Pneumoconiosis

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Abstract

The term 'pneumoconiosis' is used to describe a set of lung diseases caused by the repeated inhalation of small particles in which long-term retention in the lung is a key causative factor. The site of damage within the lung is a function of both the size and the toxicity of the inhaled dust, fume or fibre. The ability of different types of particles to cause fibrosis varies widely: crystalline silica is highly fibrogenic, whereas iron oxide is not. In susceptible individuals, pneumoconiosis usually develops after many years of exposure, sometimes presenting after retirement. Where cases are detected during employment (e.g. during health surveillance), reduction or cessation of further exposures should be the goal. Prevention is of prime importance as the lung damage caused by pneumoconiosis is irreversible, and the retained substance may continue to cause harm many years after exposure has ceased.

Keywords Coal; complicated; COPD; dust; lung; occupation; pneumoconiosis; silica; simple

Key points

- Pneumoconiosis usually takes many years to develop and affects only a proportion of similarly exposed workers
- The potential for harm depends on a range of factors including the particle size of the dust or fibre, cumulative inhaled dose, clearance rates and inherent toxicity
- Common types of pneumoconiosis include coal worker's pneumoconiosis, silicosis and asbestosis
- The radiological severity of pneumoconiosis can be graded by using the International Labour Organization classification (Table 2)
- Treatments for pneumoconiosis are only supportive, making early detection by health surveillance very important
- Where appropriate, management of existing cases should focus on prevention of further exposure, smoking cessation and compensation advice

Introduction

The term 'pneumoconiosis' is used to describe a set of occupational lung diseases associated with inhalation of an agent (dust, fume, fibre) in which retention in the lung is a key causative factor. The term is most commonly used in the context of coal worker's pneumoconiosis (CWP) and silicosis, but a number of other agents are implicated. Pneumoconiosis generally develops in susceptible individuals after many years of relevant industrial exposure, and can progress after exposure has ended.

The ability of different types of retained particles to cause lung damage varies widely; for example, silica dust is highly fibrogenic, whereas iron oxide dust is not. This depends on the size and toxicity of the inhaled particle, as well as the lung's ability to clear it. Generally, particles with a median diameter of 0.5–10 micrometres can penetrate into the alveoli, and those that are toxic to host cells can cause permanent harm. Many mechanisms are likely to be specific to the individual causative agent; in general, however, release of proinflammatory cytokines, initially from alveolar macrophages, causes fibroblast formation and eventual fibrosis.

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Only a proportion of similarly exposed workers develop pneumoconiosis, suggesting that genetic factors can be relevant in this type of disease. No specific therapies are available, so prevention of exposure and early detection of disease are of prime importance. Selected patients should be referred for consideration of transplantation.

This article focuses on the major causative exposures responsible for pneumoconiosis in the UK (coal, silica, asbestos). Other causes are listed in Table 1.

Coal worker's pneumoconiosis

Epidemiology

Although the global consumption of coal as fuel has continued to rise,[1] coal production in the UK has dramatically decreased over the last few decades. This has been associated with a falling mortality and incidence of CWP over the past 10–15 years (with approximately 140 deaths and 250–300 new cases each year). The risk of CWP relates to the duration and level of exposure, as well as the rank (carbon content) of the coal. In addition, miners exposed to dust with high quartz content (10–15%) can develop a disease similar to silicosis. CWP can be simple or complicated depending on the absence or presence of large opacities.

Clinical features

Simple coal-worker's pneumoconiosis is associated with small rounded nodules predominantly in the upper zones of the lung. These coal macules represent collections of dust-laden macrophages and are visible on a chest X-ray. In the absence of coexisting chronic obstructive pulmonary disease (COPD), simple CWP is not usually associated with symptoms, physical signs or abnormal physiology.

Other causes of pneumoconiosis

Disease	Agent	Notes
Non-fibrogenic[a]		
Siderosis	Iron oxide	Causes no symptoms, signs or physiological abnormality When co-exposure with silica occurs, it can attenuate the fibrosis seen (so-called 'mixed-dust fibrosis')
Stannosis	Tin	Marked radiological abnormality as a consequence of high atomic number No symptoms, signs or physiological abnormality
Others	Barium sulphate (baritosis)	Marked radiological abnormality as a consequence of high atomic number No symptoms, signs or physiological abnormality
	Chromite, zirconium, antimony	Marked radiological abnormality as a consequence of high atomic number No symptoms, signs or physiological abnormality
Fibrogenic		
Berylliosis	Beryllium	Sarcoid-like condition with fibrosis May respond to corticosteroids

[a] Not thought to cause significant pulmonary fibrosis.

Table 1

Complicated coal-worker's pneumoconiosis usually occurs on a background of the simple form, although it rarely occurs *de novo*. It is associated with slowly progressive upper lobe mass formation and fibrosis (previously termed progressive massive fibrosis). Complicated forms are commonly associated with cough, breathlessness and, in certain cases, progression to hypoxia and right heart failure.

International Labour Organization classification of small and large opacities

Small opacities

Category

- 0 None (or fewer than category 1)
- 1 Small opacities, few in number
- 2 Small opacities, numerous
- 3 Small opacities, very numerous, plus obscure lung markings

Large opacities

Category

- A Greatest diameter 10—50 mm
- B One or more opacities larger than category A, with combined area less than that of the right upper lobe
- C One or more opacities with combined area more than that of the right upper lobe

Table 2

Investigation

Radiology: the diagnosis of simple or complicated CWP is based on typical radiological findings and an occupational history of appropriate coal dust exposure. If available, previous radiology is particularly helpful if it shows relatively stable appearances over many years (Figure 1). Patients may also recall being told that they had 'signs of dust' on health surveillance X-rays taken while they were still working. Where radiological features are not typical, particularly if there is a significant smoking history, patients with lung nodules and mass lesions should be discussed with the lung cancer multidisciplinary teams. Particular diagnostic difficulty may occur in Caplan's syndrome (CWP plus rheumatoid arthritis), in which cavitating nodules are seen that mimic other conditions such as lung metastases, Wegener's granulomatosis and tuberculosis.

Serology: in CWP, both rheumatoid factor and antinuclear antibody may be present in the serum, although neither is sufficiently sensitive to be helpful in diagnosis.

Management and prevention

There are no effective medical treatments for CWP, other than management of hypoxia and right heart failure. Coal dust is also a cause of COPD, and coexisting airways obstruction should be treated. The importance of smoking cessation should also be highlighted. Identifying simple CWP by health surveillance and reducing further exposures, in an attempt to prevent complicated disease, is key.