Pleural Asbestosis

H. B. EISENSTADT, M.D.
Medical Clinic, Port Arthur, Texas

Asbestosis is a form of pneumoconiosis occasionally encountered. The author points to the pleural reaction which may result with no gross or x-ray evidence of involvement of the parenchyma. The presence of asbestos was histologically proven in three of the reported cases.

ASBESTOSIS is a respiratory disease caused by the inhalation of asbestos particles, mineral substances composed of magnesium iron silicates that form long flexible parallel fibers. These fibers possess a great tensile strength, are resistant to heat, alkalies and acids, can be spun into yarn, and woven into textiles.[18] Such properties make asbestos an important material for various industrial uses. Asbestos is needed in the manufacturing of blankets, clothing, threads, rope, tape, braided tubing, brake lining and brake blocks, wallpaper, wallboard, shingles, firebricks, floor covering and plastics. Asbestos is mixed with cements and plasters. It is used as insulation material for houses, pipes, boilers, ranches, wire, heaters, ironing boards, heating pads, automobile and machinery parts, etc.[3, 5, 14, 16] Due to the rapid expansion of its use, an ever-increasing number of craftsmen and laborers are exposed to this mineral. Recently, for instance, an automobile mechanic was reported to have acquired asbestosis while undercoating vehicles.[3] In addition to personnel handling the material, others not directly in contact may be exposed through air pollution.[21]

Fortunately, not all workmen acquire this disease. It has been estimated from animal experiments that[19] at least five million particles per cubic foot have to be present in the air to produce symptoms. Only fibers of a certain length (not less than 20 and not more than 50 microns) seem to be dangerous.[6] The exposure must be prolonged, and an individual sensitivity must be present. Nevertheless, a greater number of patients suffering from asbestosis can be expected in the future.

Many of them may not obtain a correct diagnosis. The latter depends essentially on the history of exposure; in addition, the clinical picture of the disease will aid in its recognition. The discovery of the rod- and club-shaped asbestos bodies in the sputum is of great significance because it proves that contact with asbestos dust has been made. Unfortunately, these bodies which consist of mineral fibers covered by proteinaceous material are not expectorated very often. The final proof of the disease depends on the demonstration of these particles inside the lung tissue[5, 17] (Fig. 1). This requires lung biopsy or autopsy.[5] The history of exposure to asbestos is often obscure because workmen are not aware that they are handling this material. In addition, it takes five to ten years of contact to develop symptomatology. Therefore, a patient may not realize that his ailment is connected with his occupation. Furthermore, the symptoms appear gradually and are nonspecific.[15] They consist of fatigue, anorexia, weight loss, weakness, dyspnea, cough and expectoration. The physical examination may show a few basal rales, a mild cyanosis and some clubbing of

FIGURE 1

fingers.[16] Initial chest x-rays will be negative.[17] A defect of pulmonary function, however, may already be present in the form of an alveolar-capillary diffusion block. Unfortunately, special equipment is needed to demonstrate such a deficiency, and the customary spirometric tests for lung volume reduction and airway obstruction are usually non-revealing.[4,5,16,18] In the more advanced stages of asbestosis the chest film will show some abnormalities. There may be bilateral basal fibrosis, groundglass clouding or honeycombing of the lower portion of the lungs. Large nodulations similar to those of silicosis are seldom seen, and localized areas of consolidation—usually caused by superimposed infection—are still more uncommonly encountered.[19]

All investigators agree that pleural changes occur frequently in asbestosis, particularly obliteration of the phrenicocostal sinuses, pleural "tents," plaques and calcifications as well as pleuromediastinal adhesions that are responsible for the so-called "shaggy heart."[11] However, it has not been sufficiently emphasized that asbesto-

sis can present itself in the form of pleural disease without any obvious parenchymal manifestations. This pleurisy may be acute, subacute, recurrent or chronic. It may be unilateral or bilateral. It usually affects the lower lung fields, but may involve an upper portion.[5] The character of this pleurisy varies considerably. It may be a mild, self-limited disease; it may appear as a prolonged or recurrent disorder; or it may be a primary malignancy of the pleura.

Such pleural forms of asbestosis frequently escape recognition as shown in the following three illustrative cases.

Case 1. A 54-year-old white male had worked as an insulator for a number of years.

In September, 1960, he suffered from a left-sided pleurisy. An x-ray film seemed to reveal a small pleural effusion. However, this diagnosis was not verified by thoracentesis because of the benign course of the disorder. The patient recovered completely from this episode.

In March, 1961, a similar attack of pleurisy appeared on the other side. At this time the symptoms were more severe, consisting of chest pain, nonproductive cough, anorexia and weight loss. A loud

FIGURE 2

[illegible] ... with a flat percussion note and suppression of breath sounds. [illegible] showed pleural effusion (Fig. [illegible]) [illegible] abnormalities were [illegible]. [illegible] streptococci and staphylococci [illegible] sedimentation rate was [illegible] (Westergren). The Latex agglutination was positive [illegible] 80 dilution. L.E. cells were absent [illegible] blood smear, the tuberculin skin test was [illegible]. A thoracentesis yielded 350 [illegible] brown-colored fluid. It contained neither malignant cells nor acid-fast bacilli. The patient was placed on a medical regimen consisting of bed rest, antituberculous medications and high caloric diet. About one month later, however, a second pleural tap became necessary. The same thoracentesis fluid was encountered.

Soon afterwards a thoracotomy was carried out because of persistent symptomatology. The surgical exploration revealed pleural adhesions with partial obliteration of the pleural cavity. The visceral pleura was as thick as an orange peel; therefore, a decortication was performed in addition to a lung biopsy. All symptoms disappeared after this operation, and the patient was restored to good health shortly thereafter.

The pathologist (Dr. Stuart Wallace, St. Mary's Hospital, Port Arthur) found "large amounts of dense connective tissue with infiltrates of lymphocytes and eosinophiles in the pleural sections. Along the pleural edge were giant cells surrounding small tags of necrotic tissue (Fig. 3). The lung section revealed thickening of the alveolar walls with macrophages and asbestos bodies (Fig. 1)."

FIGURE 3

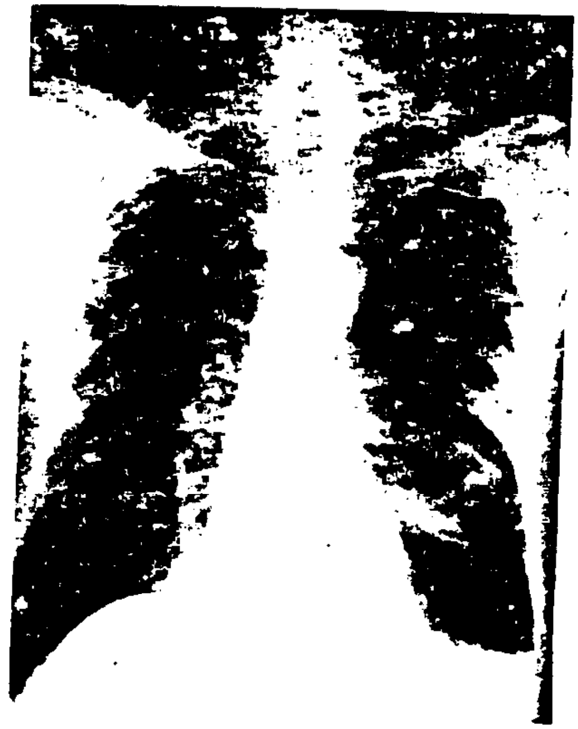

FIGURE 4

Case 2. A 57-year-old refinery foreman noticed a pleuritic type of pain in his left side for a number of years. His discomfort was attributed at first to coronary artery disease and later to an irritable bowel syndrome. However, there was never a typical anginal pain nor a characteristic gastro-intestinal dysfunction. In addition, nitroglycerin, antispasmodics, diet and bowel regulation brought no relief. On the contrary, the pain became gradually unbearable.

One day, unexpectedly, a chest film showed a left-sided pneumothorax with effusion (Fig. 4). No cause of this pleural complication could be elicited. Over a period of several months this pneumothorax was gradually replaced by a fibrothorax (Fig. 5). Since the pain persisted a thoracotomy was undertaken which revealed only connective tissue thickening. However the patient continued to go downhill after the operation, and finally destruction of a rib was discovered during a follow up study. A biopsy of this region revealed pleural mesothelioma. The patient died shortly afterwards, but no autopsy permit was granted. This case was originally reported be-

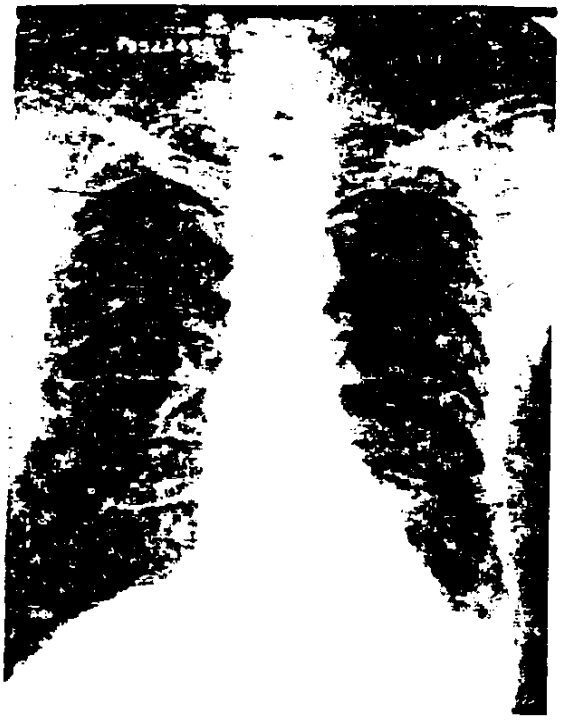

FIGURE 5

cause of the rarity of the malignant lesion and the difficulties of its diagnosis.[2] However, in retrospect, the patient was exposed to asbestos work for many years and apparently suffered from asbestosis. This disease had produced a partial symphysis of the pleura[10] which separated the cavity entered by the surgeon from the site of the malignant tumor. Unfortunately, no lung biopsy was obtained in any of the explorations. Such a specimen would have been necessary to confirm the diagnosis.

Case 3. In January, 1959, a 58-year-old oil refinery foreman who had worked with asbestos insulation for many years suffered from an acute left-sided hemorrhagic pleural effusion. Ten years earlier he had been hospitalized for a long time because of bilateral pleurisy. He made a complete recovery; however, ever since this episode, bilateral pleural calcifications (Fig. 6 →) were visible on x-ray pictures.

In 1959, increasing chest pains, progressing weight loss and atypical cells in the thoracentesis fluid lead to an exploratory thoracotomy.[2] This procedure revealed pleural symphysis with plaques and adhesions. Microscopic examination of the pleural specimen showed only non-specific granulomas.

Ten months later, however, a rib destruction was demonstrated similar to that of the previous patient (Fig. 6 →), and a biopsy of this region as well as an

[illegible] associated with [illegible] asbestosis.

Comment

In these three cases asbestosis presented itself as an "idiopathic pleural disease." The first patient suffered from a benign effusion. The second revealed a spontaneous pneumothorax changing gradually into a massive fibrothorax and terminating as a primary pleural malignancy. The last patient had initially bilateral benign pleurisy healing with pleural calcifications and died many years later of a unilateral malignant pleural disorder. This course of events is typical of asbestosis. At no time did these persons reveal any prominent clinical and roentgenological findings of parenchymal lung disease. Unfortunately, pulmonary function studies were not performed in these patients except in Case 1 after decortication. At that time normal results were encountered. Apparently, *pulmonary* asbestosis does not manifest itself clinically without marked impairment of respiratory function while *pleural* disease may

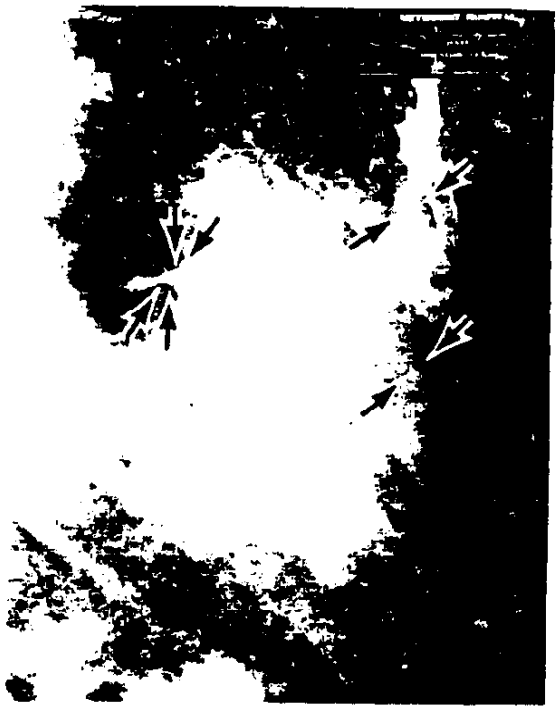

FIGURE 6

occur in the absence of respiratory insufficiency. The diagnosis of pleural asbestosis may be easily missed if only a pleural biopsy is performed because the characteristic asbestos bodies are not present in the diseased pleura itself.[19] Apparently, they are too large to reach the pleura through the lymphatic channels. They usually remain in the small bronchial ducts[5, 6] and do not even enter the alveoli.[6, 19] However, they seem to release some fibrogenic toxin responsible for the severe connective tissue reaction surrounding them.[6, 12] This pleuro-pulmonary fibrosis has been suspected to be an immunological response because it becomes apparent after many years of latency.[9, 10] The presence of many eosinophile cells in the peripheral blood as well as in the tissue section, the rapid sedimentation rate and the positive Latex agglutination test of Case 1 seemed to be best explained by such a mechanism. Changes in serum protein and rapid sedimentation rates have been previously found in asbestosis.[21] However, a positive Latex test has not been reported. This laboratory finding may represent a link to rheumatoid pleurisy[20] and rheumatoid pneumonoconiosis.[4] However, our patient never had any form of joint involvement that is so typical for the latter two disorders.

In the light of modern experiences the old dictum "idiopathic pleurisy is due to tuberculosis unless proven otherwise" must be modified. It is true that a considerable number of such instances will still be caused by the tubercle bacillus. However, in recent years exploratory thoracotomy has revealed a great variety of other causes of idiopathic pleural disease. Asbestosis will have to be added to this group. Only a thorough surgical exploration with pleural and parenchymal biopsies will permit differentiation of these lesions. Such a procedure can always be followed immediately by decortication and partial resection of the lung which are required to accomplish a cure in the majority of cases. However, in asbestosis even such aggressive therapy will not completely eliminate the danger of reactivation of the pleural disease on the other side nor the development of pulmonary fibrosis, respiratory insufficiency, cor pulmonale and primary pulmonary malignancy.[1, 2, 17] Therefore, the best medical approach to asbestosis still consists of taking proper preventive measures in the form of pre-employment and periodic health examinations of workers, limitation of working periods, ventilation of work rooms, and safe protective respiratory devices.

Summary

The widespread use of asbestos makes asbestosis an important occupational health problem. This type of pneumonoconiosis is not easily recognized. A history of exposure to such mineral dust is a significant clue; however, it may not be obtainable and, even if elicited, it may be misleading. Only a lung biopsy demonstrating asbestos bodies in the pulmonary parenchyma can clinch the diagnosis, particularly if this disease presents itself primarily as an idiopathic pleurisy.

Acknowledgment

The author expresses his appreciation to Dr. E. C. McRee, Port Arthur, for the permission to report his patient (Case 1).

Bibliography

1. Anderson, John, and Francis A. Campagna: Asbestosis and carcinoma of the lung. Arch. Env. Health, 1:39, 1960.
2. Bader, Mortimer E., Richard A. Bader, and Irving J. Selikoff: Pulmonary function in asbestosis of the lung. Am. J. Med., 30:235, 1961.
3. Brugsch, Heinrich G., and Harold Bayley: Asbestosis in a worker engaged in automobile undercoating. New Eng. J. Med., 265:379, 1961.
4. Caplan, A.: Certain unusual radiological appearances in the chest of coal-miners suffering from rheumatoid arthritis. Thorax, 8:29, 1953.
5. Castleman, Benjamin, and Betty U. Kibbee: Case records of the Massachusetts General Hospital, weekly clinicopathological exercises, case 73-1961. New Eng. J. Med., 265:745, 1961.
6. Clinicopathological Conference: Complications of asbestosis, demonstrated at the Postgraduate Medical School of London. Brit. Med. J., No. 5182:1345, 1960.
7. Clinicopathologic Conference. U. S. Naval Hospital, Philadelphia, Penn. U. S. Armed Forces Med. J., 2:203, 1960.
8. Eisenstadt, H. B.: Malignant mesothelioma of the pleura. Dis. Chest, 30:549, 1956.
9. Eisenstadt, H. B.: Primary malignant mesothelioma of the pleura. Journal-Lancet, 80:511, 1960.

4. [illegible] Brian E. and Roger Williams: The physiology of asbestosis with reference to lung function. Thorax, 16:294, 1961.
5. Harwitz, M.: Roentgenologic aspects of asbestosis. Am. J. Roentgenology, Radium Therapy and Nuclear Med., 85:[illegible], [illegible].
12. [illegible] Kenneth: [illegible] Physiology of asbestosis. A. M. A. Arch. Ind. Health, 12:185, 1955.
13. McGrath, Margaret [illegible] and L. Thomson: Pulmonary [illegible] and lung volumes in asbestosis and chronic bronchitis with emphysema. Clin. Sci., 21:15, 1961.
14. Mitchell, Jean: Health progress in an asbestos textile works. Arch. Env. Health, 3:42, 1961.
15. Sanders, O. A.: Asbestosis as differentiated from other pneumoconiosis. A. M. A. Arch. Ind. Health, 11:298, 1955.
16. Smith, Kenneth W.: Pulmonary disability in asbestos workers. A. M. A. Arch. Ind. Health, 12:195, 1955.
17. Teleschi, M. and A. L. Rubenstone: Pulmonary asbestosis. Arch. Path., 72:274, 1961.
18. Thomson, M. L., Margaret McGrath, W. J. Smither and J. M. Shepherd: Some anomalies in the measurement of pulmonary diffusion in asbestosis and chronic bronchitis with emphysema. Clinical Science, 21:1, 1961.
19. Vorwald, Arthur J., Thomas M. Durkan, and Philip C. Pratt: Experimental studies of asbestosis. Arch. Ind. Hygien. and Occup. Med., 3:1, 1951.
20. Ward, R.: Pleural effusion in rheumatoid disease. Lancet, 2:1336, 1961.
21. Williams, Roger, and P. Hugh-Jones: The significance of lung function changes in asbestosis. Thorax, 15:109, 1960.

The Author——

Dr. H. B. Eisenstadt is a native of Berlin, Germany. He formerly served as German specialist for internal diseases. Since 1938 he has practiced Internal Medicine in Port Arthur. He is a Diplomate of the American Board of Internal Medicine and a Fellow of American College of Physicians, Chest Physicians, Cardiology and Gastroenterology.