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ml of the buffer described in Appendix 1, and continue as in Appendix 1. Normally for blood specimens the final washing must be performed four times, instead of thrice as for urine specimens.

APPENDIX 3—COPROPORPHYRIN ESTIMATION

To 5 ml freshly voided urine in a stoppered test tube add 0.2 ml 3 per cent hydrogen peroxide solution, 1 ml glacial acetic acid and 3 ml ether. Shake gently, release pressure, and place in a lightproof box overnight. Observe the red fluorescence under ultraviolet light, and grade the specimen 0-++++ by degree of fluorescence.

APPENDIX 4—HEMOGLOBIN DETERMINATION

Take 0.05 ml blood, and make up to 9 mls volume with 0.4 per cent ammonium hydroxide. Measure absorption on a colorimeter, using a standard filter (EEL 404), and convert the reading to hemoglobin percentage to the nearest even number from a prepared calibration graph.

APPENDIX 5—PUNCTATE BASOPHIL COUNTS

Make a thin blood smear on a slide of 0.8-1.0 mm thickness, fix for about one minute in methyl alcohol, allow to dry and stain for 30 seconds in Sellars stain (1 g methylene blue, 6 g sodium bicarbonate in 100 ml deionized water). Wash in tap water until the slide is a sea green color.

Using a dark ground condenser and an Ehrlich eyepiece set to a 6-mm square, count the punctate cells in ten consecutive fields. (Although in counting, the punctate cells are classified as large, medium, small, very fine, or polychrome, according to the size of the intracellular structure, this differentiation is for further reference on an individual case, and normally only the total count is given.) Close the eyepiece to a 2-mm square, and count the R.B.C. in this square. Repeat both counts five times on different parts of the slide, to give a punctate count of 50 large fields and an R.B.C. count of five small ones. Calculate punctate per 1000 R.B.C.

NONOCCUPATIONAL ASBESTOSIS

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Introduction

Pleural reaction is very often observed in asbestosis. In the advanced cases of this pneumoconiosis pleural fibrosis has been almost always found, and a clear correlation between pleural changes and the severity of the pulmonary fibrosis has been observed by several investigators. Observations on the occurrence of pleural calcification in asbestosis have also been made. Reports of pleural calcification as a roentgenologic sign or complication of asbestosis have especially been made by the German authors (1). In addition to advanced cases of asbestosis, special cases have been presented in which pleural calcification has been the chief finding (2). Except in asbestosis, pleural calcification has also been observed among workers exposed to talc and mica (3, 4) or to various dusts including a short exposure to asbestos (5).

The diagnosis of pneumoconiosis is primarily based on occupational exposure to dust. Only very few reports of pneumoconioses without occupational exposure may be found in the literature (6, 7, 8). In Northern Karelia, Finland, during few past years interest has been attracted by the high incidence of cases of pleural calcification showing a rather curious and characteristic roentgenologic picture, very similar to that observed in pneumoconioses. The cases have been mainly found among the residents of a certain area, and in these cases no previous history of adequate pulmonary or pleural disease could be elicited. The occupations of the patients showed no uniformity, and the main part of the patients had no clinical symptoms of pneumoconiosis. A study of the areal distribution of cases revealed that they were grouped round two anthophyllite-asbestos mines. Asbestos constituted the common external factor sought. Even from a solely roentgenologic point of view, asbestos dust inhalation had been considered the most probable etiology.

Pleural calcification resulting from hemothorax, tuberculosis, or empyema usually occurs unilaterally and is situated on the visceral pleura. In most cases marked pleural thickening and displacement of the intrathoracic organs is observed. The calcification resulting from dust inhalation usually occurs bilaterally and is most often situated on the parietal pleura. In advanced cases calcium deposits are found in the lateral parts of the chest, basally and mediastinally. The calcium deposits take various forms—large confluent plaques, bizarre figures, or roundish spots.

The Roentgenologic and Clinical Picture of Cases Observed in Northern Karelia

In the advanced cases calcium deposits are found bilaterally in the central parts of the diaphragm; on chest walls, especially anterolaterally but also dorsally; and on the mediastinal surfaces. In the incipient cases the calcium deposit takes place either on the anterolateral chest wall, more often on the left, or in the central parts of the diaphragm, apparently more often on the right. Pericardial calcification is sometimes present.

The calcium deposits are located on the parietal pleura. In a series of 150 cases no interlobar calcium deposits were observed. The parietal location has been demonstrated by diagnostic pneumothorax examinations, and it has also been noted in some autopsied and surgical cases. Using a tangential roentgen beam, the parietal location of solitary plaques may be observed, these plaques having their bases toward the inner chest wall or toward the diaphragm. In advanced cases the lack of soft tissue mass between the calcium deposit

and inner chest wall due to the parietal location is apparent, and diagnostical pneumothorax examination is seldom needed.

The majority of cases of pleural calcification described above have been clinically symptomless. In a series of 150 cases the ordinary chest roentgen examination by P-A and lateral projections revealed pulmonary fibrosis, corresponding to the anteprimary or primary stage of pulmonary asbestosis in only about 14 per cent. Even the main part of these cases had no subjective respiratory symptoms.

The series mentioned above has been selected solely according to the roentgen diagnosis without reference to other factors. The concentration of the cases around two asbestos mines in Kuusjärvi and its neighboring communes has as yet been the only way to confirm the roentgen diagnosis. The majority of 25 cases living outside this area had previously lived there. According to these facts, any other etiology would be very difficult to credit. In very few cases a previous occupational exposure to asbestos dust has been observed, which lends slight support to the roentgen diagnosis. The diagnosis has as yet not been confirmed by other methods. In the sputum of 20 cases no asbestos bodies were found, and asbestos fiber in one case. Small amount of anthophyllite asbestos was found in the lung of an autopsied case and in the lung of a cow from the vicinity of the asbestos mine.

Discussion

The total number of cases of pleural calcification around the two asbestos mines amounts to over 800, as previously reported by the author. This number of cases was found in a mass survey of about 10,000 adult inhabitants and may include cases with previous occupational exposure to asbestos dust. In the history of the main part of cases examined at the Central Hospital of Northern Karelia the lack of occupational exposure has, however, been apparent. Some of the patients have not had any idea how the dust inhalation could have happened; the same cases have presumed that the dust rising on the roads might be responsible. In Northern Karelia the roads (highways) do not have pavement. Without mineralogic examinations it is, of course, impossible to decide whether these calcifications are resulting from inhalation of anthophyllite-asbestos dust originating from the asbestos mines or from surface deposits of other fibrous dusts, such as tremolite-asbestos, which is a very common mineral in Northern Karelia. The first alternative seems to be more probable.

In occupational pneumoconiosis some authors have called attention to the absence of pulmonary fibrosis besides the prominent appearance of the pleural calcification (2, 5); on the other hand, a clear correlation has been observed between the severity of pulmonary fibrosis and pleural calcification (9). In noncoccupational asbestosis it is apparent that extensive calcification may occur without any roentgenologically detectable pulmonary fibrosis. It seems possible that even very small amounts of asbestos fibers may cause pleural calcifications.

Anthophyllite is more stiff than serpentine asbestos. In animal tests brittle mineral fibers have not caused pulmonary fibrosis (10), but fibrosis has been observed in workers exposed to anthophyllite-asbestos dust (11, 12). Further studies are needed to reveal the part possibly played by asbestos as an etiologic factor in pulmonary fibrosis among residents around the two asbestos mines.

Some investigators have pointed out the difficulty of attributing the calcification to inhalation damage in view of its parietal location, few, if any, changes being observed in the lung or the visceral pleura (2, 5). It seems possible that the asbestos fibers move with the lung and visceral pleura in relation to the parietal pleura, thus causing capillary hemorrhages on this only, as previously presented by the author. The hemorrhage coagulates and organizes, forming a collagenous plaque which slowly calcifies. This theory could explain the course of the process resulting from inhalation of small quantities of fibrous mineral dust.

The clinical significance of the pleural calcification seems to be slight, the majority of cases showing no clinical respiratory symptoms. Further investigations on this point are needed.

From roentgenologic point of view, this endemic pleural calcification is of some significance. The roentgen diagnosis is in typical cases easy but in some cases difficult. Early pleural changes on the anterolateral chest wall may simulate infiltrations, and large calcified plaques may cause difficulty in interpretation of pulmonary changes by hiding infiltrations. The high kilovoltage technique can hereby be especially recommended. In chest roentgen examinations of workers of asbestos mines in Northern Karelia and of Outokumpu Copper mines located in the same area attention must be paid to the fact that even extensive bilateral pleural calcification does not have to be a sign of severe pulmonary changes resulting from occupational dust exposure.

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