PATHOLOGICAL STUDIES OF ASBESTOTIC PLEURAL PLAQUES —PRELIMINARY EXPLORATIONS OF HISTOGENESIS

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Pleural plaques were present in 33 of 55 autopsy cases of asbestos workers. Their exposure periods to asbestos were 5–23 years (mean 18.1 years). Twelve of them employed as miners, 21 millers. They were mainly exposed to chrysotile.

Pleural plaque is local patchy thickening with sharp borders from the surrounding normal pleura, yellow-white, harder texture. The surface may be smooth, nodular or navel. No adhesion between visceral and parietal pleura is a conspicuous feature. Pleural plaques are often found on the parietal pleura, particularly on bilateral, posterior and diaphragm pleura. In a few cases they can be seen on visceral pleura or parietal pericardium. Pleural plaques are not encountered at the apex or the costophrenic angles. Plaques are wide variety of shapes and sizes. In order to grade severity of plaques, the total area of pleural plaques is surveyed and expressed as cm^2. The total area has been graded into 3 degrees according to less than 100 cm^2; and 100–300 cm^2; and more than 300 cm^2. The degree 1, 2, and 3 were recognized in 11, 10, and 12 cases, respectively. To determine whether degrees of plaques related to exposure periods to asbestos we divided 33 cases into groups according to the interval of 10 years. There appears to be no significance to correlation between the degrees of plaques and exposure periods to asbestos. A man with degree 1 or less had been exposed to asbestos for more than 20 years. Conversely, degree 3 plaques can be seen in a case of less than 10 year standing. The maximum area of plaques was 916 cm^2 in the present reported cases. Degrees of pleural plaques were not related to standings; it could be conceivable that individual differences, especially, the differences in the sensitivity to asbestos stimulation on pleura play a role in the occurrence of the plaque.

We analyzed previously 15 lung tissues with plaques by the bleach digestion technique and carried out asbestos body counts, SEM-EDXA for core fibre elemental component of the fibre. The results of asbestos body counts are given in Table I. These results showed that the extent of the degree of plaque was not also related to asbestos body counts in the lung tissues.

Typical pleural plaques are made up of bundles of collagen fibres. They are arranged in basket-weave, or concentric circle, avascular and having few cellular elements. Sometimes, a mesothelial cell lining can be seen on the plaque surface (Figure 1). Fibrocytic nuclei were found in collagenous fibre bundle. The structure of thinner plaques was different from this. They consist of the mesothelial lining on surface and beneath loose connective tissue, fibroblasts and monocytes; these changes can be also found on some portions of typical plaques. We refer to the changes as an earlier stage of plaques. On the other hand, a massive chronic inflammation cellular infiltration of lymphocytes and plasma cells and vascularity were often found in deeper portions or periphery of plaque (Figures 2,3). There are many polarizing particles

Table I

Results of Asbestos Body Counts in 15 Cases with Various Degrees of Pleural Plaques

Grades of plaque	Case number	No. of Bodies per gram of dried tissie	G	SD_{Lg}	SE_{Lg}	T test Compare with grade0–I	T test Compare with grade I
I	5	3.3×10^3–95×10^3	21.253	0.568	0.254		
II	7	0–218×10^3	1.626	2.270	0.858	T=1.602 P>0.05	
III	3	6.7×10^3–114×10^3	3.228	0.674	0.389	T=0.411 P>0.05	T=0.961 P>0.05

in intercollagenous fibres, the deepest and beneath mesothelial lining in polarized light microscopy. Most of them are needle-like, free, a few in dust-cells. A few fibres can be found in deposits digested plaques and on sections of plaques *in situ* in SEM. Their elemental compositions are mainly Si, Mg and a few Fe, similar to that of chrysotile, while other fibre compositions are mainly Si and Ca. To classify these fibres is difficult only according to EDXA. On the basis of above-mentioned results, we regard initial formation portion of plaque was beneath the mesothelial lin-

Figure 1. Mesothelial lining on the surface, lower earlier plaque changes. H.E. x 200.

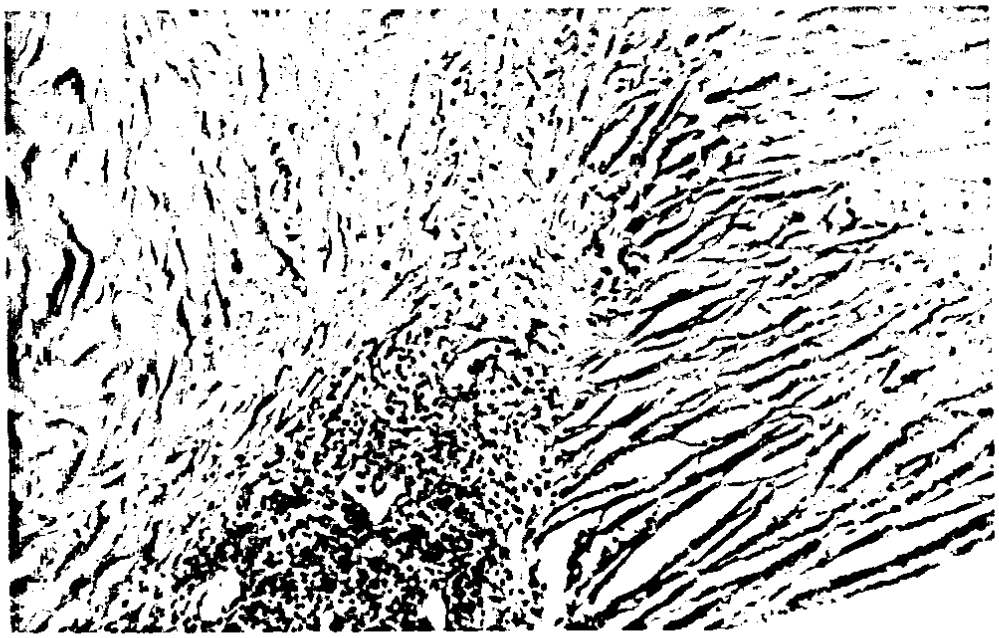

Figure 2. A massive infiltration of chronic inflammation cells on periphery of the plaque. H.E. x 100.

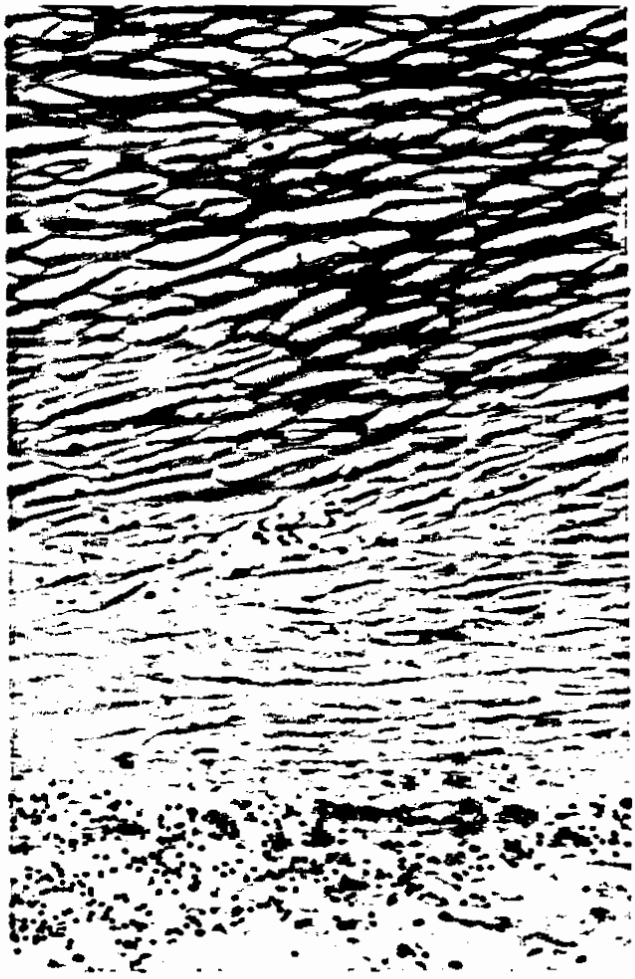

Figure 3. The plaque was clearly divided into 3 zones from base to top: lymph-like cells and vascularity, fresher connective tissue, and hyalinized collagen fibres. The picture showed that development of the plaque was from base of it. H.E. x 100.

ing. Earlier stage changes were dust-fibrous reaction then typical plaque pictures occurred owing to increasing and hyalinization of collagenous fibres. An important point to note is similarity in histopathological pictures of pleural plaques and chronic pleurisy (suppulative or tuberculous). It is not easy to distinguish among them even in light microscopy. But, there is a mesothelial cell lining on surface of plaque; only for this reason, pleural plaques surface was smooth. On the contrary, initial changes of chronic pleurisy occurred in pleural cavity, mesothelial cells desqamate firstly, then adhesion of parietal and visceral pleura

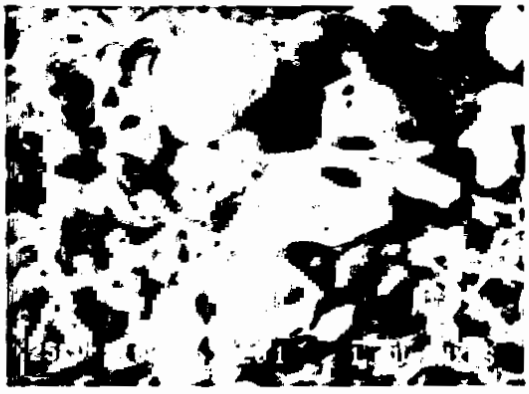

Figure 4. Asbestos fibre and its energy dispersive X-ray spectra, elemental composition. The fibre seems to be chrysotile.

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 ELEM    K       Z       A       F

 MG K  0.189   1.022   0.406   1.008
 SI K  0.440   1.022   0.460   1.002
 P  K  0.014   0.988   0.304   1.002
 S  K  0.014   1.014   0.405   1.002
 K  K  0.021   0.974   0.704   1.009
 CA K  0.037   0.997   0.767   1.009
 TI K  0.020   0.913   0.861   1.019
 FE K  0.264   0.914   0.966   1.000
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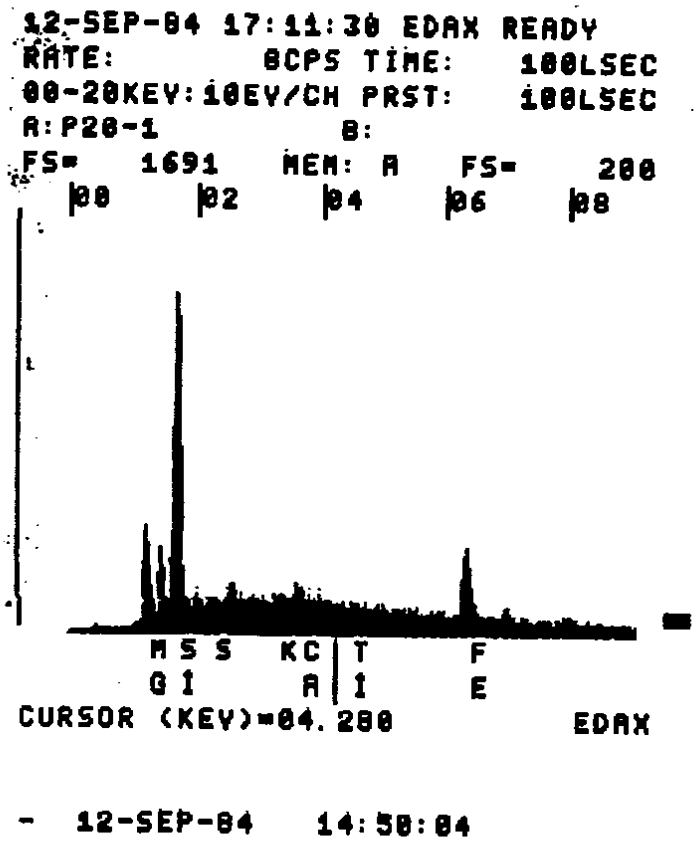
ELEM	CPS	WT % ELEM
MG K	39.790	25.315
SI K	147.999	44.455
P K	4.800	2.964
S K	4.730	2.187
K K	6.470	1.953
CA K	10.420	2.989
TI K	4.680	1.572
FE K	43.020	18.565



developed, and resulted in the cavity disappearing. It is true, for Thomson noted the mesothelial cells play no part in plaque formation,[1] but, because the mesothelial cell lining had still remained it is possible to explain the peculiarity of plaques; i.e., no adhesion, smooth. In addition, infiltration of cbronic inflammation cells in basic or peripheral portions of plaques might play a role in development of plaques, because there are general changes seen in chronic inflammation fibrosis. Yet fibrocytic nuclei were most in base of the plaque, and the fewer, the more surface. Therefore, portion of plaque formation is beneath mesothelial cell lining, origin of growth is the base of plaques. Of course, reformation of plaque can also occur beneath mesothelial lining of typical plaque. We have found that infiltration of chronic inflammation cell was sandwiched between collagenous fibres, and earlier changes were beneath mesothelial lining of typical plaque.

The exact route by which the inhaled fibres reach the parietal pleura is yet unknown. Three possible routes have been drawn by Bignon.[2] 1) Asbestos fibres penetrated directly into pleural cavity, 2) by lymph vessels, 3) by blood system. Our cases have mainly been exposed to chrysotile, but the fibres extracted from lung tissues belong to amphibole according to EDXA results. These fibres were long and straight. The penetration of amphibole is stronger than chrysotile because the latter is curved. It is suggested the first hypothesis seems impossible. It seems possible that inhaled chrysotile fibres broke into thinner and shorter fibres in lung tissue, then they were transported to pleura by lymph vessels, and induced formation of pleural plaque. In view of these reasons, we may understand the presence of chrysotile fibres in pleural plaques (le Bouffan).[3] That is the reason, why chrysotile was hardly found, and amphibole easier seen in our deposits of digested lung tissues.

SUMMARY

In order to assess the severity of pleural plaques the degree of the plaques has been reported. It is adaptable for asbestos workers that pleural plaques were graded into 3 degrees by the area interval of 100 cm^2 and 300 cm^2. Because the degree of plaques have no relation with asbestos standing, individual differences might play a role in occurrence of plaque. It has been observed that initial portion was beneath mesothelial cell, and origin of growth was the base of plaques. Some aspects, such as absence adhesion, smooth surface, asbestos body counts in lung tissues were not concerned with degrees of plaques and can be explained by the findings. Studies seem to suggest that inhaled asbestos fibres can be transferred from lung tissue to parietal pleura, but exact routes have yet to be demonstrated.

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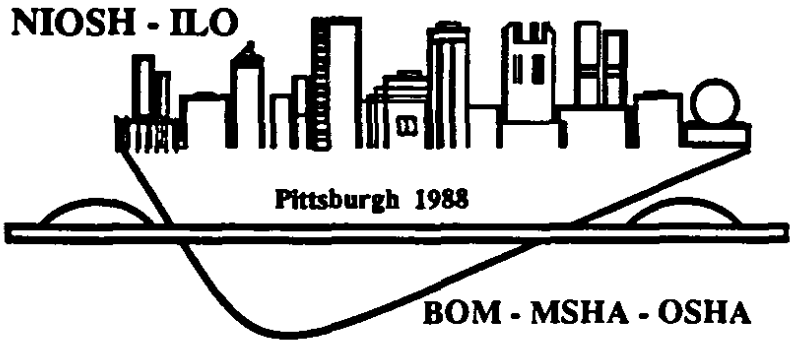

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