Medical Memoranda

A Case of Pulmonary Asbestosis

Asbestosis is a form of silicosis, the asbestos fibres consisting mainly of a fibrous magnesium and calcium silicate. These fibres are found in the fine dust present in factories engaged in the manufacture of heat-resisting materials, etc., from crude mineral asbestos.

The lesions produced in the lungs by the inhalation of asbestos fibres take the form of a diffuse pulmonary fibrosis affecting particularly the lower lobes of the lungs. The characteristic "asbestos bodies" may be found in the sputum, but it is only when they are present in clumps or masses that they indicate parenchymal involvement (Beattie and Dickson, 1943). Emphysema and bronchiectasis are commonly present; the pleura is thickened and may be adherent to the diaphragm. Pulmonary tuberculosis may be associated with asbestosis, although not so commonly as with silicosis.

The following case is of interest in view of the lapse of time between exposure to industrial hazard and the diagnosis of the disease, and because of the isolation of "asbestos bodies" in the sputum 20 years after exposure.

CASE REPORT

The patient, a man aged 45, was admitted to a sanatorium on April 4, 1949, with a diagnosis of pulmonary tuberculosis following a mass miniature radiography examination in September, 1948. He gave a history of chronic productive cough of at least ten years' duration accompanied by breathlessness on exertion for approximately the same period. The sputum was white and frothy and has never been blood-stained. Although spare in build his weight has remained stationary at about 8 st. 12 lb. (56.25 kg.). There have been no fresh symptoms during the last few years.

At the age of 12 he had a colectomy performed for rheumatoid arthritis, and has limited movement of the spine, hips, and elbow-joints. He has been in the habit of taking liquid paraffin daily for 35 years.

Occupational History.—From 1924 to 1929 the patient worked as an asbestos crusher. The crude dry mineral asbestos was crushed by mechanical hammers into its constituent fibres, which passed into hoppers and thence into sacks. During this process the air was continuously filled with dry asbestos which entered the workpeople's mouths and noses in spite of the simple precautions they took. Respirators were not provided, so the men were in the habit of tying their pocket-handkerchiefs over their mouths and noses while working. The patient worked in an atmosphere such as this for 48 hours weekly. He gave up this employment in 1929 and has not worked with asbestos since.

On examination he was seen to be of spare build and poorly nourished. His pulse, temperature, and respirations were within normal limits. The chest was emphysematous, with an expansion of only 1½ in. (4.4 cm.). Vital capacity was 2,300 c.cm. The percussion note was not impaired. A few moist sounds were present at both bases. There was no cyanosis or clubbing. Repeated examination of the sputum and stomach washings both by culture and by animal inoculation was negative for tubercle bacilli.

Asbestos bodies were found in the sputum on April 12. Oil droplets were never demonstrated. An x-ray film taken on April 7 showed a fine diffuse infiltration affecting mainly the mid-zone and lower zone of the right lung. There was some very fine scattered infiltration in the left mid-zone. The apices were clear, the bases emphysematous, and the right costophrenic and cardiophrenic angles were obliterated. There was considerable increase in the size and density of both hilar shadows. Screen examination showed little interference with the move-

ments of the diaphragm, although there was some peaking of the right cupola on inspiration.

The case was referred to the Pneumoconiosis Medical Panel as a matter of interest, but as the man had not worked in the asbestos industry for 20 years he was adjudged ineligible to recover compensation under any existing scheme.

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REFERENCE

Beattie, J. M., and Dickson, W. E. C. (1943). *Textbook of Pathology*, 4th ed. Heinemann, London.

Effects of Nitrogen Mustard on the Intraocular Pressure

Nitrogen mustard is an unstable vesicant studied during the war as a possible successor to ordinary mustard gas. When dissolved in water it forms mainly a hydrochloride (S-hydrochloride), which is stable for long periods. It is this product that has been used in the present study. When applied to the eye of the rabbit (for example, two drops of a 1% solution) its most prominent effect is a breakdown of the barrier between the blood and aqueous humour, so that plasma proteins appear in the latter fluid (Davson and Quilliam, 1947); associated with this change there is a pronounced meiosis, a severe inflammatory conjunctivitis with chemosis, and corneal oedema. So far, no study of its effects on the intraocular pressure have been recorded. The results described below show that the effects may be very severe; they will be published in detail later, it being our purpose here mainly to emphasize the severe consequences that are likely to ensue should a little of this material accidentally reach the eye—a matter of some importance in view of the use of the substance in the treatment of Hodgkin's disease.

EXPERIMENT

A rabbit was anaesthetized with pentobarbitone sodium, and after cocainization of the right eye a needle was inserted into the anterior chamber, connected to a tambour-type manometer, and the intraocular pressure recorded. Slit-lamp observation of the aqueous humour was made at the same time. The changes in intraocular pressure with time are shown in the Graph. At A three drops of nitrogen mustard were placed in the conjunctival sac; it will be seen that the intraocular pressure rose from its original value of 16 mm. Hg. to one of 55 mm. Hg. within about 25 minutes—that is, the changes so far as intraocular pressure is concerned are akin to those occurring in an acute inflammatory attack of glaucoma. The constricted pupil, however, and the breakdown of the blood-aqueous barrier are reminiscent of an acute iridocyclitis.

In general, the constriction of the pupil runs parallel with changes in intraocular pressure, the maximum constrict-