MEDICAL EDUCATION SYSTEMS, INC.

Asbestos Toxicity

Asbestos Toxicity

Goals and Objectives

The goal of the CEU is to increase the primary care provider's knowledge of hazardous substances in the environment and to aid in the evaluation of potentially exposed patients.

After completion of this course, the reader should be able to explain why asbestos may be a health hazard, describe the factors contributing to asbestos toxicity, identify potential environmental and occupational sources of exposure to asbestos, identify evaluation and treatment protocols for persons exposed to asbestos, and list sources of information on asbestos.

To receive credit for this course, submit your answers for the pretest and the challenge test items.

Case Study

- A 10-year-old boy has shortness of breath and was recently exposed to asbestos

A 10-year-old boy appears at your office with a chief complaint of shortness of breath. Exertional dyspnea has been present for the previous month and is associated with intermittent dry cough. The patient has no associated fever, chills, or chest pain. Chart review indicates no history of asthma or other pulmonary disease, although the patient has been seen several times for "hay fever."

The patient is accompanied by his mother, who appears quite anxious. The mother emotionally relates that her 65-year-old cousin has recently been diagnosed with mesothelioma and is dying. Furthermore, he had been a custodian at the patient's school for the previous 3 years, after retiring from his career as a longshoreman. His work at the school involved general cleanup and boiler room maintenance. The mother is afraid that her son's dyspnea and cough are related to asbestos exposure at the school and that he might be developing mesothelioma, because he often helped her cousin after school. Recent asbestos removal in the school boiler room has increased the mother's concern.

On physical examination, the patient is in no acute distress. Respirations are unlabored. Lung auscultation reveals a diffuse, expiratory wheeze.

Spirometry performed in the office shows a forced vital capacity (FVC) of 95% of predicted value and a forced expiratory volume in 1 second (FEV_1) of 88% of predicted value, with an FEV_1/FVC of 70%. The remainder of the examination is within normal limits. A chest radiograph is normal.

Pretest

(a) Discuss whether the patient's symptoms are related to asbestos exposure.

(b) Is the patient at risk for future disease? Explain.

(c) Can the cousin's mesothelioma be related to his work as a custodian in the school? Explain.

Who's At Risk

In the past, asbestos exposure was associated mainly with mining and milling of the raw material and with workers engaged in product manufacture. Because industrial use has decreased over the last 40 years, these occupational exposures have declined. Today, most exposures occur during repair, renovation, removal, and maintenance of asbestos that was installed years ago. The number of new exposures to the general population from in-place asbestos, however, may be greater in number than the exposures experienced by all earlier workers combined.

In detailed interviews in industrialized countries, 20% to 40% of adult men report some past occupations and jobs that may have entailed asbestos exposure at work (International Expert Meeting on Asbestos, Asbestosis, and Cancer 1997). An estimated 27 million workers in the United States were exposed to aerosolized asbestos fibers between 1940 and 1979. Over 30 million tons of asbestos have been mined, processed, and applied in the United States since the early 1900s (Kamp and Weitzman 1999). In industrialized countries, about 10,000 mesotheliomas and 20,000 asbestos-induced lung cancers are estimated to occur annually in a population of about 800 million people (International Expert Meeting on Asbestos, Asbestosis, and Cancer 1997).

- Workers in the construction trades are most heavily exposed to asbestos.

The most heavily exposed people in the United States are those in the construction trades. In 1988, there were 6,300,000 active construction workers in the United States. Because most asbestos has been used in construction, and two-thirds of asbestos produced is still used in this trade, risk to these workers can be considerable. Carpenters, utility workers, electricians, pipefitters, steel mill workers, sheet metal workers, boiler makers, and laborers are at risk for exposure of asbestos through construction materials; insulation coverings of pipes, boilers, and industrial furnaces; and other sources. Mechanics working with brake and transmission products also can be exposed to asbestos.

- Spouses and family members can be exposed through asbestos dust on workers' skin and work clothing.

Secondary exposure occurs when fibers released to the air are inhaled by persons not directly handling asbestos.

For example, 4 to 5 million shipyard workers were exposed when a relatively small number of insulation workers applied asbestos to ships' pipes and hulls. Domestic and environmental asbestos exposures can also occur indirectly. Asbestos-related diseases have occurred in family members whose only contact was dust from an exposed worker's clothing. In some circumstances, exposures in household members might approach occupational levels. Similar diseases were also found in persons who grew up within one-half mile of an asbestos factory.

People in contact with work clothes of asbestos workers or with asbestos-containing household products have developed pleural abnormalities. An asbestosis prevalence of 11% in wives, 8% in sons, and 2% in daughters was reported in families of asbestos-exposed shipyard workers. Low exposures from work-related, household, and natural sources can induce pleural plaques. For diffuse, pleural thickening, higher exposure levels might be required (International Expert Meeting on Asbestos, Asbestosis, and Cancer 1997).

According to Hillerdahl (1999), no known truly unexposed group exists in the world. No proof of a threshold value exists—that is, a minimum lower limit below which asbestos fibers cannot cause a tumor—and thus it is plausible that even such low exposure can cause mesothelioma (even if the risk is extremely low). However, on the basis of studies of nonoccupational exposures, it seems probable that occasional high-level exposure situations are those that are most harmful and should be avoided. The cumulative risk of background exposures is probably minor, and these concentrations cannot be reduced. Any source of pollution by asbestos that releases significant amounts of fibers should be eliminated, using correct equipment and techniques, as soon as it is discovered. If the asbestos is well contained and not disturbed, it is usually better to leave it in place. In many cases, encapsulation is better than removal.

- Cigarette smoke increases the risk of asbestos-associated lung cancer.

Cigarette smoking and exposure to other carcinogens greatly increase the risk of asbestos-associated lung cancer. A comparison of the experiences of 17,800 asbestos insulation workers with matched controls showed that asbestos workers who did not smoke suffered five times the number of lung cancer deaths than did controls who neither smoked nor worked with asbestos (55 deaths per 100,000 person-years for asbestos workers who did not smoke compared to 11 deaths per 100,000 person-years for controls who were neither asbestos workers nor smokers). Persons who smoked but did not work with asbestos had a death rate of 122 per 100,000 person-years; and among persons with both exposures (asbestos and cigarette smoking), 601 deaths occurred per 100,000 person-years. Evidence shows that cigarette smoking in asbestos workers is also associated with increased risk of cancer of the esophagus, oropharynx and buccal cavity, and larynx. However, other cancers that might occur in excess in asbestos-exposed persons, such as those of the stomach, colon-rectum, and kidney, do not appear to be synergistically affected by smoking and asbestos exposure, because smoking and nonsmoking asbestos workers suffer equal incidences of these health effects. Smoking appears to have no influence on the risk of mesothelioma. Although cancer, when established, can be irreversible, cancer risk due to smoking is reversible. Data indicate that risk diminishes when smoking ceases.

There might be genetic polymorphisms in various detoxifying enzymes (e.g., for reactive electrophilic molecules such as reactive oxygen radicals or nitrous oxide) that increase susceptibility to asbestos disease (Agency for Toxic Substances and Disease Registry 2001).

Challenge

> *(1) On questioning the mother, you learn that the father of the boy described in the case study is a master carpenter who specializes in restoring Victorian-style homes. What are the potential sources of asbestos exposure for the child?*
>
> *(2) The spouse of the mother's cousin is reportedly in good health. Should she be screened for asbestos-related disease? Explain.*

Exposure Pathways

- Asbestos exposure occurs primarily through inhalation of fibrous dust.

Asbestos is a generic term for a group of six naturally occurring fibrous minerals. The basic unit of asbestos-class minerals is the silicate combined in varying proportions with magnesium, iron, calcium, aluminum, and sodium or trace elements.

There are two major classes of asbestos: **serpentine**, which contains a magnesium silicate called chrysotile, and **amphiboles**, which represent a small portion of the world's commercial asbestos use and include crocidolite, amosite, anthophyllite, and tremolite. Chrysotiles are curly stranded fibers, and amphiboles are straight, rodlike fibers. Chrysotile, the sole member of the serpentine group, accounts for 93% of the world's commercial, purposeful asbestos use. The different classes and types of asbestos also appear to differ in toxicity, the basis of which might depend on fiber size, shape, and persistence in the lung (e.g., ability to clear the fiber, solubility, and durability). Contamination of other products such as vermiculite and talc from asbestos fibers is a larger problem than once thought.

Asbestos has been used in >3,000 products because of its high tensile strength, relative resistance to acid and temperature, and varying textures and degrees of flexibility. It does not evaporate, dissolve, burn, or undergo significant reactions with other chemicals, which makes asbestos non-biodegradable and environmentally cumulative.

Although many applications have been phased out of production, uses of asbestos have included the following:

Commercial

- Boilers and heating vessels
- Cement pipe
- Clutch, brake, and transmission components
- Conduits for electrical wire
- Corrosive chemical containers
- Electric motor components
- Heat-protective pads
- Laboratory furniture
- Paper products
- Pipe covering
- Roofing products
- Sealants and coatings
- Textiles (including curtains).

Homes and Buildings

- Duct and home insulation
- Fire protection panels
- Fireplace artificial logs or ashes
- Furnace insulating pads
- Fuse box liners
- Heater register tape and insulation
- Joint compounds

- Patching plaster
- Pipe or boiler insulation
- Sheet vinyl or floor tiles
- Shingles
- Textured acoustical ceiling
- Underlayment for sheet flooring.

Asbestos can also be a contaminant in other products such as vermiculite, which is used in gardens, landscape products; and home insulation; and talc, which is used in cosmetics.

Asbestos fibers can result from mining, milling, and weathering of asbestos-bearing rock, and from the manufacture, wear, and disposal of asbestos-containing products. Because of the widespread use of asbestos, its fibers are ubiquitous in the environment.

- Insulating materials produced before 1975 commonly contain asbestos.

In industrialized countries, commercial use of asbestos peaked in the 1970s. Although bans and voluntary phaseouts have contributed to declining production of asbestos since the early 1970s, it is still used in construction materials—mostly asbestos-cement products. Building insulation materials manufactured since 1975 may no longer contain asbestos; however, products made or stockpiled before the ban remain in many homes. Vermiculite-contaminated asbestos was produced as late as 1990.

Indoor air can become contaminated with fibers released from building materials, especially if they are damaged or crumbling. Common sources of asbestos in homes are sprayed asbestos ("cottage cheese") ceilings, pipe insulation, boiler coverings, wallboard, and floor and ceiling tiles. Although it is important to repair damaged asbestos as soon as possible, homeowners should not undertake repair or removal of asbestos-containing materials without professional guidance or services.

Although measurable asbestos levels in schools are usually 100 to 1,000 times below the permissible exposure limit for work environments (0.1 fibers/cc [8-hour time-weighted average, or TWA]—see Standards and Regulations section), public concern has led to widespread removal and abatement programs. However, some facilities have higher levels of airborne asbestos after removal than before, indicating that it is essential that any removal of asbestos be done properly.

Street dust can contain fibers from brake linings or crushed asbestos-containing rock used in road construction. Fibrous tremolite, the asbestos commonly found in talc, has also been found in play sand.

The air pathway is the most important route of exposure, but ingestion is possible, both from the water supply and other sources, and from swallowing what is cleared from the lungs. Drinking-water supplies might become contaminated with asbestos from erosion of natural land sources, discarded mine and mill tailings, asbestos cement pipe, and disintegration of other asbestos-containing materials transported via rain. Most water supply concentrations are <1 million fibers per liter, but in some cases have exceeded 100 million fibers per liter. The maximum contaminant level proposed by the U.S. Environmental Protection Agency (EPA) for asbestos in drinking water is 7 million fibers (>10 microns in length) per liter.

Challenge

> *(3) The patient and his family live in a home built in 1955. Pipes in the basement are covered with asbestos insulation. Should the family consider the removal of all asbestos pipe coverings in their home? Explain.*

Biologic Fate

The primary route of asbestos entry into the body is through inhalation. Ingestion of asbestos fibers can occur from drinking contaminated water (or ingestion from other sources) or after mucociliary clearance from the lungs and swallowing of the fibers. The fate of ingested asbestos is still being debated. However, it appears that a few ingested fibers pass through the gastrointestinal (GI) wall and reach blood, lymph, urine, and other tissues. (Fibers can also enter the lymphatic system via the lungs and migrate to other tissues.) Most ingested fibers will not be absorbed, but will be cleared in the feces. Asbestos fibers can also lodge in the skin and create hyperkeratoses or corns.

Asbestos fibers act differently from most types of inhaled particles in terms of the aerodynamics of reaching the depths of the lung. For most inhaled nonasbestos particles, generally only particles between 0.5 and 5 microns in diameter with a length-to-width ratio of 3:1 will be deposited in the respiratory regions (alveoli and terminal bronchioles) of the lungs. Larger particles of any kind tend to be filtered out in the upper airway and nasopharynx. Smaller particles tend to remain suspended in the inspired air, and the majority are exhaled. However, asbestos is an exception: Fibers ranging from 5 to 10 microns or more in length can also penetrate to the lower respiratory regions of the lungs, where they can have destructive effects.

In addition, asbestos fibers can fracture or split and break down into smaller diameter fibrils. Electron microscopy reveals that fibrils result from longitudinal and cross-sectional fragmentation of asbestos fibers. A single asbestos fiber can fracture into hundreds of submicroscopic fibrils. Research indicates that these uncoated fibrils might be the form that migrates into the peritoneal and pleural spaces.

- A significant proportion of inhaled asbestos fibers can be retained in the lungs.

The fibrous nature of asbestos renders the lungs' defense mechanisms ineffective. Smaller, nonfibrous particles to which the lungs are exposed are normally engulfed by macrophages and removed by lymphatic or mucociliary mechanisms. However, attempts by the macrophages to engulf fibers might not always be successful. One result is an eventual deposition in various tissues of ferrous material in a drumstick configuration called a ferruginous or asbestos body. The release of various chemicals and messengers by macrophages, as a result of the inability to engulf the fibers, is discussed below.

- The size and shape of asbestos fibers affect the lung's ability to effectively remove them.

The size of the fiber appears to play a role in its toxicity. According to Lippman (1990), asbestosis is most closely related to the number of fibers longer than about 2 micrometers (µm) and thicker than about 0.15 µm; mesothelioma to the number of fibers longer than about 5 mm and thinner than about 0.1 µm; and lung cancer to the number of fibers longer than about 10 µm and thicker than about 0.15 µm. Durability also plays a role in toxicity. Once inside the lungs, fibers can translocate along epithelium and ciliated epithelium, lymphatic drainage, or after ingestion by alveolar macrophages, if the fiber is short enough to be fully ingested.

Asbestos fibers can penetrate to the terminal bronchiolar level and enter the peribronchiolar space, resulting in a fibrogenic response. Because the fibers concentrate in the lower lung fields, there is a tendency for fibrosis to occur first in the lungs' bases, and for pleural effects to be confined to the lower two-thirds of the thorax. However, location is not diagnostic, because lesions can occur in all lung fields.

The mechanisms of fibrosis and carcinogenesis due to asbestos have been the target of much investigation. Fibrosis results from persistent release of inflammatory mediators such as lysozymes, interleukins, and fibroblast growth factors at the site of asbestos fiber penetration and deposition. It appears that fibers, because of a combination of physical/mechanical and chemical properties, stimulate cellular responses and enzyme secretions at critical target sites, leading to alterations in cell functions, differentiation patterns, quantities, and distributions. When the fibers are sufficiently durable in the lung, or at the pleura after translocation, the stimulation can continue for a sufficient length of time to produce chronic structural alteration and disease.

According to Mossman and Churg (1998), both inflammation and fibrosis, as well as expression of genes linked to cell proliferation and antioxidant defense, occur in a dose-related fashion after inhalation exposures to asbestos. Reactive oxygen species (ROS) and free radicals could also play a role. It appears that longer, more fibrogenic asbestos fibers cause a frustrated, ineffective phagocytosis and more protracted elevations in the release of ROS; activated inflammatory cells such as alveolar macrophages (AMs) might release increased amounts of oxidants. Oxidants generated by fibrogenic dusts such as asbestos might induce uptake of a variety of particle types, lipid peroxidation, stimulation of cell-signaling cascades and transcription factors, and release of cytokines such as tumor necrosis factor-alpha. These interrelated events are important in inflammation and fibrogenesis. A variety of cell types conventionally have been regarded as key participants in the inflammatory process: the AM, mast cell, T lymphocytes, and neutrophils.

Communication via elaboration of chemokines or cytokines by these cell types and their interactions with epithelial cells and fibroblasts may govern the eventual outcomes of cell injury and proliferation in response to pathogenic minerals.

Kamp and Weitzman (1999, 1997) hypothesize that free radicals activate signaling cascades and cause DNA damage that results in altered gene expression and cellular toxicity, which is important in the pathogenesis of asbestos-associated pulmonary diseases. The authors discuss the roles of ROS and reactive nitrogen species, apoptosis, and tumor promotion. The evidence shows that asbestos-induced free radical production is closely associated with the onset of DNA damage, signaling mechanisms, gene expression, mutagenicity, and apoptosis. The pathogenesis of asbestos-induced diseases probably derives from the long-term interplay between persistent free-radical production and the expression of cytokines, growth factors, and other inflammatory cell products.

It is likely that few asbestos fibers cross from the GI lumen into the blood, although several animal studies have revealed that asbestos fibers are capable of penetrating the GI tract. The risk of noncarcinogenic injury to tissues such as lung, heart, muscle, liver, kidney, skin, or eyes from GI absorption of asbestos should therefore be negligible (Agency for Toxic Substances and Disease Registry 2001). Another possible route of distribution of asbestos fibers in the body is inhalation exposure: Fibers that enter the lymphatics are presumably able to reach other tissues of the body. This is supported by the finding that people with high levels of asbestos fibers in the lung (measured as asbestos bodies) also had asbestos bodies in kidney, heart, liver, spleen, adrenals, pancreas, brain, prostate, and thyroid tissues. (This could also have been due to GI absorption following mucociliary clearance.) Data do not clearly relate GI tumors or peritoneal mesotheliomas to direct ingestion of asbestos fibers, although in occupational studies, workers exposed to asbestos by inhalation have been reported to have a twofold greater risk of colorectal cancer than unexposed workers. Some investigators believe this malignancy is caused by fibers removed from the lungs' upper respiratory regions by ciliary mechanisms and then swallowed. Asbestos bodies have been identified within some human specimens of colorectal adenocarcinomas.

Physiologic Effects

- Asbestos primarily affects the respiratory system. The immune and cardiovascular systems, and possibly the GI system, are also affected by asbestos exposure.

The respiratory, immunologic, cardiovascular, and GI systems might be adversely affected by asbestos inhalation and by ingestion of contaminated media or subsequent to mucociliary removal from the respiratory tract. Skin nodules (corns) from handling asbestos-containing materials can also occur.

No deaths due to acute exposure to asbestos have been reported, but even brief (<1 year) high exposures increase risk for future disease. Chronic inhalation exposure can cause death due to asbestosis and cancer.

The risk of developing asbestos-associated disease continues even after exposure has ceased. Fibrosis in the lung can lead to increased resistance to blood flow through the pulmonary capillary bed, resulting in pulmonary hypertension and compensatory hypertrophy of the right side of the heart.

Immunologic abnormalities, such as increased concentrations of autoantibodies and depressed lymphocyte responsiveness (Immunologic Effects section), are usually mild or absent in persons who have not developed clinical signs of asbestosis. Cardiovascular effects are secondary to pulmonary changes.

Respiratory Effects

- Asbestos exposure can result in asbestosis, mesothelioma, or carcinoma.

Inhalation of asbestos fibers can cause parenchymal (lung) asbestosis, pleural asbestosis (now termed "asbestos-related pleural abnormalities"), pleural mesothelioma, and lung carcinoma. All four syndromes can be present in a patient. Exposure to other carcinogens, dose, intensity and duration of exposure, individual susceptibility, and elapsed time since initial exposure (latency) all can play a role in disease development. Short-term high-level or chronic low-level asbestos exposure have been associated with lung cancer, mesothelioma, and pleural disorders; higher doses are more likely to produce parenchymal asbestosis. Even brief or relatively low exposures from work-related, household, and natural sources can induce pleural plaques or mesothelioma. In some circumstances, exposures in household members can approach occupational levels. One year of heavy exposure (e.g., manufacture of asbestos products, asbestos spraying, insulation work with asbestos materials, or demolition of old buildings) or 5 to 10 years of moderate exposure (e.g., construction or shipbuilding) could increase the lung cancer risk twofold or more. In some circumstances of extremely high asbestos exposure, a twofold increase of lung cancer can be achieved with exposure of <1 year (International Expert Meeting on Asbestos, Asbestosis, and Cancer 1997). Smoking and exposure to other toxicants increase the risk of asbestos-associated lung cancer.

Kamp and Weitzman (1997) report that there is general agreement that histologic or radiologic asbestosis is associated with a significant increase in the risk for lung cancer. However, disagreement exists as to whether asbestosis is simply a marker for high-dose exposure, or whether the interstitial fibrosis of asbestosis is the cancer-producing factor. It is also not necessary to have asbestosis to develop asbestos-related lung cancer.

According to Rosenberg (1997) and Kamp and Weitzman (1997), of workers certified as having asbestosis, about 20% died of pneumoconiosis, 39% died of asbestos-related lung cancer; 9% died from mesothelioma, and 32% died from other causes; 50% of the deaths occurred within 10 years after diagnosis.

Asbestosis

- Asbestosis is pulmonary fibrosis of the pleura or parenchymal interstitial tissue.

Inhalation of asbestos fibers can lead to a characteristic pneumoconiosis or diffuse interstitial fibrosis, termed asbestosis. Either heavy exposure for a short time or lower level exposure over a longer period may result in asbestosis; some cases have resulted from intense 1-day exposure. The disease can affect the lung parenchyma or pleural tissue. Clinical manifestations typically appear 20 to 40 years after onset of exposure; however, radiologic changes can occur in <20 years.

Parenchymal asbestosis is characterized as a lung disease involving a restrictive pattern, with obstructive features due to small airway disease, as well as gas exchange abnormalities. It is usually associated with higher exposure levels and radiograph changes, but mild fibrosis can occur at lower exposure levels, and pulmonary function changes can occur even without radiographic changes. Mossman and Churg (1998) feel that the development of asbestosis requires heavy exposure, possibly even involving a minimum threshold of about 25 to 100 fibers/mL/year. Latency is inversely proportional to exposure, and is now about 12.6 to 20.2 years; at lower doses, a longer latency would be expected. Smoking can worsen the result of asbestos exposure, possibly because of the increased particle retention (leading to decreased lung defenses) that takes place in smokers.

Asbestosis patients typically have elevated levels of antinuclear antibody and rheumatoid factors and a progressive decrease in total lymphocyte count with advancing fibrosis. Self-perpetuating host responses might affect the progression of fibrosis, even after exposure ceases. Fibroinflammatory patterns other than conventional asbestosis have also been described for workers with occupational exposure to asbestos. Differentiation of treatable diseases from asbestosis is very important. The differential diagnosis might include the collagen vascular diseases, radiation fibrosis, and rheumatoid arthritis.

- Pleural plaques have not been shown to be premalignant.

Pleural effects can occur even in the absence of parenchymal asbestosis. The incidence of pleural abnormalities in persons employed in asbestos-related occupations can be high (20% to 60%). Asbestos-related pleural abnormalities are found as pleural plaques, mainly involving the parietal pleura, sometimes with calcification; and diffuse pleural thickening, which is a collective name for pleural reactions involving mainly the visceral pleura. These abnormalities include benign asbestos-related pleural effusions, blunted costophrenic angle, crow’s feet or pleuroparenchymal fibrous strands, and rounded atelectasis (International Expert Meeting on Asbestos, Asbestosis, and Cancer 1997). Pleural plaques are oval areas of acellular collagen deposits, usually located bilaterally on the inferior and posterior surfaces of the pleura; they are usually asymptomatic and without clinically important findings (International Expert Meeting on Asbestos, Asbestosis, and Cancer 1997). Pleural plaques are not lung cancer precursors, although persons with pleural plaques have an increased incidence of lung cancer. Migration of inhaled asbestos to the pleura is the most likely cause of plaques.

In regions where plaques are not endemic, 80% to 90% of the plaques that are radiologically well defined are attributable to occupational asbestos exposures (International Expert Meeting on Asbestos, Asbestosis, and Cancer 1997).

Diffuse pleural fibrosis refers to noncircumscribed fibrous thickening of variable cellularity, usually found in the parietal, but mainly the visceral, layers. In occupational asbestos exposures, such diffuse fibrosis is probably a result of benign asbestos pleuritis with effusion; it might or might not be associated with rounded atelectasis. Diffuse pleural thickening, which is observed radiologically, can be associated with mild or, rarely moderate to severe restrictive pulmonary function deficits such as decreased ventilatory capacity (International Expert Meeting on Asbestos, Asbestosis, and Cancer 1997).

Mesothelioma

- Mesothelioma is a signal tumor for asbestos exposure and can appear after relatively low-level exposures.

Mesotheliomas are tumors arising from the thin serosal membranes that surround internal organs. The majority of mesotheliomas are due to asbestos exposure, although the National Cancer Institute (NCI) states that up to 30% have unknown causes. Pleural and peritoneal mesotheliomas are uncommon in the general, unexposed population. Although all asbestos types can cause mesothelioma, several studies have suggested that in humans the amphibole mineral form might be more likely to induce mesothelioma than the serpentine form. Evidence shows that peritoneal mesotheliomas are associated with higher levels of asbestos exposure than pleural mesotheliomas. Unlike asbestos-related bronchogenic cancer, mesothelioma risk does not appear to be influenced by smoking.

Mesothelioma can occur with low asbestos exposure; however, very low background environmental exposures carry only an extremely low risk. The dose necessary for effect appears to be lower for asbestos-induced mesothelioma than for pulmonary asbestosis or lung cancer. However, an extremely short exposure period might be sufficient to cause this rare tumor. A long latency period is typical—a minimum of 10 years from the first exposure is required to attribute the mesothelioma to asbestos exposure. Latency periods have been up to 57 years, although more intense exposures can result in latencies as short as 20 to 30 years. In most cases the latency interval is 30 to 40 years (International Expert Meeting on Asbestos, Asbestosis, and Cancer 1997). Some studies indicate that risk of mesothelioma from a given level of asbestos exposure depends primarily on elapsed time since exposure, with risk increasing dramatically after a lag period of about 10 years.

An estimated 1,500 cases of mesothelioma per year occur in the United States (compared with an average of 130,000 cases of lung cancer per year, mostly due to smoking). Data on death rates from pleural or peritoneal mesotheliomas over the past 10 to 20 years indicate that mesotheliomas are increasing in males over 65 years of age who have histories of occupational exposure to asbestos.

Rom (1998) states that the incidence of mesothelioma in the United States is increasing; the incidence of mesothelioma is peaking in their exposure-related mesothelioma incidence from 1940 to 1970. In the United Kingdom, where imports of asbestos peaked in the 1960s and 1970s, a peak in mesothelioma deaths is expected in 2020.

Lung Cancer

- Latency for lung cancer is 10 to 30 years or more.

There is little doubt that all types of asbestos can cause lung cancer. A latency period of 10 to 30 years or more exists between the onset of asbestos exposure and occurrence of the tumor. Whether asbestos exposure will lead to lung cancer depends not only on cumulative exposure, but also on other underlying lung cancer risks. The incidence of lung cancer from all causes is high in the general population, so asbestos as a causative factor is difficult to prove in an individual patient. The presence of asbestosis is an indicator of high exposure, but lung cancer can occur in its absence as well. Pleural plaques occur at lower levels of asbestos exposure, and diffuse pleural thickening occurs at moderate to high levels of exposure.

- It is unclear whether a threshold asbestos dose exists for lung cancer.

All four major histologic types of lung cancer (squamous cell carcinoma, adenocarcinoma, large-cell carcinoma, and small-cell carcinoma) are associated with asbestos exposure. Although asbestos-associated lung cancer tends to occur in the lower lung fields, histologic type and anatomic location are of no help in determining whether the tumor is due to asbestos. As stated previously, even 1 year of heavy exposure or 5 to 10 years of moderate exposure can increase lung cancer risk twofold or more. The relative risk of lung cancer is estimated to increase 0.5% to 4% for each fiber per cubic centimeter per year (fiber-years) of cumulative exposure (International Expert Meeting on Asbestos, Asbestosis, and Cancer 1997). At very low levels of asbestos exposure, the risk of lung cancer appears to be undetectably low (International Expert Meeting on Asbestos, Asbestosis, and Cancer 1997).

Other Carcinogenic Effects

- Increased incidence of GI cancers has been reported among asbestos workers.

Some mortality studies of asbestos workers have revealed small increases in the incidence of death from cancer at one or more extrathoracic sites, including the larynx, the kidneys and the GI system—notably the esophagus, stomach, colon, and rectum. Presumably, these cancers (other than the larynx) are due to swallowing asbestos fibers.

- The consequences of ingesting asbestos fibers are a subject of controversy.

In contrast, other epidemiologic studies have not detected statistically significant associations between asbestos ingestion and extrathoracic cancers. Various researchers and regulatory groups have reviewed the weight of evidence and have not been able to reach a consensus on the effects of ingested asbestos fibers.

Whether GI neoplasms can be induced by ingesting asbestos-contaminated drinking water (or other ingestion sources) remains controversial. In humans, asbestos bodies have been identified in extrapulmonary tissues including tonsils, thoracic and abdominal lymph nodes, pleura, peritoneum, liver, spleen, kidneys, adrenals, small intestine, pancreas, and bone marrow, as well as the lungs. In any case, oral exposure to asbestos should be avoided.

Cardiovascular Effects

- Cardiovascular effects are secondary to pulmonary fibrosis.

Fibrosis of the lung can lead to increased resistance to blood flow through the capillary bed, resulting in cor pulmonale. This condition can also occur with less severe fibrotic disease, especially if chronic obstructive lung disease is simultaneously present, as commonly seen in cigarette-smoking asbestos workers. Pulmonary hypertension can occur before decreased respiratory function is clinically detectable. Limited data from case reports suggest that constrictive pericarditis due to fibrous thickening can also result from asbestos exposure (Agency for Toxic Substances and Disease Registry 2001).

Immunologic Effects

- Immunologic abnormalities have been noted in persons with asbestosis.

Immunologic abnormalities have been observed in asbestos workers with clinical signs of asbestosis and have also been reported in environmentally exposed persons. Despite some variability, most studies indicate that cell-mediated immunity can be depressed in workers who have radiologic evidence of asbestosis. Autoantibodies (rheumatoid factor, antinuclear antibodies) are typically present in these workers. Caplan syndrome (the coexistence of pneumoconiosis with rheumatoid changes) also has been noted in asbestos workers, although it is more common in coal miners and workers with other pneumoconiosis. The implications of these immunologic changes are difficult to assess, but they are of special concern because depressed immune function might be a factor in the etiology of asbestos-induced cancer.

Challenge

> *(4) Is the mesothelioma of the patient's cousin likely to be related to his school custodial work? Explain.*
>
> *(5) How will you address the mother's concern about future health risks for her son?*

Clinical Evaluation

History and Physical Examination

The medical evaluation of persons exposed to asbestos should include a thorough medical and occupational history, physical examination, chest radiograph, and pulmonary function tests. The same protocol has been recommended for evaluating an asymptomatic patient with a history of asbestos exposure. If indicated, more specialized radiologic and laboratory testing such as high-resolution computerized (axial) tomography scan (HRCT), bronchoalveolar lavage (BAL), or lung biopsy might be helpful. Pertinent historical information includes the source, intensity and duration of exposure, time elapsed since first exposure, and work history of household members. Asbestos accumulates in the body, and even relatively minor exposures can be important. Workplace dust measurements or estimates, and a cumulative fiber dose, as expressed in fiber-years per cubic centimeter, are important parameters of asbestos exposure (International Expert Meeting on Asbestos, Asbestosis, and Cancer 1997). Smoking history is clearly important.

- Dry bibasilar rales, auscultated in the mid-axillary line, are the most common lung findings associated with asbestosis.

The physical examination should focus primarily on the patient's lungs, and particular attention should be paid to pulmonary auscultation. Fine inspiratory rales in the posterior and posterolateral lung bases, audible on deep inspiration, might be the earliest sign of interstitial fibrosis. These basilar crackles are characteristic in their sound ("fine," "cellophane," "Velcro," or "close to the ear") and occur in a bilateral basilar distribution. There is pan-inspiratory or end-inspiratory accentuation. The basilar crackles start at the bases at the midaxillary lines, spread to the posterior bases and, as disease progresses, to higher levels up from the bases. They can be difficult to distinguish from congestive heart failure (CHF) rales, but are distinct from bronchitis. The differential diagnosis can be difficult when CHF, chronic obstructive pulmonary disease, or other chronic lung disease is present, as these may be unrelated to asbestos exposure but might present similar symptoms. Generally, a chest radiograph is more sensitive than auscultation in asbestos-related disease.

Examination should also assess stigmata of other diseases that might confound the diagnosis of asbestosis. For instance, rheumatoid arthritis is sometimes associated with interstitial fibrosis. Chest-wall configuration, evidence of thoracic surgery, and cardiac status can also alter the differential diagnosis.

Signs and Symptoms

Asbestosis

- Significant clinical syndromes include asbestosis, lung cancer, and mesothelioma.

Asbestosis can manifest as pleural or parenchymal fibrosis or both. Pleural asbestosis, more properly termed "asbestos-related pleural abnormalities," is the most common finding in asbestos-induced pulmonary disease and, as described previously, involves pleural thickening,

often manifested as discrete pleural plaques. Pleural plaques can be seen radiologically as bilateral images of hyalin scar formation on either the visceral or, much more commonly, the parietal pleural surfaces. The specificity of pleural plaques is low on radiographs unless the plaques are well defined. The most common differential diagnosis is subpleural fat. Well-defined asbestos-related pleural plaques on radiographs include bilateral circumscribed plaques, bilateral calcification, and diaphragmatic plaques. Pleural plaques rarely cause symptoms. Diffuse pleural fibrosis, seen as visceral pleural thickening, can be associated with mild or, rarely, moderate or severe restrictive pulmonary defects, with dyspnea and restrictive changes on pulmonary function tests. There can be a benign pleural effusion.

- Progressive dyspnea on exertion is a common symptom of asbestosis.

A patient with parenchymal asbestosis commonly develops fatigue, weight loss, and insidious onset of dyspnea on exertion. As the disease progresses, the dyspnea worsens, regardless of any further asbestos exposure. A dry cough typically occurs, but a productive cough, even in a nonsmoker, is not uncommon. Patients often describe a “tight” feeling in the chest. Common findings are bibasilar fine end-inspiratory crackles (32% to 64%) and clubbing of the fingers (32% to 42%) (which occurs at a later stage of the disease). In the advanced stages of the disease, signs of cor pulmonale are common. Functional disturbances can include gas exchange abnormalities (e.g., diffusing capacity), a restrictive pattern, and obstructive features due to small airway disease (International Expert Meeting on Asbestos, Asbestosis, and Cancer 1997). The interstitial disease is radiographically demonstrated as a reticular fibrosis located predominantly in the lower lung fields. Radiologic evidence is often not present until at least 5 years after exposure. The American Thoracic Society states that there is convincing evidence that an asbestos-related pulmonary abnormality can occur in the absence of definite radiologic change (American Thoracic Society 1986). The detection of asbestosis by standard films (chest radiography) should be guided by standard reading methods such as those of the International Labour Organization (ILO) classification system and read by certified B readers trained to use this classification system. Early changes not seen on chest radiography can be found using HRCT in selected cases.

Fibrosis found symmetrically in the lower aspects of both lungs is typically caused by asbestos. Fibrotic lung disease due to asbestos inhalation is often associated with pleural plaque formation, which eliminates other etiologic possibilities such as drugs; radiation; sarcoidosis; collagen vascular disorders; Goodpasture syndrome; hemosiderosis; idiopathic pulmonary fibrosis secondary to lung infections; and inhaled silica, coal dust, or organic dusts. Smoking effects should be considered in the evaluation of early asbestosis, lung function tests, and respiratory symptoms. Health-care practitioners should be alert for a differential diagnosis of treatable fibrotic diseases, where intervention may be of benefit.

Lung Cancer

- Asbestos-associated lung cancers produce the same symptoms as cancers due to other etiologies.

Lung cancer caused by asbestos exposure cannot be differentiated from cancer caused by other environmental factors. The histologic type of lung cancer and its anatomic location are of no significant value in deciding whether or not an individual lung cancer is attributable to asbestos. Clinical signs and symptoms of asbestos-related cancer do not differ from those of lung cancer of other causes (International Expert Meeting on Asbestos, Asbestosis, and Cancer 1997). The differential diagnosis of lung cancer in a patient exposed to asbestos should include other possible etiologies, such as exposure to cigarette smoke, arsenic, chloromethyl ethers, chromium, nickel, and ionizing radiation. Clubbing of the distal phalanges or cyanosis of the nail beds can occur.

Mesothelioma

- The latency period for mesothelioma is 20 years or more, but the onset of symptoms is sudden.

Both pleural and peritoneal mesotheliomas can be seen in a patient exposed to asbestos, although peritoneal mesotheliomas are very rare. These tumors are rapidly invasive locally. Although onset of mesothelioma is not sudden, symptoms of the disease can be. Mesothelioma frequently presents with pleural effusion, dyspnea, and chest pain. Less common presenting symptoms are cough, weight loss, and fever. Peritoneal mesotheliomas are more difficult to diagnose by noninvasive means than pleural occurrences are. They are frequently detectable by abdominal palpation as an expanding “doughy” feeling. Early diagnosis is essential to maximize potential for successful intervention. If caught early and treated, there is a greater chance of survival. Pleural effusion can precede the mesothelioma; if pleural effusion is detected, the patient should be evaluated aggressively. Mesothelioma is seldom associated with etiologies other than asbestos exposure.

Laboratory Tests and Special Procedures

- Chest radiograph and pulmonary function tests are important procedures in diagnosing asbestos-associated disease.

Established tests and procedures helpful in diagnosing asbestos-associated disease include radiographic techniques, pulmonary function tests, and possibly computerized tomography scanning. Neither sputum studies nor blood chemistry studies are useful in diagnosing asbestos-associated disease in the clinical setting.

Quantification of the amount and type of asbestos fibers and asbestos bodies in lung tissue or bronchoalveolar lavage fluid or both might be useful in individual patients, where indicated, to determine the level of past exposure to asbestos, and aid in evaluation of differential diagnosis.

Histopathologic confirmation is required for suspected asbestos-related malignancies.

Radiographic Techniques

- Radiographic results should not be used preferentially in diagnosing asbestosis.

The chest radiograph is the basic tool for assessing asbestos-associated parenchymal and pleural disease. Radiographic findings may include interstitial fibrosis in the lower lung fields and thickening of both the parietal and visceral lung pleura. Parietal pleural thickening generally appears as a lobulated prominence of the pleura adjacent to the thoracic margin. Visceral pleural thickening is generally more diffuse and appears as interlobar fissure thickening on lateral films. Further, according to Kamp and Weitzman (1997), the chest radiograph in cases of asbestosis usually reveals small parenchymal opacities with a nodular or reticular pattern or both. The interstitial process characteristically begins in the lower lung zones and is associated with bilateral midzone parietal pleural plaques. In the early stages, combined interstitial and pleural involvement can cause a hazy, ground-glasslike appearance that blurs the heart border ("shaggy heart" sign) and the diaphragm on the chest radiograph. The pleural thickening might entrap the lung parenchyma and form a benign pleural-based mass (rounded atelectasis) that mimics bronchogenic carcinoma. Honeycombing and upper lobe involvement generally do not develop until advanced stages of asbestosis. Hilar and mediastinal lymphadenopathy are not typically present. Pulmonary effusion can be present. A system has been developed by ILO for radiographic rating of the changes in pneumoconiosis. Persons certified to use this rating system are referred to as "B readers."

The diagnosis of asbestosis should be made in the context of the overall clinical presentation and should include, but not emphasize, radiographic findings. The association of pleural thickening and calcification enhances diagnostic accuracy. Although open lung biopsy is a definitive diagnostic test for asbestosis, it is rarely used in the clinical setting.

The radiologic appearance of asbestos-induced lung cancer does not differ from that of other cancers. Asbestos-related malignancies predominantly involve the lower portion of the lungs, but they are not restricted to this location.

Computed Tomography and Other Imaging Techniques

- CT scanning is expensive, but can be helpful in individual cases.

Computed tomography (CT) and HRCT can facilitate the detection of asbestosis, asbestos-related pleural abnormalities, and asbestos-related malignancies. CT and HRCT are particularly sensitive and specific means of differentiating asbestos-related pleural plaques from soft-tissue densities.

These two imaging techniques can be invaluable when used for specific indications in individual clinical evaluations. The cost-effectiveness and long-term efficacy of using these imaging techniques as screening tools has not been established.

New imaging techniques, such as digital radiography, are under development. The utility of other current techniques, such as ultrasound, gallium scanning, magnetic resonance imaging, ventilation-perfusion studies, or positron-emission tomography, are not yet established for asbestos-related disorders.

Pulmonary Function Testing

- Small airway disease and restrictive defects are typical in nonsmoking patients with asbestosis; a combined obstructive/restrictive pattern is more typical in smokers.

Nonsmoking patients with asbestosis typically have spirometric changes that are indicative of small airway disease and restrictive defects; smokers with asbestosis might have a combined obstructive/restrictive pattern. Decreased diffusion (carbon monoxide diffusion capacity) might be expected if fibrosis is present. Small airway disease is a common early finding and is reflected in a 25% to 74% reduction of forced expiratory flow rates. This might reflect either inflammatory changes or early fibrosis in the peribronchiolar areas. Restrictive defects are observed as a reduction in FVC. Because such reduction might also occur in obstructive airway disease, an apparent combined pattern of restrictive and obstructive disease on spirometry should be followed up with further pulmonary studies including carbon monoxide diffusion capacity and static lung volumes. True restrictive disease generally manifests as a decrease in total lung capacity with normal or less residual volume, which can be determined using both the plethysmographic and helium dilution methods. Consider consulting a pulmonologist as needed.

A reduction in the vital capacity (< 88% predicted) was noted in 27% of insulation workers with a “normal” chest radiograph, and was detected as early as 5 to 9 years after exposure (Kamp and Weitzman 1997).

Sputum Studies

- Sputum studies are not useful for most patients, but might be useful as a diagnostic test for neoplasia and lung cancer.

Sputum inspection for asbestos fibers or ferruginous bodies has been advised, but most investigators now agree that the lack of sensitivity and specificity contraindicates their use for screening. BAL may be useful in individual patients. Sputum cytology also remains useful as a diagnostic test for neoplasia and lung cancer.

Other Tests

Recent studies suggest that lymphocyte (particularly T cell) abnormalities correlate with both asbestos-related malignancies and asbestosis. However, because these findings are in the early investigative stage, they are not clinically useful.

No blood test is useful for diagnosing asbestos-associated diseases. However, a patient with asbestos-related disease should be evaluated for immunologic abnormalities.

A stool hemoccult test should also be considered.

Attribution

For the purposes of diagnosis, differential diagnosis, or attribution to asbestos of potential asbestos-related disorders, the diagnostic methods described above should be used. Laboratory confirmation of significant asbestos exposure and diagnosis of other asbestos-related disorders in the same person aid in attribution of findings to asbestos. Kamp and Weitzman (1997) state that histopathologic evaluation is not necessary for compensation purposes. An ad hoc committee of the Scientific Assembly on Environmental and Occupational Health concluded that in the absence of lung tissue, a clinical diagnosis of asbestosis is established by 1) a reliable exposure history, 2) an appropriate latency period, 3) a characteristic chest radiograph, 4) reduced lung volumes and/or diffusing capacity for carbon monoxide (the mnemonic DL_{CO}), and 5) end-inspiratory crackles (Murphy et al. 1986). The quantity of asbestos bodies and uncoated fibers in the lungs correlates with the severity of fibrosis and is generally 10- to 20-fold higher in patients with asbestosis, compared with normal individuals. The number of asbestos bodies or fibers in lung tissue necessary for the diagnosis is not clear.

Challenge

> *(6) Is the father (50 years old) of the patient described in the case study at risk for asbestos-associated disease? If so, what medical evaluation should be undertaken?*

Treatment and Management

According to Mossman and Churg (1998), regulation of occupational exposures to minerals and removal of symptomatic persons from the workplace are important measures for prevention or amelioration. However, there has been little advancement in effective therapeutic strategies for patients. Ideopathic pulmonary fibrosis, silicosis, and asbestosis have traditionally been treated with corticosteroids or immunosuppressants, with discouraging results in terms of morbidity and mortality. Recent work has focused on the mechanisms of mineral-induced inflammation and fibrosis to develop novel treatments. Recent studies have addressed: administration of antioxidants or iron chelators, inhibition of tumor necrosis factor and interleukin factor-1, inhibition of phospholipases, and modification of mineral surface properties.

Follow-up of asymptomatic patients exposed to asbestos is recommended to facilitate early diagnosis and intervention. Periodic pulmonary function studies can be helpful in diagnosing early signs of asbestosis.

- Patient education is an important factor in managing asbestos-associated diseases.

Management of asbestos-associated diseases begins with patient education regarding smoking cessation and avoidance of pulmonary infections. Awareness of early symptoms of other neoplasms, including hoarseness, sores in the mouth, blood in the urine, blood in the stool, and GI symptoms, is important. Persons exposed to asbestos should be advised of the increased risk for lung cancer and the synergistic effects of cigarette smoking, although smoking does not affect the development of mesothelioma. In general, explaining environmentally related cancer risk is difficult because extrapolation of risk from workplace data to environmental exposures is difficult or impossible for many substances. Maintaining a balance between appropriate concern and avoidance of undue alarm is the goal.

Asbestosis

- Asbestosis patients should avoid pulmonary irritants and guard against lung infections.

Asbestosis is an irreversible pulmonary condition. Respiratory infections should be treated aggressively because they often prove fatal in patients with advanced fibrotic lung disease. Patients should be strongly advised to avoid all pulmonary irritants, including cigarette smoke. Influenza and pneumococcal vaccines highly recommended. In the later stages, pulmonary rehabilitation might be helpful. The patient should be advised to consult a physician when the first signs and symptoms of respiratory infection occur, so that early treatment can be instituted.

Although most investigators consider the pleural plaques associated with asbestosis to be benign, they can result in pulmonary impairment. Patients with pleural asbestosis are also more likely to have or develop parenchymal asbestosis and should be appropriately monitored. Patients should be informed that pleural plaques represent evidence of significant asbestos exposure.

Mesothelioma

- Patients with mesothelioma have a 1-year survival rate of <30%.

The prognosis in this disease is difficult to assess consistently because there is great variability in the time before diagnosis and the rate of disease progression. However, the prognosis for patients with mesothelioma has traditionally been poor; they seldom live longer than 12 to 18 months after diagnosis. The 1-year survival rate of mesothelioma patients is <30%. Some indications show that early diagnosis and multimodal or new therapies might have an impact on survival. Among specialists at major cancer centers, statistics have shown some improvement: 5-year survival has approached 40% in selected patients. Clinical trials are also ongoing and might be useful for selected patients. (The National Cancer Institute Web page [www.nci.nih.gov] EXIT can provide more details.)

Health-care providers should vigilantly monitor patients at risk for mesothelioma to find it as early as possible, especially when pleural effusion is present, and should consider consulting a specialist as indicated.

Lung Cancer

- Treatment of asbestos-associated cancer does not differ from treatment for cancers due to other causes.

Treatment of asbestos-associated cancer should include appropriate combinations of surgery, chemotherapy, and radiation, according to accepted surgical and oncologic standards.

Challenge

> *(7) If examination of the father of the child described in the case study is entirely normal except for bilateral pleural plaques, what follow-up will you recommend?*
>
> *(8) As a concerned family physician, you are identified as a community resource on asbestos exposure and invited to speak at a Parent-Teacher Association meeting. What will you tell your audience?*

Standards and Regulations

Workplace

- The Occupational Safety and Health Administration (OSHA) standard for asbestos in the workplace is 0.1 fibers/cc of air as an 8-hour TWA.

Widespread evidence of asbestos-associated disease in workers was found in the 1930s. A standard for exposure was not established in this country until 1960, but only in selected industries. In 1971, the standard was extended industry-wide. A 1968 British study judged that exposure to 2 fibers per cubic centimeter of air (fibers/cc) for the duration of a person's work life would result in an approximate 1% risk for asbestosis. This was an underestimation, but nonetheless led to the establishment of the 1976 U.S. standard of 2 fibers/cc as a time-weighted average (TWA). Further study of carcinogenicity resulted in the OSHA standard of 0.2 fibers/cc (8-hour TWA) that became effective in 1986. The level at which employers must take action to reduce employee exposure (termed "action level") is 0.1 fibers/cc (8-hour TWA).

Environment

- The EPA maximum contaminant level (MCL) for asbestos in drinking water is 7 million fibers per liter of water.

The difficulties of controlling asbestos exposure in the workplace are paralleled in the general environment. EPA recommends “no visible emissions.” In 1973, EPA banned spraying of asbestos in building interiors. No regulations for asbestos in potable water exist. The EPA proposed MCL for asbestos in drinking water is 7 million fibers (>10 microns in length) per liter of water.

The Asbestos in Schools Identification and Notification Act of 1982 requires that local education agencies inspect for friable material, analyze these materials for asbestos content, post results and notify parents and employees if asbestos is found, and maintain appropriate records. A recent study indicating that power-buffing and power-stripping of asbestos-tile floors in schools produces significant airborne-asbestos levels prompted an EPA warning to school communities. Floor maintenance will henceforth be performed by hand to prevent the release of fibers.

To protect both themselves and the environment, asbestos remediation workers should be trained to handle asbestos properly.

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Answers to Pretest and Challenge Questions

Pretest

(a) The patient's symptoms are unlikely to be related to asbestos exposure. The patient's afterschool activity occurred for a period of 3 years, which is much less than the typical latency period for asbestos-associated diseases. Asbestos levels measured in the general indoor air in schools also tend to be well below the OSHA permissible workplace level. A more likely cause of the boy's symptoms would be onset of bronchial asthma.

(b) The patient's potential exposure could place him at risk for future asbestos-related complications. Even low-level environmental asbestos exposures can eventually result in disease.

(c) The cousin's mesothelioma is unlikely to be related to his 3-year history of school custodial work; however, a number of cases of mesothelioma in long-term school custodians have been documented. In several recent studies, school custodians were also found to have asbestotic chest radiographs. Exposure to airborne asbestos while working as a longshoreman is the more likely cause of the cousin's disease.

Challenge

(1) The patient might be exposed to low levels of asbestos at home, school, and play. Asbestos materials adequately contained and not airborne are not likely to be a significant hazard, but asbestos does tend to be liberated from aging materials such as wall and ceiling insulation or pipe and duct coverings. Asbestos-containing materials aggressively abraded can also release fibers: power-buffing of asbestos-containing floor tiles is an example. The father's occupation suggests the patient and family could be receiving secondary asbestos exposure from dust brought home on his father's work clothes and person.

(2) Yes. Workers exposed to asbestos can bring fibers home on their clothes, skin, and hair, inadvertently exposing others in the household.

(3) If the pipe coverings are visibly in good condition and air sampling indicates no release of fibers, it is probably safer to leave them intact. Application of a substance to encapsulate the intact asbestos can be considered. If the pipe coverings are deteriorating, however, the family should seek professional advice from a qualified and licensed contractor specializing in asbestos abatement.

(4) See (c) in the Pretest answers above.

(5) For the child described in the case study, the physician should clearly state that the child's symptoms are not likely to be attributable to asbestos, but should not minimize the possible long-term risks of asbestosis or cancer. The synergistic effects of smoking and exposure to other carcinogens should be discussed.

If either or both parents smoke cigarettes, the child might be more likely to become a smoker himself, and thereby increase his risk of asbestos-related lung cancer. In addition, parental smoking could expose the child to secondhand smoke. The MD should encourage parents to quit smoking.

(6) Yes, the father might be at increased risk for asbestos-related disease. Homes built before 1975 were typically constructed with asbestos-containing products. Removing or repairing these materials could liberate asbestos fibers that might be inhaled if appropriate respiratory protection is not worn.
A thorough medical and occupational history; a physical examination, including auscultation of the heart and lungs; chest radiograph; and spirometry to assess possible restrictive or obstructive pulmonary disease or both might be indicated. Stool hemoccult testing is also advised.

(7) It would be prudent to have periodic evaluations including chest radiograph, pulmonary function testing, and yearly screening for colorectal cancer.

(8) Parents often feel resentful that they were not informed earlier of an asbestos hazard. A respected physician in the community is often able to put the risk of disease due to asbestos into perspective for such an audience. Before making public statements, however, it would be advisable to consult with state and local public health officials on the potential for asbestos exposure in local schools.

Sources of Information

More information on the adverse effects of asbestos and the treatment and management of persons exposed to asbestos can be obtained from ATSDR, your state and local health departments, and university medical centers. For clinical inquiries, contact ATSDR, Division of Health Education and Promotion, Office of the Director, at 404-498-0101

Asbestos Toxicity Examination

Select the best answer to each of the following items. Mark your responses on the Answer Form.

1) The most heavily exposed people in the United States are those in the construction trades.

   a) True
   b) False

2) Secondary exposure occurs when fibers released to the air are inhaled by persons not directly handling asbestos. People in contact with work clothes of asbestos workers or with asbestos-containing household products have developed pleural abnormalities. An asbestosis prevalence of 11% in wives, 8% in sons, and 2% in daughters was reported in families of asbestos-exposed shipyard workers.

   a) True
   b) False

3) Low exposures from work-related, household, and natural sources can induce_____. For diffuse, pleural thickening, higher exposure levels might be required.

   a) blackened moles
   b) pleural plaques
   c) large welts
   d) None of the above

4) Cigarette smoke increases the risk of asbestos-associated lung cancer.

   a) True
   b) False

5) There are two major classes of asbestos: serpentine, which contains a magnesium silicate called chrysotile, and amphiboles, which represent a small portion of the world's commercial asbestos use and include _____.

   a) crocidolite
   b) amosite
   c) tremolite
   d) All of the above

6) Asbestos has been used in >_____ products because of its high tensile strength, relative resistance to acid and temperature, and varying textures and degrees of flexibility.

   a) 575
   b) 1,200
   c) 3,000
   d) None of the above

7) Asbestos can also be a contaminant in other products such as vermiculite, which is used in _____.

   a) gardens
   b) home insulation
   c) talc, which is used in cosmetics
   d) All of the above

8) Drinking-water supplies might become contaminated with asbestos from _____.

   a) erosion of natural land sources
   b) asbestos cement pipe
   c) discarded mine and mill tailings
   d) All of the above

9) Asbestos fibers act differently from most types of inhaled particles in terms of the aerodynamics of reaching the depths of the lung. For most inhaled non-asbestos particles, generally only particles between 0.5 and 5 microns in diameter with a length-to-width ratio of 3:1 will be deposited in the respiratory regions (alveoli and terminal bronchioles) of the lungs.

   a) True
   b) False

10) No deaths due to acute exposure to asbestos have been reported, but even brief (<1 year) high exposures increase risk for future disease. Chronic inhalation exposure can cause death due to asbestosis and cancer. The risk of developing asbestos-associated disease continues even after exposure has ceased.

   a) True
   b) False