pulmonary function abnormalities.

Chest X-ray – Simple silicosis – innumerable small rounded opacities less than 10 mm in diameter, in the upper lung zones. Outline may be rounded or irregular. PMF – occurs when these opacities coalesce into large upper or mid-zone opacities (larger than 10 mm in diameter). As these enlarge, hila are retraced up with upper zone fibrosis and lower zone hyperinflation. Opacities are irregular and may mimic a neoplasm. Cavitation may be seen within the opacities in advanced disease or in association with TB. Hilar adenopathy with prominent calcification may be seen in some cases.

Diagnosis is based on:

i. History of exposure sufficient to cause the degree of illness seen, and appropriate latency

ii. Chest X-ray findings
iii. Absence of other causes for symptoms.

Treatment: No proven specific therapy except avoiding the source of exposure or use of optimal respiratory protection. Supportive therapy includes smoking cessation (if indicated), treatment of airflow limitation with bronchodilators, vaccination against influenza and pneumococcus, and use of supplemental oxygen if indicated, to prevent complications of hypoxaemia. A short course of steroids have been shown to reduce mortality when given during severe breathlessness.

Patients should be referred to the appropriate person for benefits, compensation etc.

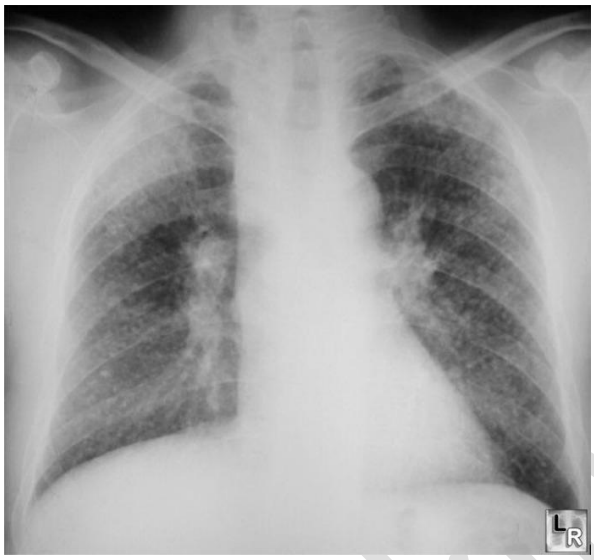


Figure 2 Chronic simple silicosis-fine granules <10mm, more in upper lobes.

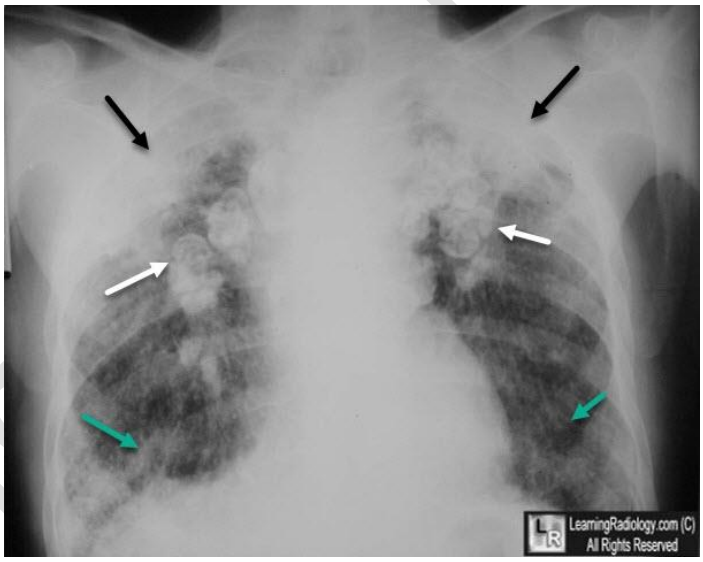


Figure 1 Silicosis with PMF - large, >10 mm nodules on apex, calcified hilar nodes with egg-shell calcification. Basal scarring. Hila pulled up due to fibrosis at apex.

c. Accelerated silicosis

This is associated with high levels of exposure and disease develops within 10 years of exposure. Radiography shows features of both acute and chronic disease.

Clinical presentation is variable. Patient may be asymptomatic with only an abnormal radiograph. Symptomatic patients have chronic cough and dyspnoea, which worsens with worsening X-ray abnormalities.

Diagnosis is based on a history of high level of exposure within the preceding 10 years, and X-ray features of both acute and chronic silicosis.

Management is similar to that of chronic silicosis – avoidance of exposure to silica dust, smoking cessation, bronchodilators if spirometry shows airflow limitation, vaccination against influenza and pneumococcus, and oxygen supplementation if indicated.

Silico-tuberculosis

Mycobacterial infection, especially tuberculosis, is a long-recognised and well-established complication of silicosis. It should be suspected when a patient with silicosis develops systemic symptoms, worsening respiratory impairment, hemoptysis, or changes in the Chest X-ray. Cavitation in a PMF lesion is particularly suspicious of MTB infection. Risk factors and predisposing factors for TB in silicosis patients are not well understood, though underlying HIV infection, previous TB, cumulative exposure and more intense exposure to silica dust, increase the risk.

The body's immune mechanism against tuberculosis is reduced in silicosis as macrophages are unable to effectively clear TB bacteria in the presence of crystalline silica particles. Also, drug penetration into fibrotic nodules is poor, hence treatment is required for a longer duration. Sub-optimal concentrations of anti-TB drugs within the nodules also increases the likelihood of drug-resistant TB.

Evaluate all silicosis patients for TB by sputum smear, culture and CBNAAT. A positive Multi-drug therapy for eight months have shown a lower relapse rate than treatment for six months in some studies.

A study among glass and non-glass factory workers in Firozabad[v] showed 46% of glass factory workers to have TB as against 4% of non-glass factory workers. Among silicotics workers, 66.6% were found to have TB, while only 20.8% of non-silicotics had tuberculosis. The same report refers to the existence of sub-radiological silicosis and the findings of a study in South Africa that showed that out of every 100 silicosis patients, 57 were sub-radiological (detected on autopsy), and that sub-radiological silicotics had 2-3 times higher risk of developing TB compared to non-silicotic patients. The authors advocate for early diagnosis of silicosis and to integrate silicosis control programmes with the national TB programme to achieve better results.

A retrospective study of treatment outcomes among patients with tuberculosis with and without silicosis in Khambat district of Gujarat by Rupani[vi] showed that TB patients with silicosis had a 2.3 times higher odds of unfavourable treatment outcomes. (Unfavourable treatment outcomes were defined as a patient stopping treatment for one month; or a positive sputum smear at the end of treatment, or the death of a patient while on treatment. Increasing age, male gender, having sputum positive TB, being previously treated for TB, and drug-resistant TB increased the chances of unfavourable treatment outcomes in silicosis patients when compared to TB patients without silicosis.

The study also included interviews of experts in silicosis for their opinion about TB patients with silicosis. They identified various factors responsible for unfavourable outcomes in patients with silico-tuberculosis (see figure below). They recommend collaborative TB-silicosis activities to achieve better outcomes. All silicosis patients should be evaluated for TB and treated as per national guidelines if needed. All TB patients should be assessed for occupational exposure to silica dust and be evaluated for silicosis. They recommend various actions to improve outcomes in patients with silico-tuberculosis, which are shown below.

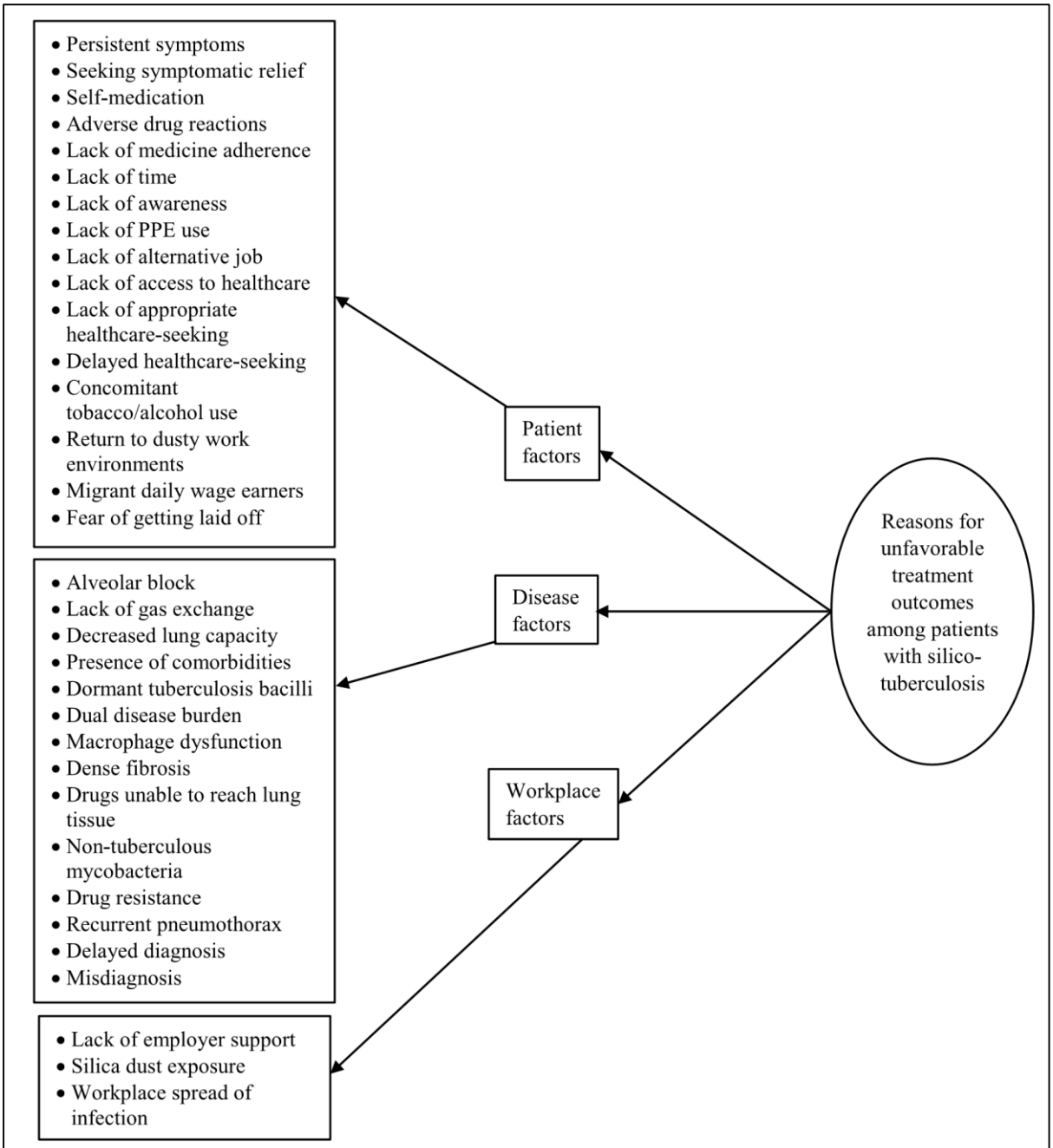


Figure 3. Reasons for unfavorable treatment outcomes among patients with silico-tuberculosis as perceived by experts during April–July 2022.

Reasons for unfavourable treatment outcomes in silico-TB patients

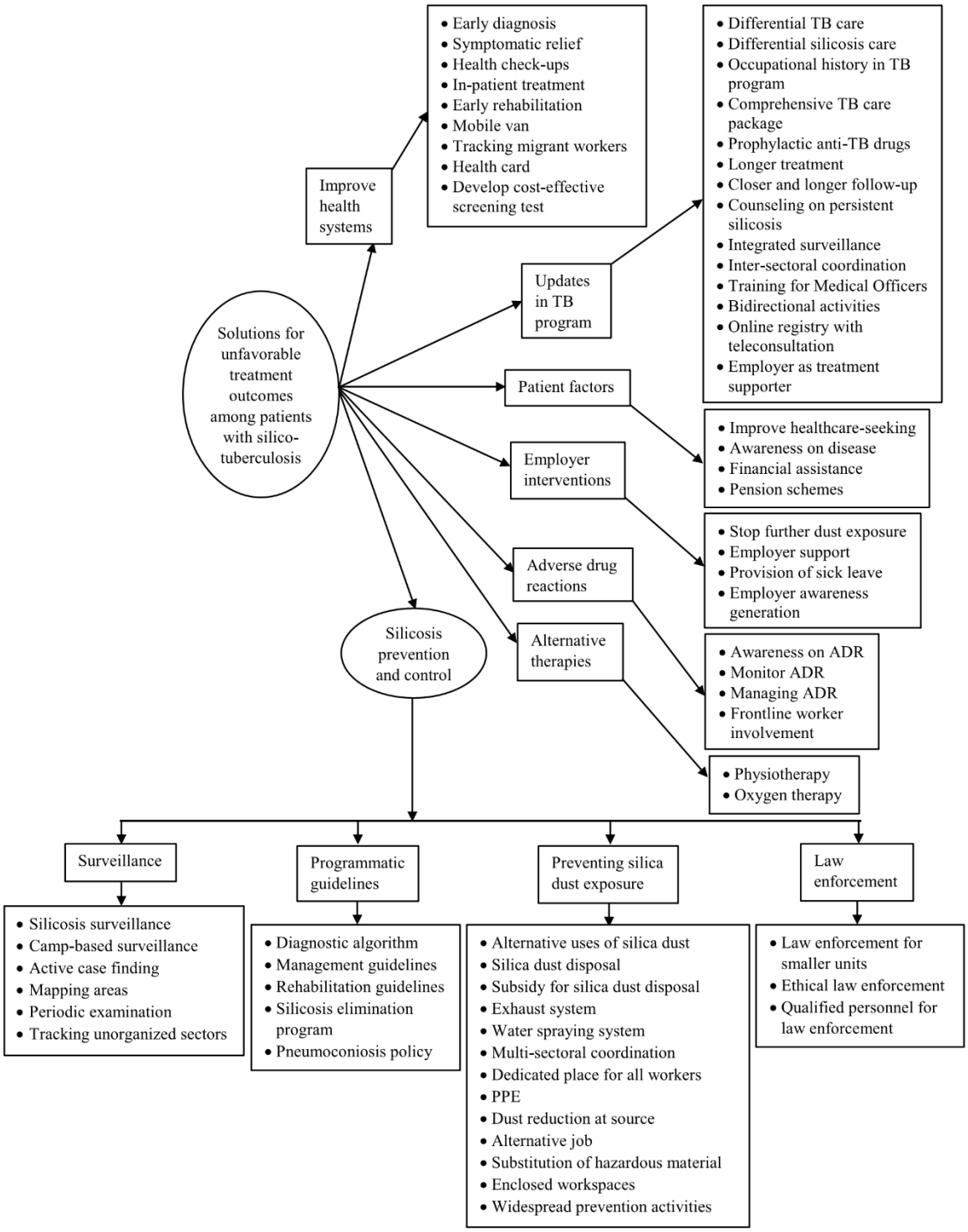


Figure 4. Solutions for unfavorable treatment outcomes among patients with silico-tuberculosis as perceived by experts during April–July 2022.

Suggested solutions to improve outcomes in silico-tuberculosis patients

Early Diagnosis of Tuberculosis in Patients with Silicosis

Silicosis patients are diagnosed on the basis of symptoms (fever, cough, breathlessness, weight loss, fatigue), as well as typical radiological findings. However, at this stage the disease is already advanced and little can be done to prevent further deterioration of the patient's condition. It is also often difficult to isolate TB bacteria from the sputum of silico-tuberculosis patients because the fibrotic tissue prevents / inhibits discharge of TB bacteria in the sputum.

It is therefore necessary to have a simple test that can detect silicosis early in workers exposed to silica dust, so that further damage can be prevented. Recent research by the ICMR-National institute of Occupational Health (ICMR-NIOH) has found serum Club Cell protein 16 (CC16) to be a useful proxy screening marker for early silicosis.

Club cell protein 16 (CC16) is the most abundant protein in bronchial secretions, and is produced by club cells that form about 22% of the cells in respiratory bronchioles. CC16 secreting cells are also present in ovaries, pancreas, uterine endometrium and mammary glands.

Chronic pulmonary inflammatory diseases like silicosis, COPD, asthma etc cause a decrease in CC16 as these conditions cause a degeneration of club cells.

Currently CC16 is detected with commercially available imported ELISA kits which are expensive. These tests also need expensive instrumentation.

The ICMR-NIOH test[vii] is a simple, point-of-care test, that uses a lateral flow assay for a semi-quantitative estimation of CC16. The assay can be performed periodically at intervals to assess the development of silicosis in workers exposed to silica dust. This assay gives an idea of lung injury before radiological changes can be seen.

The more severe the silicosis, the lower the CC16 levels.

The interpretation is done by comparing with the control line. The control line indicates a valid test. With very low CC16 levels (6ng/ml or less), only one red band is seen. With CC16 levels between 6.1 to 9 ng/ml, two red bands are seen. With normal or very early stage silicosis, CC16 levels are at 9.1ng/ml or higher. Sensitivity of the test is 100% and it is 95% specific.

The test results were checked against the ELISA test and correlated well.

Sr. No	Clinical significance	CC16 value range by ELISA
1	Suspected moderate to severe silicosis	6 ng/ml or less
2	Suspected early silicosis	6.1–9 ng/ml
3	Healthy or very early stage of silicosis that usually remains undetectable by chest x-ray	9.1 ng/ml or high

Classification of the three categories of detection using the rapid test LFA.

All X-ray confirmed silicotic patients had CC16 levels less than 9ng/ml and healthy controls had CC16 concentration >9ng/ml.

Figure	Description	Figure	Description
	**Negative control** **Observation**: **Control line**: One band was observed **Test line:** No band was observed		**CC16: 0 to 6 ng/ml (Suspected moderate to advanced Silicosis)** **Observation**: **Control line**: One band was observed **Test line:** Single band was observed
	**CC16: 6.1 to 9 ng/ml (Suspected early Silicosis)** **Observation**: **Control line**: One band was observed **Test line:** Two bands were observed		**CC16: > 9 ng/ml (Healthy person or early silicosis, not detectable by X-ray)** **Observation**: **Control line**: One band was observed **Test line:** Three bands were observed

Figure 1. Detailed description for interpretation of results for semi-quantitative lateral flow assay for detection of CC16.

Point-of-care assay for detection of CC16.

Other conditions in a silicotic patient

Silicosis is associated with an increased risk of

- mycobacterial infection,
- chronic necrotizing aspergillosis,
- lung cancer, rheumatic disorders,
- chronic kidney disease,
- chronic airflow obstruction and
- chronic bronchitis.

MANAGEMENT OF SILICOSIS

The main treatment for silicosis patients after removal from dust exposure is to provide them symptomatic relief and to enable them to lead as productive and active a life as possible. Initial presentation of the patients will be with dry cough and chest pain and there may be symptoms of underlying tuberculosis. On later stages, there will be significant cough, breathlessness on exertion, chest pain weight loss and fatigue on varying degrees. Also, in later stages due to limitation in lung expansion due to fibrosis as well as reduction in surface area for oxygen exchange, patients lead to COPD-like symptoms.

a. Remove from exposure
b. Treat the underlying COPD – bronchodilators, short-term steroids for exacerbations, supplement oxygen
c. Treat co-morbidities like TB
d. Watch for disease progression

Treatment of silicosis-associated conditions like chronic obstructive pulmonary disease, Tuberculosis, autoimmune conditions and chronic kidney disease are principally like that of patients without silicosis.

Even though for silicosis consistently effective therapy is not yet to be developed and the effective cure not yet exists, depending on the severity of the disease proper use of bronchodilators and pulmonary rehabilitation will helps in improving the quality of life. At later stages use of LTOT – Long Term Oxygen Therapy will help in respiratory distress.

Medical Management

Mild to moderate disease (breathlessness on moderate exertion, BMI normal, PEFR - Peak Expiratory Flow Rate - 150-400)

- Regular inhalation with inhaled bronchodilators like Ipratropium + Levo-Salbutamol - 2 puffs two times a day with rotahalers. DPI (Dry powder Inhaler) is effective in early stages, since it is cheaper and ease to use. Also, in early stages the respiratory effort of the patients is preserved which will make rotahalers equally effective as MDI (Metered dose Inhalers).
- Use of oral broncho dilators like Deriphylline 150mg 2 times daily will help as an adjuvant.
- Avoid Steroid Inhalers as they increase the infective exacerbations of underlying COPD.

Severe Disease **(**breathlessness on rest or while performing activities of daily living, PEFR <150, BMI<18.5)

a. **Regular inhalation** of a combination of Ipratropium (40 micrograms) + Levo-Salbutamol (100 micro-grams) puff twice a day through metered dose inhalers (MDI).
b. **MDI with Levo salbutamol is used to cover the noon** should be used when symptoms are getting worse.
c. Immunosuppressive therapy with corticosteroids has varying results. Improvement in symptoms is reported but mortality is not affected. When there is underlying tuberculosis treating steroids for initial few months will be helpful in better weight gain and recovery.
d. **Acute exacerbation:**
   i. Give Oxygen: Keep the target oxygen saturation around 94%.
   ii. Nebulization with Salbutamaol, 2.5-5 mg, add inhalational ipratropium or add inhalational budenoside.
e. **In case of severe breathlessness: Give Injectable Hydrocortisone 10 mg/ kg stat, followed by** 30–40 mg of oral prednisolone, in 3 divided doses, for a period of 10–14 days.

> Steroids can reduce mortality in cases of severe breathlessness. Give injectable Hydrocortisone 10 mg / Kg followed by oral prednisolone.

f. **Add antibiotics if any of the two are present:**
   - Breathlessness has increased
   - Increased sputum production
   - Sputum has become purulent
   - High grade fever

> Antibiotic treatment
>
> a. Cap Amoxycillin 500mg-1gm thrice daily for 7-10 days
>   OR
> b. Cap Doxycycline 100 mg twice daily for 7-10 days

g. Long term oxygen therapy (LTOT): Patients was started with home-based oxygen to keep the oxygen saturation around 94% (with nasal cannula and oxygen concentrator)

If patient's SpO2 remains below 92% consistently associated with,

- Severe breathlessness
- Bluish tint to lips and nails(cyanosis)

- Extreme fatigue
- Severe headaches
- Coughing or wheezing
- Fast pulse / heart rate

LTOT should be given to maintain the saturation at 94%

h. Patients need referral if:

- Confusion setting in
- RR>= 30 despite oxygen and inhalational salbutamol
- Systolic BP < 90 mm Hg
- SpO2 below 90% despite of oxygen

Other management options include

1. **Pneumococcal and Influenza Vaccination:** Patients with silicosis should be vaccinated with pneumococcal vaccine and booster dose every 5 years and influenza vaccine for every year.
2. **Nutrition:** Patients should be advised to consume energy-dense and protein-rich food to have a completely balanced diet as they would be experiencing muscle atrophy in their lungs.
3. **Breathing Exercise:** Teach COPD exercises – pursed lip breathing and belly/ diaphragmatic breathing

In all cases, teach the patient to

1. Blow air through straw in a bottle filled with water, combined with effective coughing.
2. Blow air in balloon.
3. Stop smoking
4. Avoid indoor cooking
5. Avoid exposure to cold air
6. The patient must be informed that drugs will be required lifelong. There may be addition or deletion of drugs, but unlikely that you they will be off drugs
7. If engaged in an occupation that exposes to inhalation of dust, they should discontinue immediately

Treatment of Silicotuberculosis

1. Patients suffering from silicosis and tuberculosis should be treated with steroids for the first 2 months: Prednisolone 0.5-1mg/kg weight divided doses
2. ATT for patients suffering from Silicotuberculosis should be continued for 9 months

PREVENTION OF SILICOSIS

a. Source reduction
b. Personal protection
c. Periodic screening for lung function

Reducing exposure to Respirable Crystalline Silica (RCS)[viii]

Occupational safety and health (OSH) standards[ix] put the Permissible Exposure Limit (PEL) of respirable silica dust at 0.05 mg/m3 (50mcg/m3) of ambient air, averaged over and 8 hour period. Actionable level is 25mcg of silica / m3 of air. (The American Conference of Governmental Industrial Hygienists – ACGIH- puts the threshold limit value (TLV) at 0.025mg/m3).

However, **in India**, the Director General of Mines Safety recommends a PEL for silica dust of 3 mg/m3 when the free silica content of airborne dust is 5% or less. This is equivalent to a PEL of 0.15mg/m3, which is three to six times higher than the US standards. A study conducted by the Occupational Health department of the National Institute of Miners' Health in Nagpur[x] showed that even asymptomatic miners exposed to dust levels less than 0.1 mg% showed silicotic changes in their lungs on chest X-ray. Thus there is need to review the present PEL standards set by the Government of India.

Source reduction

The most preferable option would be to eliminate the silica containing material, or to substitute it for one that it less likely to cause silica exposure.

Engineering controls like local exhaust ventilation (LEV) with a collecting hood and vacuum dust collector or the use of water sprays can also help to keep down RCS exposure.

Exhaust hoods in workplaces

Though drilling tools with in-built water sprays are not commonly available commercially, NIOSH has tried to design one such tool (see figure below). The angle of the water outlet as well as the amount of water / minute are crucial in determining how well the dust is kept under control.

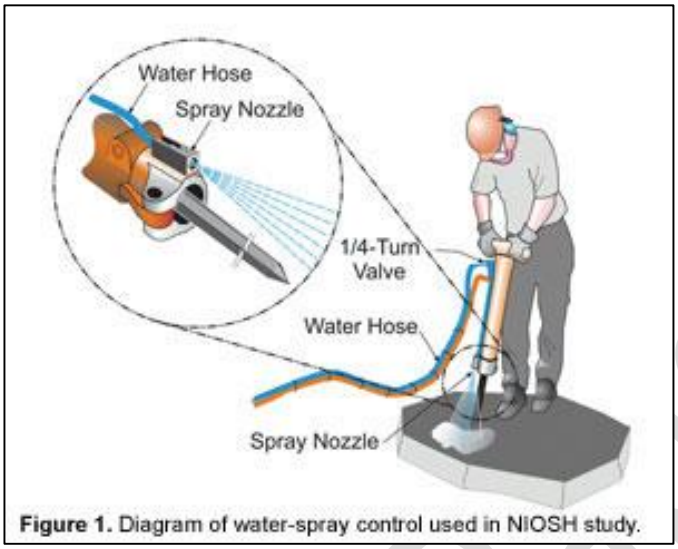


Figure 1. Diagram of water-spray control used in NIOSH study.

Spraying atomized mist over the work area can also help to precipitate airborne RCS particles. Pressurised water is sprayed as millions of tiny droplets into the work area, raising its humidity and binding with dust particles. It reduces use of water as compared to normal spraying by sprinklers.

Atomised mist sprayed over work area to reduce dust.

Personal protection

Air-purifying respirators[xi] are used along with engineering measures for protection of the worker from silica dust. They work through removing gases, vapours and aerosols (droplets and solid particles) through the use of filters, cartridges or canisters. These respirators do not

supply oxygen so cannot be used in a place that is oxygen deficient. The appropriate respirator depends on the type of environmental contaminant.

Filtering Facepiece Respirator (FFR)

- Disposable
- Covers the nose and mouth
- Filters out particles such as dust, mist, and fumes
- Select from N, R, P series 95, 99, 100 efficiency level
- Does NOT provide protection against gases and vapors
- Fit testing required

Where engineering controls cannot keep the RCS below 0.5 mg/m3, a respirator with an Assigned Protective Factor (APF) greater than 10 is required.

Protective factor = (concentration of harmful substance outside the mask) / (concentration of substance behind the mask)

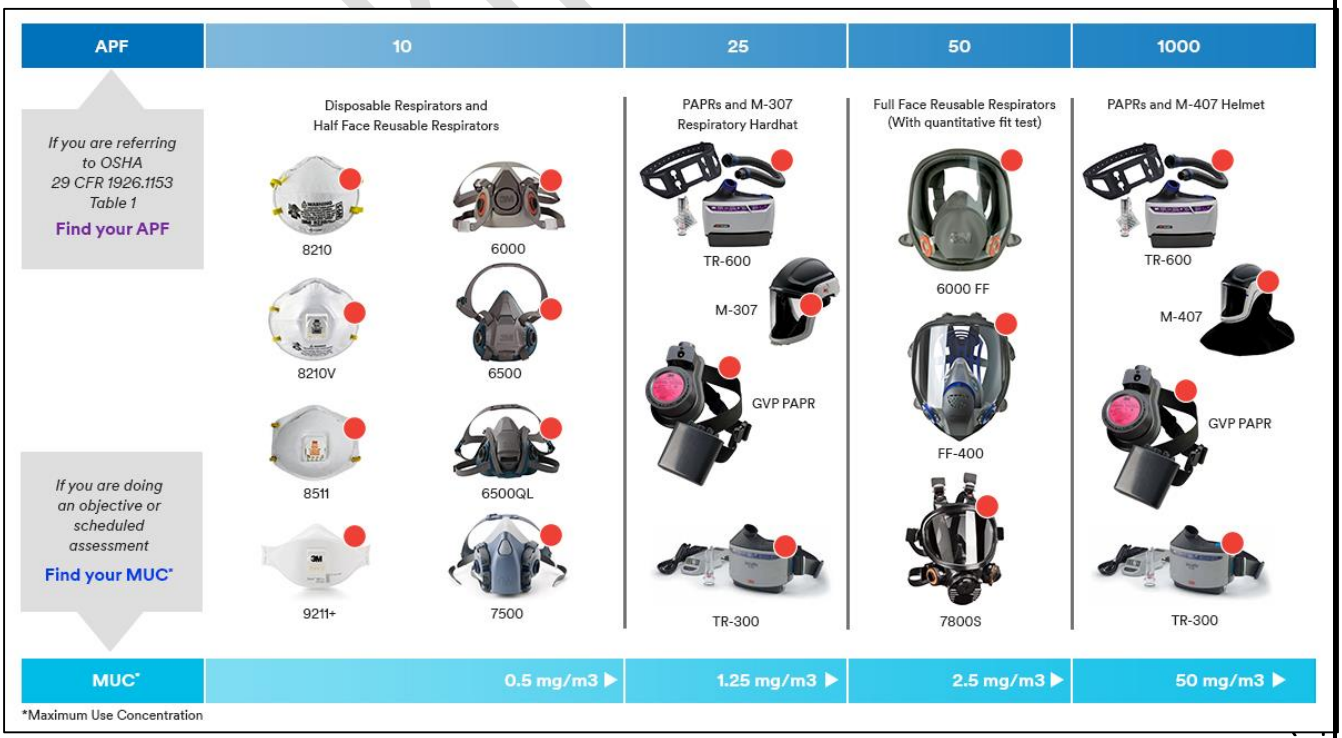
Assigned Protection Factor (APF) is the decrease in concentration of harmful substances in the inhaled air, that is expected to be provided with timely and proper use of a certified respirator of specific types, by taught and trained workers. The specific respirator is selected with a tight-fitting mask and a fit-testing[xii].

For exposures exceeding an RCS concentration of 2.5 mg/m3, only a supplied-air respirator, such as a self-contained breathing apparatus (SCBA), with an APF of at least 1,000 will be protective enough. However, since many tasks whether in construction or stone carving / mining have such high levels of exposure, it makes sense to focus on engineering controls to reduce the amount of RCS exposure.

Disposable respirator for low concentration of silica dust

Respirator with APF 10 for protection, medium RCS exposure

Hierarchy of hazard control[1][2] [hide]	
1.	The use of alternative substances which are less hazardous.
2.	The substitution of a given substance in a form that is less hazardous, e.g. replacing a fine powder by a coarser powder, pellets, or by a solution
3.	The substitution of a process by an alternative process likely to generate lower airborne concentrations of substances
4.	Total or partially enclosed process and handling systems
5.	Partial enclosure with local exhaust ventilation
6.	Local exhaust ventilation
7.	General ventilation
8.	Reducing period of exposure
9.	The introduction of appropriate working practices and systems of work, e.g. to close and store containers securely when not in use
10.	Use of monitors and warning devices to give a clear indication when unsafe airborne concentrations are present
11.	Good housekeeping
12.	Provision of a respiratory protective device program



Types of respiratory masks required for different levels of dust exposure, with different APF.

Complying with OSHA Silica Regulations

a. Check whether and to what extent the workers are exposed to silica
b. Introduce engineering controls – purchase and install dust-control equipment or specialized cutting tools
   All housekeeping to be done with wet methods and no dust removal using compressed air to be allowed.
c. Inform employees – workers exposed to silica dust must be informed of the hazards and trained on how to use PPE properly and use tools to reduce exposure risk. Additionally, any worker required to wear a tight-fitting respirator must be medically fit and trained on the use, cleaning and proper storage of their respiratory equipment.
d. Monitor air levels to see that silica dust concentrations are within permissible limits. Maintain detailed records of all air quality monitoring and employee exposure over PEL. Appoint a "competent person" at each work site who can identify silica exposure hazard and implement steps to address it.
e. Maintaining employee health records.
f. Ongoing silica management.

Measuring Respirable Silica Dust Levels

Sampling is done using a sample pump that samples air by pulling it through a cyclone. Different cyclones allow differently sized particles to pass through. The pump catches larger particles while the smaller particles collect on a filter. Collected particles are then sent for analysis. This is called gravimetric sampling, and the collection, processing and analysis can take weeks.

Real-time, direct reading instruments are now available that provide immediate information about exposure levels. They may not directly measure respirable silica levels but use light-scattering photometric technology to measure the quantity of dust in the air. It can be calibrated to determine the amount of crystalline silica in the aerosol sample, which is quite close to the levels determined by the gravimetric method. Though available commercially in India, they are too expensive for general use.

Compensation for silicosis injury

Any worker who develops silicosis is entitled to compensation by the Government.

Silicosis is a notifiable disease under Mines Act, 1952, Factories Act 1948 and Building and Other Construction Workers (BOCW) (Regulation of Employment and Conditions of Service) Act 1996. It is also a compensable disease under Employees Compensation Act 1923 and Employees' State Insurance Act 1948. While Mines Act is implemented by the Central Government through Director General of Mines Safety, the Factories Act and Building and Other Construction Workers (Regulation of Employment and Conditions of Service) Acts are implemented by state governments through the Chief Inspector of Factories.

What is the Policy in Rajasthan?

The Rajasthan Policy on Pneumoconiosis, including Silicosis Detection, Prevention, Control and Rehabilitation[xiii] spells out in detail about the steps the state government intends to take with regard to pneumoconiosis. The following schemes are available:

S. No.	Welfare Scheme	Assistance
1.	Rehabilitation Assistance	Rs3,00,000 as one-time assistance payable to the affected person after certification
2.	Assistance on death	Rs2,00,000 to the legal heir / nominee in the event of death of the Pneumoconiosis victim.
3	Pneumoconiosis Rehabilitation Pension	Pension equivalent to persons with disability, would be sanctioned irrespective of income criteria.
4.	Provision for sustenance of family	Widow pension to wife, and/or benefits under Palanhar would be provided irrespective of income criteria.
5.	Funeral Assistance	On death of the Pneumoconiosis victim, Funeral Assistance of Rs.10,000 will be given to the dependents where such assistance is not availed from any other source.

Future directions for silicosis prevention and control in India

Policy level

- Redefine permissible exposure limits (PEL) for silica dust in workplaces with safe exposure levels below 0.05mg/m3.
- Mandate periodic testing of workers for early diagnosis of silicosis through CC16 assay.
- Define and strictly mandate compliance to safety standards in different work environments with silica exposure.
- Incorporate silicosis diagnosis and management into the National TB program.

Research level

- Search for materials that can substitute for granite and reduce silica dust production.
- Affordable tools for hand-held drills and saws which will keep dust levels down (using water or vacuum suction).
- Cheaper ways to monitor air quality at different work sites having silica dust exposure risk.
- Low-cost respiratory protective equipment for use by the unorganized sector workers.
- Development of cheaper spirometers for use in the field.

Programmatic level

- Train staff at all levels about silicosis and the various occupations where this is a serious hazard.
- Periodic assessment and documentation of health of workers in the stone-cutting, mining and construction industries, for early onset of silicosis.
- Periodic assessment of conditions of work including dust control measures in place and their efficacy.
- Ensure diagnostic facilities for silicosis and COPD detection (CXR, PFT) are available at the CHC level for easier access.
- Spirometers from PFT to be made available even at PHC level for better access for patients.

References

[i] https://apps.who.int/iris/bitstream/handle/10665/271977/PMC5043201.pdf Silicosis and silicotuberculosis in India – WHO bulletin Bull World Health Organ 2016;94:777–778 | doi: http://dx.doi.org/10.2471/BLT.15.163550

[ii] https://www.downtoearth.org.in/news/governance/india-s-migrant-construction-workers-an-analysis-of-their-welfare-framework-79551

[iii] https://www.uptodate.com/contents/silicosis

[iv] https://emedicine.medscape.com/article/302027-overview

[v] Dharmendra Singh, Bidisa Sarkar, Kamalesh Sarkar, Elimination of tuberculosis requires prior control of silicosis including sub-radiological silicosis, Indian Journal of Tuberculosis, 2023, ISSN 0019-5707, https://doi.org/10.1016/j.ijtb.2023.05.003

[vi] Rupani, M.P. A mixed-methods study on impact of silicosis on tuberculosis treatment outcomes and need for TB-silicosis collaborative activities in India. *Sci Rep* **13**, 2785 (2023). https://doi.org/10.1038/s41598-023-30012-4

[vii] Nandi, S.S., Lambe, U.P., Sarkar, K. *et al.* A rapid point of care CC16 kit for screening of occupational silica dust exposed workers for early detection of silicosis/silico-tuberculosis. *Sci Rep* **11**, 23485 (2021). https://doi.org/10.1038/s41598-021-02392-y

[viii] https://synergist.aiha.org/202204-engineering-controls-silica

[ix] https://www.osha.gov/sites/default/files/publications/OSHA3681.pdf

[x] Dhatrak, Sarang & Nandi, Subroto. (2019). Assessment of silica dust exposure profile in relation to prevalence of silicosis among Indian sandstone mine workers: Need for review of standards. American Journal of Industrial Medicine. 63. 10.1002/ajim.23077

[xi] https://www.cdc.gov/coronavirus/2019-ncov/hcp/ppe-strategy/air-purifying-respirators-infographic.html

[xii] https://en.wikipedia.org/wiki/Respirator_assigned_protection_factors