IN-DEPTH REVIEW

Occupational respiratory disease in mining

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Abstract

This review is based on research-based literature on occupational lung disease in the mining and related industries, focusing on conditions of public health importance arising from asbestos, coal and silica exposure. Both ‘traditional’ and ‘new’ concerns about occupational respiratory disease in miners are addressed, with the inclusion of practical evidence-based findings relevant to practitioners working in developed and developing countries. Mining is not a homogeneous industry since current miners work in formal and informal operations with numerous, and often multiple, air-borne exposures. A further occupational health challenge facing primary care practitioners are ex-miners presenting with disease only after long latency. The sequelae of silica exposure remain an occupational health priority, particularly for practitioners who serve populations with concomitant HIV and tuberculosis infection and even when exposure is apparently below the statutory occupational exposure level. Coal workers’ pneumoconiosis, asbestos related diseases, lung cancer and other occupational respiratory diseases remain of considerable importance even after mining operations cease. While mining exposures contribute significantly to lung disease, smoking is a major factor in the development of lung cancer and chronic obstructive airways disease necessitating a comprehensive approach for prevention and control of mining-related occupational lung disease.

Key words

Asbestos; coal; lung disease; mining; silica; smoking.

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Background

The relationship between mining and occupational lung disease has been documented since the 1500s, when Agricola described dust with corrosive qualities eating away the lungs and implanting consumption in the body. In the twenty-first century, this age-old relationship between silica exposure and tuberculosis (TB) has acquired a renewed importance with the growing epidemic of HIV in developing countries, where the informal or non-regulated mining sector is significant and dust control less than optimal.

The relative prevalence and severity of mining-related occupational lung diseases are a function of the commodities mined, airborne hazard exposure levels, the period of exposure and co-existing illnesses or environmental conditions and lifestyle. The diseases and exposures are too numerous to do justice to in a short review. Thus, this review focuses on asbestos, coal and silica exposures since these are the commodities of major public and occupational health importance for occupational practitioners serving ex and current mineworkers [1,2] and are of considerable medico-legal significance. The accent of the review is on recent research findings that have implications for best clinical practice. This review is based on recent comprehensive reviews of best practice by South African physicians [1,2], classic undisputed findings, research with which the authors are associated and literature of the past 10 years, identified using Google database search for the key words: mining occupational lung disease with asbestos, coal, silica, smoking and cancer.

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Asbestos

Producers and users of asbestos

Control and prevention of asbestos-related lung diseases are recognized public health issues [3] and many countries have either banned all types of asbestos or severely restricted its use [4].

The mining and use of amphibole forms of asbestos (primarily amosite and crocidolite) has ceased worldwide, but chrysotile (the serpentine form) is still mined, e.g. in Canada and Russia, and is utilized by many countries (mainly African, Asian and South American) for, among other things, water reticulation and housing. Chrysotile-producing countries have been strong protagonists for chrysotile, and its unique properties, the lack of inexpensive substitutes, plus the scientific evidence that it is less harmful than the amphiboles [5] have prevented a blanket international prohibition of chrysotile.

Sources of exposure

Given the widespread past use, potential sources for asbestos exposure are many, even today [3]. Although the number of miners still exposed to asbestos is minimal, the manufacturing or removal of asbestos products, such as ceiling boards, remains a hazard. People living in the vicinity of asbestos mines, mills or manufacturing plants may be exposed to the fibres environmentally. For example, there is a high incidence of mesothelioma in women who live or lived in the Quebec chrysotile mining area [6] (the contamination of chrysotile, in this region, by the amphibole tremolite, increases its toxicity). Exposures from non-industrial sources have also been documented in regions where the soil is contaminated with asbestos fibres [7].

Presentation, diagnosis and clinical features

Pleural plaques

Pleural plaques continue to be the most frequent, and often only, manifestation of asbestos exposure and asbestos exposure is the commonest cause of pleural plaques. In the period 1992–1999, pleural plaques (benign pleural disease) constituted 28–35% of occupational respiratory disease cases reported in the UK [8].

Pleural plaques, in the absence of parenchymal disease, often do not cause signs and symptoms and are commonly detected as an incidental finding on a routine chest radiograph. In the past, pleural plaques were labelled as ‘visiting cards’ for asbestos exposure, implying that they had no impact on lung function. More recently, however, pleural plaques have been associated with respiratory disability [9]. A Swedish study showed that pleural plaques on the chest radiograph indicated significant exposure to asbestos, as well as an increased risk of developing mesothelioma (and possibly also lung cancer) [10].

Asbestos

As exposure levels in workplaces around the world are increasingly meeting recommended control levels, the rates of certification of death or disability from asbestosis are falling in many countries [11]. However, asbestosis deaths in the USA increased >10-fold from 1968 to 1999, with no apparent levelling off of this trend [19].

Clinical diagnosis of asbestosis depends on establishing the presence of pulmonary fibrosis with asbestos exposure of sufficient intensity. The radiological features of well-developed asbestosis seldom present a diagnostic problem; interpretation of less marked changes is more subjective, as ageing, smoking and chronic obstructive pulmonary disease (COPD) can affect the specificity of the chest radiograph. There is increased certainty when rales and a low carbon monoxide diffusion capacity are also present [13].

Although asbestosis is usually associated with restrictive lung function, some patients exhibit a mixed or obstructive lung function profile [14]. When radiological or lung function changes are marginal, high resolution computed tomography (HRCT) often shows parenchymal fibrosis and the presence of pleural plaques provides additional evidence that the parenchymal disease is asbestosis related [15]. Asbestosis is not a risk factor for TB [16].

Diagnosis of asbestosis for the purpose of legal attributability calls for greater certainty and the use of criteria that vary according to the relevant legal system.

At an international meeting in Helsinki in 1997, the criteria for diagnosis and attribution of asbestos-related diseases were reviewed and guidelines produced; in addition to radiological studies, analysis of lung tissue for asbestos fibres and bodies may be of assistance [17].

Malignant disease

The emergence of malignant asbestos-associated diseases as important causes of death in asbestos-exposed individuals can be attributed, in part, to workplace controls, resulting in a decrease in the competing risk from asbestosis and greater chance of survival into the cancer age group.

Mesothelioma

In the UK, the number of mesotheliomas among males increased almost 3-fold over the last 20 years, with 6475 cases in the 5 year period 1996–2000 [18]. In 1999, there were 2500 deaths from mesothelioma in the USA, 20% of which were in females and 15% in construction workers [19]. Given the long incubation period of mesothelioma
(20–40 years), one can realistically expect cases to continue to present well into the twenty-first century [3]. Although in the UK it has been estimated that the mesothelioma rates would decline by only the second decade of the twenty-first century [20], there is some evidence [21,22] that the rates may be dropping already.

Despite many years of clinical research, there is still no effective therapy for malignant mesothelioma; untreated the median survival time is 9 months. Chemotherapy, radiotherapy and surgery have not shown consistent improvements in survival [23].

**Lung cancer**

The association of lung cancer with asbestos exposure is well known. There is currently debate about whether asbestos exposure in itself is a cause of increased lung cancer risk, or whether it is through the development of asbestosis in association with asbestos exposure [24,25], as most people believe.

**Smoking, asbestos exposure and prevention of lung disease**

The risk of lung cancer in asbestos exposed individuals is greatly enhanced by cigarette smoking, but the multiplicative model has recently been challenged [26]. Other factors influencing the risk of lung cancer in exposed workers include intensity of exposure and fibre type. Fibre length may or may not play a role in the pathogenesis [27].

Pathologic studies do not show evidence of an association between asbestosis grade and smoking, although there is evidence to suggest that smoking enhances the retention of fibres in the lungs [28]. Both the possible role of smoking in the initiation and progression of fibrotic parenchymal disease and its established role in increasing the risk of lung cancer are strong indications for anti-smoking advice to be given to asbestos-exposed individuals and for instituting smoking cessation programmes in workplaces contaminated by asbestos dust.

**Coal workers’ pneumoconiosis**

**Occupations at risk**

There has been a steady decrease in the numbers of coal miners in Western Europe and the USA. However, the mining industry in these countries still employs large numbers (~200 000 in the USA) [29], as in Eastern Europe, India, China, Africa, Australia and South America. As the number of miners has decreased, however, mechanization has increased potential dust exposure. Hence, former and current coal workers are likely to develop coal workers’ pneumoconiosis (CWP) for the foreseeable future.

During the last 30 years, the prevalence of CWP has fallen consistently in the USA. In active underground miners included in the National Institute for Occupational Safety and Health (NIOSH) Coal Workers’ X-ray Surveillance Program [19], rates fell from >10% in the early 1970s to <2% in the late 1990s (consistent with the estimated current rate in South Africa of 2.6%) [30]. Similar trends have been noted in Europe [3,8].

Nevertheless, dust levels in coal mines are still high. A quarter of coal mine dust exposures recorded by Mine Safety and Health Administration (MSHA) in the USA exceeded the NIOSH recommended exposure limit (REL) of 1 mg/m³ [19]. The permissible exposure limit (PEL) for respirable coal dust is 2 mg/m³ in the USA. However, since new cases are occurring even among miners who have worked exclusively under current dust exposure limits, an evaluation of the dust levels in mines where these workers were employed is underway [31].

In addition to coal mining per se, other occupations at risk include coal trimming (which involves loading and stowing coal in stores or ships’ holds) and the mining and milling of graphite.

**Risk and prevalence**

Fibrosis associated with coal dust exposure is considerably less intense and extensive than that evoked by the more bioactive dusts, such as silica and asbestos [32]. The risk for CWP depends on the total dust burden in the lungs and is also related to the coal rank, which is based on its carbon content (anthracite has a higher rank than bituminous, followed by sub-bituminous and lignite) [33]. In the higher ranking coal, there may be a greater relative surface area of the coal dust particles, higher surface-free radicals and higher silica content [34]. Silica exposure and hence silicosis is more common in mines with a high grade of coal [35] and in workers such as roof bolters who work outside of the coal seams in quartz-containing rock [36].

Both the presence and stage of CWP, and the development of progressive massive fibrosis (PMF), appear to be related to the intensity of dust exposure, age [34], proportion of inhaled massive fibrosis (PMF), appear to be related to the intensity of dust exposure, age [34], proportion of inhaled silica in the dust and its surface bioactivity, individual immunological factors, and the presence of tuberculosis.

**Presentation, diagnosis and clinical features**

For many years, simple CWP was regarded as a disease without symptoms or physical signs. However, current evidence shows that coal mining, even in the absence of CWP and controlling for smoking, is associated with chronic bronchitis, chronic airflow limitation and emphysema [33,37]. In their comprehensive review, Coggon and Newman Taylor [38] concluded that:
• the balance of evidence points overwhelmingly to coal mine dust being a cause of impaired lung function;
• this can be disabling;
• the best estimate of the loss of $FEV_1$ (forced expiratory volume in 1 s) in relation to exposure of coal miners is 0.76 ml/g h/m$^3$; and
• the combined effects of coal mine dust and smoking on $FEV_1$ appear to be additive.

Symptoms of cough and sputum reported by most coal miners are likely to be a consequence of dust-induced chronic bronchitis. Breathlessness on effort is usually caused by associated chronic airflow limitation or by the development of PMF [33]. Respiratory impairment and disability increase as PMF progresses.

The chest radiograph remains a cornerstone of the diagnosis of CWP. However, because it is insensitive to the presence of macules and nodules [39], CT (computed tomography) may be more useful in individual cases.

Most evidence suggests that coal mining is associated with a decreased risk of lung cancer, but an increased risk of stomach cancer [40]. CWP alone does not carry an increased risk for mycobacterial infection, either by Mycobacterium tuberculosis or nontuberculous mycobacteria (NTM), but treatment of latent tuberculosis infection should be considered for coal workers who are thought to have had significant silica-dust exposure or who have evidence of silicosis [41]. The adverse respiratory effects of CWP and tobacco exposure necessitate active smoking cessation counselling for exposed individuals.

Silicosis

Risk and prevalence

Silicosis continues to be a major disease world wide, even in developed countries, affecting workers in mining and other occupations, including construction and foundries [3,19]. This avoidable disease remains a significant cause of morbidity and mortality [42]. Furthermore, reported cases are a gross underestimate of the total cases of silicosis [43], and in some settings, the prevalence of silicosis is rising [44].

Although silica dust is one of the most documented workplace exposures, there is controversy over the precise quantitative relationship between dust inhalation and the development of disease. Scientific evidence is increasingly demonstrating that exposure over a working lifetime to the commonly used standard of 0.1 mg/m$^3$ will result in a significant burden of radiological silicosis and also death from silicosis and lung cancer [45]. The evidence is insufficient to indicate whether or not halving this level to 0.05 mg/m$^3$ would be protective and hence it has been suggested that the level be lowered to 0.01 mg/m$^3$ [46,47].

Although the major determinant of silicosis is the lung dust burden, there is accumulating evidence that other variables such as freshly fractured silica, admixtures of other minerals, e.g. clay components which can coat the surface of the silica [29] and peak exposures also may be important [36]. Individual susceptibility to the disease determined using biomarkers may also play a role [48].

Diseases associated with silica exposure

An excellent review by Mossman and Churg [49] summarizes the processes whereby silica produces inflammation and fibrogenesis in the lung. Although silicosis continues to be the most common disease associated with silica exposure, the recent scientific literature has drawn attention to other silica-associated diseases.

Lung cancer

Recent authoritative reviews concluded that there were sufficient data to support an association between silicosis and lung cancer [12,42,47]. However, there remains debate about whether silica exposure, in the absence of silicosis, carries an increased risk for lung cancer [42,47,50]. The cancer risk may also be increased by smoking and exposure to other carcinogens, such as diesel products and radon, in the work place [51].

Tuberculosis

The association between silicosis and tuberculosis has long been recognized. Rates for active tuberculosis in silicotic subjects range from 2- to 30-fold more than those in the same workforce without silicosis [52]. Factors which influence the development of tuberculosis include the severity of silicosis, the prevalence of tuberculosis in the population from which the work force was drawn, as well as their age, general health and HIV status [2,52]. Exposure to silica, without silicosis, may also predispose individuals to tuberculosis [52,53]. Although M. tuberculosis is the usual organism, NTM account for a large proportion of the mycobacterial disease in some populations [42,54]. The prevalence of HIV infection in developing countries has and will increase the burden of tuberculosis in miners exposed to silica dust. A recent study [55] of the effect of HIV infection on silicosis and tuberculosis incidence in black South African gold mineworkers found that HIV infection increased the incidence of tuberculosis by five times and silicosis increased the incidence of tuberculosis by three times. The co-existence of HIV and silicosis increased the incidence of tuberculosis multiplicatively by fifteen times.
Connective tissue diseases and renal disease

Associations have been reported between exposure to silica and certain connective tissue diseases including progressive systemic sclerosis, systemic lupus erythematosus, rheumatoid arthritis, and renal disease [12,56].

Chronic obstructive airways disease

Chronic obstructive airways disease (emphysema) and chronic bronchitis are common manifestations of long term occupational exposure to silica dust and can develop in silica-exposed individuals with or without radiological signs of silicosis. In their comprehensive review, Hnidzo and Vallyathan [57] provide evidence that smoking can potentiate the effect of silica dust on airflow obstruction.

Presentation, diagnosis and clinical features

Silicosis is diagnosed on the basis of a history of exposure and the characteristic radiographic changes. Occasionally, however, even advanced silicosis (diagnosed by histology) may not be recognized on a chest radiograph [43]. It also remains unclear whether smoking predisposes exposed miners to silicosis or nonspecific radiographic changes from smoking are misinterpreted as silicosis [58].

People with silicosis are usually asymptomatic. The appearance of breathlessness is usually associated with a complication such as progressive massive fibrosis or tuberculosis, or may reflect associated airway disease. Cough and sputum production are common symptoms and usually relate to chronic bronchitis but may reflect the development of tuberculosis or lung cancer. The presence of cough, haemoptysis, weight loss, fever or any new radiographic feature should be pursued with culture of sputum or bronchoalveolar lavage fluid or with culture and histological examination of tissue. It should be noted that those with silicosis are also at risk for extrapulmonary tuberculosis [42,52].

Treatment and prevention

There is currently interest in the use of therapeutic agents to treat silicosis and in lung lavage to remove silica from the lung but a favourable impact on progression of acute or chronic silicosis has not been demonstrated [59]. Management of all forms of silicosis should also be directed toward control of mycobacterial disease. All subjects with silicosis should have a tuberculin skin test and, if it is positive, be offered treatment for latent tuberculosis infection [41].

The adverse effects of smoking and the interaction between silica exposure and smoking in the development of COPD, make smoking cessation programmes particularly important for silica-exposed miners [56,57].
References

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