

ASBESTOSIS, AN OCCUPATIONAL DISEASE NEW TO SWEDEN*

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Our report is occasioned by nine cases of asbestosis, including one death, to our knowledge the first in Sweden from this occupational pneumoconiosis. The first patient applied in 1953 and occasioned an examination of the 60-odd workers employed in asbestos work in the Göteborg industry; the other eight cases were found in that group examination.

— Asbestosis has been a subject of research abroad for a long time. For further study of this the reader is referred to Hunter, 1955, and his bibliography, and to Noro, 1946, and Wegelius, 1947, who described in detail the occurrence of the disease in Finland. Here we shall only relate our experience and give a brief survey of the conditions under which the disease occurs in Sweden and of its clinical symptoms and profylaxis.

Our own material, as said above, consists of 9 male workers aged 43 to 63, with times of exposure to asbestos dust of from 5 to 17 years. During this time, however, they have also been employed at other work or with different insulation material, so that the degree of exposure is very hard to judge. Seven of the cases got their asbestosis after insulation work and two after work at asbestos spraying. One of the latter, the one with lethal outcome, could be classed upon first examination as asbestosis Stage III (see below), the other as Stage II. Of the others, four may be regarded as being in Stage I-II and the rest in Stage I or incipient asbestosis.

Subjective complaints that can be attributed to the disease were reported only by the patient with Stage III. For that reason his case history will be recounted here in detail.

At the time of examination he was 63 years old and had been employed at asbestos work for 17 years, mostly at asbestos spraying, and used respiratory protection only sporadically. A year ago he was bothered by increasing shortness

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Asbestos

Asbestos is a mineral consisting chiefly of magnesium or iron silicate and varying somewhat in composition depending on the locality--Canada, South Africa, Finland, and several other countries. It consists of flexible fibers, so that it can be carded, spun, and woven into tapes or cloths. Considerable strength, high melting point, and poor heat-conducting capacity are the basis for its use as a friction material (brake bands) and insulation material.

In technology we are now dealing with extremes, in temperatures as elsewhere, and it is therefore not surprising that the use of asbestos increases every year. During a 5-year period before the war Sweden imported 17.6 tons; in a corresponding time after the war, since the shortage after the blockade was compensated for, the figure was 46.5 tons.

In Sweden asbestos is chiefly used as insulating material around pipes and the like on vessels, as fire-resistant material in ship's spaces or assembly rooms, in the manufacture of brake bands, and as a reinforcing material in cement and plastic.

In asbestos spraying for insulation against fire, the asbestos is first carded in a machine and then blown with air through a special spray pistol together with water and a binding medium against the surface of the wall, so that the mass forms a layer up to a decimeter in thickness. The dust formation is considerable.

The insulation work on ships is done with imported asbestos which as a rule has been carded. From this an insulating mass is made--often at the work site--consisting of asbestos, water, and some binding agent; this may either be allowed to dry into blocks that can be sawed or smeared directly on the part to be insulated, or else woven into a sort of ticking which is then filled with carded asbestos, sewn together, and installed on tanks or other large objects. The greatest dust production occurs in mixing the asbestos mass and in making the tickings, but considerable exposure to dust can also take place in wrapping pipes with asbestos cords and in tearing away old material.

In installations on land, safe insulating materials such as glasswool, rockwool, or cinder wool are often used, but shocks and vibrations on board, unfortunately, make their use impossible on most vessels.

Asbestosis results from inhalation of asbestos dust, which consists in large part of needles ca. 1 μ thick and from 1 to 70 μ in length. The exposure time for occurrence of asbestosis naturally varies with the degree of exposure, but is said to be about half of that for silicosis. Hunter, 1955, recounts a case in which the time between beginning of exposure and death from asbestosis was only 18 months. The irritation due to inhaled asbestos fibers gives rise to a progressive fibrosis of the lungs. In experiments with animals it has been found that short fibers give rise to an interstitial peribronchiolitic fibrosis of more diffuse type, while longer fibers produce nodules of approximately the same type as silicosis nodules (King, Klegg, and Rae, 1946). The progressing fibrosis is usually accompanied by atelectasis, emphysema, bronchiectases, and pulmonary heart.

In contrast to the conditions in silicosis, the modifications occur primarily in the lower $\frac{2}{3}$ of the lungs, with lowering of the hilus area and development of thickenings of the pleura.

From extensive experimentation with animals, King et al. in particular reached the conclusion that the fibrosis in asbestosis is due to purely mechanical irritation, in contrast to the fibrosis in silicosis, where there are strong reasons to suspect a purely chemical effect. For they could only induce asbestosis in the laboratory animals in mobile organs and by means of fibers that have retained their flexibility and sharpness and that have a certain length. Furthermore, they found no great signs of phagocytosis and no damage to the lymph glands. The particles thus are not removed from the lung to any great extent, and this contributes to the unfavorable prognosis in asbestosis, even if the patient promptly discontinues the work.

A characteristic feature is the presence in the lung and in sputum of what are called asbestosis bodies (Figure 1), which contain asbestos needles centrally and are covered at the pointed ends with a substance stainable with hematoxylin. The authors are inclined to interpret the formation of asbestos bodies as an attempt by the body to protect itself against the sharp fibers. It has also been possible to show in experiments with animals that inhalation of asbestosis bodies does not give rise to asbestosis. They are evidence not of asbestosis but of exposure to asbestos.

of breath under exertion, by a dry night cough, and by evening fatigue, and at the examination he displayed conversational dyspnea, cyanosis of the lips and prominent veins, watchglass nails, and drumstick fingers. The lung examination showed damping and râle increasing toward the bottom of the lungs. The X-ray picture of the lungs corresponded to what is found in asbestosis Stage III, and in the sputum there were abundant asbestos bodies (Figure 1, p 6). He had tachycardia but otherwise no sign of heart disease in the physical examination of the heart; roentgenologically the heart showed general enlargement, and the electrocardiogram at the first examination showed nothing remarkable. Later a branch block developed.

He was treated with antibiotics and theophyllamine, whereupon the subjective symptoms grew less and the vital capacity rose from 1,700 to 2,900 ml. He could be discharged and cared for at home, but he returned after a few months with a pneumonia that resisted all treatment.

The autopsy (performed by Sourander) showed among other things a diffusely thickened, leathery visceral pleura on both sides. The upper parts of the lungs showed edema and signs of pneumonia. In the lower $\frac{2}{3}$ of the lungs the consistency was uneven, granular, and noticeably firm, in section markedly tough. The fibrous transformation of the parenchyma of the lungs increased in the caudal direction and exhibited a coarsely porous, trabeculated appearance with rounded cavities up to the size of hazel nuts between the reticular, slate-gray, fibrous stretches. Under the microscope the parenchyma of the lung was found to be strewn with broad strands of connective tissue and foci of a locally nodular appearance. The alveoli were found to be atelectatic. In the connective tissue there was an abundance of asbestos bodies and around them a rather heavy cell infiltration, consisting of alveolar cells, macrophages, and other multinuclear giant cells. In the bronchial glands, on the other hand, no asbestos bodies could be found.

PAD: Pneumoconiosis + pneumoniae lobaris et oedema lobi sup. pulm. amb. + bronchitis mucopurulenta + dilatatio sin. et hypertrophia dextr. cordis + sclerosis a. coron. cordis + stasis chron. hepatis.

The case that we classified as Stage II was discovered in 1953 and the patient desisted then from work with asbestos. In spite of that he has shown progression radiologically, but still without subjective complaints. For the other seven we still have too short a period of observation.

Clinical Picture

Clinically, asbestosis differs little from other lung fibroses. The subjective complaints usually do not appear until Stage III, in the form of shortness of breath, dry coughing, especially at night, and fatigue. Watch-glass nails and drumstick fingers may be found. The physical pulmonary findings are most often limited to rale; after the onset of pleura thickening and lung fibrosis, also increasing damping basally. Complications are commonly in the form of bronchitis, pneumonia, and pulmonary heart with their specific symptomatic flora, and are often the immediate cause of death. Tuberculosis as a complication is by no means as common in asbestosis as in silicosis.

Besides these complications, in very recent times a high frequency of lung cancer has been observed among patients with asbestosis. In a study of the causes of death in 130 asbestos workers, Doll, 1955, found that the supermortality was substantial, or 39 cases against 15 expected. The excess was due partly to vascular diseases, 22 cases against 7 expected, and partly to lung cancer, with no fewer than 11 cases against 0.8 expected. This relationship, too, makes the prognosis for asbestosis more serious than for silicosis.

Diagnosis is based on the work anamnesis and X-ray findings and can be completed by the finding of asbestosis bodies in the sputum.

The X-ray picture in asbestosis has been described in detail, among others by Wegelius, 1947, on the basis of 126 cases. The roentgenological lung modifications in our patients correspond to those described by Wegelius, with the exception that in one of our cases we found suspected calcifications of the pleura.

Like other pneumoconioses, asbestosis has been subdivided into three stages. In the literature two such stage classifications are defined, one from Germany by Saupe, 1938, and the other from America by Shull, 1936.

Shull in his stage classification puts greater weight on the pleura thickenings than Saupe does. Like the former, we observed such thickenings in several of our cases and for that reason considered that we got the most use out of his stage divisions in classifying our material. According to his classification the stages are characterized as follows:

Stage I. Incipient asbestosis. Slight interstitial fibrosis in both lungs down toward the bases.

Stage II. Moderately advanced asbestosis. More pronounced interstitial fibrosis, which extends to the surface of the lung in the basal $\frac{2}{3}$ of the lungs.

Slight thickening of the pleura and pericardium to the same height. Slight damage to the heart on the right side and moderate apical emphysema.

Stage III. Advanced asbestosis. Very pronounced interstitial fibrosis in the basal $\frac{2}{3}$ of the lungs, very marked apical emphysema, considerable dextral heart hypertrophy, but the contours of the heart do not appear clearly because of condensations in the lungs. Almost always a marked thickening of the pleurae and pericardium.

In accord with the animal experiments referred to earlier showing that asbestosis occurs only in mobile organs, X-ray modifications are found primarily in the basal, most mobile part of the lungs. The pleura modifications are also found there, parietally and in the pleura diaphragmatica. In one of our cases, classified as Stage II, these pleura thickenings were so impervious to X-rays that it must be assumed that calcifications had occurred in them. Figure 2 shows a schematic view of their spread in a patient who had worked with asbestos for 10 years and was subjectively free of complaints.

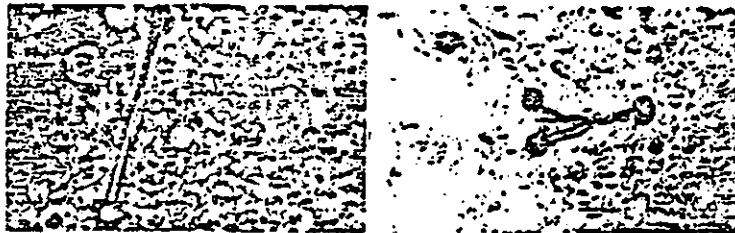


Fig. 1.

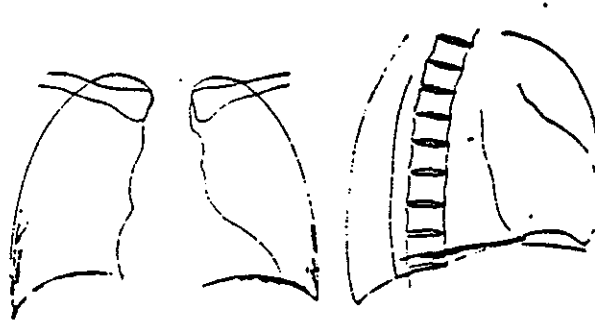


Fig. 2.

Because of the fibrous destruction of the basal parts of the lung, emphysema develops in the apices. In more advanced cases the condensations are so pronounced that normal lung tissue cannot be observed at all. The heart-lung boundaries are then largely blotted out.

As usual in pneumoconioses, the roentgenological differential diagnosis must be supported by the work anamnesis, and just as in the case of silicosis,

repeated examinations are needed before it can be said with certainty whether there is an incipient asbestosis or not. In asbestosis the conditions of exposure are somewhat easier to learn in anamnesis than is the case with silicosis, inasmuch as a workman is aware of whether he has worked with asbestos, but he has a hard time saying whether the mineral he is working on contains substances that cause silicosis or not.

The treatment, of course, is only symptomatic. In the progressing case, cortisone treatment may perhaps be considered, which might possibly arrest the advancing fibroses. Thus far, however, the risk of spread of latent infections in the already greatly reduced respiratory tissues has restrained us from such therapeutic experiments.

Prophylaxis

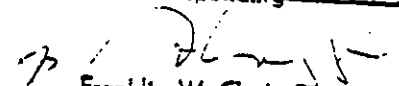
Obviously, an effective prophylaxis against this disease is needed. In England, where the processing and use of asbestos was begun very early, it has been possible through prophylactic occupational hygiene measures to reduce the morbidity from around 3 percent of asbestos workers to practically zero for the time being. In Sweden, too, similar measures have been taken since attention was called to the danger. Thus the first of our cases was reported in the fall of 1953 to the Royal Medical Board with a request for regulations concerning preventive measures similar to those in effect for silicosis. Proposals are now being worked out concerning periodic X-ray examinations of all who are employed in asbestos work.

Extensive dust surveys have been carried out by the State Institute of Public Health and are now being evaluated.

As soon as the first cases became known, the factory inspector in Göteborg served notice that protective masks were to be worn by all workers at places where the slightest risk of inhaling asbestos dust could be imagined. But that safety measure must be regarded more or less as provisional. The factory inspection service is now engaged in working out, wherever possible, other technical protection measures, which will come down in the main to ventilation and local removal of dust by suction at certain moments in the work. More detailed studies in countries where the asbestos risk was observed earlier would be necessary, however, before it would be possible to arrive at specific regulations for the asbestos industry in Sweden.

Through such occupational hygiene measures it can be hoped that this first asbestosis case in Sweden will also be the last. Asbestosis is too serious a disease for anything other than complete protection for workers employed in asbestos work to be acceptable.

I certify that the staff member who translated the foregoing is thoroughly familiar with the Swedish and English languages and that it is a true and complete translation of the corresponding Swedish document.


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