Coal Workers' Pneumoconiosis

ICD-10 J60

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Coal workers' pneumoconiosis (CWP) is one of the lung diseases arising from inhalation and deposition of respirable coal mine dust in the lungs, and from the reaction of the lungs to the dust. It is a chronic, irreversible disease of insidious onset, usually—but not always—requiring 10 or more years of dust exposure before appearing on chest x-ray. It is characterized by abnormalities visible as small or large opacities (spots) on chest x-ray. When only small opacities are present, the condition is called chronic or simple CWP. Complicated CWP or progressive massive fibrosis (PMF) are the terms used when opacities greater than 1 centimeter attributable to coal dust exposure are present on x-ray. Obsolete terms applied to the same conditions include anthracosis, anthracosilicosis, miners' phthisis, and miners' asthma.

For purposes of compensation, various jurisdictions define pneumoconiosis differently. For example, in the U.S. federal compensation system for miners, pneumoconiosis is defined as "a chronic dust disease of the lung arising out of employment in an underground coal mine" [Federal Mine Safety and Health Act, Section 402(b)]. Thus, eligibility for benefits under the federal act is not limited to dust effects visible on chest x-ray. Disease definitions delineating eligibility for state workers' compensation benefits also vary from state to state.

The ILO disseminates a conventional method for x-ray classification that is employed in the recognition and categorization of the pneumoconioses, including CWP. This method classifies opacities according to their shape, size, location, and profusion. Profusion is determined by comparing the miner's film with "standard" ILO films. There are four major categories of increasing profusion of opacities: 0, 1, 2, and 3. Whichever standard film most closely matches that of the miner determines the "major category" of profusion. If the film is in a border area between two major categories, both categories are noted, with the category most like the film noted first. For example, a film that shows a higher profusion of opacities than the category 1 standard film, but that is more like the 1/1 standard than like the 2/2 standard, is classified 1/2. PMF is classified as category A, B, or C, depending on the size of the large opacities.

The ILO system was originally established to achieve consistency in film interpretation during the conduct of health surveillance or epidemiological investigations. In the United States, readers trained in this method of interpretation who pass a competency test administered by NIOSH are designated as B readers. However, despite efforts to achieve standardized interpretations of chest x-rays through use of the ILO system, significant variability is often found

between and within readers regarding the presence and severity of CWP.

Coal workers' pneumoconiosis has characteristic pathological features, which can be seen on autopsy or biopsy and are described in standard reference works. Tissue examination is not necessary for the diagnosis of the disease.

Occurrence

The prevalence of CWP increases with increasing dust exposure and also varies with coal rank (essentially, the age of the coal). Prevalence data from a federal underground coal miner medical monitoring program for 1996-2002 show an overall prevalence of category 1/0 or greater CWP of 3.2%, and a prevalence of PMF of 0.2%. The prevalence of CWP varies around the US, with rates in Virginia and West Virginia being about eight times higher than in Illinois and Alabama. CWP is more prevalent in workers in small mines. The data also indicate that about 6% of underground miners with 25 years or more mining experience had category 1/0 or greater CWP. These statistics are very consistent with some projections based on British data suggesting that 6.2% of miners will have simple CWP and 0.4% will develop PMF after 35 years of exposure at the current US limit of 2 mg/m^3. In the U.S., about 1000 coal miners died with CWP noted on their death certificates in 1999. Surface coal workers are also at risk of developing lung diseases from dust exposure. This risk is increased for certain workers such as drillers, as well as for surface miners with prior underground experience.

Causes

Respirable coal mine dust causes CWP. Respirable dust is any dust that is small enough to be deposited in the terminal bronchioles or alveolar airspaces (generally less than 5 μm in diameter).

Coal mine dust is a mixed dust consisting mostly of coal particulate but also including other minerals found in mines, particularly silica. Certain coal mining jobs, such as surface drilling and underground roof bolting, may involve high concentrations of silica dust, and give rise to silicosis (see Silicosis) in addition to CWP. On autopsy or biopsy examination of lungs, findings of CWP and silicosis may coexist. However, the x-ray appearances of CWP and silicosis are virtually identical, and the separate diseases typically cannot be distinguished radiographically.

Pathophysiology

Inhaled fine particles of coal dust are scavenged by specialized lung cells (macrophages). These cells and dust particles accumulate deep in the respiratory tree near or in the air exchange units (alveoli). Abnormal fibrotic material (reticulin and collagen) may develop. Localized areas of tissue destruction (focal emphysema) may also occur. The areas demonstrating a combination of these abnormalities are called coal macules. In some cases, nodules consisting of macrophages and a greater quantity of abnormal fibrotic tissue are present.

These can clump together (coalesce) to form the large lesions of PMF. Generally, there is significant destruction of lung tissue as the nodules coalesce.

The definition of the exact physiological abnormalities resulting from the development of CWP has been complicated, in part, by the diversity of pulmonary responses to coal mine dust. Increasing dust exposure has been associated with progressive loss of lung function, resulting in the development of obstructive lung disease. This loss appears similar in magnitude to that caused by regular cigarette smoking. Miners have increased rates of emphysema and chronic bronchitis.

Coal workers' pneumoconiosis is itself an effect of exposure to dust. When PMF destroys and distorts lung tissue, it is typically associated with loss of lung function that can have predominantly obstructive, but also restrictive, or combined patterns of abnormality. Simple CWP alone may or may not be associated with diminished function in any particular individual. Nevertheless, individuals with simple CWP may experience significant loss of lung function as a result of the same exposure (coal mine dust) that caused the development of CWP (see chapters on Emphysema and Chronic Bronchitis).

Prevention

Primary prevention of CWP is achieved by reducing exposure to coal dust through improved ventilation and dust suppression supported by enforcement of strict dust control standards. Feasible and effective engineering dust controls exist for surface and underground coal mining operations. Current preventive efforts focus on suppressing the "respirable" fraction of coal mine dust (less than 10 μm in diameter) that appears to cause CWP. In the US, the permissible exposure level is a time-weighted average of 2.0 mg/m^3 measured as a personal sample. NIOSH has recommended improved prevention through an exposure limit of 1 mg/m^3 for respirable coal mine dust and a 0.05 mg/m^3 limit for respirable crystalline silica. Control of other lung diseases associated with dust exposure in mining which may be caused by larger dust particles may or may not be achieved by suppressing the respirable dust fraction.

Prevention of pulmonary impairment associated with PMF has been based on an assumption that PMF almost invariably develops on a background of advanced simple CWP; therefore, secondary prevention efforts have been directed at identifying miners with early simple CWP (category 1) and at further reducing the dust exposure for these miners. Later reports have brought that assumption into question, with the finding that significant numbers of PMF cases were developing among miners with early simple CWP.

Coal workers' pneumoconiosis does not resolve or improve with the elimination of exposure to coal dust, and may, in some cases, progress from simple CWP to PMF in the absence of additional exposure. Rehabilitation efforts (tertiary prevention) are the same as those employed for anyone with disabling lung disease: elimination of adverse environmental exposures; immunization against influenza and pneumococcal infection; early recognition and treatment of infec-

tion; education directed at improved levels of self-care; graded exercise; and consideration of medications such as bronchodilators.

Other Issues

Some miners develop disabling lung disease in the absence of PMF. Periodic pulmonary function testing might be useful in early disease recognition and secondary prevention.

Efforts to control CWP through the monitoring and reduction of respirable dust exposure may not be adequate to control the development of pulmonary impairment in miners. The absence of a positive finding for CWP on x-ray does not ensure the absence of significant disease from coal mine dust exposure.

Preventive strategies directed toward eliminating PMF through early identification of miners with simple CWP may be inadequate.

Coal mining communities are often isolated, with few local alternative sources of employment. Miners developing lung disease are reluctant to eliminate exposure to dust by leaving the industry when unemployment is the only alternative.

Cigarette smoking has no apparent effect on the formation of the coal macule, but adds to the miner's risk of developing emphysema, chronic bronchitis, and airway obstruction.

Modern underground mining techniques such as longwall mining result in high levels of coal mine dust being generated and thereby requiring careful identification of sources of dust and attention to control methods.

Dust regulations in the U.S. assume a 5-day, 8-hour-per-shift workweek; however, other considerations have resulted in increasing numbers of shifts per worker in some areas. The impact of these changes on health has not been evaluated.

Further Reading

Attfield MD, Wagner GR. Respiratory disease in coal miners. In *Environmental and Occupational Medicine* (3rd ed.). Philadelphia: Lippincott Raven, 1998, pp. 413-434.

ILO. Guidelines for the Use of the ILO *International Classification of Radiographs of Pneumoconioses*, 2000 edition, Geneva: International Labor Office 2002 (Occupational Safety and Health Series, No. 22 [rev. 2000]).

Kleinerman J, et al. Standards for the pathology of coal workers' pneumoconiosis. *Archives of Pathology* 1979; 103: 375-432.

Kissell FN (Ed.). *Handbook for Dust Control in Mining*. IC 9465. (DHHS [NIOSH] Publication No. 2003-147). Washington, DC: NIOSH, 2003.

National Institute for Occupational Safety and Health. *Occupational Exposure to Respirable Coal Mine Dust: Criteria for a Recommended Standard*. Cincinnati, OH: NIOSH, 1995.

National Institute for Occupational Safety and Health. *Work-related Lung Disease Surveillance Report*, 2002. Cincinnati, OH: NIOSH, 2003.

Weeks JL, Wagner GR. Compensation for occupational disease with multiple causes: the case of coal miners' respiratory diseases. *American Journal of Public Health* 1986; 76: 58-61.

Preventing Occupational Disease and Injury Second Edition

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Printed and bound in the United States of America
Set In: Palatino and Helvetica Condensed
Interior Design and Typesetting: Terence Mulligan
Cover Design: Michele Pryor
Printing and Binding by Automated Graphic Systems, Inc., White Plains, MD

ISBN 0-87553-043-5
2M 11/04