Asbestos Pleurisy

H. B. EISENSTADT, M.D., F.C.C.P.*
Port Arthur, Texas

ASBESTOSIS IS A DISEASE OF THE RESpiratory system caused by inhalation of asbestos material. The latter name is applied to a group of minerals occurring naturally in the form of flexible fibers. Of the more than 30 known substances with such properties, only six have a commercial value.[1] Each asbestos mineral has a different composition determining its physical and chemical properties. However, all of them are characterized by incombustibility, flexibility, tensile strength and resistance to heat and chemicals.[1] Due to these peculiar properties, asbestos has more than 3,000 different industrial applications.[1] This widespread use accounts for the great number of workmen coming in contact with the material.

Recently it has been stated that "silicosis is not just one disease but many, all of which we may call with this name because of lack of a better term."[2] A similar statement seems to be applicable to asbestosis. Differences of mining and milling procedures, manufacturing and usage of the end product may easily explain the varieties of the clinical appearances of this pneumoconiosis. The classic form of pulmonary asbestosis is described as a bilateral fibrosis which progresses over a number of years and leads to a shrinkage of lung and pleural spaces. From the clinical point of view there is an initial asymptomatic stage lasting a long time; this is followed by the gradual appearance of shortness of breath, anorexia, weight loss, weakness, dry or productive cough, rhonchi, decreased breath sounds, cyanosis and clubbing of fingers. Finally, respiratory failure appears, frequently associated with cor pulmonale and right-sided heart failure. Corresponding to this clinical appearance, the x-ray films of the chest are initially negative. However, gradually fine bilateral reticulation and stippling may be noted, particularly in the middle and the lower lung fields. This picture is followed by a diffuse ground-glass haziness and by coarse streaking of practically all lung portions except the apices. In the final stage, pulmonary contraction may be seen with fibrothorax. Diaphragmatic and pleuropericardial adhesions, as well as parenchymal densities, are responsible for blurring and irregularity of heart margins and diaphragm characterizing the terminal state. These various x-ray changes so well described by Pendergrass[3] have been correlated by Lynch[4] and others with the pathologic findings. During the first stage, only asbestosis bodies are observed in the otherwise normal appearing tissue section. These club- or spear-shaped bodies representing asbestos fibers covered with proteinaceous material must be present to make the pathologic diagnosis of the disease. Later, various degrees of pulmonary inflammation follow, terminating in complete disappearance of lung structures with formation of hyaline and fibrous masses. Sometimes, however, asbestosis does not show these typical features. For instance, an interesting case study has been presented by Castleman,[5] where asbestosis involved mainly the upper lobe of the left lung. This author assumed that an associated burned-out pneumonia was responsible for such an unusual localization. However, he was unable to establish the exact mechanism producing the lesion even after necropsy. A completely different form of asbestosis presenting itself mainly as pleural disease has rarely been mentioned in the literature. To be sure, involvement of the pleura has been described in a number of cases. However, this has been considered to be a late and insignificant complication associated with extensive pulmonary disease.

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Figure 1: Benign

The following case illustrates a form of asbestosis appearing primarily as idiopathic pleurisy which differs greatly from the classic picture.

In September, 1961, a 54-year-old white man, an asbestos insulator, was seen suffering from an acute pain in the left hemithorax. The pain was aggravated by coughing and deep breathing. Apart from simple colds, he had never suffered from any respiratory disorder before this time. The patient did not look seriously ill. He had a temperature elevation of 99.6°F. No abnormality could be detected in his nose and throat. There was a dullness to percussion and a suppression of breath sounds in the left lower chest. An x-ray picture demonstrated a density over the left base compatible with a diagnosis of pleural effusion. Lungs and heart appeared otherwise perfectly normal on the chest plate. Laboratory examinations included a slightly accelerated sedimentation rate and a normal blood count and urinalysis. Skin tests for tuberculosis and fungus infections were negative. Studies for L.E. cells and electrophoresis were non-revealing. Only a small amount of mucoid material was expectorated; it did not contain pus cells, eosinophiles, tubercle bacilli or other pathogenic micro-organisms. No paracentesis was performed because of the excellent general condition of the patient. After two weeks of bedrest, he recovered completely and his chest x-ray film looked normal. However, six months later, he suffered from a similar episode in his right side (Fig. 1). At this time, the onset of the disease was more insidious with soreness, nausea, anorexia, weakness and weight loss. There was a splinting of the right hemithorax; a loud friction sound could be heard on respiration. Thoracentesis was performed and 550 ml. of brown-colored fluid were aspirated. The fluid contained red cells, lymphocytes and reticulocytes in the sediment; no tumor cells and no pathogenic organisms could be detected. A culture of the fluid showed no growth. The mucoid sputum contained staphylococci and streptococci, but no tubercle bacilli. The sedimentation rate was 83 mm. per hour (Westergreen). A latex agglutination test was positive in a 1:80 dilution. The blood count revealed a moderate eosinophilia, but was otherwise normal. The patient was placed on a medical treatment with antituberculosis drugs and bed rest. However, he showed little progress after a period of one month when the pleural tap had to be repeated. It yielded 450 ml. of fluid similar to that previously encountered. Finally, after an additional month of waiting, thoracotomy was carried out. Extensive pleural adhesions were found obliterating part of the pleural cavity. There was considerable thickening of the visceral pleura (Fig. 2). Decortication was performed removing all the abnormal tissue. In addition, a specimen of the adjacent lung was obtained for microscopic examination. The latter revealed thickening of the alveolar walls with macrophages and asbestosis bodies (Fig. 3). The patient recovered completely after the operation; he has remained in good health ever since this time.

FIGURE 1: Benign pleural asbestosis (right).

FIGURE 2: Dense connective tissue with infiltrates of lymphocytes and eosinophiles.

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Comment

Asbestosis presenting itself clinically and roentgenologically primarily as a pleural disease has apparently not been described in the literature. Such a form of asbestos disease has not been mentioned in the recent textbooks on this subject.[6,7] However, it is not difficult to understand why the condition could have been overlooked. Before the advent of modern thoracic surgery, any idiopathic pleurisy was considered to be caused by tuberculosis until proved otherwise and treated routinely with an antituberculosis regimen. Benign asbestos pleurisy resembles tuberculosis closely in its appearance; it is apparently a self-limited disease of several weeks' or months' duration and therefore, may give the erroneous impression of being benefited by antituberculosis therapy. Apart from the history of exposure, nothing distinguishes this disease from pleurisy of any other etiology. Surgical exploration with pleural and pulmonary biopsy is needed to verify the diagnosis. Marked pleural thickening, hyaline and fibrous plaques, nonspecific granulomas and extensive adhesions may alert the experienced investigator to consider the disease. However, the final diagnosis rests in the demonstration of asbestosis bodies in the lung parenchyma. Unfortunately, these needle- or club-like formations are hardly ever found in the pleural specimen.

Our own patient would probably have completely recovered with prolongation of this conservative management. However, the exact etiology of his pleurisy would never have come to light without thoracotomy. Our suspicion of primary pleural asbestosis in this case was partly aroused by the history of prolonged exposure to this material. In addition, we were alerted by our previous experiences with two cases of malignant pleural asbestosis, i.e., with malignant pleural mesothelioma associated with and probably caused by asbestos disease. One of these patients suffered from unilateral pleuritic pains for a period of 12 years prior to the development of the malignant growth. These pains must have been caused by a benign process preceding the malignancy. Another case of asbestosis and malignant mesothelioma[8,9] previously described by the author had benign bilateral pleural effusions ten years before his terminal illness. His chest roentgenogram revealed bilateral streak-like basal calcifications of the pleura considered characteristic of asbestos disease. At necropsy, both patients had shown pulmonary and pleural changes typical of benign pleural asbestosis (Figs. 2 and 3) in those portions of the

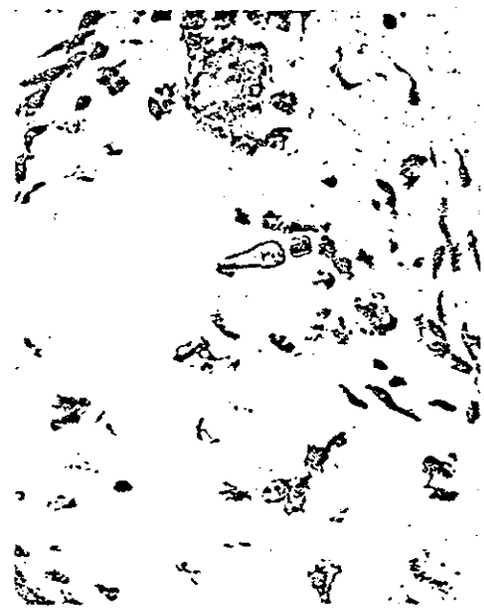

Figure 3: Alveolar walls thickened and infiltrated with inflammatory cells. Asbestosis body in center.

lungs not involved by the malignancy. Similar patients suffering from self-limited pleural effusions many years prior to the development of a pleural mesothelioma have been observed among the asbestos miners of South Africa.[10] However, the proof of benign asbestos pleurisy has been lacking in these cases.

It stands to reason that only a minority of the persons exposed to asbestos develop mesothelioma as a terminal event. However, there seems to be a large group of persons exposed to the mineral suffering from benign pleural involvement in the

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absence of any significant pulmonary abnormality. A great number of such cases are clinically entirely asymptomatic. Their chest x-ray films appear negative: their pulmonary function tests are normal. Again, another group should be expected to present themselves with signs and symptoms of acute, subacute, recurrent or chronic, unilateral or bilateral pleurisy.

Summary

A case of benign asbestos pleurisy has been described. This disease differs greatly from the classic picture of pulmonary asbestosis. It is probably not as uncommon as it would appear from the sparsity of reports in the literature. Asbestos pleurisy is usually not recognized because it has a benign, self-limited course requiring no specific therapy. It can be suspected in a person having been exposed to this substance for a prolonged period of time. Such contact may have taken place recently or a number of years ago. The correct diagnosis requires not only pleural, but also lung biopsy for the demonstration of asbestos bodies. Asbestosis must be considered in the differential diagnosis of any form of idiopathic pleurisy.

Acknowledgment: The author expresses his appreciation to Dr. E. C. McRee for his permission to report his patient.

Resumen

Se describe un caso de pleuresía por asbestosis. Esta enfermedad difiere grandemente del cuadro clásico de la asbestosis. No es tan poco común como parecía según la escasez de informaciones de casos en la literatura. La pleuresía asbestósica no es habitualmente descubierta porque es benigna, y su curso, que se limita por sí, no requiere tratamiento específico. Puede sospecharse en una persona que ha estado expuesta a asbestos por largo tiempo. Tal contacto puede ser reciente o haber tenido lugar cierto número de años antes. El diagnóstico correcto requiere no sólo biopsia pleural sino pulmonar para [illegible] de asbestos. La asbest[illegible] el diagnóstico diferen[illegible] pleuresía idiopática.

Resume

L'auteur décrit un cas de pleurésie asbestosique bénigne. Cette affection diffère grandement du tableau classique de l'asbestose pulmonaire. Elle n'est probablement pas aussi exceptionnelle qu'il le semblerait d'après la rareté des rapports dans la littérature. La pleurésie asbestosique n'est habituellement pas reconnue parce qu'elle a une évolution bénigne, en elle-même limitée, qui ne nécessite pas de traitement spécifique. Elle peut être suspectée chez une personne ayant été exposée à cette substance pendant une période de temps prolongée. Un tel contact peut avoir pris place récemment ou un certain nombre d'années auparavant. Le diagnostic correct nécessite une biopsie non seulement pleurale, mais également pulmonaire, pour la mise en évidence de corps asbestosiques. L'asbestose doit être considérée dans le diagnostic différentiel pour toute forme de pleurésie idiopathique.

Zusammenfassung

Beschreibung eines Falles von benigner Asbestose-Pleuritis. Diese Erkrankung weicht erheblich von dem plastischen Bild der pulmonalen Asbestosis ab. Sie ist wahrscheinlich nicht so ungewöhnlich, wie es nach der geringen Zahl von Literaturberichten den Anschein haben könnte. Die Asbestose-Pleuritis wird gewöhnlich nicht erkannt, weil sie gutartig ist und Dank ihres umschriebenen Ablaufes keine spezifische Therapie erforderlich macht. Man kann daran denken, bei Personen, die gegenüber dieser Substanz während einer längeren Zeitspanne exponiert waren. Solche kontakte können kürzlich oder auch eine Anzahl von Jahren zuvor erfolgt sein. Die korrekte Diagnose erfordert nicht nur eine pleurale, sondern auch eine Lungen-Biopsie zum Nachweis der Asbestose-Körper. Die Asbestose muß bei der Diffenrential-Diagnose in jeder Form der idiopathischen Pleuritis in Erwägung gezogen werden.

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For reprints, please write Dr. Eisenstadt, 2301 Procter Street, Port Arthur, Texas.