# British Thoracic Society Clinical Statement on Silicosis

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# British Thoracic Society Clinical Statement on Silicosis

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## INTRODUCTION

The aim of this clinical statement is to provide a practical UK based approach for the recognition, diagnosis and management of silicosis in order to improve patient care. Clinically relevant aspects of prevention, risk assessment and health surveillance are also considered. This statement is based on a combination of available scientific evidence and, where this is lacking, consensus expert opinion.

The target readership of the statement is respiratory and general clinicians, and allied healthcare professionals, radiologists, pathologists, GPs and occupational health providers.

## METHODOLOGY

The Clinical Statement Group (CSG) was co-chaired by Dr Johanna Feary and Dr Jennifer Hoyle. Membership was drawn from clinicians working in specialist occupational lung disease services across the UK by application through the BTS. After identifying the key areas to address, individual sections of the Clinical Statement were drafted by group members. The co-Chairs compiled these into a single draft which was then commented on and revised by the CSG members. A final edited draft was reviewed by the BTS Standards of Care Committee before posting for public consultation and peer review on the BTS website.

## SUMMARY OF CLINICAL PRACTICE POINTS

**Section 1 – background**

- Respiratory health care professionals should be aware of common jobs where silica may be encountered
- Silicosis occurs in chronic, accelerated and acute forms depending on exposure history and can have different radiological appearances
- Silicosis can occur with short term higher intensity exposure
- Silicosis may present to non-respiratory specialialities including rheumatology, nephrology, infectious diseases and general practice
- Artificial stone workers are at risk of accelerated silicosis

**Section 2 - diagnosis**

- Silicosis can usually be diagnosed based on history of exposure and compatible radiology
- Misdiagnosis as sarcoidosis or TB is common due to overlapping features e.g. elevated serum ACE, lymphopenia
- Small biopsies (e.g. from EBUS) may show granulomas but are unlikely to show silicotic nodules and birefringent crystals
- Patients should be asked if coworkers have silicosis

**Section 3 - comorbidities**

- Silicosis and silica exposure are associated with increased risk of lung cancer, TB, renal disease and rheumatoid, connective tissue disease (CTD) and systemic vasculitis

- Diagnoses of these comorbidities can be complicated due to overlapping clinical and radiological features with silicosis

**Section 4 – management**

- All patients should be referred to, or discussed with, an occupational lung disease specialist where possible
- Silicosis is incurable and treatment is supportive
- In patients with silicosis, the aim is to prevent, or minimise, further exposure to silica where possible
- There is currently no established evidence to support the use of immunosuppression or antifibrotics in silicosis
- Patients should be counselled about smoking cessation
- Patients should be screened for comorbidities
- Patients with silicosis should be provided with written information confirming their diagnosis, the implications this has on their current and future jobs, and information about Industrial Injuries Disablement Benefit (IIDB) and civil compensation

**Section 5 – progression and prognosis**

- The prognosis of silicosis depends on cumulative silica exposure, disease severity, rate of progression, and host factors such as smoking
- Disease progression can occur despite removal from exposure
- In general, mortality is higher in those with comorbidities including TB, systemic sclerosis and lung cancer.

**Section 6 - Prevention**

- Prevention is key through employers and employees using exposure controls
- Health surveillance offers the opportunity to detect early disease

## SECTION 1: BACKGROUND

Silicosis is a preventable form of pneumoconiosis caused by the inhalation of respirable crystalline silica (RCS) – hereafter referred to as silica - which exists in several natural crystalline forms including quartz, cristobalite, and tridymite (1). Silica in the form of quartz is found in substantial quantities in sand, sandstone, granite, clay, shale, slate, and some concretes and mortar (table 1). Cristobalite and tridymite are used in the ceramic, refractory, and diatomaceous earth industries. Silica also occurs in both natural and synthetic amorphous (e.g. silica gel) forms which are not considered to cause silicosis and will not be discussed further.

Silicosis has been described for centuries. Today, workers employed in a range of occupations remain at risk of developing silicosis (see box 1); these include traditional industries such as masonry and mining, and outbreaks in newer, industries like denim sandblasting (2) and fabricating engineered or artificial stone (AS) worktops (3) .

**Box 1: Example occupations associated with silica exposure**

| Category | Jobs titles patients may report |
|---|---|
| **Stone cutting and masonry** | stonemason, stone cutter/dresser, engraver, saw or Computer Numerical Control (CNC) operator, labourer, cleaner. |
| **Artificial stone and worktop fabrication** | worktop fabricator, kitchen or bathroom worktop fitter, CNC operator, workshop labourer, cleaner. |
| **Construction, demolition and civil works** | builder, construction worker, labourer, demolition worker, road worker, landscaper, sandblaster, cleaner. |
| **Bricks, ceramics and potteries** | brickworker, pottery worker/potter, kiln operator, plant operative, brick cutter, glazing or finishing worker, cleaner. |
| **Dental, jewellery and precision material manufacturing** | dental technician, dental laboratory worker, jeweller, gemstone or diamond grinder/polisher, stamper/etcher, factory operative |
| **Mining and tunnelling** | miner, tunneller, driller, shot blaster, labourer, machine operator |
| **Quarrying and stone extraction** | quarry worker, driller, machine operator or dumper. |
| **Metal casting and foundries** | foundry worker, metal worker, moulder, core maker, fettler, grinder, welder, furnace operative |

Footnote: Occupations are listed in order of the most frequently reported jobs and industries from Surveillance of Work-related Occupational Respiratory Disease (SWORD) data between 2018 and 2025(4)

Silicosis can be indistinguishable radiologically from sarcoidosis, chronic beryllium disease and tuberculosis (TB). In the absence of an appropriate occupational history, the risk of misdiagnosis and delayed diagnosis is increased (5).

Silicosis is often progressive, despite cessation of silica exposure (6) and there is no effective treatment. Furthermore, the diagnosis may be career ending for the worker, and the impact of the diagnosis extends beyond the health of the individual to include wider social and economic consequences. All patients with possible, suspected or confirmed silicosis should be referred to, or discussed with, an occupational lung disease specialist.

## Epidemiology

The number of people in the UK currently exposed to silica is unknown but is likely to be more than 500,000 (7). Similarly, the UK prevalence of silicosis is unknown for many reasons which may include (i) asymptomatic early disease (ii) not seeking medical attention for symptoms (iii) misdiagnosis and (iv) under-reporting of cases to the UK voluntary reporting scheme (SWORD)(8). Since 2024, cases of silicosis in artificial stone workers are increasingly recognised in line with global trends(9).

## Risk of disease

The risk of developing silicosis relates to the cumulative exposure (duration (years) x intensity ($mg/m^3$)) to respirable crystalline silica. Particles must be small enough to reach the distal

airways and alveoli (<10 micrometers) to cause disease. The proportion of crystalline silica varies considerably depending on the type of stone or material used (table 1).

**Table 1: Amounts of silica in different types of stone**

| Type of stone | Percentage of silica |
|---|---|
| artificial / engineered stone | up to 95% |
| sandstone, gritstone, quartzite | more than 70% |
| concrete, mortar | 25% to 70% |
| shale | 40% to 60% |
| China stone | up to 50% |
| slate | up to 40% |
| brick | up to 30% |
| granite | up to 30% |
| ironstone | up to 15% |
| basalt, dolerite | up to 5% |
| limestone, chalk, marble | up to 2% (but these can contain silica layers) |

**Adapted from:** Silicosis – causes and risk controls - HSE (10)

In the UK, under Health and Safety Law, the workplace exposure limit (WEL) to respirable crystalline silica over an 8-hour work period is currently 0.1 mg/m$^3$ (10). This amount may not be visible to the naked eye, and even at this level of exposure there is a residual risk (estimated to be ~30%) of silicosis occurring over a 40 years working lifetime (11). Many workers may be regularly exposed to levels above this.

**Definitions**

The different forms of silicosis are categorised based on duration of exposure to silica and these vary in both in their clinical picture and radiological appearances(12) (table 2). The distinction between these different forms is not always apparent and conventional definitions may need revision in the future. Historically, the International Labour Office (ILO) classification was used to provide standardised method of reporting silicosis and other pneumoconiosis on chest x-rays. This was primarily used for research and ILO classification is not currently used in routine clinical practice in the UK (13) (figure 1).

**Table 2: Comparison of different forms of silicosis(14, 15) (adapted from Barnes et al Respirology 2019)**.

| Disease | Exposure intensity and duration | Clinical and lung function test findings | Radiological findings |
|---|---|---|---|
| Simple chronic | Lower intensity exposure; > 10 years | Asymptomatic in early stages. Generally no lung function defects. | Round or irregular discrete nodules < 1cm, upper lobe predominant +/- calcification, +/- hilar adenopathy or calcification |
| Complicated chronic or Progressive Massive Fibrosis (PMF) | Variable intensity of exposure; > 10 years | Can be asymptomatic but often chronic cough, dyspnoea.<br><br>Obstructive, restrictive or mixed lung function defect<br>Reduced TLCO and TLC | Conglomerate upper lobe masses >1cm, usually (not universally) bilateral retracting to hilum and hilar adenopathy +/- calcification. Typically symmetrical.<br>Emphysema develops near nodules (centrilobular or para-cicatricial). |
| Accelerated<br>May include features of simple, complicated, or acute disease | High intensity exposure; 2-10 years | Can be asymptomatic but often dyspnoea, cough, respiratory failure.<br><br>Obstructive, restrictive or mixed lung function defect<br>Reduced TLCO and TLC | Bilateral hilar adenopathy, +/- symmetrical +/- calcified. Rapidly progressive nodules and PMF features +/- ground glass change, +/- surrounding emphysema |
| Acute silico-proteinosis | High intensity exposure; generally < 2 years | Rapid onset of dyspnoea, cough and respiratory failure<br><br>Obstructive, restrictive or mixed lung function defect<br>Reduced TLCO and TLC | Bilateral perihilar consolidation, ground glass changes, +/- 'crazy paving' appearances from co-existing interlobular thickening +/- centrilobular nodules. |

**Key:** Progressive Massive Fibrosis (PMF); TLC: Total Lung Capacity; TLCO: Transfer factor of the Lung for Carbon Monoxide

*Chronic silicosis comprises simple silicosis and complicated silicosis (or Progressive Massive Fibrosis (PMF)* if radiographic changes develop after 10 years or more of silica exposure. Simple

chronic silicosis is characterised by the presence of nodules <1cm diameter and in complicated silicosis nodules are >1cm diameter. Initally lung function may be normal thus radiographic changes may pre-date the onset of symptoms, but with progression, symptoms and lung function changes may develop.

*Accelerated silicosis,* develops generally after months or up to 10 years of exposure. Radiological appearances are similar to those of chronic silicosis but in addition ground glass changes may be present.

Both chronic and accelerated silicosis are associated with hilar and/or mediastinal lymphadenopathy +/- calcification. Mediastinal and hilar lymphadenopathy can also occur in silica-exposed individuals in the absence of parenchymal lung changes. The consensus view of the CSG is that the term 'silica associated lymphadenopathy (SAL)' should be used for such cases where alternative causes have been ruled out, and that these cases should be followed up and future research directed to understanding the natural history of this condition. In particular this should be seen as an opportunity to consider and correct potential silica exposure levels in the workplace.

*Acute silicosis*, also referred to as silico-proteinosis, occurs generally after weeks or months of very high intensity silica exposure. The onset of symtoms may be the presenting feature; radiographic changes tend to lack the nodular appearances seen in chronic disease and are characterised by alveolar-proteinosis type changes, largely based on case reports alone.

**Mechanisms of injury.**

Silica is known to be highly cytotoxic to the human lung, yet the full mechanisms of lung injury are still not completely understood.

*Chronic and accelerated silicosis*

Silica particles in the lung are ingested by alveolar macrophages, however macrophages cannot digest the silica particles and thus cannot clear them from the lung. In response reactive oxygen species are released that damage macrophage lysosomes and activate the inflammasome. This causes cytokine and chemokine production and cell apoptosis/pyroptosis resulting in acute and chronic inflammation, lung fibrosis and the activation of autoimmunity (16).

*Acute silicosis*

When silica particles reach the lung in high quantities, alveolar macrophages release cytokines and reactive oxygen species which stimulates surfactant production as a protective mechanism. Surfactant is naturally broken down by local enzymes, which subsequently triggers more surfactant production and accumulation of denatured protein. This leads to a build-up of excessive proteinaceous debris in the alveoli (16) .

## SECTION 2: DIAGNOSIS

The diagnosis of silicosis is based on a history of appropriate exposure to silica and radiological (normally CT) appearances compatible with silicosis, in the absence of a more likely diagnosis. Occupational history plays a key role in making a diagnosis in silicosis. Clinicians should have a low index of suspicion of silicosis in patients who have worked in relevant jobs (box 1) with

radiological changes and should discuss or refer to a occupational lung disease specialist. Additional time is required to take a full occupational exposure history; one-hour appointments are the recommended standard for new patient consultations in specialist clinics (17).

**Clinical features and investigations**

Early simple silicosis is often asymptomatic. With disease progression, patients may develop non-specific respiratory symptoms including cough, wheeze, chest tightness and exertional breathlessness. Such symptoms may also be due to development of associated comorbidities (see section 4). Fever, weight loss or haemoptysis should prompt consideration of alternative or co-existing diagnoses, particularly TB and lung malignancy. Previous diagnoses, or symptoms or signs of rheumatoid, connective tissue disease (CTD) and systemic vasculitis, and assessment of co- or pre-existing renal disease should be sought (see box 3).

In the context of an appropriate occupational exposure history, typical radiological features are usually sufficient to support a diagnosis of silicosis. However, these appearances can mimic those of sarcoidosis, chronic beryllium disease, TB and lung cancer and so clinicians should specifically enquire about occupation and possible exposures in all patients with suggestive radiological changes. (see boxes 1 and 2) CT changes may be less typical where there is co-exposure to other dust (e.g. construction workers co-exposed to asbestos and coal workers co-exposed to coal dust) as recognised in mixed dust pneumoconiosis (5, 18, 19). [see appendix 1] Patients presenting with non-specific respiratory symptoms and an abnormal chest x-ray may have been referred through alternative diagnostic pathways, e.g. lung cancer or ILD, and interpretation of imaging in the absence of a detailed occupational exposure history increases the risk of misdiagnosis.

Sarcoidosis is a multisystem granulomatous disease of unknown aetiology (20) which can present with similar clinical, radiological and histopathological features as seen in silicosis, and misdiagnosis between them is well recognised (5). Whether sarcoidosis develops as a consequence of silica exposure in susceptible individuals is not clear. A clinician should consider the diagnosis of silicosis where there is a history of silica exposure in a patient who presents with features consistent with sarcoidosis and should seek specialist occupational lung disease specialist input.

Silicosis can develop many years after removal from exposure so details of historical, and not just current occupations, must be sought. A short list of key questions will be sufficient in most cases to identify those with suspected disease (see box 2). The use of respiratory protective equipment should not be assumed to confer adequate protection, as it represents the least effective level of exposure control. Baseline clinical, radiological, imaging and serological assessment of patients with suspected silicosis is suggested in box 3. Laboratory findings may include lymphopenia and elevated serum angiotensin-converting enzyme levels making them unreliable tests to differentiate silicosis and sarcoidosis. Up to 25% of individuals may have positive autoantibodies, without a consistent serological pattern and with or without clinically apparent autoimmune disease. Positron Emission Tomography (PET)-CT is not routinely recommended. Lymphadenopathy due to silica and silicotic nodules can demonstrate increased metabolic uptake. This also makes the diagnosis of coexisting lung cancer challenging (21).

Echocardiography and CT pulmonary angiography are appropriate where there is compression of the pulmonary arteries by silicotic masses and/or suspicion of pulmonary hypertension.

**Box 2: Key questions to ask in an occupational history for a patient with possible silicosis**

Current or historical exposure in a high risk job (box 1) and duration of exposure (years)

Types of substance worked with e.g. stone/artificial stone/ 'quartz'/sand

Whether there was visible dust on work surfaces

Use of respiratory protective equipment and its type and if they have a beard

Whether co-workers in current or previous jobs have respiratory illnesses (if known)

Whether they have had occupational health provision and if this included surveillance (chest x-ray and / or breathing tests organised through work) and if so, what this showed

**Box 3: Recommended baseline assessment of patients with suspected silicosis**

| | |
|---|---|
| **Occupational history** | See boxes 1 and 2 |
| **Respiratory symptoms** | Cough, sputum production, wheeze, dyspnoea, haemoptysis, chest infections |
| **Other symptoms** | Weight loss, fever, night sweats, TB contact/history of TB,<br>Raynaud's, sicca symptoms, rash, arthralgia, swollen joints muscle weakness, swallowing problems, hair loss |
| **Smoking history** | |
| **Clinical examination** | Clinical signs uncommon; examine for features of Rheumatoid, CTD and vasculitis. |
| **Imaging** | All patients should have a CT scan, preferably high resolution CT |
| **Blood tests** | FBC, renal and liver function, calcium, CRP, immunoglobulins<br>Serum ACE, Rheumatoid Factor, anti-CCP antibodies, CTD screen, ANCA, IGRA<br>Consider HIV test if appropriate |
| **Urinalysis** | Urine dipstick; if microscopic haematuria or proteinuria present, urine protein-creatinine ratio |
| **Sputum (if productive cough)** | For Microscopy, Culture and Sensitivities and AFB |

**Key:** ACE: Angiontensin Converting Enzyme; AFB: Acid Fast Bacilli; ANCA: Anti-Neutrophil Cytoplasmic Antibody; CCP: cyclic citrullinated peptide; CRP: C-Reactive Protein; CT: Computed Tomograph; CTD: Connective Tissue Disease; FBC: Full Blood Count; HIV: Humman Immunodeficiency Virus; IGRA: Interferon Gamma Release Assay; TLC: Total Lung Capacity; TLCO: Transfer factor of the Lung for Carbon Monoxide

**Histopathology**

Histology is generally not required in the context of typical exposure history and radiology. Bronchoscopy with lavage, endobronchial ultrasound (EBUS) biopsy or cryobiopsy may be considered where there is diagnostic uncertainty or to exclude malignant disease although

none are considered gold standards for diagnosis of silicosis. Biopsy samples are often too small to identify silicotic nodules and foreign body granulomas (as seen in silicosis) may be misinterpreted as other granulomatous diseases or pulmonary manifestations of autoimmune conditions. Pathologists should be asked to use polarised light microscopy to visualise birefringent silica particles.

Although chronic silicosis is primarily a fibrotic or nodular lung disease, granulomatous reactions around silica particles are frequently described (22). This leads to development of silicotic nodules consisting of central, circumscribed, hyalinized, and whorled collagen surrounded by chronic inflammation and fibrosis. Complicated silicosis, is typified by coalescence or growth of these individual nodules.

Acute silicosis is similar in histologic appearance to primary pulmonary alveolar proteinosis and is characterised by airspaces filled with eosinophilic, granular, lipoproteinaceous material admixed with foamy macrophages. Interstitial fibrotic changes are usually minimal (19).

## SECTION 3: COMORBITIES

### Airflow obstruction and airways disease

Silica exposure is associated with excessive FEV1 decline, airflow obstruction and emphysema. These associations are influenced by multiple factors including smoking prevalence which is often higher among silica-exposed populations than in the general populations (23). The negative effect of smoking on lung function decline is at least additive to that of silica dust (24, 25).

Despite confounders such as occupational co-exposures e.g. coal dust (26), the available evidence supports that silica exposure causes loss of $FEV_1$ and FVC, in a dose-dependent manner (27); this may occur without radiological evidence of silicosis (27, 28) .

Asthma is also reported in silica exposed workers and there may be evidence of a work-related variability in peak flows (27-30).

### Lung cancer

Silica is a Group 1 carcinogen (31) and there is a dose-response relationship between silica exposure and lung cancer (32, 33). The relative risk of developing lung cancer is doubled in the presence of silicosis independent of smoking history. There are no specific lung cancer subtypes linked to silica exposure. Diagnosis and staging of lung cancer may be more difficult due to the presence of complicated silicosis, lymphadenopathy and avidity of silicotic nodules or lymph nodes on PET scan. Treatment for lung cancer from silica exposure does not differ from standard therapy.

### Connective Tissue Disease

There is a well described association between silica exposure and rheumatoid arthritis, connective tissue diseases, especially systemic sclerosis (known as Erasmus syndrome), systemic lupus erthyromatosis and vasculitis. These diseases may occur without radiological changes of silicosis (32, 34, 35). Among 1238 workers from the artificial stone industry without silicosis almost one quarter (24.6%) had detectable anti-nuclear antibodies (ANA) compared to

6% in a comparison cohort. 4.6% had a detectable extractable nuclear antigen (ENA) and 2.6% were positive for rheumatoid factor (36).

**Tuberculosis**

The risk of pulmonary and extrapulmonary TB, and non-tuberculous mycobacteria (NTM) infection, is increased in silica-exposed individuals and those with silicosis (37, 38). This is thought to occur through impairment of macrophage function by silica. The risk of pulmonary TB in silicosis is estimated to be increased four-fold (39). TB and NTM infection are also more common in acute and accelerated silicosis (37, 40), in people with previous TB and/or HIV and, for NTM, in structural lung disease.

**Renal disease**

Silica exposure has been associated with renal disease, including focal, crescentic, necrotising and rapidly progressive glomerulonephritis (41-44) which may occur through autoimmune antibody formation (45).

## SECTION 4: MANAGEMENT

There is currently no evidence based disease-modifying treatment for silicosis, however smoking cessation and removal from exposure may improve disease trajectory. A holistic, patient-centred approach should be used. The patient should be advised that there is a dose-response between silica exposure and disease severity, cessation of further silica exposure, management of symptoms, and prevention and treatment of complications should be discussed. Management also includes advice regarding specific government benefits, medicolegal compensation and any potential employment risks to the worker when considering cessation of exposure.

**Disease modifying therapy**

*Immunosuppression*

Oral corticosteroids and/or other immunosuppressive therapies have not been evaluated in large trials of clinical effectiveness in silicosis and currently have no proven role in its long-term treatment.

*Antifibrotics*

Anti-fibrotic drugs, efficacious in other ILDs, are under investigation but, to date, there have been no conclusive appropriately powered studies demonstrating benefit in silicosis although studies are underway (46, 47).

*Whole lung lavage*

Whole lung lavage (WLL) has been used in patients with acute silico-proteinosis and accelerated silicosis, and may transiently improve oxygenation, symptoms, and radiological findings but there is no long-term data on outcomes (48-50). Optimal timing of and which patients might benefit from WLL is unclear.

**Lung transplantation**

Lung transplantation may be an option for some patients with advanced disease and declining lung function. Those with accelerated or acute disease are likely to progress rapidly and suitable patients should be referred early for assessment. Whilst the surgery can be more technically challenging with longer operative times, outcomes from transplant for silicosis have similar survival rates to those with non-silicosis ILD (51, 52) and the general lung transplant population (53, 54).

**Supportive management**

There is a high prevalence of current (and ex-smoking) in silica-exposed workers (55, 56) and smoking increases the risk of both silicosis and co-morbidities (57, 58). All current smokers should be strongly supported to stop smoking at every opportunity (59).

As with other ILDs, symptom management may include pulmonary rehabilitation programmes which can help improve breathlessness (60). Involvement of the multidisciplinary team includes clinical nurse specialists, physiotherapy, palliative care services, dieticians, oxygen assessment, and signposting for psychological support as appropriate. Support for self-care techniques (5) including education about staying active and maintaining supportive social networks are recommended. Regular vaccinations should be offered for pneumonia, influenza and COVID-19 and respiratory syncytial virus in keeping with national guidance (61).

**Monitoring**

Each clinical review should include assessment of symptoms as in section 2. In addition, patients should be assessed for complications and comorbidities of silica exposure including lung cancer, TB, autoimmune rheumatological diseases, airflow obstruction and renal disease. New or worsening respiratory or systemic symptoms such as weight loss, haemoptysis or change in cough and finger clubbing in patients with silicosis should prompt screening for mycobacterial infection (62) and/or lung cancer.

Clinically, distinguishing silicosis from TB and NTM infection can be challenging because of overlapping clinical features (appendix 1). All patients with silicosis should be screened for active TB, particularly where immunosuppression is considered e.g. for co-existing CTD. More invasive screening may be considered if clinical concern is high. If screening for active disease is negative, a host response test, such as TST or IGRA, should be used to guide preventative treatment as per NICE guidance (63). Diagnosis and treatment of NTM and TB (active and latent disease) should be in line with diagnostic and treatment algorithms (64).

All patients with silicosis should be seen by, or discussed with, an occupational lung disease specialist. Patients should be followed up annually as a minimum with review of symptoms, imaging (CT or CXR) and measurement of spirometry and gas transfer. Frequency of follow up will vary depending on if they remain exposed to silica and the severity of disease and rate of progression. Three to six month follow up may be needed initially and a review system put in place (e.g. patient initiated follow up) in case of unexpected clinical deterioration. Shared care with general respiratory or ILD clinics for follow up appointments may be appropriate.

## Financial and legal considerations

The financial impact of a diagnosis of silicosis should not be underestimated. Many patients may face redeployment to a less skilled job with lower pay or may need to take early retirement. For example, stonemasons are highly skilled craftspeople and options for work in a non-silica exposed role are limited. Increased costs of lifestyle changes demanded by a chronic disease, including travel to frequent hospital appointments can put significant financial strain on patients (65).

In the UK, various financial support systems are available, and clinicians should signpost patients promptly to services which can provide financial advice, for example, Citizens Advice Bureau and local patient support groups (see Appendix 5).

### Benefits and compensation advice

In the UK, patients with silicosis are eligible to make a claim for IIDB(66). This is a statutory benfit payable, without consideration of fault, to employed earners who have acquired a 'prescribed' workplace disease such as silicosis. Claims can be made by post or on-line and are followed by a face to face assessment by a doctor who will confirm (or otherwise) the diagnosis. The IIDB form can be downloaded from the IIDB Government website and submitted by post or requested by telephone (https://www.gov.uk/industrial-injuries-disablement-benefit/how-to-claim). If lung cancer or TB is diagnosed with coexisting silicosis then additional claims for IIDB can be submitted. If in doubt, a patient should apply as the Department for Work and Pensions will advise on eligibility.

Patients may also be eligible for other benefits including disability benefits and should be signposted appropriately to resources including the Citizens Advice Bureau, online calculators, or in some cases a social worker (see appendix 5).

Patients with silicosis may want to consider a legal claim against their employer for a "personal injury". Patients should seek advice from a solicitor with experience of industrial disease and/or their Trade Union representative. Clinicians should explain that claims for personal injury are subjected to the statute of limitations and must be submitted within 3 years of knowing that they have silicosis. Clinicians should also document giving this advice at diagnosis and provide a written copy to the patient.

## Communications with patients, their carers and other health care professionals

All patient communications should be clear, accurate and easy-to-understand. This includes written information on diagnosis, disease progression, tests/investigations, discussions on treatment and follow ups and on financial and medicolegal aspects. Consultations should be facilitated with a translator if necessary.

Patient resources, including factsheets (see appendix 6), pictures and videos can help communicate complex healthcare information for patients in a comprehensible way (67). Materials should be provided in different languages to ensure equitable access to healthcare information. Patients should also be provided with advice on the consequences of ongoing exposure including implications on employment, and information about benefits and compensation (17).

Patients with silicosis may be from disadvantaged and/or immigrant communities and are at increased risk of social isolation (68). The importance of positive social networks (including relatives) and the broader community should be emphasised for anyone living with silicosis.

**Reporting**

There is currently no legal requirement for companies whose employees develop silicosis to report themselves to the Health and Safety Executive (HSE) via the national Reporting of Injuries, Diseases and Dangerous Occurrences Regulations 2013 (RIDDOR)(69) at the time of writing.

Any worker can make a complaint to their local HSE office about any aspect of their workplace that they feel may put them at risk of ill health or a safety issue. Workers may wish to relay their diagnosis to their employer to help with workplace investigations, reasonable adjustments and risk assessments. All communication in this context can only be done with the patients' explicit consent which should be written and kept as part of the patient record. The patient should be asked if they want to review any such communications before they are sent. This support should be given freely as part of the NHS service.

Surveillance of Work-related and Occupational Respiratory Disease (SWORD) is a voluntary reporting scheme for chest physicians and is the only national surveillance scheme for occupational lung disease in the UK (8). It provides valuable insights into case numbers, demographics, occupational and industrial groups and distribution of silicosis, as well as informing policy and regulatory responses. We recommend that all cases of silicosis should be reported to SWORD.

Healthcare professionals should explain to patients and their carers that any case of silicosis will need to be reported to the coroner as part of mandatory reporting of death. This explanation is particularly important where prognosis is poor and future death is likely to be related in any way to their diagnosis or associated complications.

## SECTION 5: PROGRESSION AND PROGNOSIS

The prognosis of silicosis varies, depending on cumulative silica exposure (70), disease severity at the time of diagnosis, rate of progression, and host factors such as smoking history. Individual disease trajectories will vary between long periods of stability and little deterioration versus progressive fibrotic lung disease with respiratory failure and death The risk of radiological progression and lung function decline is higher in those who remain exposed for longer, or who present with more advanced disease. Disease progression may occur and continue for years after exposure cessation (6, 71).

In simple silicosis the prognosis is generally more favourable; one large study from China reported a median survival of 27 years for those with early disease at diagnosis (72) .

Prognosis is particularly poor in workers with high exposures and accelerated disease such as in AS silicosis. A US cohort of AS silicosis reported a mortality of 19% (73). In AS silicosis, annual declines in $FEV_1$ and FVC range from 83-625 mL and 87-587 mL respectively over 12-48 months,

despite most patients having ceased exposure (3, 74). Radiological progression correlates closely with lung function decline (3, 75, 76).

Poor prognostic factors include co-existant pulmonary hypertension (58), active mycobacterial infection (76-78) and lung cancer (79). In systemic sclerosis, having silicosis confers a higher mortality than in those without silicosis (80).

## SECTION 6: PREVENTION

Workers have a legal right to be protected from hazard and risk in their workplace. The responsibility for this lies with employers under the Health and Safety at Work Act (81) and Control of Substances Hazardous to Health Regulations (COSHH) 2002 Regulations (82) (see appendix 3). Risk assessments should be carried out to identify any potential sources of silica exposure, decide how to prevent or minimise the risk of silicosis in their workforce, and consider whether health surveillance is required.

### Primary prevention

Primary prevention in silicosis relates to taking measures to reduce or prevent workers inhaling silica, following a workplace risk assessment (83). Various approaches within the hierarchy of control (figure 2) are usually combined to reduce exposure to silica at work (84)(appendix 2).

Factors to be considered include the silica content of the materials being used, the type of work being carried out, as well as the frequency and duration of each work task. The most effective measures should be used to reduce exposure, wherever practical.

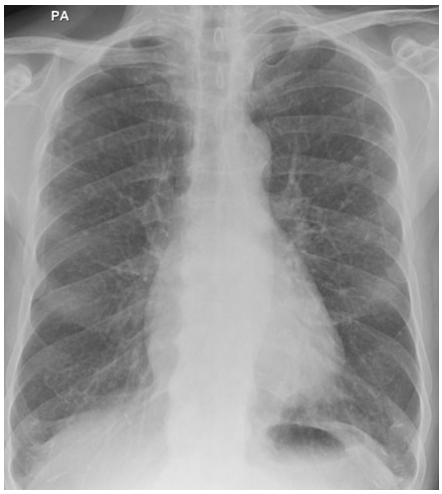
### Secondary prevention

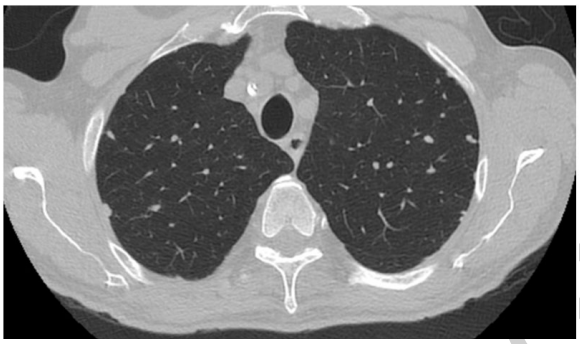
There is currently no internationally agreed strategy for screening, case finding, or health surveillance for silicosis. Secondary prevention for silica exposed workers relates to using health surveillance to detect early stages of disease.

In the UK, health surveillance is defined as a series of repeat health checks designed to identify ill health caused by work and the duty to perform health surveillance is held by the employer. Current HSE guidance requires that health surveillance is carried out for workers at risk of silicosis (85). Workers reporting respiratory symptoms, those found to have excessive lung function decline, and those with abnormal CXR findings at health surveillance should be referred to an appropriate occupational health professional and/or occupational lung disease specialist. It should not be assumed that there is universal access to health surveillance; furthermore, CXRs will miss early cases of disease (86).

**Figure 1: Radiological imaging of silicosis**

Figure 1a  Simple silicosis (CXR and CT) with small nodules (<1cm diameter))



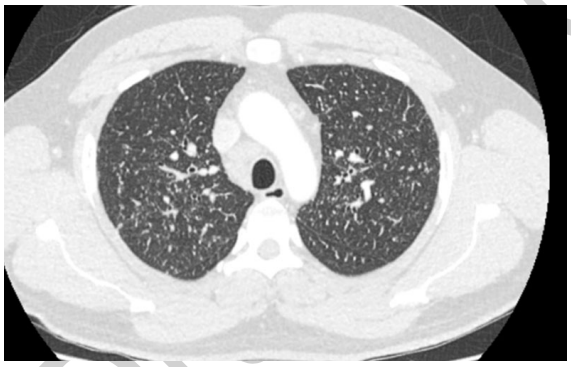
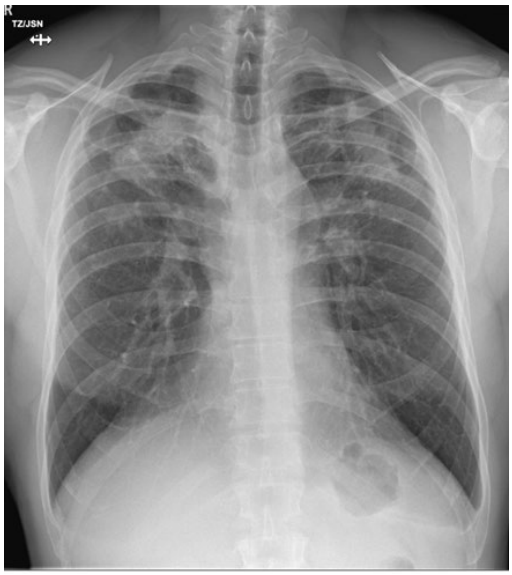
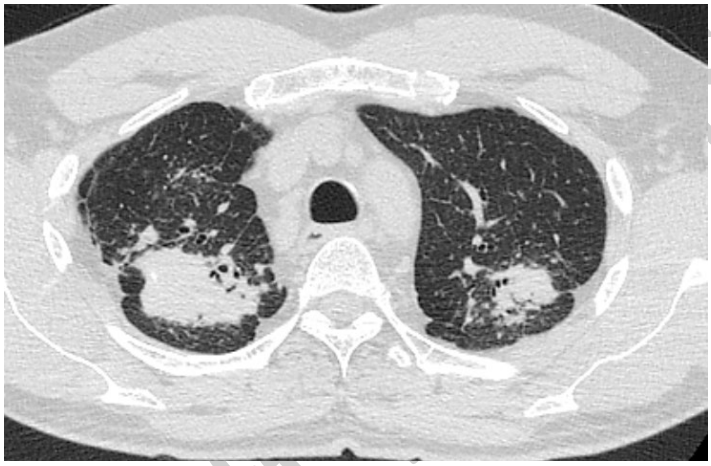


Figure 1b Complicated silicosis (CXR and CT (lung and mediastinal windows)) with bilateral symmetrical conglomerate masses (>1cm diameter) in the upper zones, para-cicatricial emphysema and calcified hilar lymphadenopathy



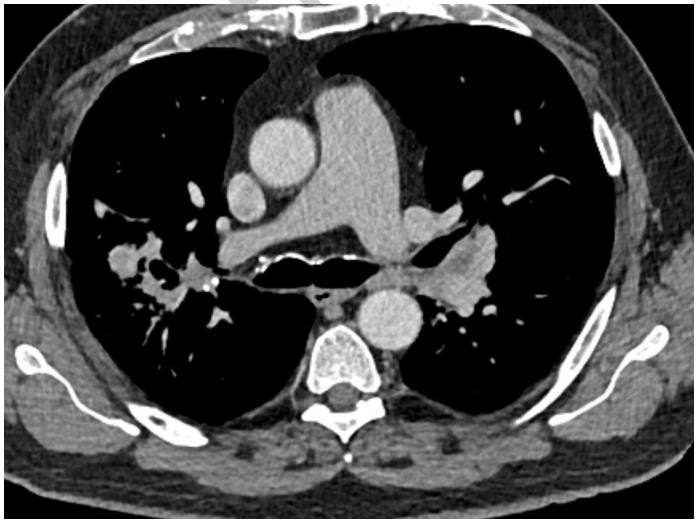
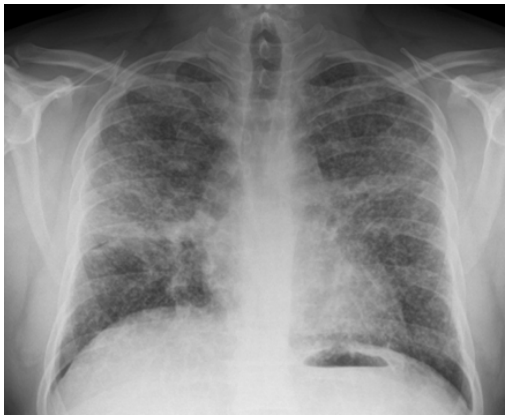
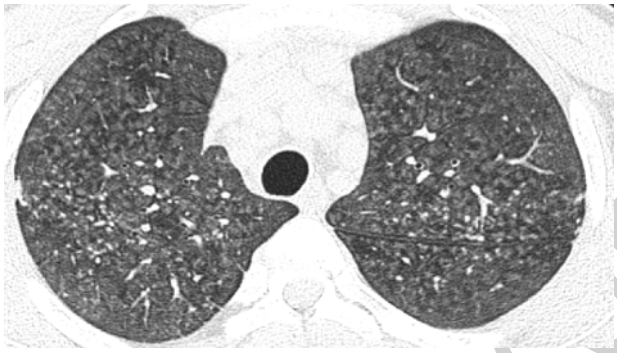
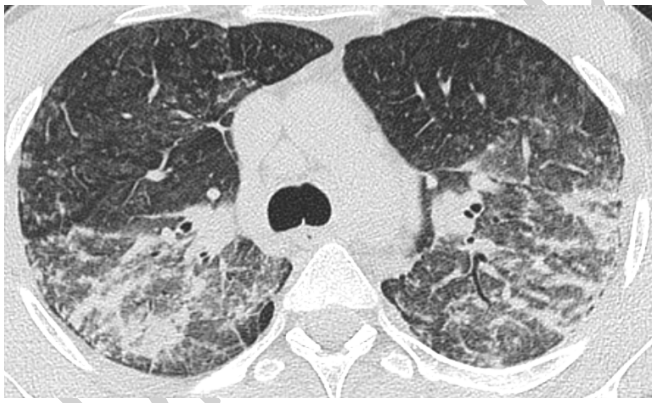


Figure 1c Accelerated silicosis (CXR and CT) with a range of different appearances including bilateral symmetrical soft centri-lobular and some hyperdense small nodules (<1cm diameter) and conglomerate nodules (>1cm diameter). Subpleural nodules and beading along the fissures also seen.







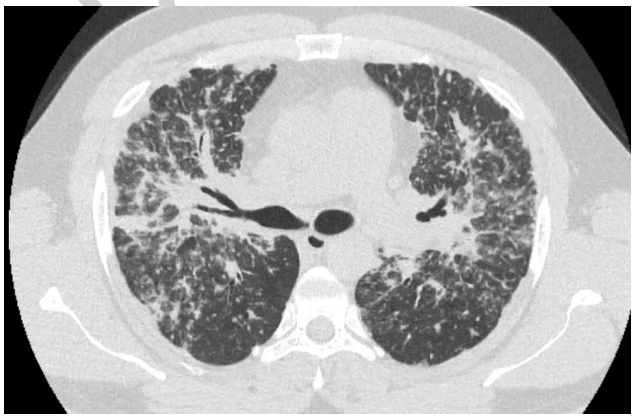
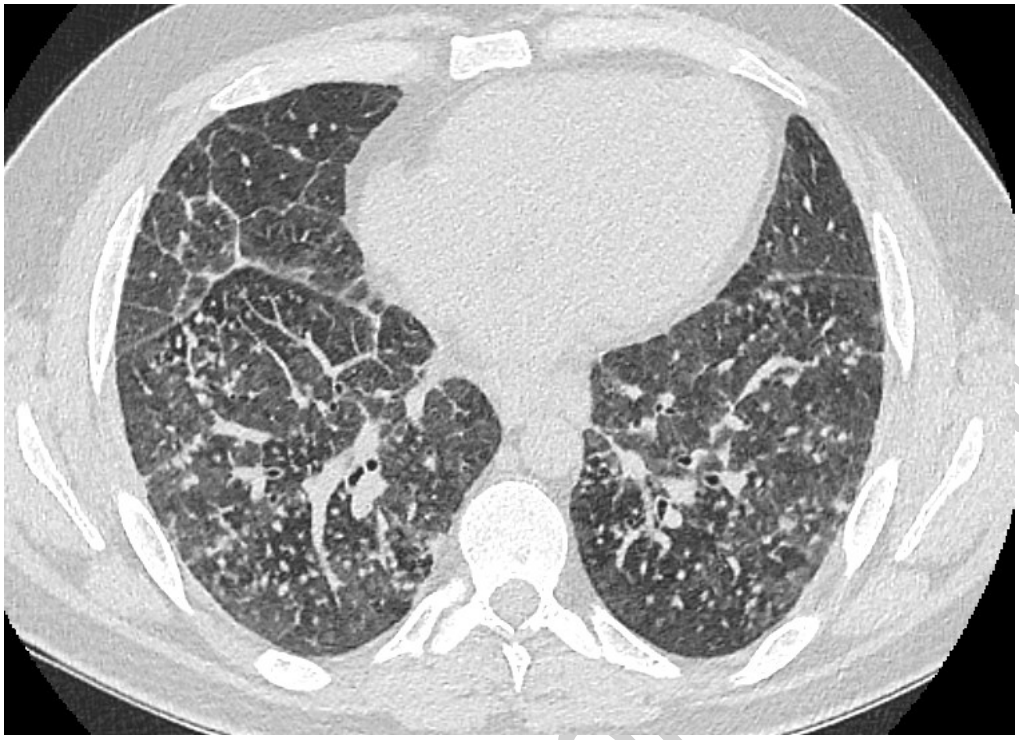
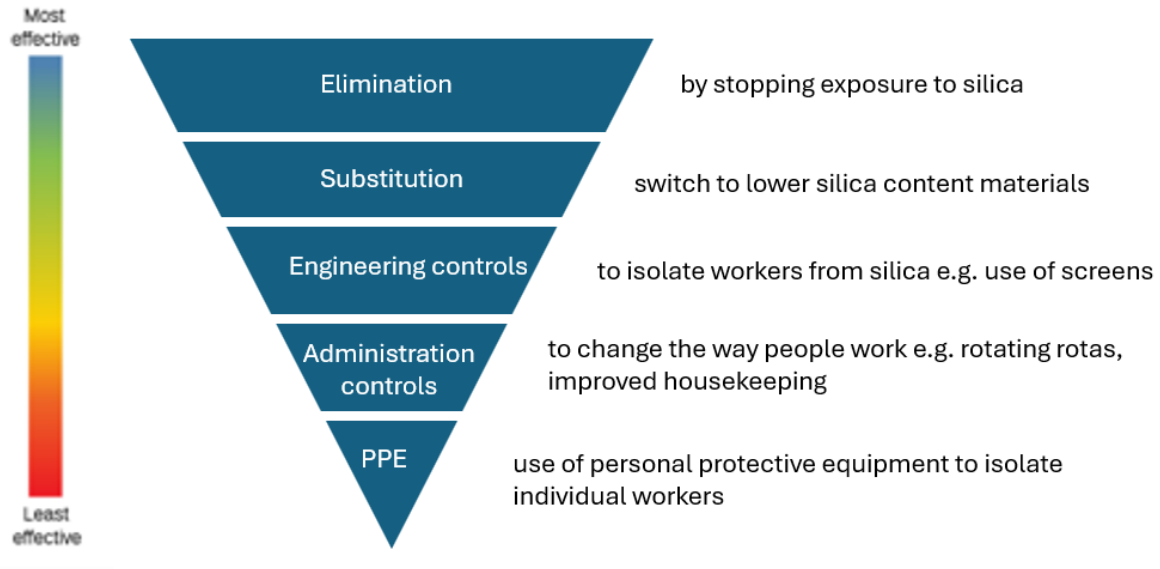


Figure 1d Acute silicoproteinosis (CT) with interlobular thickening on a background of accelerated silicosis



582 **Figure 2: Hierarchy of Control of exposure for silica**

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## APPENDIX 1: MISDIAGNOSIS OF SILICOSIS

Misdiagnosis of silicosis is common and occurs due to several factors, the overlap in diagnostic tests is explained in this table.

**Table: comparison of lab and radiology appearances of silicosis, sarcoidosis, lung cancer and TB**

| Test results | Silicosis | Sarcoidosis | Lung cancer | TB |
|---|---|---|---|---|
| **BLOOD TESTS** | | | | |
| **Peripheral lymphopenia** | Possible (87) | Possible (88) | Possible (89) | Possible (90) |
| **Raised blood calcium** | Unlikely | Incidence varies widely (91) | Most likely | Possible |
| **Raised serum ACE** | Possible (92) | Possible (92) | Unlikely but can be raised in Hodgkins lymphoma | Possible (92) |
| **Positive ANA/connective tissue screen** | Most likely and above background population levels(87) | Background population level 15%-19.5% positive depending on gender, age and cut off level. (93) | | |
| **Interferon-Gamma Release Assay (IGRA) (quantiferon)** | Negative, but may have co-existing TB | Negative | Negative | Positive (but false negatives possible) |
| **CT FEATURES** | | | | |
| **Hilar Lymphadenopathy** | Eggshell calcification of mediastinal/hilar nodes (3-6%) but more commonly non-calcified adenopathy (94). Can be present in early disease without lung nodules. | Hilar adenopathy common, can present with calcified adenopathy (5%) but eggshell calcification is rarer (94) | Nodes may be enlarged, not commonly calcified, usually asymmetrical (94) | Nodes may be enlarged, not commonly calcified in acute infection but can occur with previous/ chronic infection.<br>May be asymmetrical |
| **Upper lobe nodules (+/- hilar adenopathy)** | Usually discrete and bilateral | Can be discrete and bilateral | May be unilateral or asymmetrical distribution | Can be bilateral or unilateral |
| **Nodule cavitation** | Uncommon | Uncommon | Possible | More common |
| **Lung fibrosis** | Progressive massive fibrosis | Pulmonary fibrosis like progressive<br>massive fibrosis less common | Unlikely to be symmetrical | Following chronic infection bilateral upper lobe fibrosis possible |
| **PET scan avidity** | Possible increased FDG uptake in areas of active lung disease. | Possible increased FDG uptake in areas of active disease. | High depending on cell type and likely | Possible increased FDG uptake in areas |

| | | | | |
|---|---|---|---|---|
| | Tends to be balanced and symmetrical but can mimic lung cancer (95) | Tends to be balanced and symmetrical but can mimic lung cancer (96) | asymmetrical. Involvement of bone in disseminated disease | of active disease. Tends to be balanced in areas of infection. Can mimic lung cancer(96) |
| **HISTOCYTOLOGY** | | | | |
| **Bronchoalveolar lavage** | Possible lymphocytes >25% (97) | Possible lymphocytes >25% (97) | Lymphoid malignancies will give monoclonal cells otherwise malignant cells depending on cancer type | Possible lymphocytes >25% but expect AFB on smear or culture and neutrophil predominance |
| **Histology on small sample e.g. endobronchial and transbronchial biopsy** | Birefringent particles may be present Non- caseating granulomas in early disease possible | Non- caseating granulomas typical. Caseating granulomas rare | Rare but some cancers can have a granulomatous reaction. Cancer cell type seen (98) | Caseating necrotising granulomas typical but early TB may have little necrosis and could be non- caseating Langhans type giant cells Caseating granulomas AFB staining positive |
| **Histopathology- larger sample e.g. cryobiopsy, surgical biopsy** | Birefringent particles under polarised light. Silicotic nodules- early nodules can resemble granulomas. Dust laden giant cells | Langhans type giant cells Non caseating granulomas Asteroid bodies Schaumann bodies (non-specific) | | |
| **OTHER** | | | | |
| **Cardiac disease/ eye/ skin involvement** | Similar to background population | More likely | Similar to background population depending on cell type and treatment | Similar to background population depending on site of infection |
| **Pulmonary function pattern** | Restrictive, obstructive or mixed. Normal in simple silicosis | Restrictive or normal | Depends on previous smoking history | Depend on disease stage |
| **AFB smear or culture** | Can be positive in silicotuberculosis | negative | negative | positive |

**Key:** ACE: Angiotension Converting Enzyme; AFB: Acid Fast Bacilli; ANA: antinuclear antibodies; CT: Computed Tomograph; FDG: FluoroDeoxyGlucose; IGRA: Interferon Gamma Release Assay; PET: Positron Emission Tomography; TB: tuberculous.

## APPENDIX 2: Hierarchy of Controls with respect to Silica exposure

*Elimination*

The most effective method of preventing exposure to silica is elimination, i.e. stop using the silica-containing materials entirely. In many cases this is not possible, but examples of this that have been achieved following silicosis outbreaks, include banning the sandblasting of denim clothing to make it look "worn in" (99), and a ban on the use, supply, and manufacture of artificial stone(100).

*Substitution*

In many industrial settings, eliminating silica usage entirely is not possible, but swapping to a similar material with a lower silica content may help reduce exposure. Some types of artificial stone are made to have a very high silica content (often 90-95%) to make hard, heat resistant and durable kitchen worktops. Using a substitute material which has a lower silica content such as granite (~30% silica), marble (~5%), or silica -free artificial stone (made of amorphous silica) may help reduce the risk to workers(101).

*Engineering controls*

In many circumstances, it is necessary for work to be carried out with silica -containing materials, and engineering controls are used to separate people from the exposure. Control measures for silica are predominantly designed to reduce the level of airborne dust and include the use of enclosures, water suppression, on-tool or local exhaust ventilation, and HEPA filter vacuums. Engineering controls are only effective if they are well-designed, adequately maintained and can be used effectively by the workers (83). For artificial stone, one study found airborne levels of silica could be reduced by a factor of 10 by using water suppression and on tool exhaust ventilation(102).

*Administrative controls*

Administrative controls are designed to change the way people work with silica. These may include educating workers about the risk of silica exposure and how these can be minimised, providing signs to remind workers of best practice, promoting good housekeeping, and limiting either the number of workers exposed to silica, or the time of exposure for each individual worker(83).

*Personal protective equipment*

Where other control measures are ineffective or can't be used, PPE, is the final option to reduce silica exposure. Employers have a legal duty to ensure that the PPE provided is fit for purpose, maintained and stored properly, and correctly used by workers. If tight-fitting respirators are used, workers need to be clean shaven and be face-fit tested. For many silica related work tasks, particularly where powered tools are used, and RPE must be worn for prolonged periods, battery powered air purifying respirator masks or hoods are recommended(103).

630 **APPENDIX 3: Legislation and guidance relevant to workplace respiratory diseases.**

| | |
|---|---|
| **The Health and Safety at Work Act, 1974.** | The primary piece of legislation covering occupational health and safety in Great Britain. It sets out the general duties which:<br>• employers have towards employees and the public;<br>• employees have to themselves and to each other;<br>• certain self-employed have towards themselves and others. |
| **The Control of Substances Hazardous to Health Regulations, 2002.** | COSHH is the law that requires employers to control exposure to substances that are hazardous to health. Employers are bound to:<br>• find out what the health hazards are;<br>• decide how to prevent harm to health ('risk assessment');<br>• provide control measures to reduce harm;<br>• make sure these are used and are in good working order;<br>• provide information, instruction and training for employees;<br>• provide monitoring and health surveillance in appropriate cases;<br>• plan for emergencies. |
| **The Equality Act, 2010.** | The Equality Act legally protects people from discrimination in the workplace and elsewhere; it replaced a raft of earlier anti-discrimination legislation. It applies to patients whose silicosis has had a 'substantial' and 'long-term' negative effect on their ability to do normal daily activities. |
| **(M)SDS.** | Every chemical agent used in industry is accompanied by a '(Material) Safety Data Sheet', akin to the warnings' leaflet in a package of medicine. Safety data sheets are readily available on-line (or through a patient's employer) and can be scrutinised for the mention of their respiratory sensitising potential, through the 'risk phrase' R42 or the 'hazard phrase' H334. |
| **The 'hierarchy of control'.** | An approach to the management of workplace risk through which risk should be reduced to the lowest reasonably practicable level by taking preventative measures, in order of priority. At the 'top' of the hierarchy is 'elimination' of the hazard at work (in this case a respiratory sensitiser); the provision of respiratory protective equipment (such as a face mask) lies at the bottom of the hierarchy. |
| **The Reporting of Injuries, Diseases and Dangerous Occurrences Regulations, 2013.** | RIDDOR puts duties on employers, the self-employed and people in control of work premises to report certain serious workplace accidents, occupational diseases and specified dangerous 'near-misses'. |
| **Industrial Injuries Disablement Benefit.** | IIDB is a UK statutory compensation scheme available to employed earners who have developed one of a list of 'prescribed' occupational diseases including silicosis. |
| **Surveillance of Work-related and Occupational Respiratory Disease.** | SWORD is the national reporting scheme for occupational lung disease that is funded by the HSE and run through the University of Manchester. The Health and Occupation Research (THOR) network \| The University of Manchester |

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## APPENDIX 4: Suggested audit criteria

By first visit

- A full list of relevant occupations held, their durations, and likely exposures
- Whether their current job is likely to involve continued silica exposure
- Whether their workplace has occupational health provision
- Whether they are under health surveillance
- Whether they are aware of other workers affected in the same workplace
- Spirometry performed
- Chest radiograph performed and interpreted

By second visit

- Advanced radiology such as CT performed and reported
- Screened for TB, autoimmune and renal disease using blood tests and urinanalysis as appropriate
- Letter to patient confirming:
    - Diagnosis
    - Mitigation of ongoing exposures
    - Written compensation advice
- Reported case to SWORD scheme

## APPENDIX 5: Useful links

Information about silicosis:

NHS website: https://www.nhs.uk/conditions/silicosis/

HSE: https://www.hse.gov.uk/lung-disease/silicosis.htm

Information about benefits:

IIDB: https://www.gov.uk/industrial-injuries-disablement-benefit

CAB: https://www.citizensadvice.org.uk/

Charities and support groups:

https://www.ukata.org.uk/support/asbestos-victim-support-groups/

https://www.actionpf.org/information-support/what-is-silicosis

http://www.asthmaandlung.org.uk

671 **APPENDIX 6: Example patient information sheet**

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# Silicosis factsheet for patients

## What is silicosis?

Silicosis is a lung disease caused by breathing in dust containing silica. This causes inflammation in your lungs, which can lead to lung scarring over time. This makes your lungs become hardened and stiff, and it can become more difficult to breathe. Silicosis is a life-long (or "chronic") disease.

## What causes silicosis?

Silicosis is caused by breathing in dust which contains silica such as stone dust, sand or silica flour. Silica dust is created when you cut, sand, drill, polish or grind stones containing silica.
**The more silica you breathe in, the more likely you are to get silicosis.**

How much silica dust you breathe in, and for how long, can impact the type of silicosis you may get. You can get silicosis if you breathed in high levels of silica dust over months, or if you have breathed in smaller amounts of silica dust over years. You can develop silicosis even if you have not worked with silica for many years.
Just wearing a face mask is not enough to protect you from breathing in silica.
Not everyone who breathes in silica dust will get silicosis. Some people are more likely to get it than others.

## What are the main symptoms?

People with silicosis don't usually have any symptoms in the early stages of the disease. If the disease progresses, then you may develop breathing difficulties even if you do not work with silica dust anymore.

Silicosis can cause:

- Cough, a tight chest and breathlessness
- Feelings of tiredness and weakness
- Weight loss

Silicosis can also cause diseases which can give you other symptoms like

- Hair loss
- Joint pains
- Dry eyes and mouth
- Night sweats

Having a chronic disease like silicosis can also cause mental health problems including difficulty sleeping and low mood.

## How is silicosis diagnosed?

**Silicosis is diagnosed on a chest x-ray or a CT scan.** A CT scan gives a more detailed picture of the lungs and is done in a hospital.

At your hospital appointments will also ask you what jobs you have done, about symptoms you might have, whether you smoke and we will do breathing, blood and urine tests. We might also ask you to have a scan of your heart (echo) and use a camera to look for infection or inflammation in your lungs (bronchoscopy).

## How is silicosis treated?

**Unfortunately there is no cure or effective treatment for silicosis**. But depending on your test results we may suggest some different treatments to try to improve your symptoms. These might include:

- Medications to help your symptoms such as codeine for cough.
- Anti-fibrotic medication which are anti-scarring tablets used to treat other lung diseases and may help some people with progressive silicosis.
- Inhaler reliever medication which can help if you also have asthma or other lung diseases.
- Inhaled corticosteroids which can be helpful if you also have another lung disease.
- Washing your lungs ("lung lavage") to try to remove some silica from your lungs.
- Oxygen therapy if you have low blood oxygen levels.
- Pulmonary rehabilitation classes or exercise

We may discuss referring you to our local lung transplant service. A lung transplant is a major operation and can only be considered if you are otherwise in good health and are not smoking. We regularly speak to specialists across the world to talk about new treatment options.

## How can I help myself?

For most people with silicosis, the most important thing is to **stop being exposed to silica dust**. This might mean changing your job or workplace. We know this is not always an easy decision. Wearing a face mask is often not enough to protect you from silica dust exposure.
You should also:

- Stop smoking cigarettes and shisha – any smoking will make silicosis worse
- Keep active – aim to walk 10,000 steps a day
- Look after your mental health – speak with friends and family you trust, and let your doctor know if you would like to speak to a counsellor
- Have regular vaccinations for flu/COVID/pneumonia if your doctor tells you to
- Go to all your hospital appointments – silicosis can develop slowly or quickly, and it is important for us to look for any changes. Bring a friend or family member with you to help you remember what has been discussed.

## What support is there for me?

If you have silicosis you can get different types of financial and other support.

## Industrial Injuries Disablement Benefit

You can apply for a Government benefit called Industrial Injuries Disablement Benefit, or IIDB.
You do not need to tell your workplace if you apply for this benefit.
If you have always been self-employed, you will not be able to get this this benefit.
To apply for this benefit you can download a form here: BI100PD Industrial Injuries Disablement Benefit for a prescribed industrial disease (publishing.service.gov.uk) If you need help to write the application, please contact us. If you are based in London, you can also phone "LASAG", a charity which helps people with silicosis to apply for benefits (Tel: 0808 278 2515).

## Compensation

You may also be able to make a legal claim against your employer's insurance company. It does not matter if you have worked for more than one company. It does not matter if your company has closed.
If you want to find out more then you should speak to a lawyer who has experience working with industrial diseases. We can give you names of companies who may be helpful. Making a legal claim usually takes more than one year.
This will not cost you any money as it is done on a 'no win, no fee' basis.

## Mental health support

If you have just found out you have silicosis, it might feel like a lot to deal with. Living with a disease like silicosis can affect your mental health. So it is really important that you have the right people around you to support you on this journey.

We are here to help you take things step by step, to understand what is happening and how you might be feeling, and to connect you to support. We will also ask about your mental health during your hospital appointments. If you want to speak to a counsellor, let your doctor know.

You can also refer yourself for "talking therapies". To do this type the name of your GP surgery at this website: https://www.nhs.uk/service-search/mental-health-services/find-nhs-talking-therapies-for-anxiety-and-depression/enter-gp

# What happens next? How often will you check on me?

We will organise regular appointments to see you and tests to monitor your health.
How often we see you will be based on your individual situation and health.
Make sure you go to all your appointments – silicosis can develop slowly or quickly, and it is important for us to notice quickly if there are any changes.
You should see you GP for other health issues as normal.

**Please contact us if you have any questions about the information in this leaflet**
Phone or text: 07977 352 164 (available Monday-Friday, 9am-5pm)
Email: gstt.OCLDAdmin@nhs.net

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