The Patient With Asbestos Exposure

Robert J. McCunney, MD, MPH
Boston, Massachusetts

Attention to the hazards of asbestos has aroused concern among many healthy persons who have been exposed at some time to one of the world's most versatile materials. Present standards for exposure to asbestos, however, may not fully protect workers from asbestos-induced pulmonary malignancy. To evaluate the patient who has had some asbestos exposure, the physician should focus on the pulmonary system, take a thorough occupational history, conduct a physical examination and pulmonary function studies, and obtain chest films. In managing the patient with asbestosis, removal from further occupational exposure is advisable. For workers with only pleural changes, it is unclear whether removal from the work place is of any value.

With the widespread attention given to the health hazards of asbestos, the primary care physician will be increasingly called upon to evaluate the otherwise healthy individual who has been exposed at some time to asbestos. Issues such as the long latency period between first exposure and development of malignancies, as well as the lack of a safe level to prevent carcinogenic effects, have heightened public awareness about one of the world's most versatile materials. Since World War II, it is estimated that over 10 million people have been exposed to asbestos.

About 20,000 lawsuits, totaling nearly $45 billion, have been brought by shipyard workers against the major manufacturers of asbestos. Johns Mansville, the leading manufacturer, in turn, has filed bankruptcy proceedings, claiming the government is partly responsible for the claims, since more than one half of the individuals' exposure occurred while they were employed in government-operated shipyards. Meanwhile, the House of Representatives is considering a bill to create a national occupational disease compensation fund,[1] and the Occupational Safety and Health Administration (OSHA) is evaluating the issuing of an emergency temporary standard for asbestos. Clearly, asbestos will continue to be of concern in medical care.

In this paper focus is placed on the widely accepted adverse health effects of asbestos, and a proposed protocol is presented for the evaluation and management of asymptomatic persons with a history of exposure to asbestos.

BACKGROUND

Asbestos, known for its heat resistance, flexibility, and frictional qualities, is a mineral with over 3,000 uses. Although the manufacture of asbestos-related products has declined in the United States within the past decade, its worldwide use has increased. Asbestos is used primarily in the construction industry, in particular in cement piping, roofing, and paneling, and as a filler in architectural dead space (Table 1). Brake shoes and clutch linings continue to be made from asbestos, since suitable substitutes are unavailable.

OSHA has established federal standards that dictate acceptable levels of concentrations of asbestos in the work place, the type of respirator to be used, and the type and frequency of medical examination required.

From the Occupational Health Program, Boston University Medical Center, Boston, and Goddard Memorial Hospital, Stoughton, Massachusetts. Address reprint requests to Dr. Robert J. McCunney, Cabot Corporation, 125 High Street, Boston, MA 02110.

TABLE 1. INDUSTRIES THAT USE ASBESTOS, THEIR PRODUCTS, AND JOBS AT RISK

Industry	Products	Jobs At Risk
Textiles	Cloth, curtains, lagging, protective clothing, mailbags, padding, conveyor belts	Blenders, carders, spinners, twisters, winders, braiders, weavers, slurry mixers, laminaters, moulders, dryers
Cement products	Sheets, pipes, roofing shingles, gutters, ventilation shafts	Blenders, slurry preparers, rollers, pressers, pipe cutters
Paper products	Millboard, roofing, felt, fine-quality electrical papers, flooring felt, filters	
Friction material	Automotive products: gaskets, clutch plates, brake linings	
Insulation products	Pipe and boiler insulation, bulkhead linings for ships	
Construction		
New	Boards and tiles, putties, caulk, paints, joint fillers, cement products (tiles, pipes, siding, shingles), insulation materials	Directly: carpenters, laggers, painters, tile layers, insulation workers, sheet metal and heating equipment workers on construction sites such as plumbers, welders, electricians
Repair		Demolition workers
Shipbuilding		
Construction	Insulation materials (boards, mattresses, cloth) for engines, hull, decks, lagging of ventilation and water pipes	Laggers, refitters, strippers, steam fitters, sailmakers, joiners, shipwrights, engine fitters, masons, painters
Repair	Insulation materials, as described for construction	Directly: all above jobs in construction Indirectly: maintenance fitters and repairmen, electricians, plumbers, welders, carpenters
Automotive		
Manufacture	Gaskets, brake linings, undercoating	Installers of brake linings and gaskets
Repair	Gaskets, brake linings, undercoating	Servicemen, brake repairment, body repairmen, auto mechanics

The health aspects of asbestos-induced disease have been reviewed in detail elsewhere.[2-5] Since inhalation is the primary route of exposure, asbestosis, bronchogenic carcinoma, and mesothelioma are the major illnesses associated with asbestos. The development of asbestosis, a progressive interstitial lung disease that can lead to permanent impairment and death, depends upon the intensity and duration of exposure. Host factors are widely suspected of contributing to its pathogenesis, since increased levels of HLA-B27 antigens[6] and impaired cell-meditated immunity have occurred with greater frequency in exposed patients when compared with controls.[7,8] The fibrotic process, which can continue even after a worker has been removed from exposure, occurs more frequently in the lower lobes.

Bronchogenic malignancies (with latencies averaging over 20 years) occur more frequently in asbestos workers, especially cigarette smokers, than in other workers.[9-11] The incidence of tumors is directly related to the degree of fibrosis; the clinical presentation and management are similar to lung cancers of other causes. Nonsmokers have a much lower risk of malignancy.[12] There appears to be a linear relationship between level of asbestos exposure and risk of bronchogenic carcinoma; however, no safe level below which malignancies do not occur has been established.[13]

Mesothelioma has been described as being

pathognomonic of asbestos exposure,[14,15] although in some patients no history of exposure can be elicited. The tumor was first noted by 19th-century pathologists. Its pathogenesis still remains unknown and difficult to study because the tumor appears to occur only in humans.[16] Although rare in the general population, mesothelioma has been described in 2 to 10 percent of asbestos workers.[17,18]

To summarize, previous investigative work has revealed the following:

1. Exposure to asbestos increases the risk of lung cancer, mesothelioma, and asbestosis. Cancer of the larynx, colon, esophagus, stomach, and rectum has been described,[19,20] but "the evidence must be assessed cautiously because associations thus far reported are weak."[21] The carcinogenic potential of asbestos in the gastrointestinal tract appears to be low.[22]

2. A positive dose-response relationship exists between level of exposure to asbestos and asbestos-related disease, including asbestosis, bronchogenic carcinoma, and mesothelioma. A similar relationship exists among clinical findings, pulmonary function studies, and chest film abnormalities.

3. No safe level of asbestos below which risk of mesothelioma or lung cancer does not exist has been established.

4. Smoking greatly increases the risk of asbestos-induced bronchogenic carcinoma. There appears to be no relationship between smoking and pleural changes, including mesothelioma.

MEDICAL EVALUATION

The OSHA standard for the medical evaluation of workers exposed to asbestos includes a thorough medical and occupational history, physical examination, chest film, and pulmonary function studies.[23] The same studies are advisable when a physician evaluates an asymptomatic patient.

Occupational History

Since asbestos-induced illnesses can occur with minimal exposures, a thorough occupational history is essential. Attention should be given to the type of job and to the intensity of exposure experienced by the worker. Even indirect exposure, such as that experienced by electricians, sheet-metal workers, and welders in ship construction, has been associated with adverse health effects. The time of first exposure is important because of the long latency associated with mesothelioma and lung cancer. Place of residence and jobs of family members should be noted, since mesothelioma has been described among wives whose only exposure to asbestos was through laundering their husbands' work clothes.

Physical Examination

End inspiratory dry rales in the midaxillary line are the most common lung findings associated with asbestosis. Clubbing has also been described, but its diagnostic utility has been questioned. In mesothelioma, a unilateral pleural effusion can be present. Asbestos bodies in both sputum and lung tissue are described as histological hallmarks of asbestos exposure.[25,26] These fibers, coated by hemosiderin and glycoprotein complexes, are thought to be formed by macrophages that have phagocytized an asbestos particle. The presence of asbestos bodies in sputum or lung tissue proves asbestos exposure, but does not necessarily indicate asbestos-related disease, since some studies have demonstrated the presence of asbestos bodies in lung tissue in 50 percent of unselected autopsies.

Chest Film

The chest film is the most sensitive noninvasive test available to detect asbestos-induced pulmonary disease.[27] The most common finding is bilateral pleural thickening, which has been shown to have an 80 percent predictive value for past exposure to asbestos.[28] Pleural thickening is often manifested as multiple discrete pleural plaques rather than as diffuse thickening. These plaques, with their characteristic appearance and calcification, are virtually pathognomonic of asbestos exposure. Diffuse pleural thickening may occur in many other disorders. Pleural calcifications (usually bilateral) are a late finding. In asbestosis, small linear irregular opacities are usually noted symmetrically at the lung bases. The lesions usually involve the lower half of the lungs and assume

a reticulonodular pattern, sometimes with honeycombing.

Pulmonary Function Studies

Since asbestosis is a restrictive disease, one expects a decline in forced expiratory volume (FEV_1) with a preservation of the FEV_1 to the forced vital capacity (FVC) ratio. Changes in FVC have been heralded as being more accurate in describing the pulmonary deterioration over time as well as being the most sensitive test to determine early pathological changes. A low FVC and FEV_1 may also occur in obstructive airway diseases and are not specific for restrictive disease.

Review of Systems

There are no specific early symptoms for asbestosis, although patients can present with exertional dyspnea, nonproductive cough, and a vague feeling of being unwell. Symptoms of lung cancer caused by asbestos occur late in the disease process, as do symptoms of lung cancer from other causes, whereas mesothelioma's initial manifestations include shortness of breath and nonpleuritic pain, often referred to the upper abdomen or shoulder.[24]

DIAGNOSIS

Diagnosing asbestos-related disease can be difficult, since no uniform criteria exist. Recent guidelines for pathological diagnosis, however, have been proposed.[5] With a good exposure history and typical x-ray findings of interstitial disease, asbestosis can be diagnosed with confidence without biopsy.[29] The only definitive test, however, is the open lung biopsy, because transthoracic and transbronchial biopsies usually do not yield sufficient tissue. Usually, there are discrete foci of fibrosis in the walls of the respiratory bronchioles associated with the accumulation of asbestos bodies. These changes have been described without abnormalities in chest films. A proposed grading scheme for pneumoconiosis has been described.[30] Since fibrosis occurs in other disease states, the association of pleural thickening and calcification enhances diagnostic accuracy. Diffusion capacity can assist in difficult cases, since it has been described as being 95 percent sensitive (95 percent of patients with asbestosis have diffusion capacities lower than two standard deviations below normal).[31] Bronchoalveolar lavage has demonstrated signs of alveolitis, thought to presage asbestosis.[32]

The diagnosis, treatment, and management of asbestos-induced bronchogenic carcinoma is similar to that for bronchogenic carcinoma resulting from other causes, such as that resulting from smoking.

In the diagnosis of mesothelioma, the most crucial step is to distinguish the tumor from primary bronchogenic carcinoma and from metastatic carcinoma and sarcoma. A unilateral pleural effusion, usually an exudate, is often present. Since the tumor has a tendency to grow along needle tracts, closed lung biopsies are discouraged in favor of thoracotomies.[33,34] The computerized tomographic (CAT) scan can differentiate focal plaques from intrapulmonary modules as well as delineate pulmonary pseudotumors.[35]

The average patient among the well will not fall into any of the above diagnostic categories, but may have any number of abnormalities, including benign asbestos pleural effusion, pleural thickening, pleural plaques, and calcification.

Benign asbestos pleural effusion is defined as exposure to asbestos, confirmed by chest film or thoracentesis, with no other disease related to pleural effusion and no malignant tumor within three years. In the study of workers, Epler[36] et al found that in over 1,100 workers, benign asbestos pleural effusion was the most common asbestos-related abnormality noted during the first 20 years of exposure.

Pleural thickening is another common finding noted during the evaluation of "healthy" patients exposed to asbestos. The changes are usually bilateral and are generally not associated with an increased risk of mesothelioma. Other causes of pleural thickening include dusts, pleurisy, and old rib fractures.

Pleural plaques are discrete grey-white lesions consisting of hyalinized fibrous tissues found on the parietal pleura of the thorax, diaphragm, mediastinum, and pericardium. The plaques commonly occur in the absence of other asbestos-induced lung disease and do not appear to develop into malignancies, although some suggest such a

TABLE 2. MEDICAL FOLLOW-UP OF PERSONS WITH PAST OR CURRENT HISTORY OF EXPOSURE TO ASBESTOS

Frequency	Condition or Situation	Routine Management
Every 6 months	Asbestosis	Review of systems Physical examination Chest film with oblique views Sputum for cytologic testing* Aggressive treatment of pulmonary area Upper respiratory tract infections Current pneumococcal and influenza vaccinations
Yearly	Exposure for 10 years Ten years since first exposure Currently working in asbestos environment Clinical finding suggestive but not diagnostic of asbestosis Chest film abnormalities secondary to asbestosis	Review of systems Physical examination Pulmonary function studies Chest film (PA,** lateral, and oblique views)
Every 2 years	Asymptomatic Negative findings Exposure for 10 years, or 10 years since exposure	Review of systems Physical examination Pulmonary function studies Chest film (PA,** lateral, and oblique views)

Currently, no evidence exists to support the belief that these tests performed on a routine basis will help reduce morbidity or mortality from asbestos-induced pulmonary malignancies
***PA, posterior/anterior*

risk.[29,37] Patients with pleural plaques appear more likely to develop parenchymal disease.[38]

MANAGEMENT

Asbestosis should be managed symptomatically, with aggressive treatment of infections, which are the usual cause of death. The use of steroids is generally contraindicated, whereas influenza and pneumococcal vaccines are strongly recommended.

Bronchogenic carcinoma is managed as in other settings. For mesotheliomas, radiation and chemotherapy have offered promise, but effective treatment remains elusive. In some settings, pleural decortication has proven helpful.[39,40]

Follow-up

Table 2 proposes a scheme for the follow-up of abnormalities noted in the initial evaluation. The point of any periodic evaluation in an otherwise healthy person is to determine the presence of unrecognized disease so that early intervention will reduce long-term morbidity and mortality from the disease in question. For mesothelioma and bronchogenic carcinoma, no screening tool, including sputum cytology, has proven effective, although periodic pulmonary function studies can be helpful in diagnosing early signs of asbestosis.

References

1. Disease victims get one-third of claims sought in law suits, according to Rand study. Occup Saf Health Rep 1983; 13(10):213-214
2. Becklake M: Asbestos related disease of the lung and other organs: Their epidemiology and implications for clinical practice. Am Rev Respir Dis 1976; 141:187-227
3. Selikoff IJ, Lee DH: Asbestos and Disease. New York, Academic Press, 1978
4. Preger L, Arai DT, Kotin P, Weill H, et al: Asbestos-Related Diseases. New York, Grune & Stratton, 1978, p 271

5. Craighead JE, Abraham JL, Churg A, et al: The pathology of asbestos-associated diseases of the lungs and pleural cavities: Diagnostic criteria and proposed grading schema. Arch Pathol Lab Med 1982; 106:544-596
6. Merchant JA, Klonda PT, Soutar CA, et al: The HLA system in asbestos workers. Br Med J 1975; 1:189-192
7. Nagan E, Solomon A, Cochrane JC, et al: Immunological studies of patients with asbestosis: I. Studies of cell mediated immunity. Clin Exp Immunol 1977; 28:261-267
8. Kang KY, Sera Y, Okochi X, et al: Lymphocytes in asbestosis. N Engl J Med 1974; 291:735-745
9. Selikoff, IJ, Hammond EC: Asbestos-associated disease in U.S. shipyards. 1978; 28:2
10. Selikoff IJ, Hammond EC, Seidman H: Latency of asbestos disease among insulation workers in the United States and Canada. Cancer 1980; 46:2736-2740
11. Blot WJ, Harrington JM, Toledo A, et al: Lung cancer after employment in shipyards during World War II. N Engl J Med 1978; 299:620-624
12. Selikoff IJ, Hammond EC, Seidman H: Mortality effects of cigarette smoking among Amosite asbestos factory workers. J Natl Cancer Inst 1980; 65:507-513
13. McDonald C, Liddell FDK, Gibbs GW, et al: Dust exposure and mortality in chyrsotile mining. Br J Ind Med 1980; 37:11-24
14. Godwin MC, Jagatic J: Asbestos and mesothelioma. Environ Res 1970; 3:391-416
15. Elme PC: Mesotheliomas, minerals and man made mineral fibres. Thorax 1980; 35:561-563
16. McDonald JC, McDonald AD: Epidemiology of mesothelioma from estimated incidence. Prev Med 1977; 6:426-646
17. Selikoff J, Lilis R, Nidrovlson WJ: Asbestos disease in United States shipyards. Health hazards of asbestos exposure. Ann NY Acad Sci 1979; 330:297-311
18. Newhouse ML, Berry G: Predictions of mortality from mesothelial tumors in asbestos factory workers. Br J Ind Med 1976; 33:147-151
19. Selikoff IJ, Hammond EC: Multiple risk factors in etiology of environmental cancer. In Fraumeni JR (ed): Persons at High Risk of Cancer: An Approach to Cancer Etiology and Control. New York, Academic Press, 1975, pp 467-483
20. Elines PC, Simpson MJC: Insulation workers in Belfast 3. Mortality 1940-66. Br J Ind Med 1971; 28:226-236
21. Miller AB: Asbestos fibre dust and gastrointestinal malignancies. Review of literature with regard to a cause/effect relationship. J Chronic Dis 1978; 31:23-33
22. Craighead JE, Moosman BT: The pathogenesis of asbestos-associated diseases. N Engl J Med 1982; 306:1446-1455
23. General Industry Standards, Occupational Safety and Health Administration 29 CRF1910.1001(j). Government Printing Offices, 1978
24. Antman KH: Malignant mesothelioma. N Engl J Med 1980; 303:202-202
25. Gaensler EA, Addington WW: Asbestos or ferruginous bodies. N Engl J Med 1969; 280:488-492
26. David JMG: Further observations on the ultrastructure and chemistry of asbestos bodies. Exp Mol Pathol 1970; 13:346-358
27. Murphy R: Asbestos related disease: Difficulties in diagnosing occupationally related illnesses. Compr Ther 1980; 6:6013
28. Albelda SM, Epstein DM, Geften WB, Miller WT: Pleural thickening: Its significance and relationship to asbestos dust exposure. Am Rev Respir Dis 1982; 126:621-624
29. Becklake MR: Asbestos-related diseases of the lungs and pleura. Am Rev Respir Dis 1982; 126:187-194
30. Guidelines for the Use of the ILO International Classification of Pneumoconioses. Geneva, International Labor Office, 1980
31. Ferris BF: Recommended standardization procedures for pulmonary function testing. Am Rev Respir Dis 1978; 118:55-88
32. Rebuck AS, Braeede AC: Bronchoalveolar lavage in asbestosis. Arch Intern Med 1983; 143:950-952
33. Antman KH, Blun RH, Greenberger JS, et al: Multimodality therapy for malignant mesotheliomas based on a study of natural history. Am J Med 1980; 68: 356-362
34. Legha SS, Muggia FM: Pleural mesothelioma: Clinical features and therapeutic implications. Ann Intern Med 1977; 87:613-621
35. Goodman LR: Radiology of asbestos disease. JAMA 1983; 249:644-646
36. Epler GR, Mcloud TC, Gaensler EA: Prevalence and incidence of benign asbestos pleural effusion in a working population. JAMA 1982; 247:617-622
37. Edge JR: Incidence of bronchial cancer in shipyard workers with pleural plaques. Ann NY Acad Sci 1979; 330:289-294
38. Sheers G: Asbestos associated disease in employees of Devonport dockyard. Ann NY Acad Sci 1979; 300:281-288
39. Butchart EG, Ashcroft T, Barnsley WC, et al: Pleuropneumonectomy in the management of diffuse malignant mesothelioma of the pleura. Thorax 1976; 31:15-24
40. DeLarie GA, Jensik RJ, Faber LP, et al: Surgical management of malignant mesothelioma. Ann Thorac Surg 1978; 26:375-381