Helsinki Criteria for Asbestos-Related Disease

Antti Tossavainen
Institute of Occupational Health (FIOH), Helsinki, Finland

Abstract

The International Expert meeting on Asbestos, Asbestosis and Cancer was convened in Helsinki on 20-22 January 1997 to discuss disorders of the lung and pleura and to agree contemporary criteria for their diagnosis and attribution with respect to asbestos. The group decided to name the consensus report as ‘The Helsinki Criteria’ (Scand J Work Environ Health 1997;23:311-316).

The clinical diagnosis of asbestos-related diseases is based on a detailed interview of the patient and occupational data on asbestos exposure, signs and symptoms, radiological and lung physiology findings, and selected cytological, histological and other laboratory studies. Asbestosis is generally associated with relatively high exposure levels. Radiological findings of small opacities (ILO grade 1/0) are usually regarded as early stage of asbestosis. Smoking effects should be considered in the evaluation of lung function tests and respiratory symptoms. A histological diagnosis of asbestosis requires the identification of diffuse interstitial fibrosis in well inflated lung tissue plus the presence of asbestos bodies or uncoated fibres. Low exposures from work-related, household and natural sources may induce pleural plaques but for diffuse pleural thickening, higher exposure levels may be required.

For mesothelioma, an occupational history of brief or low-level exposure should be considered sufficient. A lung fibre count above the background range, radiological findings or histopathological evidence can also relate a case of pleural or peritoneal mesothelioma to asbestos exposure. Smoking has no influence on the risk of mesothelioma.

All major histological types of lung cancer can be related to asbestos. Clinical signs and symptoms are of no significant value in deciding whether or not an individual case is attributable to asbestos. One year of heavy exposure (manufacture of asbestos products, asbestos spraying, insulation work, demolition of old buildings) or 5 - 10 years of moderate exposure (construction, shipbuilding) may increase the lung cancer risk 2-fold or more. A minimum lag-time of 10 years from the first exposure is required. A cumulative exposure of 25 fibre-years was estimated to double the risk of lung cancer. The presence of asbestosis is an indicator of heavy exposure and can contribute some additional risk of lung cancer beyond that conferred by asbestos exposure alone. A 2-fold risk of lung cancer is related to retained fibre levels of 2 million (>5 µm) or 5 million (>1 µm) amphibole fibres per gram dry lung tissue. This concentration is approximately equal to 5000 to 15000 asbestos bodies per gram dry tissue, or 5 to 15 asbestos bodies per millilitre of bronchoalveolar lavage fluid. Tobacco smoking does not detract from the risk of lung cancer attributable to asbestos exposure.
Background

Despite national and international actions, occupational exposure to asbestos continues to be a major health hazard. Up to 20,000 asbestos-induced lung cancers and 10,000 mesotheliomas occur annually in Western Europe, Scandinavia, North America, Japan and Australia. In the most affected age groups, mesothelioma may account for about 1% of all deaths. In addition, about 5-7% of all lung cancers can be attributed to asbestos exposure. The life cycle of asbestos products begins in the primary asbestos industry and continues with secondary manufacture, installation, usage and disposal. Worldwide millions of workers have been exposed to asbestos in the workplace, most often during use, maintenance, repair and replacement of asbestos-containing materials. About 170 tons of produced and consumed asbestos will cause at least one death from pleural or peritoneal mesothelioma and contribute to several cases of lung cancer, asbestosis and pleural abnormalities [1]. Thousands of asbestos-related diseases will be diagnosed and treated by clinicians across industrialized countries in the next 20 to 30 years and even later in the developing world.

The International Expert Meeting on Asbestos, Asbestosis and Cancer was convened in Helsinki on 20-22 January 1997 to discuss disorders of the lung and pleura and to agree upon contemporary criteria for the diagnosis and attribution with respect to asbestos. The group decided to name the consensus report as 'The Helsinki Criteria' [2,3]. Accordingly, the clinical diagnosis of asbestos-related diseases should be based on a detailed interview of the patient and occupational data on asbestos exposure, signs and symptoms, radiological and lung physiology findings and selected cytological, histological and other laboratory studies. Subsequently the Helsinki criteria for asbestos-related lung cancer have met widespread acceptance and are consistent with approaches to attribution and compensation in several countries, e.g., in Germany, France, Finland and Australia [4,5].

Work History

In general, reliable work histories provide the most practical and useful measure of occupational asbestos exposure. Using structured questionnaires and checklists, trained interviewers can identify persons who have a work history compatible with significant exposure. In industrialized countries, about 20 to 40% of adult men report some past occupations and jobs that may have entailed asbestos exposure at work. The interview data may need to be supplemented with employment records and inquiries to current or past workplaces. Technical and hygienic experts can advise on the probable level of asbestos exposure using measurement data or general knowledge. The individual work histories are commonly classified in terms of exposure probability, duration and intensity. A cumulative fibre dose, as expressed as fibre-years, can be calculated from typical fibre levels at various occupations and in the use of asbestos-containing materials.

Tissue Analysis

Analysis of lung tissue for asbestos fibres and asbestos bodies can provide data to supplement the occupational history. For clinical purposes, the following guidelines are recommended to identify persons with a high probability of asbestos exposure at work:
over 0.1 million amphibole fibres (>5 µm) per gram dry lung tissue or over 1 million amphibole fibres (>1 µm) per gram dry lung tissue as measured by electron microscopy in a qualified laboratory or over 1000 asbestos bodies per gram dry tissue (or 100 asbestos bodies per gram of wet tissue) or over 1 asbestos body per millilitre of bronchoalveolar lavage fluid as measured by light microscopy in a qualified laboratory.

**Exposure Criteria and Diagnosis**

Asbestosis is generally associated with relatively high exposure levels. Radiological findings of small opacities (ILO grade 1/0) are usually regarded as an early stage of asbestosis. Smoking effects should be considered in the evaluation of lung function tests and respiratory symptoms. A histological diagnosis of asbestosis requires the identification of diffuse interstitial fibrosis in well inflated lung tissue plus the presence of asbestos bodies or uncoated fibres. Lower exposures from work-related, household and natural sources (below 0.01 fibres/cm³) may induce pleural plaques but for diffuse pleural thickening higher exposure levels may be required.

For mesothelioma, an occupational history of brief or low-level exposure should be considered sufficient. About 80% of mesothelioma patients have had some occupational exposure to asbestos. A lung fibre count above the background range, radiological findings or histopathological evidence may also relate a case of pleural or peritoneal mesothelioma to asbestos. In the absence of such markers, a history of significant occupational, domestic or environmental exposure to asbestos will suffice for attribution. A minimum of 10 years from the first exposure is required to attribute the mesothelioma to asbestos exposure, although in most cases the latency interval is longer (about 30 to 40 years). Smoking has no influence on the risk of mesothelioma.

All major histological types of lung cancer can be related to asbestos. Clinical signs and symptoms are of no significant value in deciding whether or not an individual case is attributable to asbestos. One year heavy exposure (manufacture of asbestos products, asbestos spraying, insulation work, demolition of old buildings) or 5-10 years of moderate exposure (construction, shipbuilding) may increase the lung cancer risk 2-fold or more. A minimum lag-time of 10 years from the first exposure is required. A cumulative exposure of 25 fibre-years was estimated to double the risk of lung cancer. Clinical cases of asbestosis may occur at comparable levels of cumulative exposure. The presence of asbestosis is an indicator of heavy exposure and can contribute some additional risk beyond that conferred by asbestos exposure alone. Heavy exposure, in the absence of radiologically diagnosed asbestosis, is considered sufficient for attribution. Because pleural plaques may be associated with low levels of asbestos exposure, the attribution of lung cancer to asbestos must be supported by a relevant occupational history or other evidence. A 2-fold risk of lung cancer is related to retained fibre levels of 2 million (>5 µm) or 5 million (>1 µm) amphibole fibres per gram dry lung tissue. This concentration is approximately equal to 5000 to 15 000 asbestos bodies per gram dry tissue, or 5 to 15 asbestos bodies per millilitre of bronchoalveolar lavage fluid. Tobacco smoking does not detract from the risk of lung cancer attributable to asbestos exposure.

**References**


