Occupational medicine

Asbestosis and silicosis

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Interstitial fibrosis resulting from workplace exposure to asbestos and crystalline silica persists throughout the world despite knowledge of the causes and effective means for prevention. Asbestosis and silicosis occurrence is predictable among people overexposed to dusts in various industries and occupations such as mining, construction, manufacturing, and building maintenance. Asbestosis and silicosis are incurable and may be progressive even after dust exposure has ceased, therefore early recognition and supportive interventions are important. Although current disease is a result of past exposures, effective control of current workplace exposures is the only way to prevent continued occurrence of these potentially debilitating diseases. Physicians can contribute to this effort through accurate diagnosis and disease reporting.

Asbestosis and silicosis are chronic non-malignant lung diseases caused by inhalation of hazardous dusts found in a variety of workplaces. They persist despite substantial knowledge about their causes and effective means for their prevention. Asbestosis has been recognised throughout much of this century,[1] and lung disease from silica exposure has been reported for hundreds of years. Since both diseases can occur years after initial exposure, there is a tendency to ascribe current disease incidence to historical workplace conditions. However, modern technologies used in the absence of modern controls continue to pose health risk and allow persistence of these diseases.

Surveillance and epidemiology

There is no uniform international surveillance and reporting of asbestosis or silicosis. National information about disease incidence, prevalence, and trends is sparse, lacking in comparability, and difficult to obtain. In the USA between 1979 and 1992, 4882 death certificates listed silicosis as an underlying or contributing cause of death. 8761 death certificates listed asbestosis during that period. The number of death certificates that mention silicosis has decreased during the past 25 years, whereas the number mentioning asbestosis has risen steadily.[2] Silicosis mortality rates calculated for various European countries ranged from 0·91 per 100 000 men in the UK to 7·36 per 100 000 men in Belgium during the period 1985–86.[3] These rates are in the same range as those of deaths from sarcoidosis (0·49 per 100 000 in 1992) or chronic liver disease (7·9 per 100 000 in 1993) in the USA. General population mortality rates are not necessarily illuminating for purposes of comparison, since information about the number of people at risk of disease from work is not available. Surveillance for silicosis in four States in the USA identified 447 new cases between 1988 and 1992.[4] The number of cases captured in any surveillance system is an underestimate, because many cases are undiagnosed.

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The epidemiology of asbestosis was reviewed by Becklake in 1992.[5] Exposure-response relations between fibre exposure and pulmonary fibrosis have varied widely across industries and occupations, consistent with theories that health risk can be modified by process-specific factors. By contrast, exposure-response estimates for silicosis risk in various populations fall in a narrow range.[6]

Exposure settings

The continued occurrence of new cases of silicosis and asbestosis is due, partly, to the diversity of settings in which hazardous exposures continue. Too often, employers fail to recognise and control the hazard, workers are unaware of the risk, and workplace inspectors fail to take samples and to enforce current exposure limits.

Silica

Exposure to crystalline silica—and hence the risk of silicosis—is well recognised in both surface and underground mining, as well as tunnelling and quarrying. The silicosis risk in the construction industry, although substantial, is often unrecognised: quartz dust is created in the drilling, digging, and blasting necessary for site preparation, and also many construction materials, such as cement, produce respirable crystalline silica when cut

Panel 1: **Occupations and industries associated with silicosis**

Occupations	**Industries**
Mining-machine operators	Coal-mining
Labourers	Construction
Managers and administrators	Metal-mining
Supervisors, precision production occupations	Blast furnaces, steelworks, rolling and finishing mills
Janitors and cleaners	Non-metallic mining and quarrying
Operators of moulding and casting machines	Iron and steel foundries
Supervisors and proprietors, sales occupations	Structural clay products
Operating engineers	
Machinists	
Hand-moulding, casting, and forming occupations	

or ground.[7] Crushed stone contains varying amounts of crystalline silica, which can be liberated as respirable particles in milling, grinding, or in end use such as road building or maintenance of railway tracks. Although many industrialised countries severely restrict the use of sand as an abrasive blasting agent, sand-blasting continues in the USA.[8] Use of non-quartz abrasives in decoration or shaping of materials that contain granite or other quartz can also pose health risk. Finely ground silica (sometimes known as silica flour) is used as a component of many products, including paints and toothpaste. There is risk both in the silica-milling process and in its use.[9] Panel 1 lists the occupations and industries most commonly recorded on death certificates for people with silicosis (among residents in selected USA States, aged >15 years, 1991–92).

Asbestos

Asbestos has been used in more than 3000 commercial products. World production and consumption of asbestos peaked in the mid-1970s at around 5·0 million metric tons, and has declined since to less than 2·5 million tons in 1994.[10] Although use of new asbestos has fallen substantially in western Europe and north America, consumption is increasing in countries with more rapidly expanding economies.[11] There is health risk to miners and millers of asbestos, but the unprotected use of asbestos-containing materials in manufacturing and construction places more people at greater risk. Asbestos has valuable thermal, electrical, and sound-insulating qualities, and many workers have experienced substantial exposure in the installation of various forms of insulation. Although there has been much effort to limit exposure during original installation, the maintenance, repair, and removal of asbestos-containing material can liberate high levels of respirable fibres. Buildings containing asbestos should be demolished only with appropriate safeguards. There has been much concern about the health risk to office workers, teachers, and students in offices and classrooms with asbestos in place, but the documented exposures are orders of magnitude below those to maintenance and custodial workers in whom asbestosis has been documented.[12] The health risk to building occupants where asbestos is in good repair and undisturbed is not considered to be significant. Panel 2 lists the occupations and industries most commonly associated with deaths from asbestosis (residents in selected USA States, aged >15 years, 1991–92).

Panel 2: **Occupations and industries associated with asbestosis**

Occupations	Industries
Plumbers, pipefitters, and steamfitters	Construction
Electricians	Ship and boat building and repairing
Insulation workers	Chemicals and other manufacturing
Carpenters	Non-metallic mineral stone products
Labourers	Railways
Supervisors, managers	General government
Boilermakers	Yarn, thread, and fabric mills
Welders and cutters	Other rubber products, plastic footwear, and belting
Janitors and cleaners	Trucking service

Disease descriptions

Silicosis

Silica occurs naturally in a variety of crystalline forms, alpha quartz being the most common. Heat can convert amorphous silica into more biologically active forms. The quantity of inhaled crystalline silica, size distribution, and surface characteristics of the dust may affect toxicity. Freshly fractured silica created by grinding or abrasion seems to have greater toxicity.[13]

Silicosis, the interstitial lung disease caused by the pulmonary response to inhaled crystalline silica, is described as chronic, accelerated, or acute. Chronic and accelerated silicosis are pathologically similar and are distinguished only by their time course. Chronic silicosis generally appears 10 or more years from first exposure. Accelerated silicosis may occur within 2 years of initial exposure. Both are characterised by the formation of pathognomonic silica nodules in the lung parenchyma—well-organised concentric central whorls of collagen surrounded by inflammatory cells. In both chronic and accelerated silicosis, the fibrotic nodules can coalesce into larger lesions, denoted progressive massive fibrosis, which distort and destroy the normal pulmonary architecture. By contrast, acute silicosis develops rapidly after intense exposure generally to fine particles of freshly fractured crystalline silica. Acute silicosis has many features of alveolar proteinosis rather than presenting as interstitial fibrosis.

Asbestosis

Asbestosis is a fibrotic interstitial lung disease caused by a cascade of responses to inhaled asbestos fibres.[14] Under usual exposure conditions, onset of radiographically visible fibrosis rarely occurs earlier than 15–20 years from first exposure. Although there are differences in the mineralogical characteristics, patterns of translocation after inhalation, and biological persistence of various forms of asbestos fibres, all forms can cause interstitial fibrosis and other associated diseases. In practice, much of the asbestos in place in buildings and in use in general industry includes various mineral species; therefore, exposures tend to be to mixed fibre types.

Clinical presentation

There are no symptoms or physical signs uniquely associated with the pneumoconioses. Asbestosis and chronic silicosis are generally of insidious onset with gradually progressive dyspnoea, at first noticeable only on exertion and often attributed by the patient to ageing. Cough, dry or with sputum production, may be present, especially in current or former smokers and in those who have worked in dusty environments. Sexual dysfunction is common, although patients rarely associate this problem with their pulmonary disease. Patients may be free of symptoms, with abnormalities identified by chest radiography during medical screening or surveillance activities. In accelerated silicosis, there is a greater probability of chest symptoms being present when the radiographic abnormalities are first detected. Acute silicosis can have a fulminant course with rapid progression of dyspnoea, wasting, and exercise intolerance.

Physical examination may be unremarkable with mild disease. Persistent basilar crackles or râles, often at end inspiration, are noted in many patients with asbestosis.

Wheezing, particularly with forced expiration, is associated with airways obstruction, and is non-specific. Other non-specific physical signs of pulmonary dysfunction (including clubbing of the fingers, cyanosis, and right-sided congestive heart failure) may be observed in patients with advanced disease.

Chest radiography is the primary diagnostic investigation permitting recognition of the pneumoconioses. Asbestosis presents with irregular linear opacities, primarily at the bases and the periphery, which gradually become visible in the mid and occasionally upper zones of the lung. Other radiographic signs resulting from asbestos exposure include circumscribed pleural plaques (with or without calcification) or diffuse pleural thickening, including blunting of the costophrenic angle, and, rarely, a pseudo-tumour known also as rounded atelectasis. Parenchymal fibrosis from asbestos exposure can occur with or without pleural plaques. A clinical diagnosis of asbestosis is generally made on the basis of a credible history of asbestos exposure, a reasonable delay between first exposure and detection, and radiographic findings.[15] Isolated pleural plaques, otherwise unexplained, in an individual with remote asbestos exposure are probably attributable to the exposure and may be associated with exertional dyspnoea and pulmonary dysfunction.[16,17]

Silicosis presents radiographically with small opacities, more commonly rounded but occasionally irregular, in the upper or mid zones of the lung. As disease advances, opacities may become visible throughout all lung zones and may become confluent. A diagnosis of progressive massive fibrosis is made when one or more opacities of length greater than 1 cm are present. Calcification of hilar lymph nodes is seen occasionally.

Standard approaches to the recognition and description of chest radiographic changes from mineral-dust exposure are used in medical surveillance, epidemiology, and, at times, in clinical practice. The most widely accepted scheme is published by the International Labour Office (ILO).[18] This system provides a stepwise method for assessment and description of the shape, size, location, and profusion of opacities that may have resulted from dust exposure. Radiographs are classified after comparison with so-called standard radiographs, and the results are recorded systematically. The system and its limitations have been described elsewhere.[19] There are also conventions for classification of pleural abnormalities and for recording of changes associated with other diseases. The large opacities of progressive massive fibrosis are classified by size.

10–20% of people with substantial asbestos or silica exposure and associated pathological abnormalities have chest radiographs classified within the ILO normal range;[20] thus, the plain radiograph is judged to be around 80% sensitive. Although the low level of non-specific opacities associated with cigarette smoking may confound recognition of abnormalities caused by asbestos exposure, no association has been found between cigarette smoking and grade of asbestosis at necropsy.[21] Because of the limitations of the chest radiograph, various other diagnostic methods have been used. High-resolution computed tomography shows promise.[22]

Tests of pulmonary function are not diagnostically specific, but can provide important information about the functional status of people with pneumoconiosis. No single pattern of abnormality is found associated with either asbestosis or silicosis. Pulmonary-function tests can help define and measure impairment, but none are diagnostic of asbestosis or silicosis. Patients can show restrictive, mixed, or obstructive patterns on spirometry depending on the totality of their exposure history and their individual response. Reductions in diffusion capacity to carbon monoxide are common, especially in asbestosis. These reductions can occur with normal chest radiographs in exposed individuals.[16]

Bronchoalveolar lavage or biopsy, though useful in research settings and for clarification of ambiguous clinical presentations, is generally unnecessary for clinical diagnosis and management of the patient.

Treatment

There is no effective treatment to reverse the course of asbestosis or silicosis. Prevention of these diseases through elimination of hazardous exposure conditions is therefore of primary importance. The clinical approach for people with pneumoconiosis is directed at elimination of progression, amelioration of symptoms, improvement of overall condition, and reduction of risk of associated disorders.

Asbestosis and all forms of silicosis can progress even if exposure ceases.[6,23] Both are irreversible; however, some epidemiological and laboratory investigations suggest that early identification of disease followed by withdrawal from exposure may lead to more favourable long-term outcomes.[24] Since the risk of either disease relates largely to the cumulative exposure to respirable hazardous dust, removal from continuing exposure seems likely to provide some measure of protection against progression. Evidence for this assumption is sparse, however, since the variability in disease and ethical considerations would make a structured investigation of this issue difficult. Continued exposure to the causative agent seems imprudent; however, when cases of disease are manifested primarily by chest radiographic abnormality with little or no pulmonary dysfunction, a patient may wish to make an informed choice to continue his or her normal employment while using the maximum amount of environmental and personal protection available.

The approach to management of these patients does not differ from that for patients with other chronic pulmonary diseases. Vaccination against pneumococcal pneumonia and influenza, mobilisation of secretions, bronchodilator administration, nutritional advice, breathing retraining, home oxygen therapy, and graded exercise all may be helpful in some patients. All current smokers should stop smoking.

Pharmacological approaches have not yet proven beneficial in arresting or reversing progression of fibrosis, although there remains active research interest in these measures. Whole-lung lavage to remove retained dust is being investigated as a therapy for people with moderate to severe silicosis, but its use remains experimental as yet.[25] Lung transplantation has been done for a few patients with severe advanced disease.

Associated disorders

The most ominous diseases associated with asbestos exposure are cancers of the mesothelium or lung, even in the absence of fibrosis on chest radiography.[26] The risk of mesothelioma is unrelated to tobacco use, and no modifiable risk factors have been discovered. Tobacco

Panel 3: **WHO recommendations for health screening and public-health surveillance of workers exposed to asbestos or silica**

Silica (crystalline quartz) dust exposure

Chest radiograph: at baseline, after 2 or 3 years of exposure, then every 2–5 years

Spirometry and an updated symptom questionnaire: annually after the preplacement examination; alternatively they should be done at the same frequency as chest radiography.

Health surveillance should be lifelong.

Asbestos exposure

Chest radiograph: at baseline, then every 3–5 years for workers with less than 10 years since the first asbestos exposure; every 1–2 years for workers with longer than 10 years since first asbestos exposure; and annually for workers with longer than 20 years since first exposure. (Frequency may be adjusted depending on the age of the worker and the intensity and duration of dust exposure.)

Respiratory symptom questionnaire, physical examination, and spirometry: annually; alternatively, they should be done at the same frequency as chest radiography.

Health surveillance should be lifelong.

use seems to increase lung-cancer risk synergistically in asbestos workers. This risk can be reduced over time through smoking cessation. Diligent and continuing effort must be directed toward that goal in all smokers.

Lung-cancer risk is increased in people with silicosis.[27] The International Agency for Research on Cancer has lately determined that respirable crystalline silica found in the workplace is carcinogenic to human beings and categorised it as a group 1 carcinogen (http://www.iarc.fr./publications/vol68.htm).[28]

A risk of other cancers, particularly of the gastrointestinal tract, has been associated with asbestos exposure in some but not all studies. This association is biologically plausible, since fibres cleared through mucociliary action are ingested.

Exudative pleural effusions not associated with a malignant disorder generally occur (if at all) within 15 years of first exposure to asbestos. They may resolve spontaneously, leaving visible blunting of the costophrenic angle and, in some cases, visceral pleural thickening that may itself be associated with pulmonary dysfunction. Hyaline plaques on the parietal pleura, in some cases calcified, are also a result of asbestos exposure. Thickened pleura can compress adjacent lung parenchyma, forming what appears to be an isolated lesion or pseudo tumour.

Silicosis (and possibly silica exposure without radiographically visible silicosis) is associated with increased rates of infection with *Mycobacterium tuberculosis*.[29] Non-tuberculous mycobacterial infection also occurs.[30] Rheumatic diseases, particularly scleroderma, and chronic renal disease are more common among individuals exposed to silica than in the general population.[31,32]

Prevention

Effective prevention of asbestosis is a primary responsibility of employers. Physicians have an important supportive role, and may have responsibility for health surveillance of workers.

Exposure control—either through substitution of safer materials or the adoption of control technologies—is the only effective means of prevention of asbestosis or silicosis. Workplace exposures must be monitored frequently, and processes changed when harmful levels of dust are present. Where exposure is intermittent and unavoidable, as in building maintenance and repair, respiratory protection by personal respirators (provided as part of a comprehensive programme of fit testing and training), may be a useful adjunct to proper work practices.[33] If there is concern because of friable asbestos in public buildings, offices, or schools, environmental measurements should be taken and appropriate remedial-action plans instituted. Because encapsulation or removal of asbestos can generate high concentrations of fibres, workers involved in these activities should receive specific training. Some countries require certification of asbestos-abatement workers. In the USA, regular health assessment is required for workers exposed to asbestos.[34] Recommendations of a WHO expert committee for health screening and public-health surveillance of workers exposed to asbestos or silica are shown in panel 3. These recommendations assume that workers are free of symptoms or signs of disease, and that effective exposure controls are in place.[35]

Exposure to high levels of asbestos or respirable quartz is more common than understanding of the risk, therefore many patients present to physicians without knowledge of the source of disease. New cases of silicosis and asbestosis should be viewed as sentinel health events, worth reporting to public-health authorities to stimulate workplace investigations. Even though there is a long time between first exposure and disease recognition, hazardous exposures may continue in current workplaces of people with disease.[36] In the USA, the Council of State and Territorial Epidemiologists has recommended that silicosis be added as a condition reportable to the National Public Health Surveillance System. There is voluntary or mandatory public-health reporting in some countries. In addition, physicians may have specific responsibilities to report occurrences of occupational disease through programmes designed for compensation of injured workers.

Conclusion

Asbestosis, silicosis, and associated disorders are generally refractory to medical intervention. Cases of asbestosis and silicosis seen now are the result of the failure in the past to apply available information to prevent disease. Despite progress in many industries and occupations, current workplace conditions will inevitably lead to future disease unless a comprehensive and continuing commitment is made to exposure control. Physicians can contribute to disease prevention through accurate diagnosis and reporting of these conditions and through effective health communication.

References

1 Pulmonary asbestosis. *Lancet* 1930; i: 701–02.

2 Work-Related Lung Disease Surveillance Report 1996. Cincinnati, Ohio: National Institute for Occupational Safety and Health. DHHS (NIOSH) Publication, 1996: 96–134..

3 Merler E, Biggeri A, Olmastroni L, Arfaioli C, Buiatti E. Pneumoconiosis mortality in Italy. Tenth International Symposium Epidemiology in Occupational Health, ISEOH '94, Sept 20–23, 1994, Villa Erba, Como, Italy: 220 (abstr).

4 Work-Related Lung Disease Surveillance Report 1994. Cincinnati, Ohio: National Institute for Occupational Safety and Health, 1994: DHHS (NIOSH) publication no 94-120.

5 Becklake MR. The epidemiology of asbestosis. In: Liddell D, Miller K, eds. Mineral fibers and health. Boca Raton, FL, CRC Press, 1992: 104–19.

6 Steenland NK, Brown D. Silicosis among gold miners: exposure-response analysis and risk assessment. *Am J Public Health* 1995; **85:** 1372–77.

7 NIOSH alert: request for assistance in preventing silicosis and deaths in construction workers. Cincinnati, Ohio: National Institute for Occupational Safety and Health, 1996: DHHS (NIOSH) publication no 96–112.

8 NIOSH alert: request for assistance in preventing silicosis and deaths from sandblasting. Cincinnati, Ohio: National Institute for Occupational Safety and Health, 1992: DHHS (NIOSH) publication no 92-102.

9 Johnson WM, Busnardo MS. Silicosis following employment in the manufacture of silica flour and industrial sand. *J Occup Med* 1993; **35:** 716–19.

10 US Department of Interior—Bureau of Mines. Asbestos. Mineral Industry Surveys, Annual Review—1994, May 1995.

11 Pigg BJ. The uses of chrysotile. *Ann Occup Hyg* 1994; **38:** 453–58.

12 HEI. Asbestos in public and commercial buildings: a literature review and synthesis of current knowledge. Cambridge, MA: Health Effects Institute-Asbestos Research, 1991.

13 Vallyathan V, Shi X, Dalal NS, et al. Generation of free radicals from freshly fractured silica dust: potential role in acute silica-induced lung injury. *Am Rev Respir Dis* 1988; **138:** 1213–19.

14 Begin R, Cantin A, Masse S. Recent advances in the clinical assessment and pathogenesis of the mineral dust pneumoconioses. *Eur Respir J* 1989; **2:** 988–1001.

15 Rosenstock L. Asbestosis and asbestos-related pleural disease. In: Rosenstock L, Cullen MR, eds. Textbook of clinical occupational and environmental medicine. Philadelphia: WB Saunders, 1994: 254–63.

16 Bourbeau J, Ernst P, Chrome J, Armstrong B, Becklake MR. The relationship between respiratory impairment and asbestos-related pleural abnormality in an active work force. *Am Rev Respir Dis* 1990; **142:** 837–42.

17 Schwartz DA, Fuortes LJ, Galvin JR, et al. Asbestos-induced pleural fibrosis and impaired lung function. *Am Rev Respir Dis* 1990; **141:** 321–26.

18 International Labour Office. Guidelines for the use of ILO international classification of radiographs of pneumoconioses. Geneva: ILO, 1980.

19 Wagner GR, Attfield MD, Parker JE. Chest radiography in dust-exposed miners: promise and problems, potential and imperfections. *Occup Med: State of the Art Rev* 1993; **8:** 127–41.

20 Kipen HM, Lilis R, Suzuki Y, Valciukas JA, Selikoff IJ. Pulmonary fibrosis in asbestos insulation workers with lung cancer: a radiological and histopathological evaluation. *Br J Ind Med* 1987; **44:** 96–100.

21 Hnizdo E, Sluis-Cremer GK. Effect of tobacco smoking on the presence of asbestosis at postmortem and on reading of irregular opacities on roentgenograms in asbestos-exposed workers. *Am Rev Respir Dis* 1988; **138:** 1207–12.

22 Naidich DP. Pulmonary parenchymal high-resolution CT: to be or not to be. *Radiology* 1989; **171:** 22–24.

23 Becklake MR, Liddell FDK, Manfreda J, McDonald JC. Radiological changes after withdrawal from asbestos exposure. *Br J Ind Med* 1979; **36:** 23–28.

24 Begin R, Cantin A, Masse E. Influence of continued asbestos exposure on the outcome of asbestosis in sheep. *Exp Lung Res* 1991; **17:** 971–84.

25 Wilt JL, Banks DE, Weissman DN, et al. Reduction of lung dust burden in pneumoconiosis by whole-lung lavage. *J Occup Environ Med* 1996; **38:** 619–24.

26 Wilkinson P, Hansell DM, Janssens J, et al. Is lung cancer associated with asbestos exposure when there are no small opacities on the chest radiograph? *Lancet* 1995; **345:** 1074–78.

27 Smith AH, Lopipero PA, Barroga VR. Meta-analysis of studies of lung cancer among silicotics. *Epidemiology* 1995; **6:** 617–24.

28 IARC. Evaluation of carcinogenic risks to humans: silica, some silicates, coal dust, and para-aramid fibrils. Lyon: IARC (in press).

29 Snider DE. The relationship between tuberculosis and silicosis. *Am Rev Respir Dis* 1978; **118:** 455–60.

30 Cowie RL. The mycobacteriology of pulmonary tuberculosis in South African gold miners. *Tubercle* 1990; **71:** 39–42.

31 Sluis-Cremer GK, Hessel PA, Hnizdo EH, Churchill AR, Zeiss EA. Silica, silicosis, and progressive systemic sclerosis. *Br J Ind Med* 1985; **42:** 838–43.

32 Kallenbert CGM. Renal disease—another effect of silica exposure? *Nephrol Dial Transplant* 1995; **10:** 1117–19.

33 Bollinger NJ, Schutz RH. A NIOSH technical guide . . . NIOSH guide to industrial respiratory protection. DHHS (NIOSH) publication no 87–116.

34 Federal Register, Department of Labor, Occupational Safety and Health Administration, 29 CFR Parts 1910,1001, Occupational Exposure to Asbestos; final rule, August 10, 1994.

35 Wagner GR. Screening and surveillance of workers exposed to mineral dusts. Geneva: WHO, 1996.

36 Valiante DJ, Rosenman KD. Does silicosis still occur? *JAMA* 1989; **262:** 3003–07.