The pathology of asbestosis

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Abstract

The term *asbestosis* refers to diffuse interstitial pulmonary fibrosis consequent to the excess inhalation of asbestos fibres. It is a disease associated with heavy cumulative asbestos dose and the latent period, from initial exposure to disease manifestation, is long usually 20 years or more, with an inverse correlation with dose. Because heavy industrial exposures have diminished the incidence of asbestosis has decreased. Asbestosis is a divisible disease with the frequency and severity of disease correlating with cumulative asbestos dose. Disease severity also correlates with asbestos fibre type (amphiboles more potent than commercial chrysotile), immune and genetic factors. Pathologically, there are two components to the diagnostic criteria which must be met: first, the presence of diffuse interstitial lung fibrosis of an appropriate pattern; and second, some tissue marker of excess asbestos inhalation either requisite numbers of asbestos bodies (as determined by light microscopy) or elevated asbestos fibres (as determined by mineral analysis). The clinical picture is not specific although in contrast to idiopathic pulmonary fibrosis, which is the most frequent diagnostic problem, asbestosis has a slowly progressive course over decades. Lung cancer occurs at cumulative asbestos doses similar to that necessary to cause lung fibrosis (asbestosis). The main differential diagnoses with asbestosis are idiopathic pulmonary fibrosis, pulmonary fibrosis associated with connective tissue disorders, chronic phase hypersensitivity pneumonitis and silicate (mica, talc, kaolin) pneumoconiosis.

Keywords asbestos; asbestosis; interstitial lung disease; pathology

Introduction

Asbestos is a general term applied to certain fibrous forms of various silicate minerals which have crystallized in nature producing fibres of high tensile strength and flexibility, a feature which is important in its commercial applications.[1] Asbestos occurs in one of two forms: serpentine and amphibole. Chrysotile is the only serpentine form of asbestos and comprises 90% of all asbestos used in most countries. The major amphiboles that have been used commercially are amosite, crocidolite, and—to a much lesser degree—anthophyllite. Non-commercial amphiboles include tremolite and actinolite. The physicochemical differences between amphiboles and chrysotile are distinct and underpin their significant different biological toxicities. Amphibole fibres are straight and rigid with long biopersistence in tissues (years to decades) whereas chrysotile fibres are curled, pliable with short biopersistence in tissues (weeks to months) (Figures 1—3). The persistence of respirable bio-active fibres is the single most important factor in the development of asbestos-related diseases whether neoplastic or non-neoplastic. Because of its physico-chemical properties, chrysotile asbestos is most suitable for making fabrics, other flexible items and also used in friction product manufacture. The amphiboles with superior chemical and physical stability were used often with chrysotile to make various asbestos-cement products, insulation pipes and boards, roofing and fire-proofing materials.

Historically, asbestosis was the first asbestos-related disease recognized. Dr. Montague Murray, has been generally credited as the first person to report the disease in 1899, with the term *asbestosis* coined in 1925 by Oliver and again used in 1927 by Cooke.[2] The links between asbestos and lung cancer in the presence of asbestosis were suggested and disputed a decade later and firmly established in 1955 by Doll.[3,4] For mesothelioma the association with asbestos was suggested in 1960 by Wagner and co-workers in his case series of persons in the crocidolite mining district of Griqualand West in the North West Province of South Africa.[5]

The adverse risks of asbestosis have been effectively controlled by the workplace changes introduced by the 1931 Asbestos regulations and again by the more stringent 1969 Asbestos regulations. These allowed the continued use of asbestos only if maximum allowable concentrations of dust were not exceeded and if other precautions were maintained. In the 1970s, 1980s, and 1990s further restrictions, both voluntary and statutory, were placed on the importation and use of asbestos. This has resulted in a dramatic decrease in the number of symptomatic cases of asbestosis in the developed countries. In July 1999, the European Commission announced a European Union ban on all remaining chrysotile use by 1 January 2005. In the United States, Occupational Safety and Health Administration (OHSA) introduced a range of controls for asbestos. The current OSHA airborne permissible exposure limit (PEL) (introduced in 1994) is set at 0.1 fibre/ml (for all fibre types). Life-long exposure to asbestos at these permissible levels is considered sufficient to prevent the manifestation of asbestosis.

It has long been known that amphibole asbestos dust exposure constitutes a significant danger to health. However, some critical issues still remain in scientific dispute, including the relative hazards of different types of asbestos and whether there is a safe level of exposure to any of them. Nowadays, the major adverse health effects of asbestos are related to increased risk of cancer especially malignant pleural mesothelioma following low dose exposures from brief, intermittent sources to amphibole asbestos in populations of persons previously not considered 'at risk' of developing fatal asbestos-related diseases.

Clinico-radiological features

The clinical presentation of asbestosis is typically insidious with shortness of breath on exertion and a dry cough. Auscultation may reveal late inspiratory crackles initially in the postero-lateral regions of the lower zones but later becoming more widespread as the disease progresses. In advanced stages cyanosis and cardiorespiratory failure may occur.

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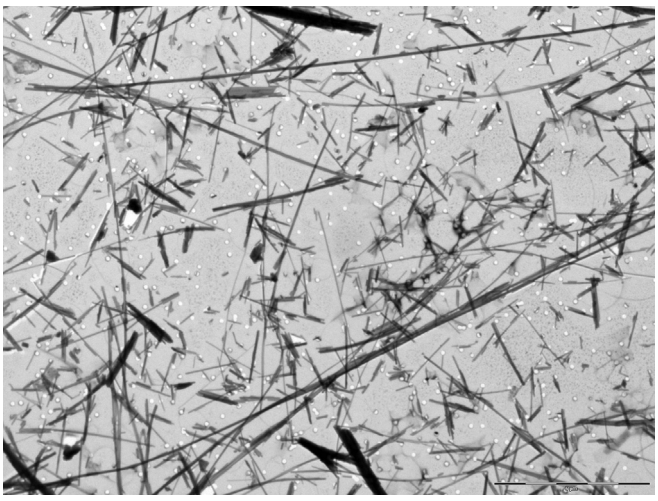

Figure 1 Chrysotile asbestos fibres (TEM).

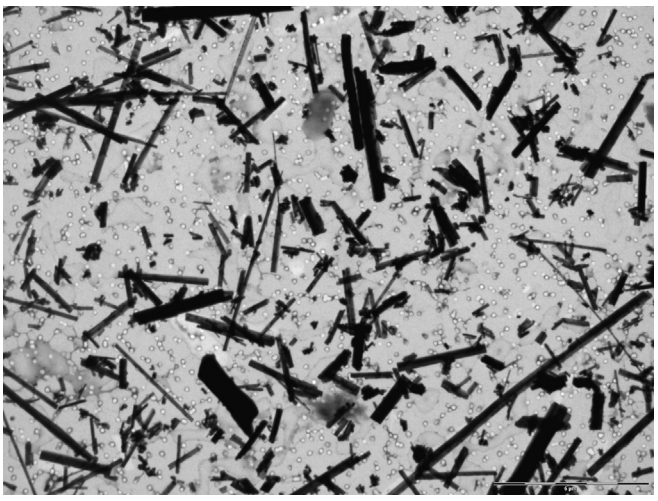

Figure 3 Crocidolite asbestos fibres (TEM).

Lung function tests show a restrictive pattern with impairment of gas transfer but there may be an element of airflow limitation.

Generally asbestosis is characterized by lower lobe involvement and small irregular opacities. Progression will result in thickening of the linear opacities and the development of honeycombing particularly in the sub-pleural region of the lower lobes. Pleural abnormalities such as diffuse pleural fibrosis or plaques may be present.

Pathology

Asbestosis is characterized by diffuse interstitial fibrosis of the lungs, particularly with involvement of the lower zones and with sub-pleural accentuation (Figures 4). In advanced cased the lungs have a firm consistency with a bosselated surface and have a grey—white cut surface with areas of honeycombing. Purulent secretions may be seen indicating complicating pneumonia. Firm mass lesions may suggest complicating neoplasia. There may or may not be associated pleural thickening and/or plaques. The presence of pleural changes is a neutral factor in determining the causation of the lung fibrosis because the pleura is far more sensitive to the injurious effects of asbestos than the lung parenchyma. Accordingly, the same lack of significance may be stated for exposed persons with lung cancer and pleural changes.

The light microscopic findings of asbestosis are now well established and defined by the recent 2010 College of Pathologists — Pulmonary Pathology Society (CAP-PPS) Asbestosis Guidelines committee.[6] First there is a requirement to confirm the presence of diffuse interstitial fibrosis *of an appropriate pattern* described as 'always acellular and collagenous rather than fibroblastic and inflammatory'. The second component is the presence of a necessary minimum number of either asbestos bodies or fibres (see below). The aforementioned *asbestosis*

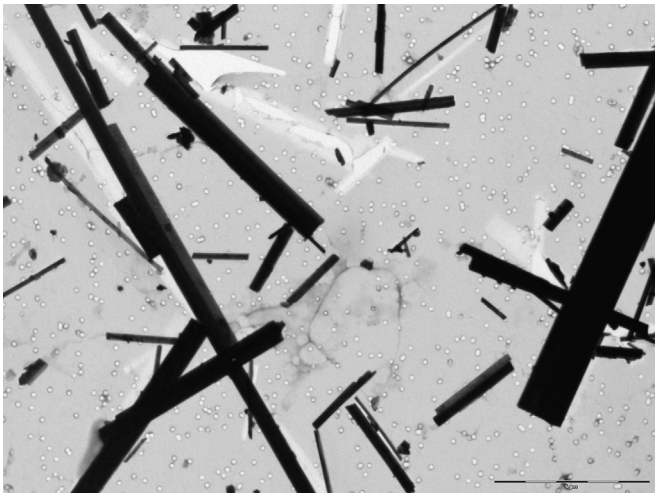

Figure 2 Amosite asbestos fibres (TEM).

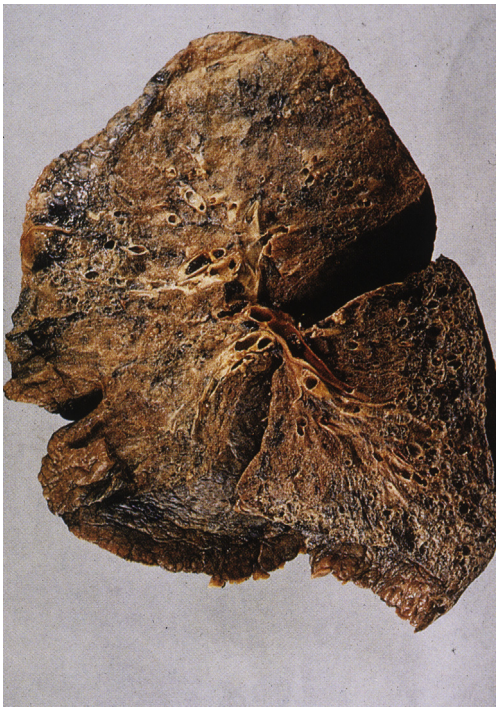

Figure 4 Lung showing fibrosis and honeycombing (asbestosis) most marked in the lower zones and sub-pleurally.