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Te Kaporeihana Āwhina Hunga Whara

ACC Review:

A DISTILLATION OF BEST PRACTICE REFLECTING ACC'S CURRENT POSITION

Asbestos-related disease

- Asbestos is a naturally occurring mineral that has been linked to human lung disease. All forms of asbestos increase the risk
- There are three types of asbestos-related lung disease, interstitial fibrosis (asbestosis), pleural plaques and thickening, lung and pleural cancer
- Latency periods of 20-50 years between exposure and disease are typical
- Standard evaluation includes work history, plain chest x-ray and lung function testing
- Treatment of asbestosis includes appropriate vaccinations, treatment of lung infections, smoking cessation, and the use of oxygen if necessary.

Background

Asbestos is a naturally occurring fibrous silicate once widely used for its heat-resistant properties. Most exposure to asbestos has been industrial, affecting workers who mine, process, or manufacture asbestos products. Workers in shipbuilding, construction, insulation, electrical, pipefitting and demolition are also among those considered at increased risk. General environmental exposures are not associated with significant asbestos-related disease.

All types and lengths of asbestos fibres irritate lung tissues and can cause inflammation, scarring and neoplastic change.[1] The primary adverse effects are pleural plaques and diffuse pleural thickening, pleural effusions, asbestosis, lung cancer and mesothelioma. Recognition of these adverse health effects led to legislative control of its use in the early 1980s. However, because of the latency period between exposure and onset of around 20-50 years, asbestos-related disease continues to be present.[2]

Adverse Health Effects

Pleural plaques develop after 20-40 years of low, intermittent exposure and affect up to 50% of those exposed. The plaques consist of slow growing collagen bundles (that sometimes contain asbestos fibres) in the parietal pleura. Plaques continue to grow even after cessation of exposure but are not considered pre-malignant. Calcification may occur after about 30-40 years. Patients are often asymptomatic with plaques found coincidentally.

Diffuse pleural thickening is less specific for asbestos exposure. The thickening is probably due to inflammation and fibrosis of the pleural lymphatics and may follow pleural effusions. It has a latency period of about 15 years. If extensive, lung expansion can be restricted causing significant dyspnoea.

Benign pleural effusions are often the earliest manifestation of asbestos-related disease, typically occurring within 10 years of exposure. Most are self-limiting and resolve within a few months but some are chronic or recurrent.[3] Typically there are no symptoms but some patients develop pleuritic chest pain, breathlessness and pyrexia.

Asbestosis is diffuse pulmonary fibrosis caused by the inhalation of asbestos fibres, generally after high-level, long-term exposure over 15-20 years. Fibrosis begins sub-pleurally in the lower lobes and may progress long after exposure to asbestos has ceased. Asbestosis may cause a dry cough and progressive dyspnoea with the clinical finding of basal inspiratory crackles. Finger clubbing is associated with advanced cases. Severe asbestosis may lead to pulmonary hypertension and respiratory failure.

Lung cancer develops in perhaps 20-25% of heavily exposed asbestos workers. It has a latency period of around 25-35 years. The predominant subtype is bronchoalveolar cell carcinoma, but adenocarcinoma and squamous cell carcinoma also occur. Smoking has a marked synergistic effect upon cancer development. Asbestosis may co-exist.

Malignant pleural (and peritoneal) mesothelioma is a rare disease strongly associated with asbestos inhalation. Pleural effusions commonly antedate development of malignancy. Even short-term occupational exposures can cause mesothelioma, suggesting that the association is not dose-related. The latency period is 20-50 years. Smoking does not appear to be a risk factor. Symptoms of localised chest wall pain and weight loss frequently tend to occur 6-8 months prior to diagnosis. Cough and dyspnoea may also be present.

Clinical Evaluation

Clinical evaluation should include a complete medical and occupational history, and physical examination with special attention to the lungs, heart and upper extremities (i.e. clubbing). A single PA chest x-ray is usually sufficient for screening purposes in previously exposed workers.[4] It will detect pleural plaques, diffuse pleural thickening and pleural effusions, although computerised tomography is more sensitive and specific.[5] Up to 30% of patients with early asbestosis have a normal chest x-ray but subtle changes will be visible on high resolution CT scan.[6] Lung function tests are indicated for suspected asbestosis and should include a diffusion capacity. Asbestosis is associated with a restrictive impairment and reduced diffusion capacity but in smokers a mixed obstructive-restrictive deficit may exist. Confirmed cases require periodic spirometry. Image-guided pleural thoracentesis or biopsy is occasionally required to investigate pleural effusions and masses. A confident diagnosis of mesothelioma is often difficult to make and usually requires immunohistochemical tests.

Management

Pleural plaques require no management or surveillance. Rarely, the restriction caused by diffuse pleural thickening may be severe enough to warrant decortication of the lung. Symptomatic patients with pleural effusions may be treated initially by therapeutic aspiration or drainage but recurrent effusions often require pleurodesis.

There is no effective treatment for asbestosis. Management consists of treating infections, vaccinating against influenza and pneumococcus, and providing supplementary oxygen during exercise or rest. Steroid and immune-based therapies have not been shown to be beneficial. Patients who smoke should be advised to stop.

The prognosis in pleural mesothelioma is very poor, with a median survival of 10 months and most patients dying within 2 years. In selected cases, complete surgical resection by extra-pleural pneumonectomy followed by systemic chemotherapy may improve survival.[7]

Issues Relevant to ACC

Patients who have pleural plaques, diffuse pleural thickening, asbestosis, malignant mesothelioma and bronchogenic carcinoma as a result of their employment are eligible for ACC cover.

References

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