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Respiratory Diseases in Iron Ore Miners and Millers

SUMMARY

Workers in iron mines are at risk of developing interstitial lung disease if the dust levels are above the threshold limit value. However, they more commonly develop the usual diseases that affect all workers. Some illnesses, such as chronic bronchitis, bronchial asthma, and the collagen vascular diseases that affect the lung, may be more severe because of the inhalation of dust. The most difficult problem is to differentiate asymptomatic sarcoidosis from pneumoconiosis. The family doctor who also acts as the company doctor must be aware of the potential conflict of interest. (Can Fam Physician 1989; 35:1517-1520.)

Key words: iron ore workers, mining-related disease, occupational hazards, respiratory system

RÉSUMÉ

Les mineurs travaillant dans les mines de fer sont à risque de développer une pneumopathie interstitielle si les taux de poussière dépassent la valeur du seuil limite. Cependant, ces travailleurs développeront plus souvent les maladies qui affectent couramment tous les autres travailleurs. Certaines maladies, comme la bronchite chronique, l'asthme bronchique et les collagènes vasculaires qui affectent les poumons, peuvent se trouver aggravées par l'inhalation de poussière. Le problème le plus difficile est de différencier la sarcoïdose asymptomatique de la pneumoconiose. Le médecin de famille, qui est aussi le médecin de l'entreprise, doit être conscient du conflit potentiel d'intérêt qui le guette.

Iron Processing

Most of the earth's crust is composed of silicate minerals. These are compounds of silicone dioxide that are in various combinations with other elements, such as iron, aluminum, gold, and so forth. Large concentrations of uncombined iron were often deposited with the silica as iron ore.

Threshold Limit Value

A safety standard for atmospheric dust, the threshold limit value (TLV), for most dusts is reviewed and published annually by the Committee on Threshold Standards of the American Conference of Government Industrial Hygienists. The separation of iron from the waste rock produces a great deal of dust, which must be removed to provide safe air for the workers to breathe.

By law, the dust levels must be kept below the TLV. This value appears to be a maximum level of dust to which the workers can be exposed during their working lives without developing pneumoconiosis. It is safe for the workers to be exposed to higher dust levels for short periods, provided that these episodes are not prolonged and their total average exposure is not above the TLV.

Dust Exposure Index

The measurement of the dust exposures of individual workers requires a
number of dust-measuring devices at fixed stations and individual personal monitors worn by the workers. Using both techniques, individual jobs can be described and their dust exposures calculated.

Dust levels must be constantly measured because some data show that dust levels are dependent upon the location of the sampling device, the time of the day during which the sample is collected, and the season, with higher dust levels tending to occur during the winter when the mill is more tightly closed than during the summer.

Pneumoconiosis

Pneumoconiosis is a general term used to describe all interstitial lung diseases caused by the inhalation of inorganic dusts. More specific terms, such as siderosis, asbestosis, and silicosis, are used to indicate the causative mineral in the dust. Pneumoconioses are preventable and occur only when a worker is exposed to high levels of dust.

The permissible dust levels in mines and mills in Canada are regulated by provincial governments, which set and enforce standards. The government usually use the standards for air published by the American Conference of Government Industrial Hygienists.

These TLVs describe the maximum dust levels to which a worker can be exposed without developing pneumoconiosis and may vary, depending upon the composition of minerals in the dust. Levels significantly above the TLVs are likely to produce accelerated pneumoconiosis and significant disability in some of the workers exposed. The higher the dust level, the more likely it is that miners will develop radiologic abnormalities and the more likely that they will develop disability.

For physicians who see the workers for diagnosis and management, the major problem is to recognize the occupational history of the worker and to quickly exclude other diseases.

Pathology

Mineral dusts vary in their ability to produce pulmonary reactions. Pure iron dust produces no harmful reaction in the lungs. Workers who are exposed to pure iron dust and fumes, such as arc welders, oxy-acetylene cutters, and silver polishers, develop radiologic changes because the iron is concentrated in the macrophages and reticuloendothelial cells in the lungs, which produces dense infiltrates on chest X-ray films. When lungs from these workers are examined, they are found to be stained red by the iron but have no fibrotic reaction.4,5

Iron, however, is not processed as a pure ore. The respirable dust caused by the process of extraction often contains quartz and other minerals. The percentage of these minerals in the dust often varies according to the location of the dust sampler in the plant.6 In the workers from western Labrador that I saw,7 the respirable dust was found to contain a number of compounds, such as cumingtonite, anthophyllite, ilelite, kaolinite, geothite, hematite, quartz, pyrolusite, magnetite, siderite, and cristobalite. Each of these has a different propensity to produce fibrous reactions in the lung.

Clinical Presentation

Most iron workers will have a known occupational history while they are still working in the mining industry. Physicians should be aware that workers can present with typical features of pneumoconiosis years after their last exposure to dust.8 Therefore, it is important to determine the occupational history of patients with an interstitial lung disease.

In the early stages, workers with siderosis will have round radiologic abnormalities in the upper halves of the lung field that are indistinguishable from those of silicosis. These workers typically have no symptoms. In contrast, most iron ore and foundry workers are exposed to dust that contains a variable amount of silica, at times mixed with other fibrogenic material.7 These cases may develop a mixed-dust siderosilicosis, the progression and outcome of which is similar to silicosis.

The earliest radiologic findings are discrete, rounded opacities, usually in the upper halves of both lung fields. Biopsy at this stage may demonstrate a typical silicotic nodule or, more likely, a diffuse mononuclear inflammatory reaction centred in the interstitium, dust, and the proliferation of fibroblasts with collagen formation. Pulmonary function test and general examination results at this stage are usually normal.

Initially the radiologic densities are discrete, but with continuation of the process, more infiltrates develop that may form large opacities as they conglomerate. At this stage, almost all patients become symptomatic with dyspnea on exertion and, in some instances, with a cough that produces a small amount of sputum.

As the disease progresses, the chest X-ray film reveals the development of linear infiltrations due to fibrosis and iron particles in the pulmonary lymphatic system with the loss of the normal bronchovascular markings. The hilar lymph nodes become more dense and later may enlarge a little. With the development of more extensive disease, the X-ray film may reveal extensive confluent infiltrates in the upper lobes as massive pulmonary fibrosis develops. The workers at this stage will have significant symptoms and may have signs of pulmonary hypertension and congestive heart failure in addition to respiratory failure.

In chronic pneumoconiosis a worker usually develops radiologic abnormalities 30 or 40 years after first exposure to the dust. In contrast, a very high dust exposure (as in sandblasting) can trigger the development of acute silicosis within a period of three to six months. Accelerated pneumoconiosis can develop about 10 years after exposure to dust levels significantly above the TLV. In Labrador, I identified an accelerated mixed-dust siderosilicosis in workers exposed to dust for eight to 10 years.7

Workers who develop accelerated or acute pneumoconiosis tend to be younger than workers with chronic disease and have developed it as a result of more intense exposure. When removed from further dust exposure, their disease often continues to progress because the large amount of dust that initiated the reaction remains within the lungs, producing further fibrosis.7 Many of these workers progress from simple pneumoconiosis to more extensive disease or even progressive massive fibrosis with the consequent development of severe pulmonary symptoms that may progress to cardiorespiratory failure.

In addition to the typical pneumoconiosis, these workers are subject to other illnesses, some of which are ac-

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centuated by their exposure to dust, and some of which occur spontaneously. These illnesses are pulmonary tuberculosis, chronic bronchitis, and some of the collagen vascular diseases, such as fibrosing alveolitis, rheumatoid arthritis, and scleroderma, that appear to have a much more aggressive pulmonary reaction to the dust. Reports have indicated a slightly higher incidence of bronchogenic carcinoma in iron ore miners in England and in France. The cause of this apparent increase has not been identified. It does not appear to be due to any radioactive gases in the mines or to any increased rate of cigarette smoking.

A small number of miners may develop signs of chronic bronchitis as the result of exposure to dust. Very few workers who are non-smokers develop industrial bronchitis. These workers can be recognized by the development of cough that produces a small amount of sputum, but no apparent radiologic changes.

The vast majority of workers who develop chronic bronchitis are smokers who may have some additive effect of the dust and fumes to their cigarette habit. The symptoms of other workers with pulmonary diseases such as bronchial asthma or bronchiectasis may worsen significantly because the dust and fumes irritate their inflamed bronchi.

A major problem for the physician is the differentiation of sarcoidosis from accelerated pneumoconiosis in younger men. This will frequently require a diagnostic biopsy unless the sarcoidosis presents in a typical manner, or with suggestive features, such as erythema nodosum, recurrent renal calculi, joint symptoms, or hilar adenopathy. In many instances, the diagnosis is not clear-cut because the workers are asymptomatic and have an interstitial pulmonary infiltrate without massive lymphadenopathy. The X-ray pattern in these cases is similar to pneumoconiosis with mild hilar lymphadenopathy.

Diagnosis

Pneumoconiosis often is diagnosed after the worker is removed from the dust. Signs and symptoms often develop late and frequently progress after the worker is no longer exposed to the dust. The physician who sees these workers must make the correct diagnosis as quickly as possible with a minimum of inconvenience and morbidity to the worker and the family.

Pneumoconiosis is diagnosed after a chest X-ray film reveals interstitial abnormalities consistent with the diagnostic abnormalities found on the standard X-ray films distributed by the International Labour Organization, a history of exposure to dust, and no other identifiable cause for the abnormal radiographic findings.

A major problem is to quickly exclude other interstitial lung diseases (such as sarcoidosis), immunologic interstitial lung diseases (such as rheumatoid lung disease and scleroderma), infections (such as tuberculosis and Pneumocystis carinii), and malignancy, which is usually metastatic. The natural history of the radiologic abnormality is important; slow development and progression are usual for pneumoconiosis. A significant regression or unusually rapid progression of the abnormality is strong evidence against a diagnosis of pneumoconiosis. For this reason, a careful review of all previous chest X-ray films is essential.

A significant systemic reaction and pulmonary signs or symptoms are also unusual in the early stages of pneumoconiosis and indicate the probability of another disease.

In some instances, when there is confusion about the diagnosis, a lung biopsy may be required. The biopsy is meant to identify potentially treatable diseases and not to confirm the diagnosis of pneumoconiosis. An open lung biopsy should be performed to obtain a satisfactory specimen for pathologic analysis. An exception would be when sarcoidosis or infections are strongly suspected and the patient is not critically ill. In these cases, multiple transbronchial lung and alveolar biopsies through a flexible bronchoscope have been found to be satisfactory. If the transbronchial biopsies are inadequate, a mediastinal lymph node or open lung biopsy will subsequently be required to complete the investigations.

Therapy

Unfortunately, there is no specific therapy for pneumoconiosis, although workers with complications, such as infection or congestive heart failure secondary to pulmonary hypertension, may benefit from antibiotics, diuretics, or oxygen therapy. The lack of specific therapy for these workers requires us to reduce their risks to an absolute minimum if we wish to continue to use these minerals. Although there is no specific medical therapy, most of the workers and their families require significant help to effectively deal with the disease and the many social problems they now must face.

Social Consequences

A major problem for the physician dealing with these workers and their families is the social consequences of the diagnosis that prevents them from continuing in their job. Most workers are highly paid and have specific skills that are not usually transferable to other occupations. With the development of an interstitial lung disease, the workers will usually have their miner's medical certificate cancelled, which prevents them from working in a dusty environment. Unfortunately, in most mines alternative job opportunities are not easily found for these workers.

The Workers Compensation Boards provide for retraining and disability payments for workers with pneumoconiosis, but frequently do not for workers with other respiratory diseases who lose their miner's medical certificate or who must leave their jobs because of increased respiratory symptoms. Because of this, the family physician may have a worker who feels well, looks well, and has normal physiologic function, but is no longer readily employable, especially at the same pay scale and with the same job satisfaction.

Potential Conflict of Interest

The family doctor who acts both as family doctor and as company doctor must recognize that there is a serious potential conflict of interest. This dual position may be essential to attract a physician to the community because the mines and mills are often remote and the only reason for the town to exist. Nevertheless, physicians who assume this position must be aware of the problem and protect both their patients and themselves.

Workers will often assume that, if they are regularly examined by a doctor and permitted to continue to work, they are safe. When a problem
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Precautions: 1. Replacement of systemic steroids with RHINALAR should be gradual and carefully monitored by the physician. 2. Although absorption sufficient to produce systemic effects has not been shown in clinical studies with RHINALAR Nasal Mist, the potential of adrenal suppression still exists and this must be considered as a possibility with prolonged excessive usage. Patients on long-term therapy should be reassessed periodically to avoid unnecessary continued use. 3. Since onset of action may be somewhat slower than oral or topical sympathomimetic amines or antihistamines, RHINALAR should be used for several days before evaluating therapy. 4. If adverse effects are not evident after approximately 7 days, the patient should be re-evaluated. 5. If hypersensitivity reactions occur during therapy, the drug should be discontinued and appropriate treatment should be instituted. 6. Continued therapy can decrease resistance to local infection. If pyogenic infections occur during therapy, appropriate treatment should be instituted. 7. Despite the very low level of absorption of flunisolide administered intranasally, the following must be kept in mind: a) corticosteroid effects may be enhanced in patients with hypothyroidism and in those with cataracts. b) in hypothyroidism, antacids should be used cautiously in conjunction with corticosteroids. 8. Patients should be advised to inform subsequent physicians of the prior use of corticosteroids. 9. During local corticosteroid therapy, the possibility of atrophic rhinitis and/or pharyngeal candidiasis should be kept in mind. 10. RHINALAR should not be used during an asthma attack. 11. Because of the inhibitory effect of corticosteroids on wound healing, in patients who have had recent nasal surgery or trauma, a nasal corticosteroid should be used with caution until healing has occurred. Adverse Reactions: Side effects noted with RHINALAR have been consistent with what one would expect from topical administration to an already inflamed nasal mucosa. The most frequent side effect observed was a mild transient nasal burning and stinging. Occasionally this was severe enough to warrant discontinuation of RHINALAR therapy. Other side effects seen in patients treated with RHINALAR, in order of decreasing prevalence were: nasal irritation, epistaxis, nasal and stuffy nose, nasal stuffiness, hoarseness, and headache. Exceptionally rare may be exacerbation of therapy. Rhinopharyngitis and Treatment of Overdose: Acute overdose has not been reported. When used at excessive doses, the potential of sternal effects such as hypercorticism and adrenal suppression does exist. Decreasing the dose will abolish these manifestations. Dosage and Administration: RHINALAR Nasal Mist is for administration by the intranasal route only. Usual Starting Dose: Adult: 2 sprays (each approximately 25 μg) into each nostril twice a day, increase to maximum 3 times a day if needed. Children: For children 6 to 14 years of age, one spray (approximately 25 μg) into each nostril 3 times daily. Maintenance Dose: After the desired clinical effect is obtained, the maintenance dose should be the smallest amount necessary to control the symptoms. Some patients may be maintained on as little as one spray (approximately 25 μg) into each nostril per day. Patients on long-term therapy should be reassessed periodically to avoid unnecessary continued use. There is no recommended daily dose. The maximum recommended dosage is one spray (approximately 25 μg) into each nostril for adults and one spray for each nostril for children 6 to 14 years of age. The effectiveness of RHINALAR is not immediate. Full therapeutic benefit requires usage. The absence of an immediate effect should be explained to the patient in order to ensure cooperation and continuation of treatment with the regular dosage schedule. In the presence of excessive nasal mucosal secretion or edema of the nasal mucosa, the drug may fail to reach the site of action. In such cases it is advisable to use a nasal vasoconstrictor for two or three days prior to RHINALAR therapy. Dose form: RHINALAR Nasal Mist is a 0.02% aqueous solution of flunisolide in a 25ml plastic bottle fitted with a metered pump device which delivers approximately 25 μg of flunisolide per spray in a mist which is inhaled into the nostril. For full information on the device see patients directions for use. Product monograph available to health professionals upon request. How to Obtain: Doig AT, McLaughlin AJG, Doig AT, Clinical, radiological and pathological studies of the lungs of electric arc welders. Lancet 1956; i:394-5. References: 1. Agricola G. De re metallico. 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