First Review of the Dust Diseases and Lifetime Care and Support Schemes

Thoracic Society of Australia and New Zealand response to questions taken on notice

The following responses have been prepared by the Occupational and Environmental Lung Disease Special Interest Group of the Thoracic Society of Australia and New Zealand

Question 1. Incidence of dust diseases

For compensation purposes, the most relevant data would be incidence, i.e. the number of new cases per year. Prevalence is all cases currently present in the community. Please note these estimates below are approximates and often based on overseas data as very little data is available in NSW.

1. Occupational asthma including reactive airway dysfunction (RADS) and occupationally exacerbated asthma

Systematic reviews of studies from various countries have concluded that 9–15% of asthma cases in working-age adults can be attributed to occupational exposures (Balmes et al. 2003; Blanc & Toren 1999; Newman Taylor et al. 2004). This includes occupational (new-onset) asthma and reactivated pre-existing asthma (asthma that has been asymptomatic for a long time). In Australia, from a survey of about 5,300 adults aged 18–49 years in New South Wales in 2000–01, we estimated 9.5% of adult-onset asthma cases were due to occupational exposures (Johnson et al. 2006). An incidence rate of 250–300 cases per million workers per year was found by (Kogevinas et al. 2007) this would equate to about 925-1110 cases of occupational asthma in NSW in 2016, given a labour force of about 3.7 million in that year (ABS Labour Force Survey).

2. Occupational lung cancers including those related to causes other than asbestos (e.g. silica, arsenic)

There were approximately 3400 new cases of lung cancer in NSW in 2016 (AIHW). Epidemiological studies suggest approximately 10% are attributable to occupational exposures (Gustavson et al., 2000), this would equate to about 340 cases of occupational lung cancers. This estimate would include those due to asbestos and silica exposure which are already covered under the scheme.

3. Dust-induced pulmonary fibrosis

Some epidemiological data in the United States indicates the prevalence of dust induced pulmonary fibrosis in those with pulmonary fibrosis to be 14% while the incidence was reported as 12% (Coultas et al., 1994). The European Registry suggests a prevalence of 4–18% of occupational pulmonary fibrosis

and an incidence of 13–19% (Thomeer et al 2001, Khalil et al 2007). In various populations, the incidence estimates for idiopathic pulmonary fibrosis have ranged from 6 per 100,000 to as high as 32 per 100,000 (Coultas et al., 1994). Approximately 10 per 100,000 is a reasonable estimate for the US (Ley et al., 2013), there is no current data in Australia available. This would equate to about 700 new cases a year in NSW, 12 to 19% may be dust induced pulmonary fibrosis, i.e. 84 to 133 cases per year

4. Chronic obstructive pulmonary disease (COPD) related to dust, fume and mist exposure

Vapors, gas, dust, or fumes on the longest held job exposure was associated with an increased risk of COPD (OR 2.11; 95% CI 1.59-2.82) and a Population Attributable Fraction (PAF) of 31% (95% CI 22-39%) (Blanc et al, 2005). After adjusting for smoking status and demography, the odds ratio for COPD related to self-reported occupational exposure was 2.0 (95% confidence interval (CI) 1.6-2.5), resulting in an adjusted population attributable risk (PAR) of 20% (95% CI 13-27%). (Trupin et al., 2003). Using these data approximately 13 to 31 % of new cases of COPD may be related to occupational exposures. The incidence of COPD is approximately 300 per 100,000 (Rycroft et al, 2012). This would equate to about 2,700 to 6500 new cases a year of COPD related to dust, fume and mist exposure.

5. Pneumonia related to occupational exposures

Among men aged 20-64 years there was increased mortality from infectious pneumonias among construction workers exposed to metal fumes (RR 2.31, 95% CI 1.35 to 3.95), inorganic dust (RR 1.87, 95% CI 1.22 to 2.87) and chemicals (RR 1.91, 95% CI 1.37 to 3.22). (Toren et al., 2011). Hospitalisations for pneumonia in NSW are about 300 per 100,000 per year. I was unable to find data on what proportion of these may be due to occupational exposures

6. Systemic diseases related to occupational dust exposures

There are several diseases which are related to dust exposure or interact with such exposures but which do not primarily affect the lung. One example is scleroderma, which has been related to occupational silica exposure, and Caplan's syndrome (rheumatoid pneumoconiosis).

In Australia the annual incidence of scleroderma is 16 per 1 000 000 with a prevalence of 233 per 1 000 000. The female to male ratio of the condition is 4:1 respectively. A review of the literature disclosed 32 published series, with clinical data of 254 scleroderma with silica exposure patients (96% males). Scleroderma with silica exposure patients represented 37.5–86% of the scleroderma males and 0–2.7% of the scleroderma females (Freire et al 2015). This would equate to about 8 to 19 new cases per year of scleroderma with silica exposure in NSW.

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Question 2. Supply of Dr. Ryan Hoy's presentation on dust diseases to DDB Board.

Attachment A – Presentation

Attachment B - Matar, E., Frankel, A., Blake, L. K. M., Silverstone, E. J., Johnson, A. R., & Yates, D. H. (2017). Lessons from practice. *Med J Aust*, *206*(9), 385-386.

Question 3. Recommendation on how to update the list of compensable occupational lung disease and associated outcomes.

TSANZ believes the current list of 13 compensable occupational lung diseases requires updating and should be expanded to include:

- Occupational asthma including reactive airway dysfunction (RADS) and occupationally-exacerbated asthma
- Occupational lung cancers including those related to causes other than asbestos (e.g. silica, arsenic, exposure to ionising radiation, bischloromethylether (BCME) etc)
- Dust-induced pulmonary fibrosis e.g due to wood dusts
- Chronic obstructive pulmonary disease (COPD) related to dust, fume and mist exposure
- Pneumonia related to occupational exposures e.g. brucellosis, psittacosis in poultry workers, Legionnaire's disease, lobar pneumonia in welders, occupationally-acquired tuberculosis in health care workers etc.

Serious consideration should also be given to including diseases affecting the upper airways and nasopharyngeal tract (such as laryngeal cancer and carcinoma of the sinuses), and including systemic diseases induced by occupational exposures e.g. connective tissue disorders attributable to silica exposure.

This list should be kept up to date as new diseases emerge. Provision needs to be made for inclusion of newly recognised occupational diseases and new causes of e.g. occupational asthma. One mechanism which allows this is by having a particular category which allows individual recognition of attribution. A Medical Advisory Panel consisting of 3 independent expert physicians would be useful in this regard.

In order to keep the list up to date we recommend establishing an independent scientific advisory committee with the long term responsibility for occupational lung disease. This committee should review the list of diseases at regular intervals (for example, every 5 years) or when requested. The committee should be primarily scientific and medical with experts from a range of relevant specialties who can make detailed assessments of the evidence. These specialties could include respiratory and occupational medicine, epidemiology, occupational hygiene, medical oncology etc. where applicable, and potentially also lawyers with special expertise in this area.

A similar model is already in Australia used for updating compensation arrangements in the Dept of Veterans Affairs. The Specialist Medical Review Council (SMRC) is an independent statutory body responsible to the Minister for Veterans' Affairs. The SMRC consists of medical practitioners and medical scientists appointed as Councilors by the Minister and selected by the Convener of the SMRC for a particular review (3-5 Councilors) on the basis of their expertise in the injury or disease relevant to the subject to review. The legislative authority for the Specialist Medical Review Council is contained in Part XIB of the Veterans' Entitlements Act 1986 (VEA).

Useful international models of this system include the United Kingdom's Industrial Injuries Advisory Council (IIAC), established in 1946. IIAC is an advisory non-departmental public body, sponsored by the Department for Work and Pensions. The Council does not have any staff of its own but DWP provides a small administrative team. The council holds an annual public meeting in a different place in the UK each year, with other council meetings are usually held in DWP offices. Various International Labour Organisation/World Health Organisation committees also consider occupational lung diseases and support the global occupational health network. Currently, for example, there is a Global Program for the Elimination of Silicosis (GOHNET).

Useful list of occupations and of current evidence relating to occupational diseases are available from these models, as well as criteria for disease attribution and disablement assessment.

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www.iiac.independent.gov.uk

www.who.int

www.smrc.gov.au

who.int/occupational_health/publications/newsletter/gohnet

ANZ

Artificial stone associated silicosis

DR RYAN HOY

Silicosis

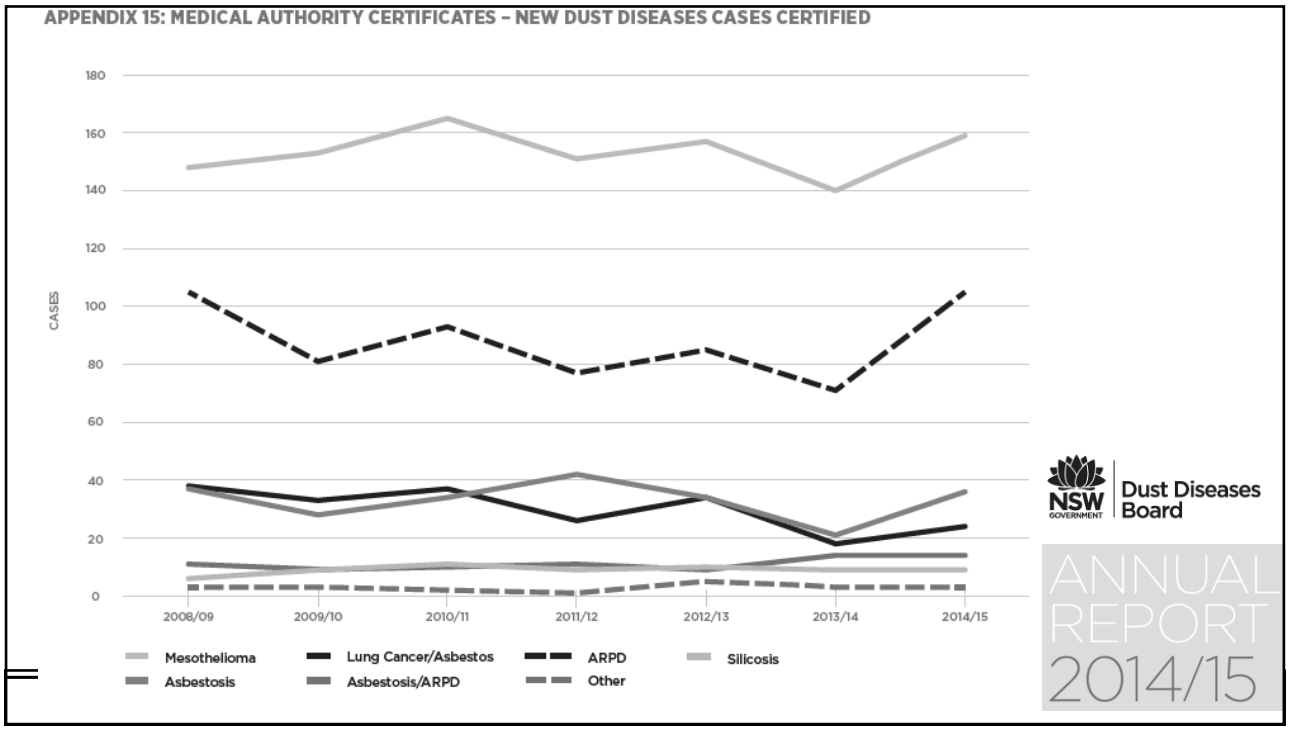
Silica (SiO_2) is a naturally occurring, widely abundant mineral that is the major component of rocks and soil.

Silicosis is a chronic diffuse interstitial fibronodular lung disease caused by inhalation of crystalline silica.

Silica also associated with COPD, tuberculosis, connective tissue diseases, renal disease and lung cancer.

Traditional occupations at risk of silica exposure include miners, quarry workers and tunnellers, foundry and pottery workers, and construction workers (such as pavers, who frequently cut or break stone, concrete or brick)

Recently in Australia several cases of accelerated silicosis in workers exposed to dust from use of artificial/engineered stone have been identified, indicating significant potential risk with this new building product.



Types of silicosis

- Acute silicosis
- Accelerated silicosis
- Chronic silicosis:
  - Simple silicosis
  - Progressive massive fibrosis

Acute silicosis

- Also known as silicoproteinosis
  - Develops after exposure to very high concentrations of respirable crystalline silica and results in symptoms within a few weeks to a few years after the initial exposure.
  - Symptoms may precede significant radiologic findings

Typically progressive and no specific therapy has been identified.

Early referral for lung transplantation

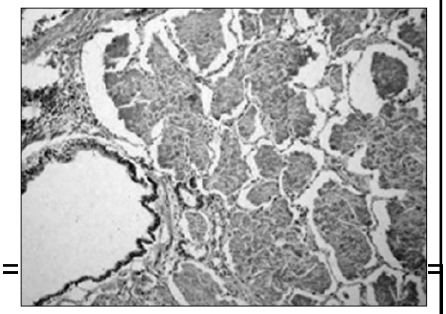


Fig. 1—Photomicrograph of autopsy specimen from male cadaver (age at death, 28 years; cause of death, silicoproteinosis) shows abundant intraalveolar proteinaceous material and positive reaction to stain. (Periodic acid–Schiff, ×100)

Accelerated silicosis

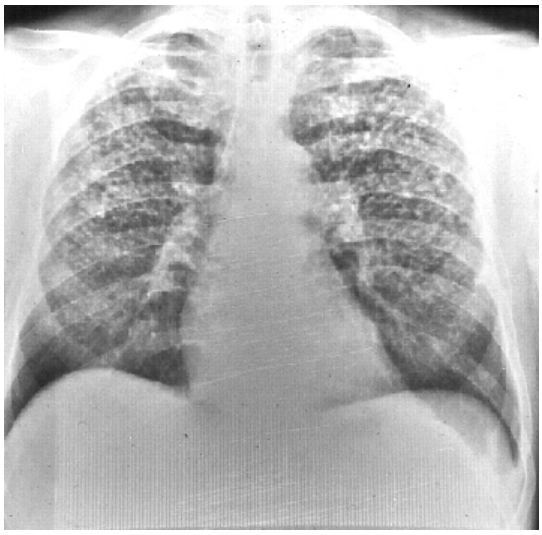
- Also associated with high-level exposure to silica
- Differentiated from chronic silicosis only by its more rapid development (within 10 years) following first exposure.
- Patients who develop silicosis after a short time period are at increased risk for the later development of progressive massive fibrosis (PMF) and may be at greater risk of complications.
- Chronic cough and dyspnoea on exertion are common and become more severe with worsening radiographic abnormalities

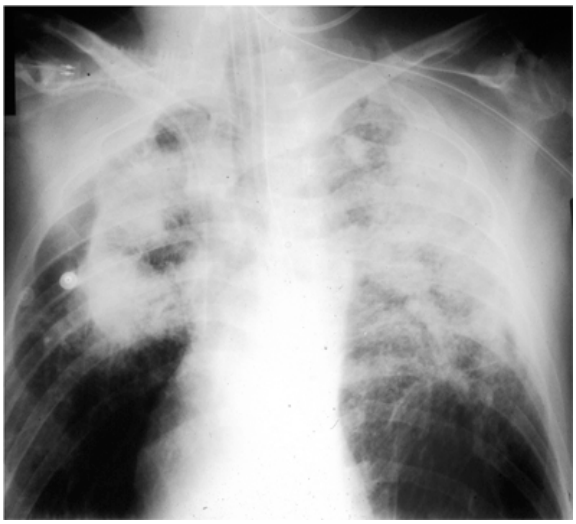
Chronic silicosis

Most common form of silicosis. Appears 10 to 30 years after first exposure. May become radiologically apparent after leaving workplace.

1. Simple silicosis: Usually asymptomatic. Radiologically characterised by presence of innumerable, small, rounded opacities (less than 10 mm in diameter). Distributed predominantly in the upper lung zones
2. Progressive massive fibrosis (PMF): Small opacities gradually enlarge and coalesce to form larger, upper- or mid-zone opacities more than 10 mm in diameter. Cavitation may develop. Associated hilar adenopathy with calcification in 5%.

Mycobacterial infection important differential diagnosis and may co-exist.



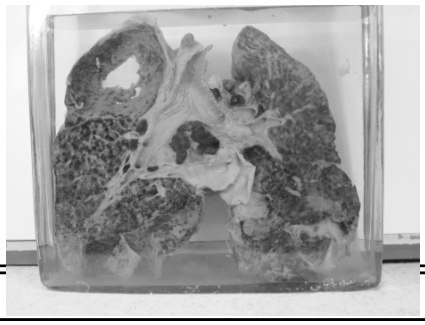


A historical disease?

Awareness and dust suppression measure in certain industries have markedly reduced the number of cases of this preventable lung disease

Ongoing exposure to silica in Australian workplaces:

- Cross-sectional survey of the Australian working population (18–65 years old)
- 6.4% of respondents were deemed exposed to RCS at work in 2012 (3.3% were exposed at a high level)



Artificial or engineered stone

Various tradenames

Developed in late 1980s and available in Australia since 2001

Cheaper then marble

Wider range of colours

Non-porous, scratch and stain resistant

4 times flexural strength and double impact resistance of granite

Artificial stone production

Source: www.caesarstone.com

>85% crystalline silica

Polymeric resin 7-15%

Additives 0-8%

Approximate crystalline silica content of different materials	
Sandstone	70–90%
Concrete, mortar	25–70%
Tile	30–45%
Granite	20–45%, typically 30%
Slate	20–40%
Brick	Up to 30%
Limestone	2%
Marble	2%

Artificial stone associated silicosis

Observational study 11 workers at a family run business manufacturing kitchen and bathroom counter tops exposed since 1995. Six cases of silicosis diagnosed, disease prevalence in this environment 54.5%. Pascual, 2011

Israel National Lung Transplant Program: 25 patients referred include 10 transplanted with silicosis associated with artificial stone used primarily for kitchen countertops and bathroom fixtures. Kramer, 2012

Spain - 46 cases of silicosis diagnosed by HRCT, 91% had simple chronic silicosis. Median age of 33 years and a median of 11 years working in the manufacturing of countertops. 1 death. Perez-Aloson, 2013

Italy: 3 cases reported, consistent with accelerated silicosis. Paolucci, 2015

No surveillance / cross-sectional studies of the industry

Outbreak of autoimmune disease in silicosis linked to artificial stone

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Occupational Medicine 2015;65:444–450

Israel National Transplant program evaluated 40 patients with advanced silicosis over a 15 year period with advanced silica-related lung disease.

All 40 male and had substantial occupational history of silica exposure working with synthetic stone material. Dry cutting and polishing stone for end use in kitchen and other countertop applications.

9 (23%) of cohort with specific immunological diagnosis. >7 fold excess compared with general population

Case	Symptoms	Physical findings	Relevant serologies	Clinical diagnosis
1	Raynaud's; Dysphagia	Sclerodactyly; Telangiectasia; Serositis	Anti-Scl-70 (+); SSA (anti-Ro) (+)	SSc
2	Raynaud's; Arthritis	Digital pitting; Arthritis; Serositis; Fever	ANA > 1:160; RNP (+); SSA (anti-Ro) (+)	MCD
3	Raynaud's; Dysphagia	Sclerodactyly; Digital pitting	ANCA (+)	SSc
4	Raynaud's	Digital pitting; Arthritis; Serositis	Anti-Scl-70 (+); RNP (+); SSA (anti-Ro) (+); SSB (anti-La) (+)	SSc
5	Arthritis, Xerostomia	Arthritis	ANA > 1:160; SSA (anti-Ro) (+)	Sjogren's syndrome
6	Arthritis	Arthritis; Fever; Rash	ANA 1:80; RNP (+); SSA (anti-Ro) (+)	MCD
7	Arthritis	Arthritis; Serositis	ANA > 1:160; RF (+)	RA
8	Arthritis	Arthritis	RF (+)	RA
9	Arthritis, Myalgia	Arthritis: Myositis	ANA (+); SSA (anti-Ro) (+); SSB (anti-La) (+); anti-JO-1 (+)	Polymyositis—anti-synthetase syndrome

The New York Times | http://nyti.ms/1ZReakJ

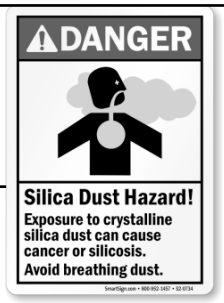
BUSINESS DAY

Popular Quartz Countertops Pose a Risk to Workers

By BARRY MEIER APRIL 1, 2016

As sleek "engineered stone" countertops grow in popularity, safety experts are warning that workers who handle them are at particularly high risk from an old workplace hazard — silica, the mineral tied to silicosis, a debilitating and potentially deadly lung disease.

Primary prevention



Respirable crystalline silica is classified as a hazardous chemical by SafeWork Australia and an occupational carcinogen by IARC.

Australian standard for exposure is 0.1mg/m^3 8 hour TWA.

NOHSC 2001: 30 minute exposure 0.3mg/m^3 and single short term exposure not exceed > 0.5 mg/m^3

Primary prevention relies on interventions before disease has developed.

Control dust at source: wet cutting

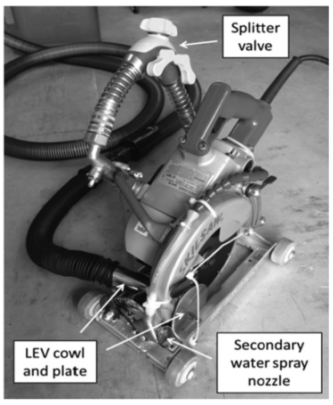
Ventilation: local exhaust ventilation, use of extraction hoods

Personal protective equipment: appropriate selection, fit testing, education, maintenance.

Respirable Silica Dust Suppression During Artificial Stone Countertop Cutting

Jared H. Cooper, David L. Johnson* and Margaret L. Phillips

Ann. Occup. Hyg., 2015, Vol. 59, No. 1, 122–126



1 A secondary water flow provided a fan-shaped water curtain sprayed normal to the path of the ejected stone dust. The secondary flow could be shut off during LEV and wetted-blade-only trials. The LEV cowl was attached only during LEV trials.

Table 2. Respirable silica dust concentrations (mg m^{-3}) averaged over nominal 30-min sampling period

Replicate	Wetted blade only	Wetted blade + water curtain	Wetted blade + LEV	Dry
1	4.846	2.944	ND[a]	44.37
2	2.563	0.920[b]	0.139[b]	
3	1.874	3.405	0.201[b]	
4	2.209	1.373	0.669	
Mean	4.934	3.813	0.604	
SEM	0.923	1.018	0.225	

ND, not detected.
[a]Measured silica mass from which this concentration was calculated was < LOD.
[b]Measured silica mass from which this concentration was calculated was < LOQ.

Secondary prevention

Health monitoring should be implemented if likely to meet or exceed 0.05 mg/m^3 8 hour TWA. Aim to identify early, pre-clinical markers of silicosis.

Baseline medical examination, annual review, final medical assessment

Incorporates:
- Work history
- Medical history
- Examination
- Respiratory function tests – according to ATS/ERS standards
- Chest x-ray reported according to current International Labour Organisation (ILO) classification

Recommendations may include review of workplace control or removal from work with silica.

Tertiary prevention

Early, accurate diagnosis by respiratory physicians to minimize medical and socioeconomic impact on the worker

Current TSANZ OELD SIG activities

Major concerns about unsafe workplace exposures and practices

Poor understanding of the risk of exposure associated with artificial stone which is a relatively new building material

Lack of adherence to current regulated standards and surveillance requirements.

TSANZ Occupational and Environmental Health SIG raised these concerned with SafeWork Australia and all ministers responsible for workplace safety nationwide

Call for greater inspection of workplaces, demonstration of performance of health surveillance and enforcement of exposure standards.

Education of apprentices, employees and employers regarding the risk.

Further research is required

Lessons from practice

Complicated silicosis resulting from occupational exposure to engineered stone products

Clinical record

A 54-year-old man, formerly a smoker, presented with a 6-year history of chronic cough and exertional breathlessness without previous respiratory illnesses. Born in Vietnam, he came to Australia as a refugee at the age of 20 years. A screening chest x-ray was performed on his arrival in Australia; as the patient was not informed about any abnormality, this was assumed to be normal. He commenced work as a labourer; he denied exposure to silica-containing materials and did not participate in activities typically associated with silica exposure (such as jack-hammering) during this period. About 15 years later, the patient started a job manufacturing stone benchtops. He cut, ground, finished and installed the benchtops, using a popular brand of engineered stone comprising > 85% crystalline silica. Occasionally, he made benchtops from granite and marble. During the first 7 years of this work, the patient did not use any respiratory protective equipment, but later used a simple paper mask. Despite some dust extraction facilities in the factory, he reported that the environment was visibly dusty and that dust suppression with water was hardly ever used.

At presentation, chest examination showed scattered fine crackles and bronchial breath sounds bilaterally in the upper zones. Spirometry showed restriction (forced expiratory volume in 1 second [FEV_1]/forced vital capacity [FVC], 1.9/2.6 L [73% and 82% predicted, respectively]) and no bronchodilator reversibility; gas transfer was reduced (diffusing capacity of the lungs for carbon monoxide [D_{LCO}], 60% predicted, carbon monoxide transfer coefficient [K_{CO}], 92%).

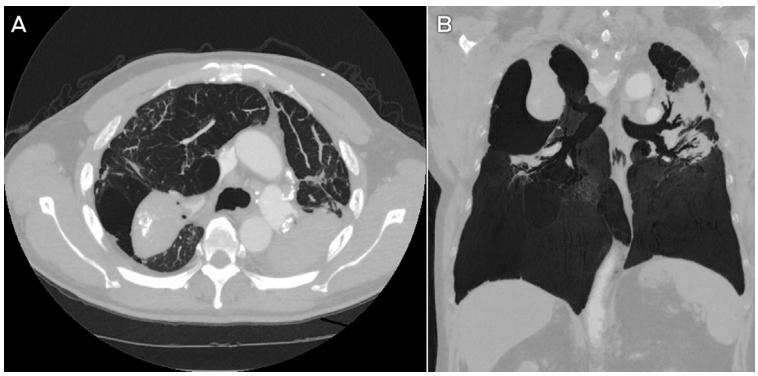
Multiple sputum specimens tested negative for acid-fast bacilli. Levels of inflammatory markers were within reference levels, and the result of a screening test for autoimmunity and vasculitis was negative. A computed tomography (CT) scan of the chest (Box 1) showed confluent bilateral calcified fibrotic masses in the upper zones, with marked volume loss, distortion and perilesional bullae. Occasional small peripheral lung nodules were present, predominantly distributed in the upper zones. There was calcified mediastinal lymphadenopathy.

Bronchoscopic washings tested negative for acid-fast bacilli and malignant cells. Positron emission tomography (PET) scanning showed intense uptake in the confluent densities (maximum standardised uptake value, 10.6) and mediastinal lymphadenopathy. CT-guided fine-needle aspiration biopsy was negative for malignant cells. Transbronchial fine-needle aspiration biopsy of lymph nodes using endobronchial ultrasound was non-diagnostic.

An open biopsy of the right upper lobe lesions and paratracheal lymph nodes was performed. Histological analysis of the lung sample showed numerous large sclerotic silicotic nodules surrounded by collections of histiocytes (Box 2). Analysis of lymph node sections confirmed prominent nodular silicosis.

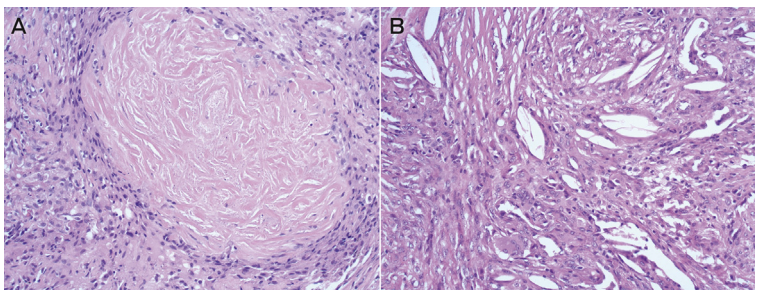
Overall, the findings, including PET, were compatible with a diagnosis of complicated silicosis with progressive massive fibrosis. The patient continued to experience worsening breathlessness, despite treatment with bronchodilators and inhaled corticosteroids. He subsequently developed bilateral pneumothoraces, requiring temporary intercostal drain insertion, and is now listed for lung transplantation. ◆

1 Axial (A) and coronal (B) computed tomography scans of the patient's lungs



The scans show confluent mass-like fibrosis in the posterior upper lung zones, with adjacent bullae and compensatory expansion of the lower lobes. Calcification is seen within the fibrotic masses as well as within the hilar and mediastinal lymph nodes. There are occasional small peripheral nodules, most obvious in the right lung. ◆

2 Typical features of silicosis on lung histopathology slides



The biopsy specimen (A) shows the whorled appearance of a silicotic lung nodule, consisting of concentric laminated collagen fibres (haematoxylin–eosin stain, magnification 10×). A higher magnification image (B) highlights clefts containing faintly visible silicate particles surrounded by macrophages and giant cells (magnification 20×). ◆

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Silicosis refers to a spectrum of progressive and debilitating occupational lung diseases caused by the inhalation of free crystalline silica. Once established, there is no effective treatment. Prevention is therefore paramount. Exposure to silica has been unequivocally associated with an increased risk of lung cancer, as well as an increased frequency of tuberculosis and possibly also autoimmune disease. Implementation of appropriate workplace standards to minimise exposure is crucial to reduce incident and severe cases of silicosis.

Silicon dioxide (silica) is the most abundant mineral on Earth and is present in almost all types of rock, sand, clay and gravel. The most common crystalline forms of silica are quartz, cristobalite and tridymite. Silica exposure may occur in many work settings. Despite a global downward trend, new outbreaks of silicosis have recently been reported, with life-threatening silicosis occurring after exposure to a relatively new type of engineered stone product used for kitchen and bathroom benchtops.[1,2] These products (known as "artificial quartz conglomerate" and "artificial stone"), which contain a high content of free crystalline silica (70–90%), are increasingly used in preference to their marble and granite counterparts because of their low cost, improved durability and hardness.

Risk of exposure to high levels of crystalline silica from engineered stone is present at all levels of this industry, from manufacturing (stone cutting, shaping and finishing) to assembly and installation.[3] In a recent study, 25 ornamental stone workers who had been employed in dry-cutting a synthetic stone product developed advanced silicosis.[1] In another report, silicosis was diagnosed in 46 men working in the manufacture of artificial stone for kitchen benchtops.[2]

Following these reports, an alert issued in the United States highlighted potentially dangerous levels of silica exposure associated with suboptimal practices in the artificial stone industry.[4] Further calls to action have come from Europe, with a report from Tuscany citing seven cases of silicosis in workers exposed to crystalline silica during benchtop manufacturing.[5] In almost all reported cases, there was little adherence to basic protection measures, such as provision of appropriate ventilation systems and use of personal protective equipment.

The Safe Work Australia workplace exposure standard for respirable crystalline silica (time-weighted average) is 0.1 mg/m^3, which is designed to prevent the occurrence of silicosis.[6] Our case reaffirms the need for vigorous enforcement of dust reduction regulations, particularly in the growing industry of engineered stone products. Benchtop stonemasonry is a potentially dangerous occupation, and medical practitioners should have a heightened awareness of this newly described occupational hazard.

Lessons from practice

- Silicosis is a disabling but entirely preventable occupational lung disease caused by exposure to inhaled free crystalline silica.
- Recent outbreaks of silicosis have been associated with the manufacture of relatively new engineered stone products that are used for kitchen and bathroom benchtops.
- Medical practitioners should be aware that cutting and installing these engineered artificial stone products can be a hazardous occupational exposure.
- Appropriate dust suppression practices and the use of respiratory protective equipment should be reinforced to prevent silica-related diseases. ◆

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