Spectrum of Asbestosis and other Asbestos related diseases— from a Radiological viewpoint

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Overview

Patient Presentation

Defining the disease

Spectrum of disease and disease characteristics

Radiological Menu of Tests

Patient follow-up

References

Acknowledgements
Our Patient: Presentation

Our patient is an 85 yr old Male with ARF, CHF s/p CABG, who was referred for evaluation of CHF exacerbation.

He presented with a history of mild cough and fever, and gradually progressive shortness of breath on exertion since the past 4-5 years, and increased O₂ requirements.

However, his imaging studies showed a chronic pulmonary process which was separate from his acute findings.
Our Patient: History and Review of Systems

Patient has shortness of breath on walking for about more than 15 minutes at a stretch (Grade 2). It develops gradually and is progressive with more exertion.

Shortness of breath is relieved when the patient rests.

Patient has mild cough as mentioned before, with minimal mucoid expectoration for the past 3 years.
Our Patient : Past Medical History

CHF
CAD
Aortic Stenosis s/p CABG and TAVI procedure
HTN

Our Patient : Medications

Albuterol 90 mcg inhaled prn
Ipratropium 0.5 mg nebulization 4 times/day
Tiotropium bromide 18mcg inhaled 1 capsule/day
Furosemide 40 mg PO daily
Metoprolol 50 mg PO daily
Our Patient: Occupational History

History of working in the Insulation section of a construction company for about 40 years (i.e. known long-term heavy exposure).

Retired at the age of 55.
Our Patient: Social History

Smoking 1 ppd from the age of 40, quit about 15 years ago
Alcohol consumption, about 2 cans of beer/week
No known tuberculosis exposure
No history of travel outside the USA

Our Patient: Family History
Both parents died of MI, exact age unknown.
No known family history of lung disease or any lung cancers.
Our Patient : Physical Examination

Vitals are all stable. No dyspnea at rest.

Patient gets dyspnea on exertion after walking on level ground for more than 15 minutes (Grade 2).

No other significant findings.
Our Patient: Spirometry

Spirometry showed:
FVC = 2.56L
FEV1 = 2.05L
Decreased lung compliance

FEV1/FVC = 80.2% (which is a normal value and indicates an underlying restrictive lung disease, as opposed to an obstructive etiology).
Our Patient: Asbestos Exposure on Frontal Chest Radiograph

- Linear calcifications along the diaphragm
- Pleural plaques
- Furled leaf appearance of pleural plaque

PACS, BIDMC
Our Patient: Asbestos Exposure on Lateral Chest Radiograph

Linear calcifications along the diaphragm

PACS, BIDMC
Asbestosis

Asbestosis is pneumoconiosis caused by inhalation of asbestos fibers.

The disease is characterized by slowly progressive, diffuse pulmonary fibrosis.
What is Asbestos?

Asbestos is the general term for a group of naturally occurring fibers made up of hydrated magnesium silicates. Due to its fire resistance, tensile strength and chemical properties, Asbestos is widely used for a range of construction and insulation applications.
Types of Asbestos fibers

Two types, based on shape:

1. **Serpentine** fibers, of which **Chrysotile** is the only type used widely, are long, curly strands.
   - more fibrogenic
   - less toxic than the amphibole fibers.

2. **Amphibole** fibers (crocidolite, amosite, tremolite, etc) are long, straight, rod-like structures.
   - more carcinogenic.

90 percent of the asbestos in commercial use in the USA is of Chrysolite variety.
Pathogenesis

2 mechanisms :-

i) Direct toxic effects of the fibers on pleura and pulmonary parenchymal cells,

ii) Release of various mediators (ROS, proteases, cytokines, and growth factors) by inflammatory cells.

Larger asbestos fibers (> 5 microns) settle in the upper airways and are cleared by mucociliary action.

However, smaller fibers settle in the lower airways where they enter lung parenchymal cells directly, and cause fibrosis.

Free radicals react with and damage cellular macromolecules and disrupt DNA to give rise to lung cancer.

Smoking interferes with mucociliary clearance.
Sources of exposure (Risk Factors):

1. **Mining** of the fibers

2. **Industrial applications** of asbestos (eg. work with textiles, plumbing, cement, pipe-fitting, insulation, shipbuilding)

3. **Nonoccupational exposure:**
   i) regular exposure to asbestos-soiled work clothes of an asbestos worker,
   ii) renovation or demolition of asbestos-containing buildings,
   iii) environmental exposure from an industrial neighborhood.

**Smoking** has been shown to be synergistic for increased risk of lung cancer.
Spectrum of disease:

The spectrum of pulmonary diseases associated with asbestos exposure includes the following conditions:

1. **Pleural disease** (focal and diffuse benign pleural plaques)
   - Asbestos exposure

2. **Asbestosis**

3. **Malignancies**:
   - i) Bronchogenic Carcinoma (Small cell and non-small cell carcinoma of the lung)
   - ii) Malignant mesothelioma
Asbestosis vs Asbestos exposure

Asbestos exposure refers to pleural fibrosis or plaquing, but absence of Interstitial lung disease.

Asbestosis specifically refers to interstitial (parenchymal) fibrosis from asbestos.

Differentiation is important, as Asbestos exposure is covered under health insurance, but not Asbestosis.
Clinical Manifestations

Signs and symptoms typically develop after an asymptomatic period of about 20-30 years after the beginning of exposure.

However, the pleura is more sensitive to asbestos-related disease process than the parenchyma, and as a result, pleural disease often present earlier, such as BAPE (benign asbestos pleural effusions)
Signs and Symptoms

1. Classical presentation is an insidious onset of dyspnea on exertion. The DOE is progressive and continues even in the absence of further asbestos exposure.

2. Cough and wheezing may also be present, but are more of a consequence of concomitant smoking.

3. Bilateral basal end-inspiratory crackles (in 50% pts)

4. Finger clubbing (in 30% pts)

5. Symptoms of Cor pulmonale in advanced cases.
Diagnosis:

3 key findings that support a diagnosis of asbestosis¹:

1. A **reliable history** of asbestos exposure with latency period of >20 years, And/Or:
   Presence of **markers of exposure** (eg, pleural plaques are virtually pathognomonic of previous exposure, or recovery of sufficient quantities of asbestos fibers/bodies in BAL or lung tissue).

2. Definite evidence of **interstitial fibrosis**, manifested by:
   - end-inspiratory crackles on chest examination;
   - reduced lung volumes and/or DLCO;
   - typical chest radiograph or HRCT findings of ILD;
   - histologic evidence of interstitial fibrosis.

3. **Absence of other** causes of diffuse parenchymal lung disease.

¹ **American Thoracic Society.** Diagnosis and initial management of nonmalignant diseases related to asbestos. Am J Respir Crit Care Med 2004; 170:691.
Complications

1. **Respiratory failure** (Cigarette smoking contributes synergistically)
2. **Malignant mesothelioma**
3. **Bronchogenic carcinoma** (Asbestos exposure and smoking act synergistically and the risk is multiplicative)
4. **Cor pulmonale**
Management
There is currently no cure or specific treatment for asbestosis. The disease once occurred is irreversible.

Treatment measures are mainly preventive:

1. Prevention of further asbestos exposure (Change of occupation).

2. Smoking cessation

3. Early detection of lung abnormalities and complications by Imaging and PFTs.

4. Supplemental oxygen when there is increased requirement, either resting or post exercise.

5. Immediate treatment of respiratory infections.

6. Pneumococcal and influenza vaccinations.
Radiologic Menu of Tests:

1. Chest X ray
   70% sensitivity in detecting interstitial pathology

2. CT Scan
   More sensitive and specific than CXR (97% sens., 100% spec.)
   Currently the Gold Standard for diagnosis of asbestos related disease

3. Fluorine-18 FDG PET / Gallium lung scanning
   Shows increased uptake of marker in tumor cells in bronchogenic CA and malignant mesothelioma)
   (for distinguishing from benign pleural disease)¹

Now, we shall see the commonly seen imaging findings in Asbestos related disease in our patient and some other companion patients…
Our Patient: Asbestos Exposure on Frontal Chest Radiograph

- Linear calcifications along the diaphragm
- Pleural plaques
- Furled leaf appearance of pleural plaques
Our Patient: Asbestos Exposure on Lateral Chest Radiograph

Linear calcifications along the diaphragm

PACS, BIDMC
Our Patient: Asbestosis Exposure on Chest CT Scan Scout Film

Linear calcifications along the diaphragm
Asbestosis: Presence of interstitial disease

The interstitial disease process usually starts in the lower lung zones. Bilateral mid-lung zone plaques are seen on the parietal pleura.

In the early stages of asbestosis, combined interstitial and pleural involvement causes a hazy, "ground glass" appearance on CXR.

This causes blurring of the diaphragm and heart border, giving rise to the "shaggy heart" sign.
Companion Patient #1: Basilar Opacities on CXR

Irregular opacities on the lower lung fields:

Source: Am J Respir Crit Care Med vol 170 pp 692, 2004; www.atsjournals.org
BAPE (benign asbestos pleural effusion)

BAPEs are usually small and unilateral.

They occur years before the onset of interstitial disease, usually within the first 15 years from initial exposure.

One of the early signs of underlying asbestos disease.

Typically resolve spontaneously after several weeks, but may leave residual blunting of the CP angle or thickening of the visceral pleura.
Our Patient: Benign Asbestos Pleural Effusion on CXR

BAPE
(Benign asbestos pleural effusion)
Pleural Plaques

Benign pleural plaques are the hallmark of asbestos exposure. (They are uncommon in other interstitial lung diseases)

Seen in 50% of persons exposed to asbestos. Usually seen >20 years after initial exposure.

 Preferentially involve the parietal pleura adjacent to ribs. Less extensive in the intercostal spaces. Only rarely occur on the visceral pleura.

They are not seen in the region of the costophrenic sulci and at the apices of the lungs.

Usually asymptomatic.
Our Patient: Pleural Plaques on CXR
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Pleural Plaques
Our Patient: Calcified pleural plaques on CT Scan
Our Patient: Calcified pleural plaques on CT Scan
Fibrotic bands superimposed on a background of widespread irregular opacities

Obscuring of the heart border by fibrosis:
“Shaggy heart” border

Blunted costophrenic angles.
Companion Patient #3 : Honeycomb Lung on CT Scan

Honeycombing and upper lobe involvement develop in advanced stages of disease (severe disease)

Source: American Academy of Family Physicians
Round atelectasis

Inflammatory reaction & fibrosis in pleura → fibrous tissue contracts → pleura folds into lung → atelectasis of a part of the lung (rounded atelectasis)

Usually associated with pleural thickening and volume loss.
Companion Patient #4: Rounded Atelectasis on CXR

Source: Dr. Paul Stark, MD. UpToDate.com
http://www.uptodate.com/contents/image?imageKey=PULM%2F82006~PULM%2F78745&topicKey=PULM%2F4307&rank=1~61&source=see_link&search=asbestosis
Asbestosis

Small bilateral parenchymal opacities (fibrosis) with a multinodular or reticular pattern; Pleural fibrosis may or may not be present. Depends on dose and duration of exposure. Usually develops >20-30 yrs. Fibrosis spreads from resp. bronchioles & alveolar ducts. Lower lobes & subpleural → middle lobe & lingula. Advanced disease: Upper lobe involvement and honeycombing.
CXR Features of Asbestosis

Hazy ground-glass opacities

Shaggy cardiac silhouette

Ill-defined diaphragmatic contours

Rounded atelectasis

Small nodules

50% cases show coexisting pleural involvement
Companion Patient #5: Pleural plaques and Interstitial fibrosis on CXR

Interstitial fibrosis (Small bilateral parenchymal opacities with a multinodular or reticular pattern)

Shaggy cardiac silhouette

Pleural plaques

Source: Dr. Roberto Schubert. Available at http://radiopaedia.org/images/1827647
HRCT for Asbestosis

Much more sensitive and specific than CXR.

Subpleural linear densities parallel to the pleura.

Basilar and dorsal lung parenchymal fibrosis, with septal fibrosis all over.

Coarse parenchymal bands (2 to 5 cm in length), which are contiguous with the pleura.

Coarse honeycombing in advanced disease.

Pleural plaques, which differentiate asbestosis from other ILDs.
Companion Patient #6: Parenchymal Fibrosis and Honeycombing on CT Scan

- Bibasilar parenchymal fibrosis
- Cystic honeycomb appearance
- Calcified pleural plaques

Source: Dr. Paul Stark, MD. UpToDate.com
http://www.uptodate.com/contents/image?imageKey=PULM%2F77568&topicKey=PULM%2F4307&rank=1~61&source=see_link&search=asbestosis
Complication: Mesothelioma

Most common primary neoplasm of the pleura

Usually caused by Amphibole fibers

Seen >30 years post initial exposure

Poor prognosis despite chemotherapy and radiotherapy,
Most patients die within a year after diagnosis.

Direct spread into surrounding mesothelial sacs,
LN, hematogenous mets

Unlike lung cancer, there seems to be no association between mesothelioma and tobacco smoking.
Companion Patient #7: Mesothelioma on CXR

CXR shows pleural effusion and pleural thickening leading to retraction of chest wall.

Source: Dr. Frank Gaillard. Available at http://radiopaedia.org/images/175244
Companion Patient #7: Mesothelioma on CT Scan

Enveloping nodular thickening of pleura

Contraction of hemithorax

Extension along fissures

Invasion

Dr Frank Gaillard. Available at http://radiopaedia.org/images/175244
Intense linear FDG uptake surrounding the right lung and extending into Rt. hemidiaphragm, corresponding to the soft tissue density on CT.

Back to our patient: Assessment and Plan

He is symptomatic and has oxygen desaturation on exertion. Hence, supplemental $O_2$ at home as per need.

Continue Nebulization prn.

Chest Physiotherapy.

Yearly follow up with the pulmonologist and yearly PFTs

Yearly CXR to screen for complications such as lung cancer/mesothelioma.

Repeat CT only if he becomes symptomatic again.

Pneumococcal and influenza vaccination
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