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Coal Workers' Lung Diseases and Silicosis

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COAL WORKERS' LUNG DISEASES

Introduction and History

Coal miners are at risk for developing several distinct clinical illnesses in relation to their occupational exposures. Historically, some names applied to these conditions were miners' asthma, phthisis, anthracosis, and in Scotland, miners' black lung. It was recognized early that these afflictions were related to the occupation of mining; however, it wasn't until the development of specialized techniques such as chest radiography, pulmonary function testing, the discovery of the tubercle bacillus, and sophisticated histological examination of tissue that respiratory diseases affecting miners could be separated and defined.

Coal workers' pneumoconiosis (CWP) is the parenchymal lung disease that results from the inhalation and deposition of coal mine dust, and the tissue's reaction to its presence. This occupational lung disease was first described in the early 1800s. In addition to CWP, coal mine dust exposures increase a miner's risk of developing chronic bronchitis, chronic obstructive pulmonary disease, and pathological emphysema. Radon gas exposures in coal mines may exceed recommended levels and represent a risk for cancers of the lung and larynx.

For a long time, the pneumoconiosis that affected coal miners was thought to be silicosis. In the 1930s, it was argued that silicosis, CWP, and bronchitis were distinct clinically and pathologically. Unfortunately, it was also suggested that coal dust was not harmful, in spite of reports of the adverse effects of coal dust among coal trimmers. It was not until washed coal, free of silica, was shown to produce a dust disease of the lungs in stevedores, who worked leveling coal in the holds of ships, that CWP was widely accepted as pathophysiologically distinct from silicosis.

In the United States, little attention was given to coal miners' respiratory diseases until the Public Health Service conducted a pilot prevalence study of CWP in the early 1960s. Since then, a large number of studies performed by

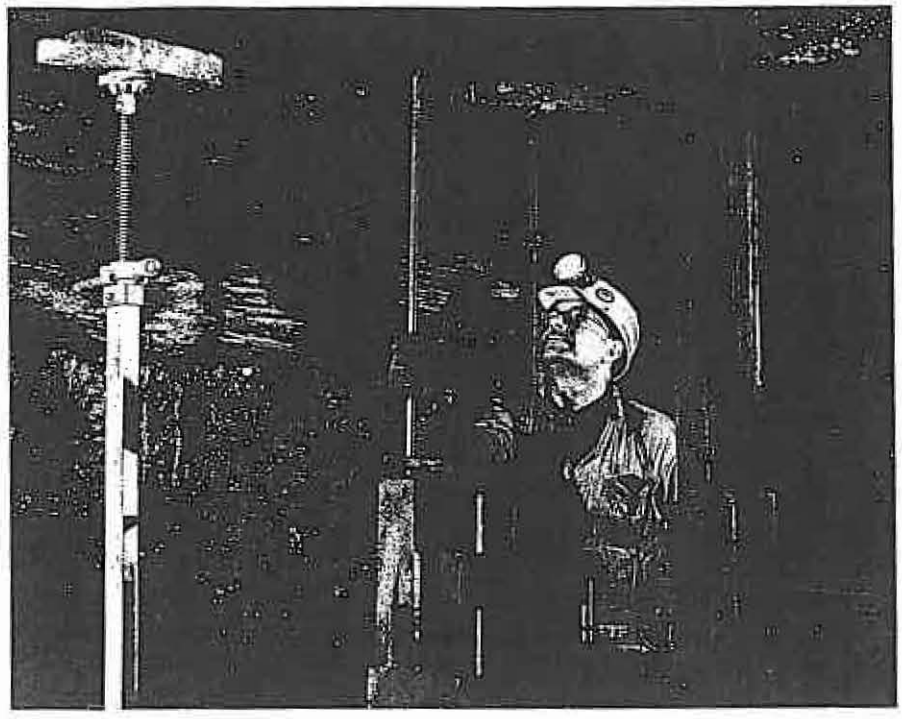

Figure 57-1 Roof bolting in underground coal mine. A potentially high-risk operation for respiratory exposures to airborne silica. *(Photo courtesy of U.S. Bureau of Mines.)*

the National Institute for Occupational Safety and Health (NIOSH) have greatly increased the knowledge and understanding of the nature and extent of lung diseases from coal mining in the United States.

Coal and Coal Mining

Coal is not a pure mineral. It is a spectrum of carbonaceous rocks derived from the accumulation of vegetation sedimented under swampy conditions and subjected to extreme pressure over long periods of time. Coals are characterized by rank, which relates to geologic age, hardness, carbon content, and the amount of heat released (BTUs) when they are burned. Thus, peat is the lowest rank (softest) coal, being geologically the newest, and anthracite is the highest rank (hardest) and oldest type of coal.

Coal may be found in outcroppings and in seams that vary from a few feet to several thousand feet below the surface. Surface or strip mining, which currently accounts for the majority of US coal production, involves removal of the overburden and mining the coal seams with large earth-moving equipment. In some areas of the eastern United States, mountaintop removal mining has become the dominant form of mining. Mountaintop mining involves first removal of all vegetation and soil, and then drilling and blasting through hundreds of feet of strata to access the coal seam. The excess rock and soil is placed in the steep stream beds along the mountainsides, creating areas called valley fills. Occasionally, surface mining is also performed by boring into coal outcrops with an auger. Dust levels in the air at surface mines are generally less hazardous than in underground mines, with a few notable exceptions (discussed below).

Deep mines produce somewhat less than half of the coal mined in the United States. Coal outcrops of sufficient size can be mined deep into the hillsides. Deep seams are accessed through vertical shafts drilled from the surface to the coal seam where the mining process then follows the seam through a series of more or less horizontal tunnels.

Not all coal mining jobs are equally exposed to respiratory hazards. In underground mines, airborne dust concentrations are highest at the coal-cutting face, where coal is removed from the intact seams. Face jobs include the loading of coal into transportation vehicles or train cars, and, depending on the techniques used in the mine, operation of continuous or long wall mining machines. Exposures to crystalline silica and thus risk of silicosis also occur in underground mines, particularly in miners involved in roof support, called roof bolting (Fig. 57-1), or drilling operations, and in motormen who operate underground coal trains and use sand for traction on the rails. Workers in some aboveground coal mining operations also may have important exposure to dusts. These include workers at tipples and preparation plants, where crushing, sizing, washing, and blending of coal is done, and coal is stored or loaded onto ships, railroad cars, or river barges. Workers at surface coal mines who work in or around the drilling rigs, to make holes in which explosives are placed, are exposed to silica and at risk for the development of silicosis rather than CWP.

Epidemiology of Lung Diseases in US Coal Miners

The first major survey of the health of American coal workers was conducted by the US Public Health Service from 1969 to

1971, evaluating symptoms, lung function, and chest radiographic findings. This study included over 9000 miners at 31 underground mines (2 were anthracite mines; 29 were bituminous mines). Participation in the survey was over 90 percent. The mines were chosen to represent different geographic areas, coal seams, and mining methods. After this initial study, subsequent surveys have been conducted to evaluate miners at these and other US mines.

Radiographic Findings

Radiographic data from the initial survey showed an overall prevalence of simple and complicated CWP of nearly 30 percent. There was variation by region of the country and the type (rank) of coal mined. Among eastern Pennsylvania anthracite (high rank) coal miners, 46 percent had simple and 14 percent had complicated CWP. In contrast, among the miners in the western plateau of Colorado and Utah mining a lower rank coal, only 5 percent had simple CWP, and none had the complicated form. Among underground miners, those working at the coal face and exposed to higher concentrations of coal mine dust, higher prevalences of CWP were found than among surface workers or those whose jobs caused them to enter the face area intermittently.

Results from multiple studies have clearly demonstrated that the prevalence of radiographic changes of simple CWP is related to the duration and intensity of dust exposure, and CWP can develop even at current dust levels. British studies also clearly showed that the attack rate (incidence of new cases) and the probability of progressing to a higher category of simple CWP were related to the mass of respirable dust to which the miner was exposed during his or her lifetime.

The same cannot be said for the complicated form of CWP, progressive massive fibrosis (PMF). Once an individual has inhaled sufficient coal mine dust into the lungs for the chest radiograph to be classified with at least International Labour Office (ILO) Category 2 pneumoconiosis (see below), the probability of progressing to the complicated form appears to be independent of any further dust exposure. The rate of progression to PMF appears to be influenced chiefly by the age at which the miner begins to show radiographic changes of CWP. Progression may also be influenced by the presence of a rheumatoid diathesis (see below for additional discussion of immunologic issues).

Enforcement of dust control measures in the United States, fully enacted in 1973, resulted in a declining pneumoconiosis attack rate. Subsequently, many miners with CWP retired, and follow-up studies have demonstrated a marked decline in the prevalence of CWP in active US miners. This was confirmed through the federally mandated chest radiograph surveillance program for underground US miners. Between 1973 and 1978, CWP was found in over one-third of the miners who participated in the program and had worked 25 years or more underground. By 1996–2002, only 1 in 20 (5.4 percent) of these miners showed radiographic evidence of CWP (Fig. 57-2). Between 1999 and 2002, chest radiographic surveillance examinations were also offered to many

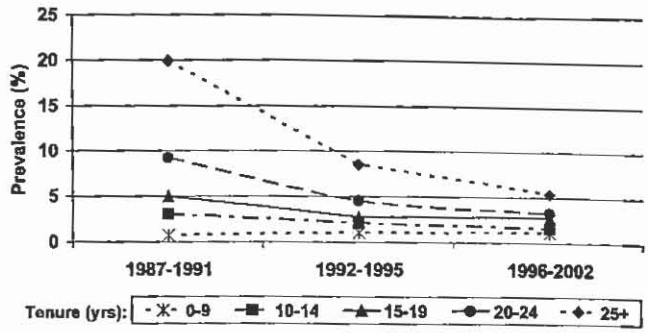

Figure 57-2 Trends in coal workers' pneumoconiosis (CWP) prevalence by tenure among examinees employed at underground coal mines, US Coal Workers' X-Ray Surveillance Program, 1987–2002.

surface coal miners, and 3.4 percent of miners with work tenure of 25 or more years demonstrated radiographic pneumoconiosis. In spite of the marked overall improvements in dust control in US underground coal mines, a recent evaluation of national surveillance data demonstrated onset of advanced CWP among miners who had worked their entire careers under the current dust enforcement regime. The authors of this report observed an increased risk of rapidly progressive pneumoconiosis among miners in smaller mines (less than 50 employees) and in certain geographic regions, and concluded that prevention measures in these settings were inadequate (Fig. 57-3).

Ventilatory Lung Function

Ventilatory function was also evaluated in the large studies of US miners mentioned above. Initial reports evaluated miners' lung function in comparison to the *radiographic findings* of CWP. Miners with complicated CWP were found to consistently show an important deficit in lung function. In contrast to the ventilatory findings associated with PMF, obstructive abnormalities were noted in miners with simple pneumoconiosis; however, the findings were not consistent, and with increasing category of simple CWP, the average functional decrement was small and variable. Subsequently, studies in the United States and Great Britain evaluated lung function with respect to the miners' *cumulative dust exposure*, and have helped to clarify the adverse effect of dust on coal miners' lung function. Miners show a progressively greater risk of lung function loss with increasing cumulative dust exposure, independent of the chest radiographic findings of CWP. The forced expiratory volume in 1 s (FEV_1) loss is most severe in those who work for many years at the dustiest jobs. Among smoking miners, the effects of tobacco smoke appear to be additive to the dust effect but no disproportionate dust effect has been noted in relation to tobacco use. Also, there is evidence that miners experience a more rapid loss in spirometric function parameters over their first few years of mining, with slower dust-related declines after that time.

In summary, the epidemiological evidence has shown that coal miners experience ventilatory lung function loss with increasing exposure to dust, either in the presence or

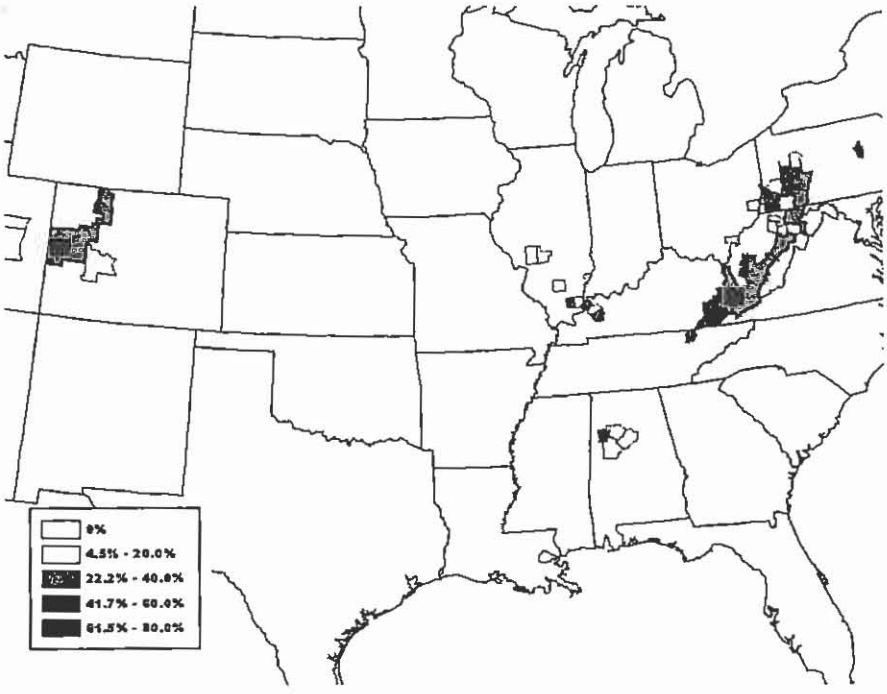

Figure 57-3 Proportion of miners with rapidly progressive CWP by county (not shown are counties with fewer than five miners evaluated).

absence of CWP. Among smoking miners, the effects of tobacco and dust appear to be additive. Although, on average, functional losses associated with dust are small, it is estimated that 35 years of work at the current dust limit will cause a clinically important FEV_1 loss in 8 out of 100 nonsmoking coal miners. When complicated CWP is present, an additional ventilatory deficit is likely.

Mortality

Studies of mortality in coal miners have been reported from the United States and Britain. Findings from both countries have been generally consistent, and reveal that the miners experience increased mortality attributable to pneumoconiosis, emphysema, and chronic bronchitis. Radiographic findings of advanced CWP (PMF) consistently affect mortality, especially in categories B and C, whereas among miners with simple CWP, decreases in survival were smaller. Accelerated FEV_1 decline is also associated with increased mortality from both cardiovascular and respiratory causes. Miners' risks of dying from the obstructive airway diseases of emphysema and chronic bronchitis exhibit a different geographic pattern than the mortality from CWP, suggesting that these dust effects have different mechanisms.

Pathology of Coal Miners' Lung Diseases

The coal macule is the primary lesion of simple CWP (Fig. 57-4). This lesion is essential for the pathological diagnosis of CWP. The lesion consists of a focal collection of coal dust in pigment-laden macrophages around the respiratory bronchioles and tapering off toward the alveolar duct. A fine network of reticulin is present in the early stages and may include a small amount of collagen depending upon the character of the dust. Centriacinar emphysema, the dilation and injury of lung gas exchange units, is observed with increased prevalence in the lungs of coal miners. The severity is proportional to the miner's cumulative dust exposure. Focal emphysema is the form of centriacinar emphysema that is seen as an integral part of the simple lesion of CWP. It is characterized by enlargement of the airspaces immediately adjacent to the dust macule. The pathological severity of the emphysema increases with increasing lung dust retention. Muscular thickening of pulmonary arteries, in conjunction with hypertrophy of the right ventricle, can be observed with both simple and complicated CWP, and is increasingly prominent when CWP is associated with other lung disorders. Pathological changes in the airways consistent with chronic bronchitis, including enlargement of mucous glands, have also been noted in miners' lungs.

With increasing dust exposure, due to the normal clearance mechanisms being overwhelmed, the lung lesions increase in size and number. These larger fibrotic lesions are called coal nodules and are palpable in lung specimens, whereas coal macules are not. Palpable coal nodules are classified as micronodular up to 7 mm in diameter and macronodular from 7 mm and larger.

Classic silicotic nodules have been found in the lungs of 12 percent of coal miners at autopsy. Other patterns of interstitial disease (usual interstitial pneumonia, UIP) have also been reported among coal miners either alone or in combination with the typical pathology of the pneumoconioses.

Complicated CWP or PMF is diagnosed when one or more nodules in a lung specimen are noted to attain a size of 2 cm or greater in diameter. The 2 cm is an arbitrary choice of a minimal diameter that permits better correlation with clinical and radiographic measurements. (In fact, when coal-induced radiographic shadows are >1 cm, PMF is said to be present.)

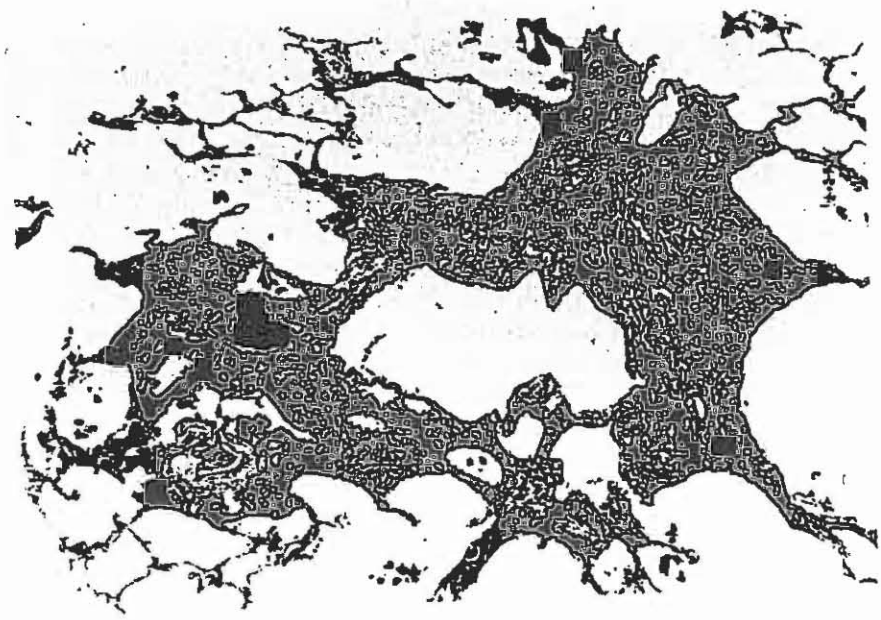

Figure 57-4 A coal macule, microscopic section. *(Courtesy of Dr. Val Vallyathan, National Institute for Occupational Safety and Health, Morgantown, WV.)*

Lesions are solid, heavily pigmented, rubbery to hard, and occur most commonly in the apical posterior portions of the upper lobes or the superior segments of the lower lobes. They tend to occur symmetrically, but may be asymmetrical, and may cavitate. Airways and vessels adjacent to the lesions may be distorted, and within the lesions, they are destroyed. PMF generally occurs in association with background pathological changes of simple CWP.

Clinical Features of Coal Workers' Lung Diseases

Chronic cough and sputum production are more common with increasing dust-exposure, regardless of the presence or absence of simple pneumoconiosis. These symptoms are likely related to bronchitic changes in the large airways, including thickening of the airway wall with mucous gland enlargement and hypersecretion that result from continued inhalation of dust particles presenting a chronic burden to the mucociliary escalator. Some miners with simple pneumoconiosis may have no related symptoms or physical signs, but with severe airflow obstruction or advanced pneumoconiosis, dyspnea, cough, and sputum production are frequent. Edema of the lower extremities, and findings consistent with cor pulmonale, may occur. Melanoptysis (expectoration of black sputum) occasionally results from excavation of a PMF lesion.

Clubbing and crackles are not generally considered features of coal miners' lung diseases, and if noted, should prompt further studies. However, a series of 38 cases of a chronic interstitial pneumonia among coal miners has recently been reported. The clinical findings in these atypical cases included crackles, finger clubbing, restrictive impairment, diffusion block, and neutrophilic bronchoalveolar lavage (BAL). CWP has not been associated with increased risk for development of coexisting mycobacterial infection, in contrast to silicosis. However, a minority of miners show classic silicotic nodules. Certainly, progressive infiltrates or cavitary lesions in PMF should prompt examination of the sputum for typical and atypical mycobacteria.

Radiology of Coal Workers' Pneumoconiosis

The diagnosis of CWP can be made with confidence, without histological confirmation, in the presence of an adequate history (at least 5 to 10 years) of coal mine dust exposure and a characteristic chest radiograph. The radiograph in simple pneumoconiosis shows small opacities, ranging in size from pinhead up to 1 cm in diameter. Rounded nodules predominate and tend to appear first in the upper zones and involve middle and lower zones as the number of opacities increase. PMF is characterized by one or more large opacities greater than 1 cm. Upper lobe predominance is also typical in complicated pneumoconiosis. High-resolution computed tomography (HRCT) scanning in coal miners may reveal parenchymal nodules and emphysema when standard radiographs are normal. In atypical cases, CT scans may show ground-glass opacities and honeycombing, at times without nodular findings typical of CWP. Radiographic evidence of bronchiectasis has also been reported in coal miners, particularly among those with CWP.

Several schemes have been used for classifying the radiographic shadows of pneumoconiosis in epidemiological studies; currently the ILO 2000 classification is the most widely accepted. When using the ILO system, simple pneumoconiosis is divided into major categories 1, 2, and 3 according to the profusion of small opacities in the lung fields. Each major category, including 0, is subdivided into 3 subcategories, providing a full range of 12 categories of simple CWP. A reading of category 1/0 indicates the definite presence of opacities consistent with pneumoconiosis. Complicated pneumoconiosis (PMF) is divided into categories A, B, and C, based on

the size of the large opacities. Findings of collapse, consolidation, and emphysema may be associated with the shadows of complicated pneumoconiosis.

The clinician may be presented with the diagnostic dilemma of distinguishing primary or metastatic lung neoplasia from an unusual presentation of PMF or Caplan's syndrome. When typical large opacities of PMF occur symmetrically and bilaterally on a background of simple CWP, one can be confident that the lesions are unlikely to represent neoplastic disease. Prior radiographs from medical screening programs are often obtainable, and can help confirm stability or progression over a long time interval. Positron emission tomography with fluorodeoxyglucose (FDG-PET) scanning may be useful in differentiating PMF lesions from malignancy when the mass lesion has a low level of glucose metabolism, although some massive pneumoconiotic lesions may demonstrate an uptake of fluorodeoxyglucose similar to neoplasms. On magnetic resonance imaging (MRI) with contrast enhancement, the pattern of change over time in signal intensity has been reported to be a differential criterion in this setting. When the imaging workup is equivocal, the differentiation of PMF from neoplasm may be impossible without a biopsy. Hemorrhagic complications may occur during biopsy of PMF lesions due to their vascular nature.

Lung Function and Respiratory Impairment in Coal Miners

Coal mine exposures may result in several pathological processes (simple and complicated CWP, silicosis, chronic bronchitis, mineral-dust airway disease, emphysema, and dust-related airflow limitation), each of which may contribute to adverse physiological consequences. In an individual miner, the pattern and severity of impairment found will be related to such recognized factors as the intensity and duration of respirable dust exposure, geologic factors (e.g., coal rank, silica content), residence time of dust in the lung, and exposure to other respiratory hazards (e.g., tobacco smoke). In miners with airway hyperresponsiveness, greater functional deficits and an increased risk of symptoms may be expected. Several other mining exposures may also contribute lung function loss in coal miners, including gases from underground explosive blasting and aerosols of potentially contaminated water used for dust control. Additional factors implicated in underground coal miners' accelerated lung function declines include weight gain, childhood pneumonia, and childhood exposures to environmental tobacco smoke.

Ventilatory Function

Epidemiological studies, as discussed above, have extensively documented the occurrence of exposure-related deficits in FEV_1 and forced vital capacity (FVC) in coal miners. The magnitude of the average dust effect has varied between studies. Over a working lifetime, average predicted losses in FEV_1 under current US dust standards ranged from 124 ml to 610 ml. Subgroups of miners experience a more severe effect, and from 6 to 8 percent of miners may be expected to develop clinically important airflow limitation. For example, a more severe effect of dust on loss of lung function was observed in a group 199 men who had chosen to leave coal mine work. These dust effects can be compared with those of another recognized respiratory hazard, cigarette smoking. For example, Attfield observed that when 1072 miners' lung function was followed over an 11-year period, a year of work at coal face jobs resulted in lung function loss essentially similar to that due to smoking for 1 year. When tenure in less dusty work was included in the analysis, mine dust exposure resulted in average lung function losses about 38 percent of that attributable to smoking (average 13 cigarettes per day).

Physiological findings consistent with small airways disease have been noted to develop in nonsmoking miners, consistent with the pathological findings with dust deposition.

Gas Exchange

Diffusing capacity has been studied in relation to radiographic changes of coal worker's pneumoconiosis. The small rounded opacities seen in miners with simple CWP have not generally been associated with measurable reductions in D_{LCO}. However, in subgroups of miners, abnormal diffusing impairment has been correlated with radiographic changes. Thus, gas transfer is often low when the large opacities of complicated CWP are present and may also be reduced in miners who show either predominantly pinpoint opacities ("p" type by the ILO classification) or small irregular opacities on their chest radiograph.

Gas exchange on exercise has also been investigated in coal miners. Many of the reports have been based in patients referred for disability evaluations, and thus suffer from ill-defined selection biases. Exposure-response relationships are also unclear with respect to findings in these series. Exertional hypoxia, pulmonary arterial hypertension, and excess ventilation have frequently been observed in miners, particularly those with complicated CWP or airflow obstruction. However, the proportion of miners who show exertional gas exchange abnormalities in the absence of either PMF or clinically important airflow obstruction is still a topic of investigation.

Immunology of Coal Workers' Pneumoconiosis

The potential role of immunologic factors in mineral dust pneumoconioses was noted by Caplan who observed the association between distinctive nodular radiographic opacities in the lungs of Welsh coal miners and rheumatoid arthritis. This observation was extended when similar radiographic appearances were described in miners without arthritis but with circulating rheumatoid factor (RF). Increased prevalence of circulating RF among miners with complicated pneumoconiosis (PMF) has also been reported.

Soutar et al reported on a study of serum antinuclear antibodies (ANA) and RF among 109 miners with radiographic evidence of pneumoconiosis attending the London

Pneumoconiosis Panel. They reported positive ANA in 17 percent and RF in 10 percent of the miners whereas about 2 to 3 percent positive ANA was expected in a healthy male population. The prevalence of ANA was 9 percent in simple CWP and 27 percent in those with category C (PMF). A similar trend was seen with RF, ranging from 6 percent in simple CWP to 18 percent in category C. Combining both ANA and RF resulted in prevalences of positive results in 13 percent of the miners with simple CWP and 45 percent of those with category C CWP.

In 1973 Lippmann et al reported a prevalence study of circulating ANA and RF among coal miners in the United States. Sera from 207 coal miners were examined. Of the 196 miners with radiographic opacities of pneumoconiosis, 9 were positive for RF, while 34 percent had positive ANA. There were regional variations in ANA that seemed to parallel the prevalences of radiographic changes; namely, prevalence was higher in anthracite miners and lower in bituminous miners.

Studies of serum immunoglobulins were conducted by Hahon et al among 155 US coal miners with chest radiographs demonstrating simple CWP, Caplan's syndrome, or PMF. They found significantly higher serum concentrations of C3, α_1-antitrypsin, IgA and IgG in anthracite miners than in bituminous miners with PMF. Compared to normal controls, the miners' C3, α_1-antitrypsin, and IgG and IgG values were elevated. There were few differences in these serum proteins among the miners with simple CWP. The authors did not find any association between the elevated immunoglobulins and FEV_1.

There have been few studies of the peripheral lymphocytes in coal miners. Dauber et al examined the lymphocyte function of 15 miners with pneumoconiosis. They found decreased numbers of both T and B lymphocytes in the peripheral blood in the miners compared to controls. They also found that cell function, as determined by response to stimulation by concavalin A, was lower in the miners with complicated CWP than in either miners with simple CWP or controls.

Autoantibodies directed at lung collagen and reticulin have been identified in the sera of coal miners. The lung autoantibodies tend to reside in the serum IgA. It is not clear whether these autoantibodies participate in the CWP reaction in the lungs or simply represent epiphenomena.

Special Studies

Bronchoalveolar lavage (BAL) has been used in studying mechanisms in the pulmonary reactions in CWP. Rom et al studied 15 symptomatic, nonsmoking coal miners with simple CWP by BAL. They found no significant difference between miners with CWP and controls in the number of cells recovered, the percentage distribution, and in the release of superoxide anion or hydrogen peroxide. This contrasted with the findings in subjects with asbestosis and silicosis whose values for spontaneous release of oxidant superoxide and hydrogen peroxide were significantly higher than controls. With regard to fibronectin and alveolar macrophage–derived growth factor, the miners with CWP had values that were elevated above controls and not different from the values obtained in subjects with asbestosis and silicosis.

Wallaert demonstrated significantly increased total number of lung cells recovered from miners with simple and complicated CWP, as well as increased percentages of alveolar macrophages, lymphocytes, and neutrophils. Alveolar cells from miners with simple and complicated CWP spontaneously released significantly more superoxide demonstrated by chemiluminescence than controls.

Management of Coal Workers' Lung Diseases

There is no specific therapy for CWP. The primary prevention of lung disease in miners must include continuing efforts at reducing coal mine dust exposure. Medical management is best directed at prevention, early recognition, and treatment of complications. The major clinical challenges are the recognition and management of airflow obstruction, respiratory infection, hypoxemia, respiratory failure, cor pulmonale, arrhythmias, and pneumothorax.

Improved mining methods and lower dust levels appear to be reducing exposures and new cases of both simple and complicated pneumoconiosis. Medical surveillance programs, using chest radiographs, allow early recognition of workers with simple pneumoconiosis. Workers with simple pneumoconiosis should be encouraged to exercise their rights to frequent dust measurements, and transfer to low dust jobs when necessary. Any worker with PMF should be carefully advised about the risks of further dust exposures.

Workers presenting with respiratory symptoms should have careful evaluation. Initial history and examination should be supplemented by chest radiograph, spirometry with bronchodilators, diffusing capacity, electrocardiogram, and resting arterial blood gas measurement as indicated. A thorough initial database allows accurate assessment of the worker's respiratory health and serves as a starting point for observing the response to therapy or progression of disease.

For miners who smoke, cessation is important regardless of symptoms, radiographic abnormalities, or functional status. Physician encouragement to stop smoking should be supplemented by support from smoking cessation groups, use of nicotine replacement, pharmacologic aids, and behavior modification techniques.

Symptomatic reversible airflow obstruction may benefit from treatment with inhaled and oral bronchodilators. Patients with severe obstruction and inadequate improvement from the usual measures should be considered for a monitored trial of corticosteroids. If improvement is objectively documented, continuation of inhaled and, rarely, oral steroids may be of benefit.

Hypoxemia can be a serious complication in advanced pneumoconiosis. It may be present at rest, with exercise, or during sleep. Chronic hypoxemia can lead to additional complications including polycythemia, pulmonary hypertension, cor pulmonale, and cerebral dysfunction. Therapy with low flow oxygen is indicated when arterial oxygen tension is less

than 55 torr. Oxygen therapy in this setting may improve exercise tolerance, reduce dyspnea, and prevent arrhythmias, polycythemia, and heart failure.

Patients with significant airflow obstruction or PMF should receive appropriate immunization with influenza and pneumococcal vaccines. Bacterial and viral episodes of bronchitis or pneumonia should be promptly recognized and appropriately treated.

Patients with complicated pneumoconiosis, especially those who have been exposed to silica as well as coal mine dust, deserve special attention with regard to mycobacterial infection. Patients with a history of weight loss, fever, sweats, or malaise should be promptly investigated with chest radiographs and sputum examination for acid-fast bacilli stains and cultures. Occasionally, the sputum may be negative and mycobacterial infection can only be documented by fiberoptic bronchoscopy with brushings and washings. Active tuberculosis in patients with CWP can, in general, be successfully treated with the usual drug regimens provided rifampin is one of the drugs used. However, some authorities would recommend that in coal miners with a significant history of concurrent silica exposure (such as motormen, roof bolters, drillers, and shaft development workers), the treatment for tuberculosis may need to be more aggressive, and long-term follow-up is indicated in view of several reports of recurrent pulmonary tuberculosis in patients with PMF after completion of apparently adequate therapy.

Respiratory failure may complicate advanced disease in coal miners, as it does in other chronic obstructive respiratory diseases. Ventilatory support measures are indicated when the failure is precipitated by a treatable complication. The application of ventilatory support measures should be clarified in advanced directives before the need arises.

Clinicians need to assess the contribution of occupational dust exposures to ventilatory impairments in their patients with a history of coal mine exposure. Factors which can assist in this include a careful work history with documentation of the mining region, duration and categories of coal mine employment, as well as the duration and intensity of any tobacco smoking. Factors associated with an increased risk of a clinically important dust effect are a history of prolonged exposures in dusty jobs, exposures to higher rank coals, a younger age at first employment, and the finding of radiographic changes of CWP. Physicians should assist their patients with job-related impairments in obtaining appropriate compensation through local and national programs.

SILICOSIS

Introduction

Silicosis is a fibrosing disease of the lungs caused by the inhalation, retention, and pulmonary reaction to crystalline silica. Despite knowledge of the cause of this disorder (inhalation of dust containing respirable crystalline silica), this serious and potentially fatal occupational lung disease remains prevalent throughout the world. Silica, or silicon dioxide, is the predominant component of the Earth's crust. Occupational exposure to silica particles of respirable size (aerodynamic diameter of 0.5 to 5 microns) is associated with mining, quarrying, drilling, tunneling, and abrasive blasting with quartz-containing materials (sandblasting). Silicosis risk is also recognized in masonry and refractory operations, cement and concrete production, and highway repair, and as well as during work in potteries, foundries, and dental laboratories. Among ornamental stone carvers in Brazil, the prevalence of disease remains over 50 percent. Because crystalline silica exposure is so widespread, and silica sand is an inexpensive and versatile component of many manufacturing processes, millions of workers throughout the world are at risk of disease. The disease is often unrecognized and underreported, and thus its true prevalence is substantially underestimated. In the United States, fatal cases of silicosis and multiple cases from the same worksite continue to be recognized.

Definition

Silicosis is an occupational lung disease attributable to the inhalation of silicon dioxide, commonly known as silica, in crystalline forms, usually as quartz, but also as other important crystalline forms of silica (i.e., cristobalite and tridymite). These forms are also called "free silica" to distinguish them from the silicates. The silica content in different rock formations, such as sandstone, granite, and slate, varies from 20 percent to nearly 100 percent.

Workers in High-Risk Occupations and Industries

Although silicosis is an ancient disease, new cases are still reported in both the developed and developing world. In the early part of the twentieth century, silicosis was a major cause of morbidity and mortality. Contemporary workers are still exposed to silica dust in a variety of occupations. When new technology lacks adequate dust controls, exposures may be more hazardous and dust levels higher than in nonmechanized work settings. Whenever the Earth's crust is disturbed and silica containing rock or sand is used or processed, there are potential respiratory risks for workers. The development of silicosis is continuing to be reported among workers from industries and work settings not previously recognized to offer a risk of this disease, reflecting the nearly ubiquitous presence of silica. The type of silica exposure appears to affect the risk of disease—settings such as drilling or sandblasting in which silica is freshly fractured, represent an increased risk of silicosis. Even brief periods of exposure to high levels can result in a clear increased lifetime risk for disease. The development and progression of silicosis frequently occurs after exposures have ceased. In countries throughout the world, mining, quarrying, tunneling, abrasive blasting, construction, and foundry work continue to present major risks

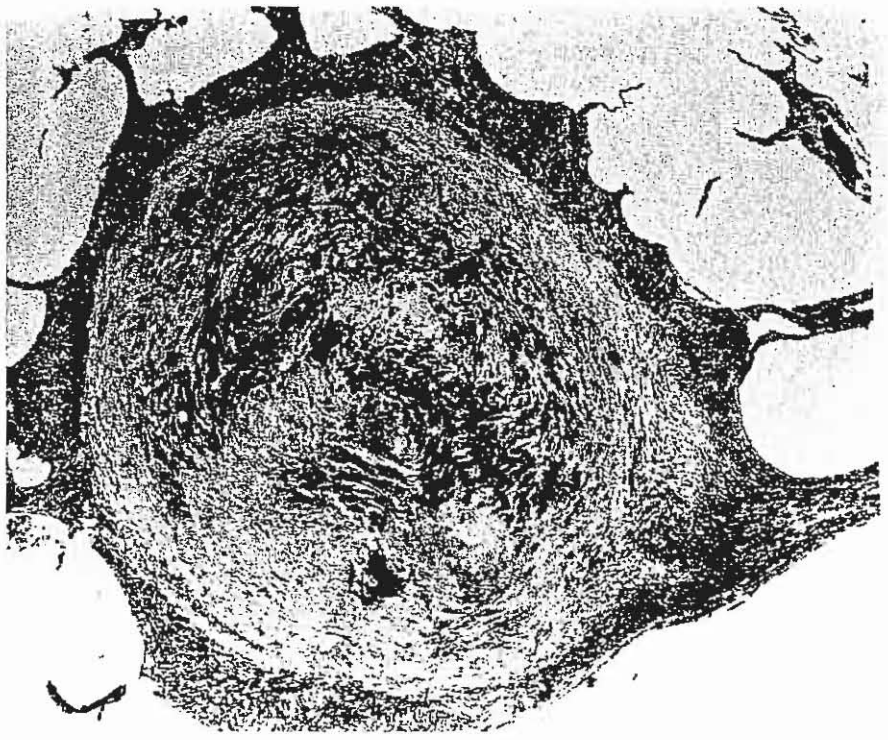

Figure 57-5 Lung pathology showing classic silicotic nodule. (See text for description.) *(Courtesy of Dr. Val Vallyathan, National Institute for Occupational Safety and Health, Morgantown, WV.)*

for silicosis, and important exposures continue to occur, even in developed nations.

Forms of Silicosis: Exposure History and Clinicopathological Descriptions

Chronic, accelerated, and acute forms of silicosis have been well characterized. These clinical and pathological expressions of the disease reflect differing exposure intensities, latency periods, and natural histories. The chronic or classic form usually follows one or more decades of exposure to respirable dust containing quartz. The accelerated form results from heavier exposures, often with a latency of 5 to 10 years. Accelerated silicosis develops more rapidly than the chronic form and generally progresses inexorably even after silica exposure is interrupted. The acute form of silicosis is a consequence of intense exposures to high levels of respirable dust which contain a significant proportion of silica. The reported exposure period is usually from several months up to about 5 years, and the clinical course is usually one of rapid progression.

Chronic (or classic) silicosis may be asymptomatic or result in insidiously progressive exertional dyspnea or cough (often mistakenly attributed to the aging process). A latency of 15 years or more since onset of exposure is common. Radiographically, it presents with small (less than 10 mm) rounded opacities predominantly in the upper lung zones. The pathological hallmark in the lungs of patients with the chronic form is the silicotic nodule. The lesion is characterized by a cell-free central area of concentrically arranged, whorled hyalinized collagen fibers, surrounded by cellular connective tissue with reticulin fibers (Fig. 57-5). When examined under polarized light, birefringent particles are typically seen most prominently in the periphery of the silicotic nodule. Electron microscopy using specialized techniques can identify the specific mineral content of the particles, but is rarely needed for routine diagnostic purposes. Silicotic nodules in the visceral pleura, regional lymph nodes, and occasionally in other organs, may also result from silica exposure. One or more groups of the small lung nodules of chronic silicosis may coalesce and result in larger shadows on the chest radiograph (greater than 10 mm), heralding the onset of complicated or conglomerate silicosis (often referred to as progressive massive fibrosis). This progressive illness may occur even after exposure to silica-containing dust has ceased.

Progressive massive fibrosis (*PMF*) is frequently associated with a clinically important compromise of lung structure and function, and as a consequence, symptoms of exertional dyspnea and reduced functional status. This form of silicosis is characterized by nodular opacities greater than 1 cm on the chest radiograph (Fig. 57-6). Common laboratory findings include a diminished carbon monoxide diffusing capacity, reduced arterial oxygen tension at rest or with exercise, and a demonstrable restrictive pattern on spirometry and lung volume measurement. Concomitant dust-induced bronchitis or distortion of the bronchial tree may also result in productive cough or airflow obstruction. Recurrent bacterial infection, not unlike that seen in bronchiectasis, may occur. Weight loss and cavitation of the large opacities should prompt concern for tuberculosis or other mycobacterial infection.

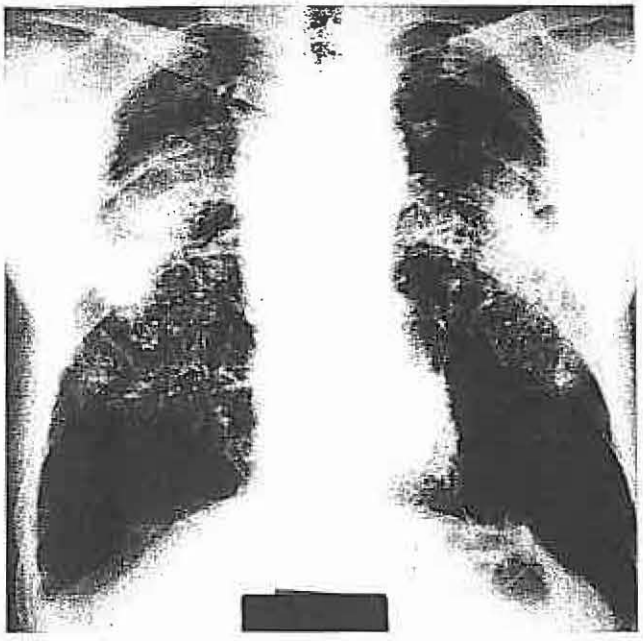

Figure 57-6 Complicated silicosis demonstrating progressive massive fibrosis.

Pneumothorax may be a life-threatening complication, since the fibrotic lung may be difficult to re-expand. Hypoxemic respiratory failure with cor pulmonale and congestive heart failure are common terminal findings.

Accelerated silicosis results from exposures that are more intense and of shorter (5 to 10 years) duration than in the chronic form, while symptoms, radiographic findings, physiological measurements, and lung pathology are similar. Deterioration in lung function is more rapid, and many workers with accelerated disease develop superimposed mycobacterial infection. Findings consistent with autoimmune diseases, including scleroderma, rheumatoid arthritis, or systemic lupus, may be seen in association with silicosis, more often in the accelerated type. The progression of radiographic abnormalities and functional impairment can be very rapid when autoimmune disease occurs with silicosis.

Acute silicosis may develop within a few months up to about 5 years after a massive inhalation of silica. Dramatic dyspnea, weakness, and weight loss are often presenting symptoms. The radiographic findings differ from those in the more chronic forms of silicosis, and are dominated by a diffuse alveolar filling pattern, with a lower lung zone predominance. Air bronchograms may be present. Histological findings similar to pulmonary alveolar proteinosis have been described, and extrapulmonary (renal and hepatic) abnormalities are occasionally reported. The usual clinical course is rapid progression to severe hypoxemic ventilatory failure and death.

Tuberculosis may complicate all forms of silicosis, but people with acute and accelerated disease may be at higher risk. Silica exposure alone, even without silicosis may also predispose to this infection. *Mycobacteria tuberculosis* is the usual organism, but nontuberculous (atypical) mycobacteria are also seen.

Even in the absence of radiographic silicosis, silica-exposed workers may also develop other diseases associated with occupational dust exposure, such as chronic bronchitis and the associated emphysema. Progressive declines in lung function have been documented in workers from inhalation of silica and other occupational mineral dust exposures.

Pathogenesis and the Association with Tuberculosis

The precise mechanism of silica toxicity is uncertain, but it is thought to be mediated by generation of reactive oxygen species, both by the surface of silica particles themselves and by activation of alveolar macrophages. The nature and the extent of the biologic response are in general related to the intensity of the exposure; however, there is growing evidence that freshly fractured silica may be more toxic than aged silica-containing dusts, perhaps related to reactive oxidant radical groups on the surface cleavage plane. An abundance of evidence implicates the interaction between the pulmonary alveolar macrophage and silica particles deposited in the lung. Release of chemotactic factors and inflammatory mediators result in recruitment of polymorphonuclear leukocytes, lymphocytes, and additional macrophages. Fibroblast-stimulating factors are released that promote hyalinization and collagen deposition. The resulting pathological lesion is the silicotic nodule, containing a central acellular zone with silica particles surrounded by whorls of collagen and fibroblasts, and an active peripheral zone composed of macrophages, fibroblasts, plasma cells, and additional free silica (Fig. 57-5).

The initiating toxic insult may occur with minimal immunologic reaction; however, a sustained immunologic response may be important in some of the chronic manifestations of silicosis. For example, ANA are noted in accelerated silicosis occurring with scleroderma, as well as in other collagen diseases among workers who have been exposed to silica. The susceptibility of silicotic workers to infections, such as tuberculosis and *Nocardia asteroides*, is likely related to the toxic effect of silica on pulmonary macrophages.

The link between silicosis and tuberculosis has been recognized for nearly a century. Again, people with acute silicosis appear to be at considerably higher risk.

Clinical Picture of Silicosis

When silicosis is symptomatic, the primary symptom is usually dyspnea, first noted with activity or exercise and later, as the functional reserve of the lung is lost, also reported at rest. However, in the absence of other respiratory disease, shortness of breath may be absent and the presentation may be an asymptomatic worker with an abnormal chest radiograph. The radiograph may at times show quite advanced disease with only minimal symptoms. The appearance or progression of dyspnea may herald the development of complications including tuberculosis, airways obstruction, PMF, or cor pulmonale. Productive cough is often present, secondary to chronic bronchitis from occupational dust exposure, tobacco use, or both. Cough may at times also be attributed to

pressure from large masses of silicotic lymph nodes on the trachea or mainstem bronchi.

Other chest symptoms are less common than dyspnea and cough. Hemoptysis is rare and should raise concern for complicating disorders, such as pulmonary neoplasms or mycobacterial infection. Wheeze and chest tightness may occur in the presence of silicosis, but usually as part of associated obstructive airways disease or bronchitis. Chest pain and finger clubbing are not features of silicosis. Systemic symptoms, such as fever and weight loss, suggest complicating infection or neoplastic disease. Advanced forms of silicosis are associated with progressive respiratory failure with or without cor pulmonale. Few physical signs may be noted unless complications are present.

Radiographic Patterns in Silicosis

The earliest radiographic signs of uncomplicated silicosis are generally small, rounded opacities. These can be categorized using the ILO International Classification of Radiographs of Pneumoconioses by size, shape, and profusion category. In silicosis, rounded opacities of the "q" and "r" type dominate. Other patterns have also been described, including linear or irregular shadows. The opacities seen on the radiograph represent the summation of pathological silicotic nodules and associated changes. They are usually found to predominate initially in the upper lung zones and may progress to involve other zones. Hilar lymphadenopathy is also noted, sometimes in advance of nodular parenchymal shadows. Eggshell calcification of the lymph nodes is strongly suggestive of silicosis, although this feature is uncommon. PMF is characterized by the formation of large opacities. These are categorized by size using the ILO classification as categories A, B, or C. The large fibrotic lesions of PMF tend to contract to the upper lung zones, leaving areas of compensatory emphysema at their margins and in the lung bases. As a result of this process, small, rounded opacities that previously were evident on the radiograph may become less visible or at times disappear. Pleural abnormalities are not common on routine chest radiographs with silicosis, however, CT scanning often documents localized pleural thickening, particularly in association with conglomerate lesions. Pleural effusions are less frequently noted. Large opacities may pose a concern regarding neoplasm. The radiographic distinction between PMF lesions and lung malignancies may be difficult, particularly if previous radiographs are unavailable for comparison. As in complicated CWP, FDG-PET scanning may sometimes be helpful in this distinction. Although ischemic necrosis may occur in large silicotic lesions, the onset of cavitation or a rapid change in the radiographic appearance should prompt a search for active mycobacterial disease. Acute silicosis may present with a radiologic alveolar filling pattern with rapid development of PMF or complicated mass lesions.

Lung Functional Abnormalities in Silicosis

Pulmonary function tests, such as spirometry and diffusing capacity, are helpful for the clinical evaluation of people with suspected silicosis. Spirometry may also be of value in early recognition of the health effects from occupational dust exposures, as it can detect physiological abnormalities that may precede radiographic changes. No specific or characteristic pattern of ventilatory impairment is present in silicosis. Spirometry may be normal, or when abnormal, the tracings may show obstruction, restriction, or a mixed pattern. Obstruction may indeed be the more common finding. Silica and mixed dust exposures may lead to clinically important airflow limitation independent of radiographic abnormality. Functional changes tend to be more marked with advanced radiologic categories. However, no good correlation exists between radiographic abnormalities and ventilatory impairment, and workers experience lung function loss proportionate to the duration and intensity of silica dust exposure. Diffusing impairment may also occur in the absence of ventilatory impairment. In acute and accelerated silicosis, functional changes generally occur earlier, are more marked, and the progression is more rapid. In acute silicosis, radiographic progression is accompanied by increasing ventilatory impairment and gas exchange abnormalities, which leads to respiratory failure and eventually to death from intractable hypoxemia.

Complications and Special Diagnostic Issues in Silicosis

With a history of sufficient exposure and a characteristic radiograph, the diagnosis of silicosis is generally not difficult to establish. Challenges arise only when the radiologic features are unusual or the history of exposure is not recognized. Lung biopsy is rarely required to establish the diagnosis. However, tissue samples are helpful in some clinical settings when complications are present or the differential diagnosis includes tuberculosis, neoplasm, or PMF. Biopsy material should be sent for culture, and in research settings, dust analysis may be a useful additional measure. When tissue is required, open or thoracoscopic lung biopsies are generally necessary for adequate material for examination, and to assure satisfactory hemostasis.

Vigilance for infectious complications, especially tuberculosis and other mycobacteria, cannot be over-emphasized, and symptoms of change in cough or hemoptysis, and fever or weight loss should trigger a workup to exclude this treatable problem. Nocardia and fungal infections are also reported in association with acute silicosis.

The International Agency for Research on Cancer has classified crystalline silica as a 2A carcinogen based on "sufficient" evidence of carcinogenicity in laboratory animals and "limited" evidence of carcinogenicity in humans. Uncertainty over the pathogenic mechanisms for the development of lung cancer in silica-exposed populations exists, and the possible relationship between silicosis (or lung fibrosis) and cancer in exposed workers continues to be studied. Regardless of the mechanism that may be responsible for neoplastic events, there is ample evidence of the link between occupational exposure to silica and lung cancer.

Prevention of Silicosis

Prevention remains the principal goal in dealing with this occupational lung disease. Effective exposure controls are available for most processes, and include process enclosure, wet abrasive techniques, and local exhaust ventilation, combined with a comprehensive approach to personal protection. Where possible, less hazardous industrial agents should be substituted for silica. The education of workers and employers regarding the hazards of silica dust exposure and measures to control exposure is also important.

If silicosis is recognized in a worker, termination of any continuing exposures is advisable. Unfortunately, the disease often will progress even without further silica exposure. The finding of a case of silicosis is a "sentinel health event" and should prompt a thorough evaluation of workplace exposures and control measures by a competent authority, with the goal of recognizing the sources of the hazard and protecting other workers who may continue to be at risk.

Medical Screening and Surveillance in Silicosis

Workers exposed to silica and other mineral dusts should be monitored on a regular basis for adverse health effects as a supplement to, but not a substitute for, exposure monitoring and control. Health screening commonly includes evaluation of respiratory symptoms, spirometric abnormalities, and radiographic changes. There is evidence that, if silicosis subsequently develops, workers who have participated in periodic health monitoring experience reduced severity of disease. Evaluation for tuberculosis infection with intradermal skin testing should also be performed. In addition to reporting of results to the individual workers, health data from all workers at a plant or operation should be periodically analyzed to assess the adequacy of prevention activities.

Therapy, Management of Complications, and Control of Silicosis

When prevention has been unsuccessful and silicosis has developed, therapy is directed largely at complications of the disease. Therapeutic measures are similar to those commonly used in the management of airflow obstruction, infection, pneumothorax, hypoxemia, and respiratory failure complicating other pulmonary disease. Historically, the inhalation of aerosolized aluminum was attempted, unsuccessfully, as a specific therapy for silicosis. Polyvinyl pyridine-N-oxide, a polymer that has protected laboratory animals, is not available for use in humans. Laboratory work with tetrandrine has shown in vivo reduction in fibrosis and collagen synthesis in silica-exposed animals treated with this drug. However, evidence of human efficacy is currently lacking, and there are concerns about the potential toxicity, including mutagenicity, of this drug. Because of the high prevalence of disease in some countries, investigations of combinations of drugs and other interventions continue. Currently, no successful approach has emerged, and the search for a specific therapy for silicosis has to date been unrewarding.

For workers with a diagnosis of silicosis, further exposure to silica-containing dusts is undesirable. If the disease is advanced, or has occurred after a relatively short exposure (i.e., less than 15 years), then further dust exposure should be assiduously avoided. Advice on job reassignment should be considered in the context of the worker's age, symptoms, functional status, and the current working conditions and measured silica exposures. Patients with silicosis may have few symptoms early in the disease; however, physicians should be aware that many states have a strict time limit dating from the physician's diagnosis of silicosis regarding application for workers' compensation and reimbursement of medical costs.

In the medical management of silicosis, vigilance for complicating infection, especially tuberculosis, is critical. The use of bacillus Calmette-Guérin (BCG) vaccine in the tuberculin-negative silicotic patient is not recommended, but the use of preventive isoniazid (INH) therapy in the tuberculin-positive silicotic patient is advised. The diagnosis of active tuberculosis infection in patients with silicosis can be difficult. Clinical symptoms of weight loss, fever, sweats, and malaise should prompt radiographic evaluation and sputum acid-fast bacilli stains and cultures. Radiographic changes with infection may be subtle and atypical. Enlargement or cavitation in conglomerate lesions or nodular opacities is of particular concern. Bacteriologic studies on expectorated sputum may not always be reliable in silicotuberculosis. Fiberoptic bronchoscopy for additional specimens for culture and study may be helpful in establishing a diagnosis of active disease. The use of multidrug therapy for suspected active disease in silicotics is justified at a lower level of suspicion than in the nonsilicotic patient, due to the difficulty in firmly establishing evidence for active infection. To obtain satisfactory results in the presence of silicosis, antituberculous treatment must be more prolonged, with regimens lasting at least 8 months. A multiplicative increase in risk of mycobacterial infection is associated with the combination of silicosis and human immunodeficiency virus (HIV) infection, as has been encountered in South African gold miners. These infections represent major clinical and public health challenges. Prolonged treatment is essential, and there is potential for both adverse drug reactions and interactions between antiretroviral and antituberculous therapy. Recommended approaches continue to evolve, and clinicians should consult the latest authoritative recommendations.

Ventilatory support for respiratory failure is indicated when precipitated by a treatable complication. Pneumothorax, spontaneous and ventilator-related, is usually treated by chest tube insertion. Bronchopleural fistula may develop, and surgical consultation and management should be considered.

Acute silicosis may rapidly progress to respiratory failure. When this disease resembles pulmonary alveolar proteinosis and severe hypoxemia is present, aggressive therapy has included massive whole-lung lavage with the patient under general anesthesia in an attempt to improve gas exchange and remove alveolar debris. Although appealing in concept,

the efficacy of whole lung lavage has not been established. Glucocorticoid therapy has also been used for acute silicosis; however, it is also of unproven benefit.

Some young patients with end-stage silicosis may be considered candidates for lung or heart-lung transplantation by centers experienced with this expensive and high-risk procedure. Early referral and evaluation for this intervention may be offered to selected patients.

The discussion of an aggressive and high-technology therapeutic intervention such as transplantation serves to dramatically underscore the serious and potentially fatal nature of silicosis, as well as emphasize the crucial role for primary prevention. The control of silicosis ultimately depends upon the control of workplace dust exposures. This is accomplished by rigorous and conscientious application of fundamental occupational hygiene and engineering principles, with a commitment to the preservation of worker health.

PREVENTION STRATEGIES FOR COAL WORKERS' LUNG DISEASES AND SILICOSIS

The control of coal workers' lung diseases and silicosis in both the developed and developing world requires comprehensive prevention strategies, including exposure control, medical surveillance, research, and education. Example approaches include:

- Major efforts must be directed to installation of effective engineering controls and improvements in work practices to progressively reduce dust exposures to acceptable levels. These efforts are labeled primary prevention. Personal respiratory protection should also be used, particularly during short-term operations or unusual/emergency conditions, and while engineering controls are being modified or improved. The use of respirators will only be effective when part of a professionally managed comprehensive respiratory protection program, and should never be relied upon outside of such a program.
- Primary prevention should involve ongoing dust exposure monitoring, and include mechanisms for feedback to modify and improve working conditions if exposures are measured above mandated levels. Even exposure at currently permissible levels has been reported to represent a risk of disease.
- Secondary prevention through medical screening and surveillance should be designed to benefit the individual worker and other potentially exposed workers. Illness identified through medical screening represents a failure of primary prevention, and thus should trigger feedback to those involved in environmental monitoring and work practice evaluations.
- Education about the respiratory health hazards from uncontrolled exposures to silica and coal mine dust must be available to workers, employers, managers, and health care providers.
- Information on the cumulative burden of disease should be monitored over time for both silica and coal mine dust.
- Research into mining-related lung diseases should be encouraged, to improve recognition, monitoring, exposure reduction, and therapy, and to increase understanding of pathogenesis. Research efforts should supplement, not displace, attention to dust control.
- Clinicians who recognize coal-related diseases or silicosis in their patients should attempt to determine whether ongoing workplace exposures present a continuing risk to current workers, while maintaining the confidentiality of the patient-physician relationship. Assistance in this can often be obtained through local or state health departments, occupational medicine groups, and federal agencies. Reporting of occupational diseases is required in many states.

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