

# Occupational lung disorders of the parenchyma: important points for clinicians

## Educational aims

- ▶ To provide guidance on how to explore the possibility of an environmental aetiology in patients with interstitial lung disease.
- ▶ To describe the main causes of the mineral pneumoconioses, including silicosis and asbestosis.
- ▶ To describe the features of lung diseases associated with hypersensitivity, including extrinsic allergic alveolitis, chronic beryllium lung disease, and cobalt lung.
- ▶ To describe some novel forms of occupational lung diseases, such as those caused by exposure to synthetic polymers.

## Summary

Occupational and environmental exposures can lie at the root of a wide range of interstitial lung diseases and should always be considered when making a diagnosis. A detailed history is an invaluable adjunct to any other diagnostic tests.

These diseases can be the direct result of cumulative exposure to the causative agent, or their occurrence may be mainly related to individual susceptibility.

As lifestyles and employment patterns change, the traditional pneumoconioses are being supplemented with diseases that may result from newer technologies and exposure profiles.

Occupational diseases of the parenchyma often pose a difficult diagnostic challenge. An occupational or environmental aetiology should always be considered in patients with interstitial lung disease. Pathology may be an important adjunct or even an essential tool for making a (differential) diagnosis, but a comprehensive and detailed history (box 1) is paramount in any attempt to find out whether a disease has an exogenous cause and, if so, what that cause is. The clinician should take into account both recent and remote exposures, investigating personal habits (for

instance smoking), medication use, work, hobbies, contact with animals, home environment and so on. Both recent and remote exposures should be considered [1]. Such an undertaking is a skill in itself, and it may be useful to consult a specialist in occupational medicine or toxicology.

Occupational diseases caused by exposure to mineral dust particles range from the traditional pneumoconioses – such as silicosis, coal worker's pneumoconiosis and asbestosis – to less common pneumoconioses, such as "welder's lung" and talcosis [2–5].

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## Key points

- ▶ An occupational or environmental aetiology should always be considered in cases of interstitial lung disease.
- ▶ A detailed history of the patient's exposures is vital in making a diagnosis.
- ▶ Depending on the condition, cumulative exposure or host susceptibility can be the key determinant.

**BOX 1. Taking an occupational history**

An occupational history needs to be comprehensive and specific. It could include the following

**Time**

- From school until present.
- Have there been any periods of military service?
- Jobs held from school until present.
- (Recent) changes in production processes.
- Describe a typical working day/week.

**Space**

- Exact name and address of the workplace.
- The layout of the workplace (make a diagram).

**Type of work**

- Function (exact job description).
- Exposures and their circumstances (use MSDS (materials safety data sheets). Many MSDS can be found using an internet search
- Personal protective equipment.
- Normal and occasional activities.
- Have there been any accidents?
- What other activities are carried out nearby?

**Other factors**

- Casual jobs.
- Hobbies.
- Domestic exposures (animals including birds, damp, mould, household members' jobs).
- Environmental exposures (home, travel).
- Smoking and other habits.
- Cosmetics.
- Medication.

## 'Traditional' pneumoconioses

### Silicosis [5, 6]

Silicosis is caused by exposure to free crystalline silica ( $\text{SiO}_2$ ). This is generally in the form of quartz, but may be cristobalite. Exposure occurs mainly in mines or foundries, but many other occupations involve work with quartz-containing materials, such as sand or other minerals [7]. The pathological hallmark of silicosis consists of the silicotic nodule, which may often appear first in hilar or mediastinal lymph nodes. Finding silicotic nodules must always raise the suspicion of silicosis, and certainly constitutes a serious challenge to a diagnosis of, for instance, sarcoidosis. The occurrence of silicotic nodules may also be an important factor in attributing an occupational aetiology in cases of bronchopulmonary cancer, since occupational exposure to free crystalline silica is a recognised cause of human cancer. On the other hand, the absence of characteristic silicotic nodules should not necessarily exclude a diagnosis of silica-induced lung disease.

In some patients, silicosis may have an accelerated clinical course. Very heavy exposures may lead to alveolar lipoproteinosis.

### Coal worker's pneumoconiosis

Coal worker's pneumoconiosis may or may not be associated with the presence of significant numbers of silicotic noduli. Consequently the systematic use of the term anthraco-silicosis for coal worker's pneumoconiosis may not always be adequate.

### Asbestosis [5, 8]

The term asbestosis should only be used to label the parenchymal disease caused by asbestos exposure, and should not be used when only the pleura is involved [9, 10]. Nevertheless, the presence of (uni- or bilateral) localised thickening of the parietal pleura (*i.e.* plaques) or diffuse pleural thickening in cases of pulmonary fibrosis strongly points to a diagnosis of asbestosis. The absence of pleural lesions, however, does not exclude the existence of asbestosis. The diagnosis of asbestosis rests on a history of a substantial past exposure to asbestos – which may be documented by the finding of asbestos bodies in sputum, bronchoalveolar lavage or lung tissue – in the presence of a compatible clinical, radiological and pathological setting.

## Other conditions

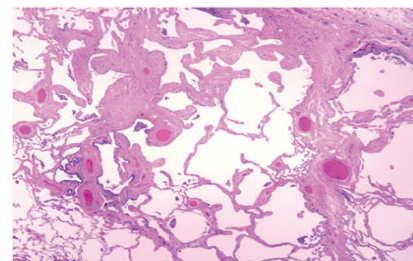
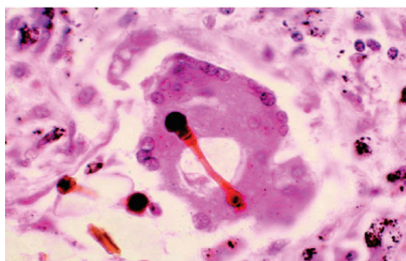
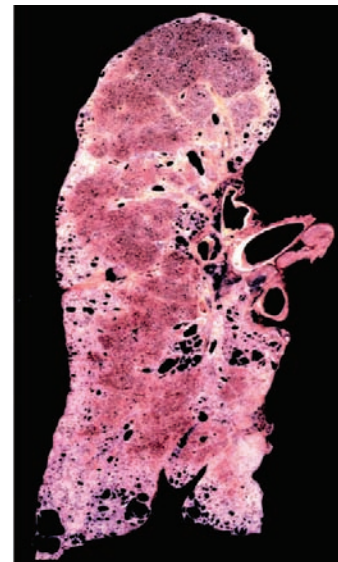
Less common mineral pneumoconioses include siderosis (sometimes found in welders), talcosis and those caused by aluminium, tin or barium. Mixed exposures to silica or asbestos should always be considered. Although individual susceptibility does play a role in the occurrence and clinical course of the mineral pneumoconioses, these disorders result mainly from excessive cumulative exposures to the offending mineral dusts. However, in some parenchymal lung diseases, individual host susceptibility – rather than exposure intensity – is the main determinant of occurrence [11].

Individual host susceptibility is obviously a major factor in extrinsic allergic alveolitis (or hypersensitivity pneumonitis). This disease is generally caused by exposure of a susceptible individual to aerosolised antigenic material of biological origin, such as microbial spores, avian antigens or other proteins. The discovery of specific (precipitating) immunoglobulin G antibodies in the serum represents an important clue in reaching an aetiological diagnosis, but this is not always feasible. The pathology of extrinsic allergic alveolitis is that of a lymphoplasmocytic

bronchiolo-alveolitis with granulomas, but typical (subacute) cases of extrinsic allergic alveolitis rarely require full pathological confirmation. On the other hand, in patients with a long-standing low-grade exposure and a silent progression towards chronic fibrosis, the typical pathological features may no longer be evident and the differential diagnosis with idiopathic pulmonary fibrosis becomes more difficult. Some synthetic chemicals, most notably some isocyanates, may also cause a clinical picture typical of hypersensitivity pneumonitis.

The pulmonary presentation of chronic beryllium lung disease is virtually indistinguishable from that of sarcoidosis [12]. Because chronic beryllium disease results from a cellular immune response against beryllium, the diagnosis of berylliosis can be made with an *in vitro* lymphocyte proliferation test using a beryllium salt. Other exposures to minerals, most notably talc, and metallic agents [13, 14], including aluminium, may also lead to parenchymal lung disease with sarcoid-like granulomas. Consequently, a potential exposure to beryllium or other substances must always be considered even in cases of pathologically documented "sarcoidosis" [15].

Hard-metal lung disease is a rare condition, caused by cobalt-containing particles [16]. It may present clinically as hypersensitivity pneumonitis. However, its pathology is characterised,



at least in typical cases, by the presence of bizarre "cannibalistic" multinuclear cells in bronchoalveolar lavage and in lung tissue (giant cell interstitial pneumonitis).

**Figure**  
Features of asbestosis. Reproduced from [22] with permission from the publisher.

### Educational questions

Please choose one correct answer per question.

- Silicosis is a disease of the lung parenchyma
  - that is generally caused by a brief occupational exposure to free crystalline silica.
  - that is caused by a prolonged occupational exposure to coal dust.
  - that is always accompanied by eggshell calcifications in the hilar lymph nodes.
  - that is characterised pathologically by the presence of nodules of whorled collagen surrounded by inflammatory cells in the lung parenchyma.
- Asbestosis is a fibrosis of the lung parenchyma
  - that is always accompanied by pleural changes visible on a high resolution CT of the chest.
  - that is always easy to differentiate clinically from idiopathic pulmonary fibrosis.
  - that is characterised by the presence of asbestos bodies in the alveoli or BAL.
  - that can be caused by environmental exposure to a variety of natural and man-made mineral fibres.
- Chronic beryllium disease
  - occurs frequently in welders of stainless steel.
  - is characterised by the presence of multinucleated giant cells in the lung parenchyma and BAL.
  - is caused by a cell-mediated immunological reaction against beryllium.
  - only occurs in nonsmokers.
- Flock worker's lung
  - has been described in workers exposed to high levels of microfibrils of nylon.
  - has been described only in workers exposed to microfibrils of nylon.
  - is caused by high exposure to organic solvents used in the textile industry.
  - is characterised by severe airways obstruction caused by bronchiolitis obliterans.

## Suggested answers

1. d
2. c
3. c
4. a

## Novel aetiologies

Recent outbreaks of severe lung disease in textile workers who had been engaged in paint-spraying ("Ardystil syndrome"), have illustrated that organising pneumonia may have an occupational origin [17–19]. Similarly, the occurrence

of interstitial lung disease in workers exposed to microfibrils of synthetic materials such as nylon or polyethylene ("flock worker's lung") show that new occupational causes of lung diseases may still arise [20–21]. Pulmonary physicians and pathologists have an important role in discovering and characterising these novel conditions.

### References

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### Further reading

#### Textbooks of occupational lung disease

Parkes WR. *Occupational Lung Disorders*. 3rd Edn. Oxford, Butterworth-Heinemann, 1994.

A classical textbook; useful information on mineralogy; good pictures; contains many old references to occupational lung diseases, including "exotic" ones.

Harber P, Schenker M, Balme JR. *Occupational and Environmental Respiratory Disease*. St Louis, Mosby, 1996.

Excellent US textbook; comprehensive.

Hendrick DJ, Burge PS, Beckett WS, Churg A. *Occupational Disorders of the Lung: Recognition, Management, and Prevention*. London, WB Saunders, 2002.

Excellent and up-to-date textbook with a novel approach, including "difficult cases".