Short reports

Asymmetric rapidly progressive lung fibrosis: a cause of pseudotumour in asbestosis

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Malignant neoplasia of the lung or pleura is a common and serious complication of exposure to asbestos¹ and necessitates careful surveillance of those at risk. The assessment of a pulmonary mass lesion after exposure to asbestos is complicated by the occurrence of pulmonary "pseudotumours." These result from pleural fibrosis and entrapment of the lung parenchyma (Blesovsky's syndrome.^{2,3}). We report asymmetric rapidly progressive fibrosis of the upper lobe of the lung as another cause of apparent lung malignancy after exposure to asbestos.

Case report

A 38 year old man was investigated for an enlarging opacity in the right upper lobe. Eighteen years before presentation he had worked for 12 months in the crocidolite mill at Wittenoom Gorge in Western Australia. Five years before presentation, a chest radiograph first showed evidence of silicoasbestosis and he subsequently had frequent radiographic examinations of his chest. He admitted to breathlessness on mild exertion but had no cough or haemoptysis. He had smoked 20 cigarettes a day from the age of 18. He had a history of renal calculi and migraine headaches but took no medication. He had worked as a barman and hotelier since leaving the asbestos industry. On examination he was fit, tall, and thin. Apart from a reduced expansion on the right there were no abnormal physical findings in the chest. There was no lymphadenopathy and no abnormalities were found on examination of the cardiovascular system or abdomen.

The haemoglobin, white cell count, and platelet

count results were all normal. Results of serum electrolytes, serum proteins, and liver function tests were also normal. The vital capacity was 5.7 l (predicted 4.5 $\pm$ 0.6 l) and the forced expiratory volume in one second was 3.3 l (predicted 3.7 $\pm$ 0.5 l). The transfer factor of the lung for carbon monoxide was 16.5 ml/min/mm Hg (predicted 30.9 $\pm$ 5.1 ml/min/mm Hg). The earliest abnormalities on chest radiography had been diffuse reticulonodular opacities in the mid and upper lung fields (fig 1). An initially symmetric confluence of opacities in both upper lobes (fig 2) had been followed by progressive and quite asymmetrical enlargement of a mass in the right upper lobe (fig 3). Needle aspiration biopsy showed bronchial epithelial cells, macrophages, asbestos bodies, and debris. No abnormality was found on rigid bronchoscopy. At thoracotomy the upper lobe was found to be shrunken, fibrotic, and contracted to the size of a cricket ball, with the middle and lower lobes correspondingly expanded. The pleural cavity was free of adhesions. There were multiple small white raised areas 1 mm in diameter scattered over the pleural surface of the right upper lobe. A right upper lobectomy was carried out.

The resected portion of lung measured 6.5 cm in diameter and was grey and firm. Microscopically, the normal lung architecture was effaced by nodules and irregular bands of fibrous tissue consisting of a mixture of dense collagenous hypocellular fibrosis with other areas showing a vigorous cellular fibroblastic proliferation. Some birefringent silica particles were present in the hyaline nodules, but the most striking observation was the large number of ferruginous bodies entrapped in fibrous tissue and also located in residual alveolar spaces in association with numerous macrophages (fig 4). The changes were attributed to silicoasbestosis with an exuberant

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Fig 1 *Fine opacities in mid and upper zones. Opacities in apices are becoming confluent and horizontal fissure is raised a little. Lung bases are normal.*

Fig 2 *Mid and upper zone opacities becoming larger and denser. Appearances are those usually associated with silicosis and early progressive massive fibrosis.*

Fig 3 Clearly defined mass in right apex. Pronounced contraction of right upper lobe evidenced by raising of hilum and further raising of horizontal fissure.

Fig 4 Complete destruction of normal architecture by hyaline fibrosis and cellular fibroblastic zones. Numerous ferruginous bodies occupy right half of field. (Haematoxylin and eosin, $\times 220$). Inset: (a) Birefringent silica particles using polarized light ($\times 100$), (b) ferruginous (asbestos) body ($\times 380$).

fibrous tissue response. There was no evidence of a neoplastic process.

Discussion

The radiographic appearances of this man's lesion mimicked carcinoma of the lung, and were sufficiently sinister to prompt thoracotomy and lobectomy. Although the radiograph suggested silicosis, with a predominance of small rounded opacities in an upper zone distribution, his industrial exposure was to asbestos fibre in far higher proportion than to silica dust.⁴ The degree of deficit in transfer factor of the lung, and the histological appearances of the resected lobe also showed asbestosis to be present.

Progressive massive fibrosis of the lungs after exposure to asbestos is uncommon^{5,6} and is often in association with some exposure to silica, as in this case. Upper lobe fibrosis in the absence of overt pneumoconiosis may also follow exposure to asbestos.^{7,8} These processes are usually bilateral and slowly progressive, by contrast with the rapidly increasing size of the lesion (despite the long period since the cessation of exposure) and the asymmetry

of the upper lobe involvement which contributed to the suspicion of malignancy in this case.

Asymmetric progressive massive fibrosis of an upper lobe of the lung should be considered as another cause of apparent malignancy in the differential diagnosis of large radiographic opacities in patients with asbestos related pulmonary fibrosis, particularly if there has been concomitant exposure to silica.

References

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