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Asbestos-Related Pleuropulmonary Disease

An APCCMPD Ambulatory-Care Curriculum Teaching Script

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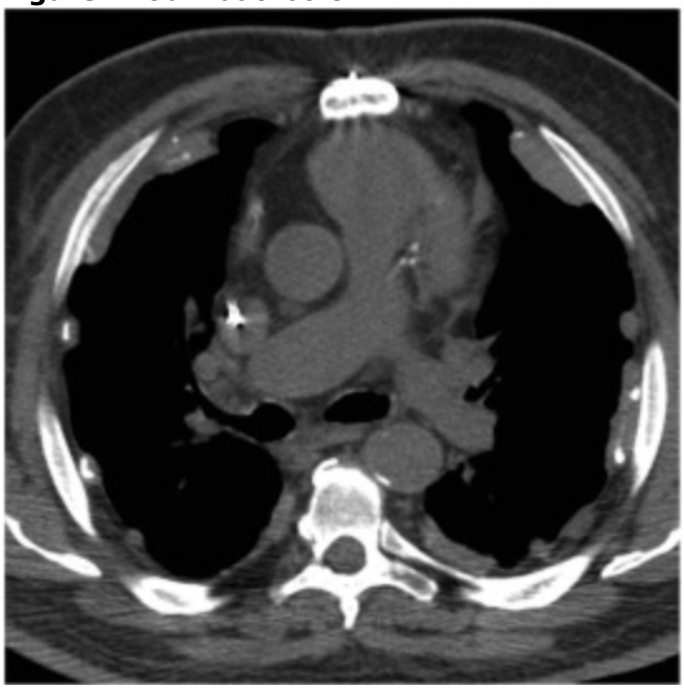
Asbestos-Related Pleuropulmonary Disease

Scenario 1:

A 65-year-old male with a history of tobacco use (45 pack-years, current smoker), undocumented chronic obstructive pulmonary disease (COPD) (has recent breathing problems, started on inhalers by primary care physician, no spirometry has been done), and hypertension presented to your office for evaluation of an abnormal chest CT scan. His primary care physician performed chest imaging as a part of a lung cancer screening program. He was told to follow up with a pulmonologist for this computed tomography (CT) abnormality to discuss next steps in management. He may have had chest x-rays in the past but doesn't remember the details. He recently retired after working as a subcontractor for the last 45 years. He mostly did weld and cut concrete without respirator use. His CT is shown below in Figure 1.

Figure 1. Our Patient's CT



Representative slice of patients CT chest.
From Lee, 2023.

Question 1: What is asbestos?

Question 2: What aspects of the occupational history would make you suspicious for possible asbestos-related lung disease?

Question 3: What are the subtypes of asbestos-related lung disease?

Question 4: What are the characteristic imaging findings of each type of asbestos-related process*?*

Question 5: What are the typical clinical presentations of patients with asbestos-related disease?

Question 6: What diagnostic studies should be considered in someone being evaluated for asbestos-related lung disease?

Question 7: What other pulmonary processes are in the differential diagnosis for patients who may have asbestos-related pulmonary disease?

Question 8: How would you manage a patient that you have diagnosed with asbestosis?

Asbestos-Related Pleuropulmonary Disease

Educational Objectives

1. Identify risk factors for asbestos exposure
2. Define the various subtypes of asbestos related lung disease
3. Discuss the components of an appropriate evaluation and follow up for patients with known or suspected asbestos related lung disease

Scenario 1:

A 65-year-old male with a history of tobacco use (45 pack-years, current smoker), undocumented chronic obstructive pulmonary disease (COPD) (has recent breathing problems, started on inhalers by primary care physician, no spirometry has been done), and hypertension presented to your office for evaluation of an abnormal chest CT scan. His primary care physician performed chest imaging as a part of a lung cancer screening program. He was told to follow up with a pulmonologist for this computed tomography (CT) abnormality to discuss next steps in management. He may have had chest x-rays in the past but doesn't remember the details. He recently retired after working as a subcontractor for the last 45 years. He mostly did welding and cut concrete without respirator use. His CT is shown below in Figure 1.

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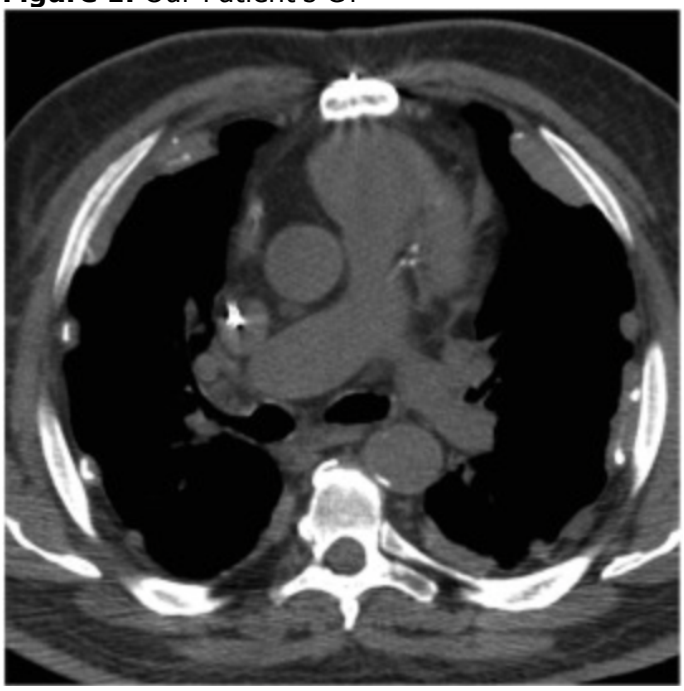


Representative slice of patients CT chest.
From Lee, 2023.

Question 1: What is asbestos?

"Asbestos" refers to a group of minerals containing hydrated magnesium silicates that form fibers. These minerals naturally occur throughout the world and the fibers are used industrially for their resistance to fire, acid and electricity with 1.2 million metric tons mined in 2024 (Flanagan, 2025). When these fibers are disturbed, they break into microscopic fibrils which can be inhaled deep into the lungs. The most common form of asbestos (~95% of US consumption) is called chrysotile (chemical formula $Mg_3(Si_2O_5)(OH)_4$), and is in the serpentine category with fibers arranged into sheets. Five other forms of asbestos are in the amphibole category, with straight needle-like fibrils. About one third of the world's countries have completely banned the use of any asbestos products. In the United States, asbestos regulation began in the 1970s, and although it is still permitted for some uses its import was banned in 2024. The major types of asbestos fibers are summarized in Table 1.

Table 1. Types of Asbestos

Mineral: Group and Form	Location of Major Deposits, Commercial or Other	Main Commercial Uses and/or Other Sources of Human Exposure
ASBESTOS MINERALS		
Serpentine		
Chrysotile[†] (white asbestos)	Canada (Quebec, British Columbia, Yukon, Newfoundland[‡]), Russia, China (Szechwan), Brazil, Mediterranean countries (Cyprus, Corsica, Greece, Italy), southern Africa (South Africa, Zimbabwe, Swaziland)	Brake lining, shipbuilding and repair, polishing of precious stones, stone cutting, whetstone cutting, foundry operations (mainly for insulation) Asbestos cement products (pipes, gutters, tiles, roofing); insulation, fireproofing, reinforced plastics (fan blades, electric switchgear); textiles; friction materials; paper products; filters, spray-on products
Amphibole		
Crocidolite (blue asbestos)	South Africa (Northwest Cape[‡]), western Australia[‡]	Used in combination, mostly in cement but also in some of the products listed above
Amosite (brown asbestos)	South Africa (Northern Province, former Transvaal) Finland[‡]	
Anthophyllite		Filler in rubber and plastics
Tremolite	Contaminates ore in certain asbestos, talc, iron, and vermiculite mines; also found in some agricultural soils	May or may not be removed in processing; has rural domestic uses (e.g., stucco)
Cummington-grunerite	Contaminates ore in certain iron mines (often not fibrous)	No commercial use

Adapted from Murray and Nadal (2015).

Question 2: What aspects of the occupational history would make you suspicious for possible asbestos-related lung disease?

Asbestos exposure may occur via asbestos mining, other occupational exposure, or outside of work (such as handling soiled work clothes, living near a geological source where asbestos is disturbed, or living near a facility that processes asbestos). Work involving maintenance or demolition of structures containing asbestos materials are at risk as well (see Table 2). Asbestos use in the United States has been very restricted since the 1970s-1980s by federal and state regulation, though its use is still permitted in some products such as brake pads. There is no significant health risk associated with occupying a building containing asbestos that is adequately maintained and undisturbed (i.e. not respirable). Internationally, bans on the commercial use of asbestos are common. However, mining of asbestos continues to occur in countries including Russia, China, Brazil, and Kazakhstan. Occupations associated with asbestos exposure are listed in Table 2.

Table 2. Professions at Risk of Asbestos Exposure

Occupations	***Industries***
Plumbers	Construction
Pipefitters	Shipbuilding and repairing
Steamfitters	Chemicals and other manufacturing
Electricians	Nonmetallic mineral stone products
Insulation workers	Railways
Carpenters	Yarn, thread, and fabric mills
Laborers	Trucking
Boilermakers	Plastic and rubber manufacturing
Welders and cutters	
Janitors	

Adapted from Wagner, 1997

There is also a significant environmental risk for people living close to facilities where asbestos is mined and used. For example, more than 200 people in Libby, Montana (about 10% of the town's population) died from asbestos related lung disease after exposure from a vermiculite mine contaminated by tremolite asbestos (Lee, 1998).

There is a long latency time from asbestos exposure to detection of asbestosis or mesothelioma, with an estimated range of about 10-40 years. Epidemiological studies have also demonstrated a dose response.

In the US, current OSHA (Occupational Safety and Health Administration) standards specify that employers should ensure no employee is exposed to an airborne concentration of asbestos greater than 0.1 fiber per cubic centimeter of air in an 8-hour time-weighted average (OSHA, 2025). They also specify that medical surveillance should occur in employees exposed to asbestos above the

permissible exposure limit, including at least annual medical and work histories and questionnaires, physical examination, CXR, pulmonary function tests, and any other evaluation deemed necessary by the examining physician.

Question 3: What are the subtypes of asbestos-related lung disease?

Pleural Plaques

- Pleural plaques, as demonstrated in Figure 2, are the most common manifestation of prior asbestos exposure, occurring in more than half of exposed individuals and considered virtually pathognomonic for asbestos exposure. They represent radiographic or histologic findings and, in isolation, do not cause pulmonary symptoms. Pleural plaques most commonly arise from the parietal pleura, particularly along the posterolateral chest wall, diaphragm, and mediastinal pleura. In contrast, involvement of the visceral pleura is less common and, when present, is more frequently associated with other asbestos-related conditions such as asbestosis or diffuse pleural thickening. There is a significant lag time between asbestos exposure and the development of plaques, as long as 20-30 years from first exposure. It is believed that sub-pleural lymphatics carry the inhaled microscopic asbestos fibers to the pleura where these remain. They are then internalized by mesothelial cells, which leads to fibrosis and hyaline membrane production (Hillerdal, 1980). Histological evaluation may demonstrate asbestos fibers within pleural plaques; however, their presence is not required for diagnosis. For patients with equivalent asbestos exposure, presence or absence of plaques does not correlate with risk of developing malignancy.

Diffuse Pleural Thickening (DPT)

- Although DPT and pleural plaques share similar names and a benign nature, they are distinct disease processes with different causes. DPT is a broader disease not limited to asbestos exposure, arising from the visceral pleura and often manifesting a few years after exposure to asbestos (Miles, 2005). Unlike pleural plaques, DPT is believed to result from fibrosis due to chronic pleural irritation rather than direct fiber internalization. In cases related to asbestos, it may follow the development of benign pleural effusion or respond to reactive oxygen and nitrogen species and inflammatory cytokines triggered by asbestos fibers. DPT is diagnosed radiographically when the pleura is measured to be >3 mm thick for 8 cm in the craniocaudal dimension and 5 cm in the lateral or anterior/posterior dimension, as demonstrated in Figure 3 (Lee, 2023). While DPT can lead to restrictive physiology and severe symptoms, the overall prognosis is generally favorable. Decortication may be considered in severe cases, but mortality from DPT alone is rare, typically occurring only in isolated cases without associated asbestosis or malignancy.

Rounded Atelectasis

- In cases of significant parietal pleural scarring, such as in pleural plaques and DPT, adjacent alveoli are prone to collapse in a pattern extending from the pleura. Unlike typical plate-like atelectasis, rounded atelectasis is by definition always adjacent to the pleura. Vessels and bronchi entering the region of atelectasis appear curved and converge toward the lesion, forming the characteristic "comet tail" sign that is typical for this condition (Lee, 2023). These findings are demonstrated in Figure 4. The collapse itself is rarely symptomatic, and when accompanied by other evidence of pleural disease should not be cause for alarm. However, if detected as a solitary lesion, further investigation is warranted to rule out an underlying peri-pleural malignancy.

Benign Asbestos Pleural Effusion (BAPE)

- Approximately 3-7% of asbestos-exposed workers develop BAPE, (Peacock, 2000) a potentially underestimated condition due to its asymptomatic nature and often found incidentally (see Figure 5). These effusions frequently spontaneously resolve in a few months, but some cases may persist or recur over several years. While predominantly unilateral, BAPE can manifest bilaterally or switch sides during resolution and recurrence. It may manifest earlier than other asbestos-related findings, such as within 10 years of asbestos exposure. The effusion is typically exudative, blood-tinged, and may be eosinophilic (Adelman,1984). Asbestos fibers are uncommonly found in the effusion but may be present in the underlying lung parenchyma. Diagnosis is based on consistent occupational history and exclusion of other causes including malignancy.

Asbestosis (Fibrosis, ILD)

- Not all asbestos-related pleuropulmonary disease is asbestosis. Asbestosis refers to interstitial lung disease and pulmonary fibrosis caused by inhalation of asbestos fibers. Fibrosis can be mild or can progress to diffuse pulmonary fibrosis. Radiologically, asbestosis presents with a HRCT (High-Resolution CT) pattern of UIP (Usual Interstitial Pneumonitis). Histologically, the fibrosis typically starts with a predominantly subpleural distribution similar to UIP but differs in that fibroblastic foci are less common in asbestosis. Asbestosis often demonstrates mild fibrosis of the visceral pleura, which is not seen in UIP. Figure 6 demonstrates the typical HRCT findings of Asbestosis. Asbestos fibers (also called ferruginous bodies, see Figure 7) may be present on histological examination if iron staining is used, and their presence support the diagnosis of asbestosis.

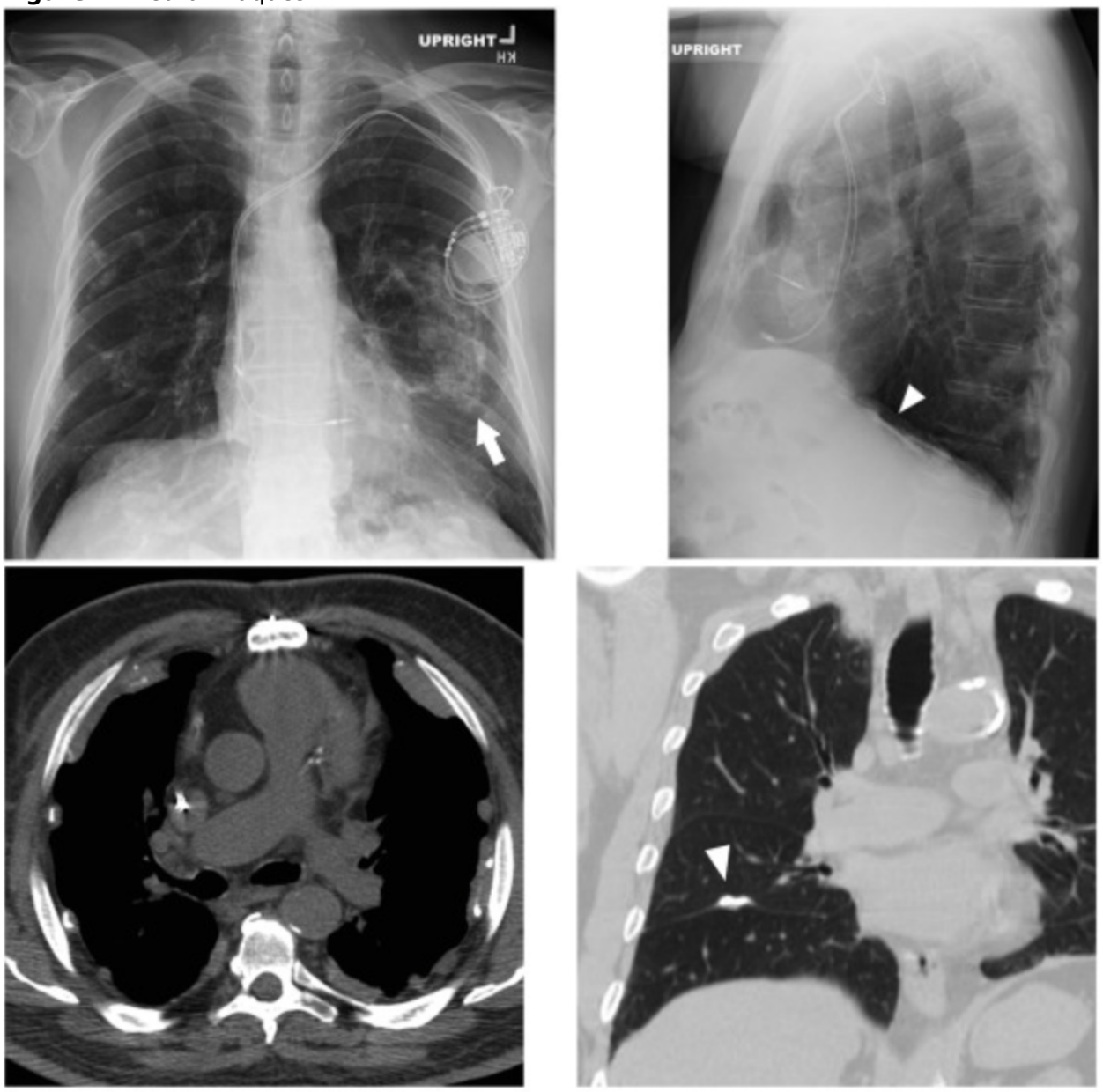
What about cancer?

- Asbestos exposure is linked to increased risk of lung cancer and mesothelioma. The risk of malignancy is dose-dependent and synergistic with smoking: compared to nonsmokers without asbestos exposure, smokers with asbestos exposure have 8-fold higher odds of developing lung cancer while nonsmokers with asbestos exposure have 1.7-fold higher odds (Lee, 1998).
- While mesothelioma is famously associated with asbestos exposure, the most common cancer associated with asbestos exposure is lung cancer (i.e. bronchogenic carcinoma). The subtypes of lung cancer associated with asbestos exposure are similar to the overall distribution of lung cancer subtypes.
- Mesothelioma should be suspected in patients with relevant exposures who exhibit diffuse pleural thickening or nodularity, especially with associated symptoms such as pleuritic pain and weight loss. Diagnosis should be made with thoracoscopy and pleural sampling with immunohistochemical investigation, not on sampling of pleural fluid alone due to high risk of error. No screening for mesothelioma is recommended.

Question 4: What are the characteristic imaging findings of each type of asbestos-related process*?*

Representative imaging of pleural plaques is shown in Figure 2.

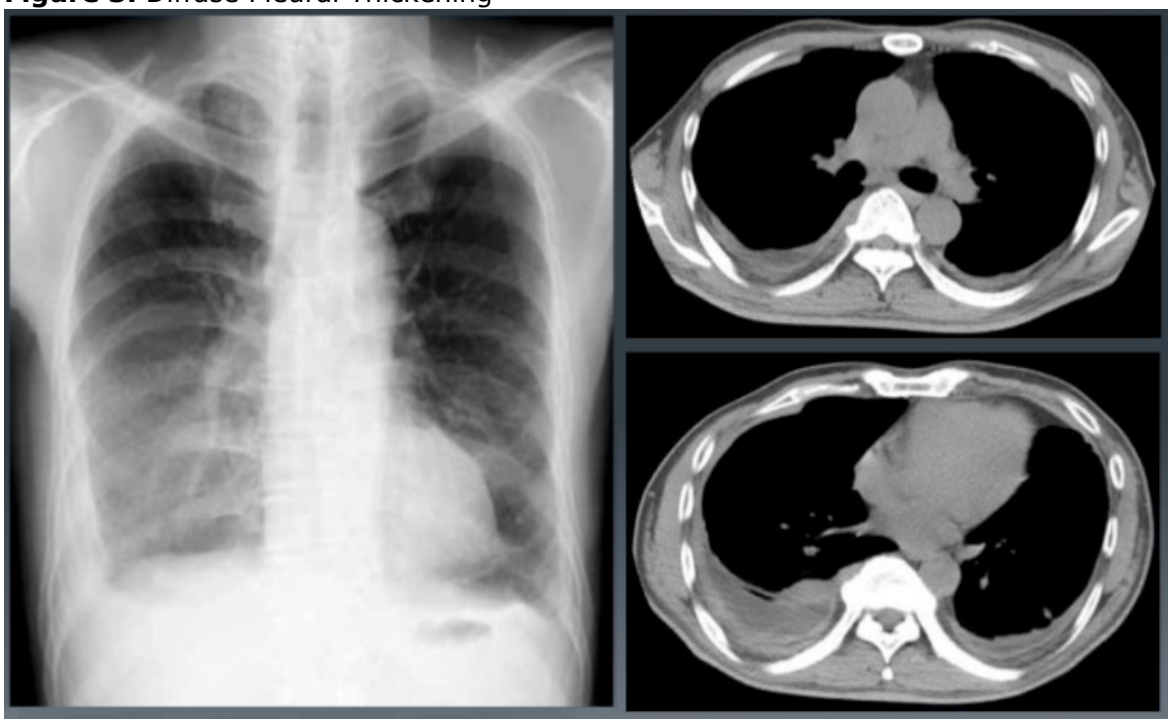
Figure 2. Pleural Plaques



Plain radiograph and CT imaging of pleural plaques. (Lee, 2023)
Image credit, Lee, 2023.

Diffuse pleural thickening is illustrated in Figure 3.

Figure 3. Diffuse Pleural Thickening



Radiographic and CT images of patients with diffuse pleural thickening.
Adapted from Kato, 2015.

Classic imaging features of rounded atelectasis is shown in Figure 4.

Figure 4. Rounded Atelectasis

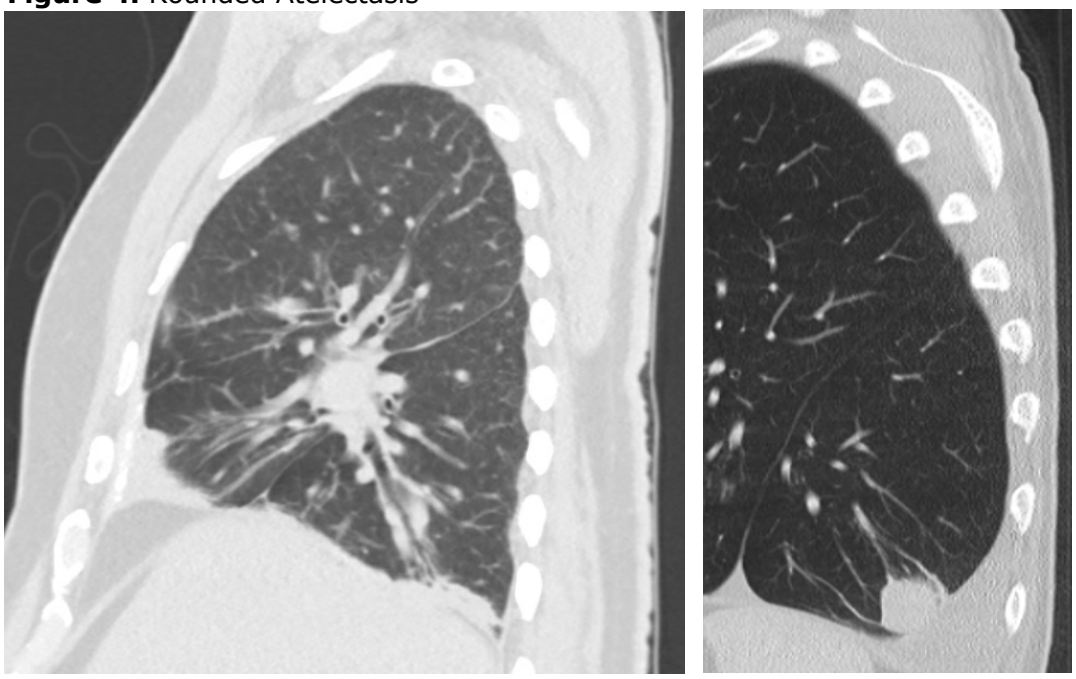
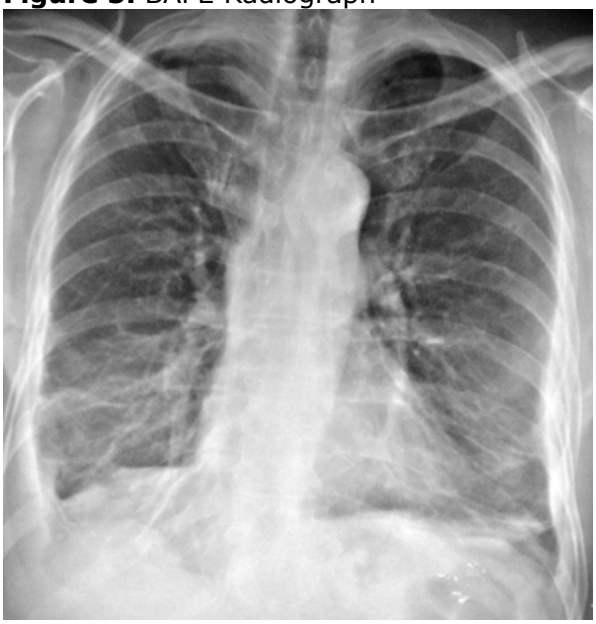


Image from Radiopedia (Weerakkody, 2025)

Radiographic finding of BAPE is shown in Figure 5.

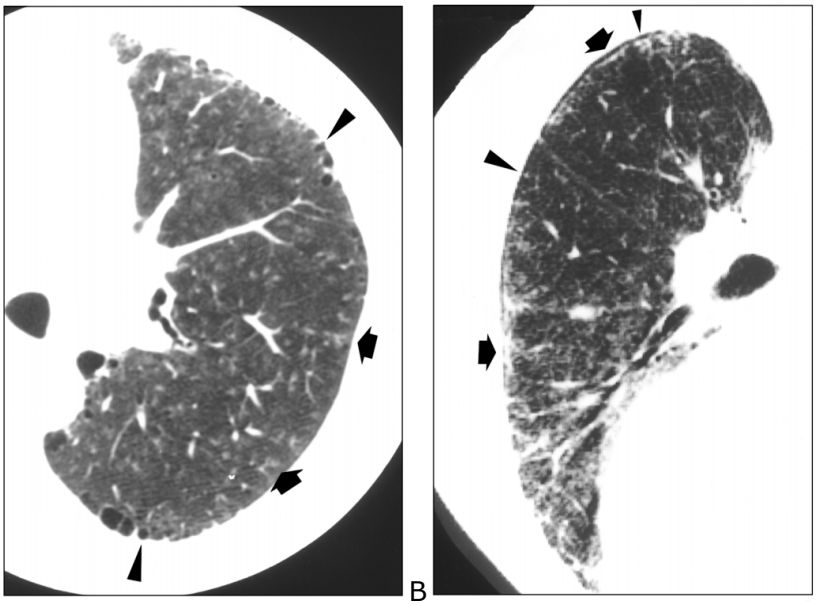
Figure 5. BAPE Radiograph



BAPE on plain radiograph.
From UpToDate (King, 2024)

High-resolution CT findings of asbestosis are shown in Figure 6 (A & B).

Figure 6. High-resolution CT Scans from Patients with Asbestosis



A B

A. Arrows point to subpleural dotlike or branching opacities located a few millimeter away from pleura; arrowheads point to paraseptal emphysema.
B. Prone images demonstrates subpleural lines parallel to inner chest wall(arrows) and subpleural dot-like opacities (arrowheads).
Borrowed from Akira, 2003.

Question 5: What are the typical clinical presentations of patients with asbestos-related disease?

Certain asbestos-related pleuropulmonary diseases such as pleural plaque, pleural thickening, and BAPE are often asymptomatic. In contrast, asbestosis often presents with nonspecific respiratory symptoms including progressive dyspnea on exertion, chest pain, and nonproductive cough. Sputum production and wheezing are less common and may be associated with concomitant COPD or a history of tobacco use. Physical exam may reveal bibasilar crackles and digital clubbing. Severe cases may be associated with hypoxemia or pulmonary hypertension and right heart failure. Evaluation should include full PFTs, which characteristically show a restrictive pattern with decreased TLC and FVC, decreased DLCO (Lazarus, 2011), and no obstruction (though there is some controversy about whether a mixed obstructive-restrictive pattern may sometimes be seen even in never smokers).

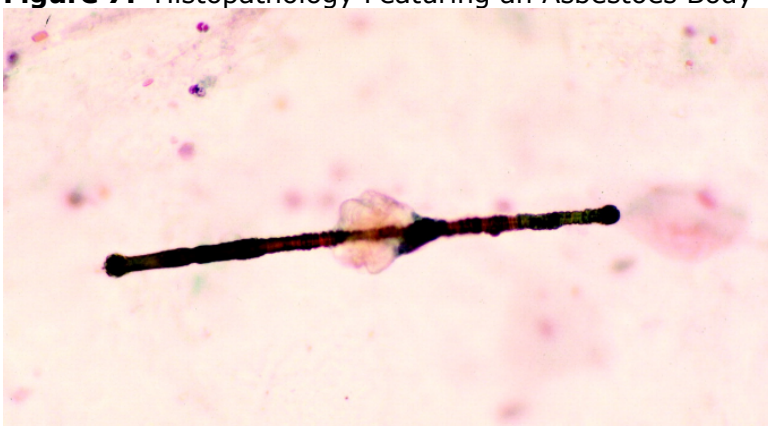
Question 6: What diagnostic studies should be considered in someone being evaluated for asbestos-related lung disease?

No specific testing is needed for incidentally discovered pleural plaques. The imaging findings are pathognomonic with an appropriate clinical history. The plaques do not cause symptoms. Patients with either more concerning or non-diagnostic plain films (as may be seen in milder disease) should be referred for CT imaging to further evaluate the pleura and lung parenchyma (Al Jarad, 1991). In patients with asbestos-related lung disease, baseline PFTs are recommended as significant restrictive physiology may warrant referral for decortication or other pleural procedures.

Patients with BAPE often undergo thoracentesis as effusions precede other lung findings. Other etiologies of effusions should be ruled out, regardless of a convincing occupational history. It is rare to find asbestos fibers in the pleural fluid, although they may be present. Increased fluid eosinophils, combined with an appropriate history, is usually adequate for diagnosing asbestos-related disease.

During bronchoscopy with bronchoalveolar lavage or thoracentesis, asbestos fibers may be seen in the pleural fluid or BAL (Broncho Alveolar Lavage). In parenchymal lung biopsy, asbestos bodies (fibers encased in collagen and iron-rich material) may be present in patients with asbestosis. These bodies are absent in pleural plaques. An Asbestos body on Histopathology is shown in Figure 7.

Figure 7. Histopathology Featuring an Asbestoes Body



An asbestos body as may be seen on histopathology.
Adapted from ATS guideline on management of asbestos-related diseases (American Journal of Critical Care Medicine [AJRCCM], 2003).

Question 7: What other pulmonary processes are in the differential diagnosis for patients who may have asbestos-related pulmonary disease?

Pleural plaques are pathognomonic for asbestos exposure, and BAPE can be diagnosed relatively confidently based on history and pleural fluid analysis.

Especially if not accompanied by pleural plaques, asbestosis can be challenging to differentiate from other conditions, with IPF being a primary radiologic mimic, as both will display a UIP pattern on CT scan. The main differentiator will be the clinical history of exposure, and the presence of other asbestos-specific findings such as pleural plaques or effusion.

Silicosis may also be an asbestosis mimic (Roach, 2002). As noted previously, the main motif in asbestos fibers is the silicate molecule. Both conditions share similar pathophysiology involving phagocytosis of particles by alveolar macrophages leading to inflammation, collagen synthesis, and fibrosis upon macrophage apoptosis. The overlap in occupational exposure to asbestos and silica dust further complicates diagnosis. Silicosis will be more likely to involve hilar nodes, have upper lung zone predominance on HRCT, exhibit less pleural involvement, and may exhibit egg-shell calcification. Ultimately, treatment (or prevention of further progression) is effectively the same.

Other interstitial diseases such as hypersensitivity pneumonitis, sarcoidosis, and radiation pneumonitis may be confused with asbestosis on imaging. Table 3 outlines specific radiographic characteristics aiding differentiation, complementing a comprehensive patient history for a conclusive diagnosis.

Table 3. Radiographic Mimics of Asbestosis

Causes of honeycombing	Comments
Idiopathic pulmonary fibrosis	Distribution predominantly bibasilar and subpleural
Collagen tissue disease	Honeycombing mainly seen in rheumatoid arthritis-ILD
Chronic hypersensitivity pneumonitis	Honeycombing can be seen predominantly in the upper/middle zones. Clues to diagnosis: mosaic attenuation, air trapping in expiratory scans
Asbestosis	Distribution predominantly bibasilar and subpleural. Clues to diagnosis: pleural plaques (±calcification), subpleural lines
NSIP	When seen, honeycombing is minimal. Clues to diagnosis: subpleural sparing, central/peribronchovascular predominance of findings
Sarcoidosis	In rare cases, the distribution is bibasilar and subpleural. Typically, honeycombing is seen in upper/middle zones extending from the periphery toward the hilum. Clues to diagnosis: perilymphatic nodules, hilar/mediastinal lymphadenopathy (±calcification), progressive massive fibrosis
Radiation	Confined to radiation port
End-stage ARDS	Usually involves anterior lung (barotrauma)

Adapted from Tzilas, 2017

Question 8: How would you manage a patient that you have diagnosed with asbestosis?

Asbestosis lacks a disease-specific treatment. Patients with asbestosis should be counseled about its occupational nature, expectations for progression, and their cancer risk in the context of asbestos exposure history and other risk factors. For progressive fibrosis, treatment with nintedanib and referral for lung transplantation can be discussed. Routine preventive care such as vaccinations and supportive care such as pulmonary rehabilitation and oxygen therapy for those with hypoxemia should be recommended.

At this time, there are no USPSTF guidelines regarding lung cancer screening related to asbestos exposure, however some feel that asbestos exposure should warrant regular lung cancer screening. Patients should be counseled to stop smoking.

When a diagnosis of asbestosis is established, patients should be counseled about potential legal avenues for compensation and may be advised to seek legal counsel. Table 4 shows 2004 ATS recommendations for management after a diagnosis of asbestosis.

Table 4. Management of Asbestosis

Recommendations for Management after Diagnosis of Asbestosis
1. Patient notification • Inform patient of work-related illness • Report to appropriate authority as occupational disease, as required by law • Inform patient that there are options for compensation
2. Impairment assessment • Conduct an assessment of functional impairment • Rate impairment in accordance with ATS criteria, which are incorporated into the AMA Guides
3. Tertiary prevention • Smoking cessation (primary prevention for smoking-related disorders) • Withdrawal from further excessive exposure • Immunization (pneumococcal pneumonia, influenza) • Management of concurrent respiratory and other diseases
4. Monitoring • Chest film and pulmonary function testing should be conducted every 3 to 5 years • Active monitoring (periodic screening for colon cancer) • Observation and elevated index of suspicion but not screening for lung cancer, mesothelioma, gastrointestinal cancers (other than colon)
5. Development of a patient-specific management plan for symptomatic disease

Adapted from ATS 2004 guidelines.

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