OCCUPATIONAL LUNG DISEASES

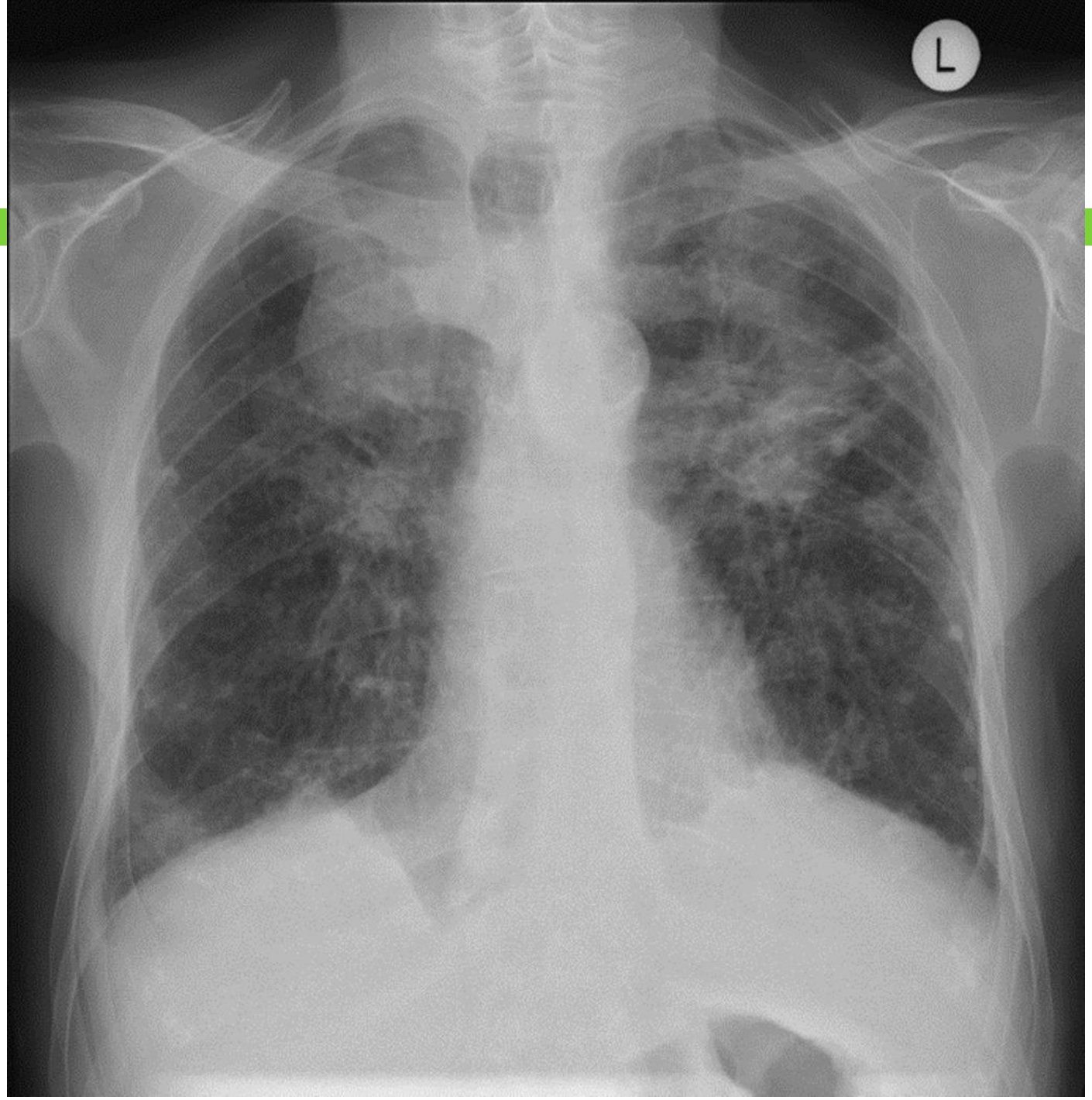
Occupational lung dis

- Exposure to dusts, gases, vapours and fumes at work can cause several different types of lung disease:
- Acute bronchitis and even pulmonary oedema from irritants such as sulphur dioxide, chlorine, ammonia or the oxides of nitrogen
- Pulmonary fibrosis due to mineral dust
- Occupational asthma (the commonest industrial lung disease).
- Hypersensitivity pneumonitis .
- Bronchial carcinoma due to industrial agents (e.g. polycyclic hydrocarbons.

- environmental/occupational history is vital . Systematically collecting information about a patient’s past employment, hobbies, and household exposures is practical. Taking a focused exposure history aimed at identifying occupational or environmental risk factors .h/o suggestiv of clustering.

When to Suspect Occupational Lung Disease

1. Patient concern.(c/0)
2. Patient report of a temporal pattern of signs and symptoms, with improvement over the weekend or during vacation.
3. Patient report that several coworkers are affected with a similar illness.
4. Patient report of known hazardous substances at work.
5. Lack of a therapeutic response to aggressive appropriate treatment.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Asbestos-associated Lung Disease

- Asbestos fibers are naturally occurring fibrous, hydrated silicate (cause asbestos-related lung diseases
- Pleural disease
- Pleural plaques (localized, often partially calcified)
- Benign pleural effusion
- Mesothelioma
- Parenchymal lung disease
- Asbestosis Lung cancer


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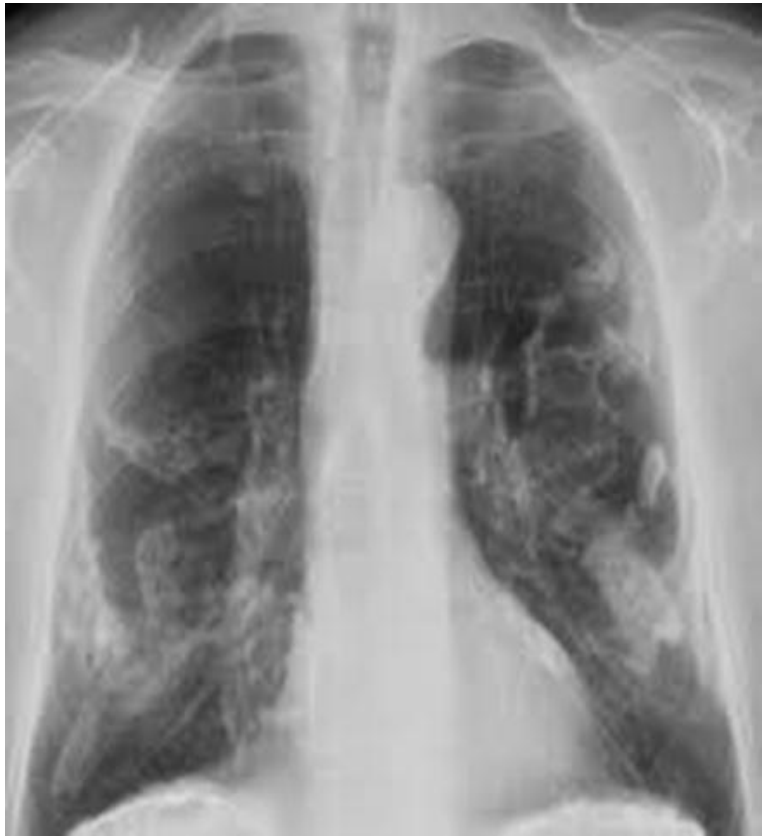
Risk Factors

- risk increase withcumulative exposure to the asbestos fiber.
- The largest number of exposed workers was in the construction industry, and the shipbuilding.

Asbestosis

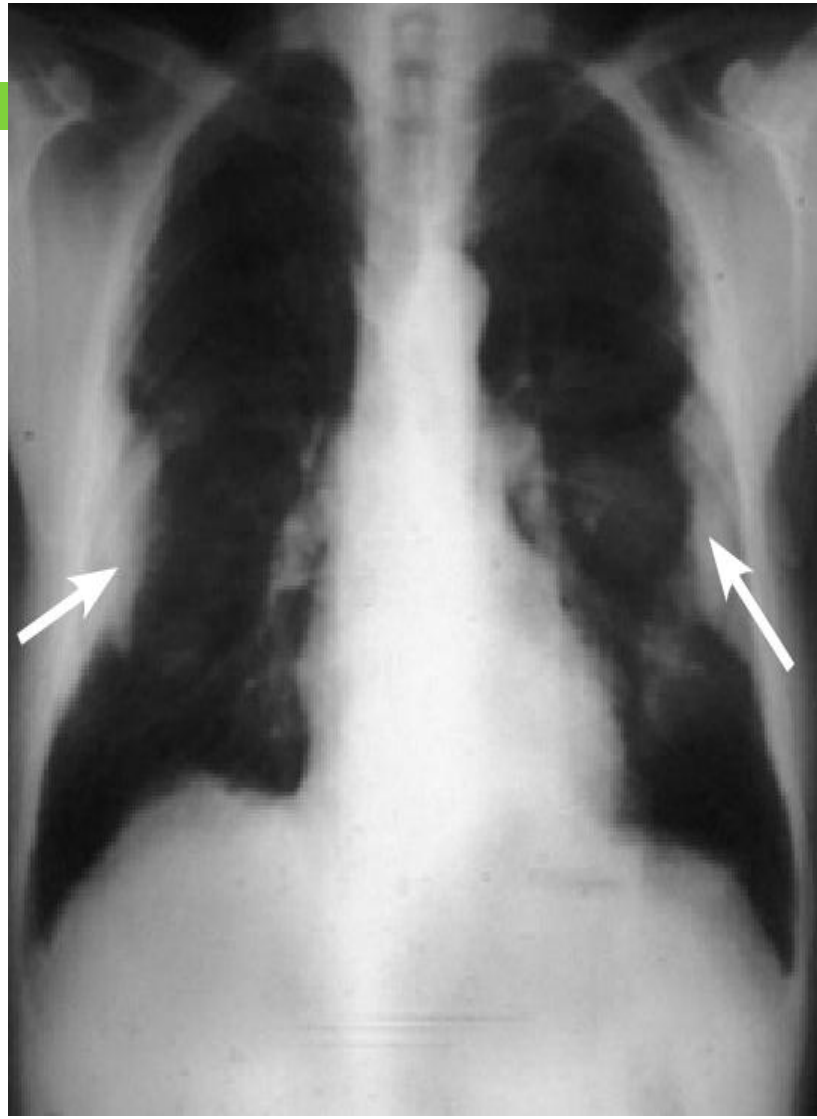
- bilateral interstitial fibrosis of the lung parenchyma caused by inhalation of asbestos fibers
- Pt c/o breathlessness,
- o/e clubbing and bibasilar inspiratory crackles, and pulmonary function testing showing a restrictive pattern
- and decreased DLCO.



- A 55-year-old factory maintenance worker falls at work. A CXR is performed to evaluate the patient for a possible broken rib. Bilateral pleural thickening is seen on CXR. Further history indicates he is very active without any respiratory symptoms. He smokes 20 cigarettes a day. There is no family history of lung disease. He does not take any respiratory medicine.

Pleural Plaques

- Pleural plaques are typically develop bilaterally, with a latency of more than 10 years. Patient should be monitored for the development of additional asbestos-related intrathoracic disease.



treatment

- There is no effective pharmacologic therapy for asbestosis.
- Steroids are of no values in treatment.
- Management includes supplemental oxygen if necessary ,influenza and pneumococcal vaccinations, smoking cessation if applicable, and aggressive therapy for intercurrent infections.

Asbestos-related Lung Cancer

- Cigarette smoke and asbestos have a synergistic (multiplicative) effect on the risk for lung cancer. but this is not true for mesothelioma.

Chronic Obstructive Pulmonary Disease

- Smoking is the most common risk factor for COPD.
- The most common occupational and environmental causative agents for COPD are
- coal, silica, and cadmium exposure.

Diagnosis and Management

- Dx and pharrmacologic management is similar to that of other forms of COPD, except that removal from exposure must also be considered.

- 38 year old male presents with 11 month history of rhinitis, followed by cough, shortness of breath and chest tightness.
- No environmental (seasonal) medication or aspirin allergy
- Ex-smoker: 5 pack years.
- Occupation: automobile painter for 3 years
- Otherwise well
- Symptoms: worse at end of the workday – improves on weekends and vacations
- Physical exam: un-remarkable. Cardiac and lung sounds were normal. There were no inspiratory or expiratory wheezes, nor a prolonged expiratory phase.

occupational asthma

- Occupational asthma (OA) is a disease characterized by variable airflow obstruction, airway and airway inflammation attributed to a particular occupational environment and not due to stimuli encountered outside the workplace
- Approximately 10 percent of asthmatic subjects identify workplace exposure factors.

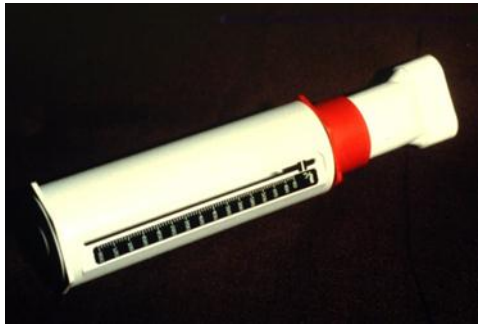
Risk factors

- Isocyanates (eg, toluene diisocyanate,) insulators, painters
- Metals (eg, chromic acid, vanadium, platinum salts)-> welders and chemical workers
- Animal proteins (eg, domestic and laboratory animals, fish and seafood)→ Farmers, veterinarians, poultry , fish and seafood processors.

Major causes of occupational asthma

	Occupation at risk
Low molecular weight chemicals	
Isocyanates (eg, toluene diisocyanate, diphenylmethane diisocyanate, hexamethylene diisocyanate, naphthalene diisocyanate)	Polyurethane workers, roofers, insulators, painters
Anhydrides (eg, trimellitic anhydride, phthalic anhydride)	Manufacturers of paint, plastics, epoxy resins
Metals (eg, chromic acid, potassium dichromate, nickel sulfate, vanadium, platinum salts)	Platers, welders, metal and chemical workers
Drugs (eg, beta lactam agents, opiates, other)	Pharmaceutical workers, farm workers, health professionals
Wood dust (eg, Western red cedar, maple, oak, exotic woods)	Carpenters, woodworkers
Dyes and bleaches (eg, anthraquinone, carmine, henna extract, persulfate, reactive dyes)	Fabric and fur dyers, hairdressers
Amines	Chemists, cleaners, plastic manufacturers
Glues and resins (eg, acrylates, epoxy)	Plastic manufacturers
Miscellaneous (eg, formaldehyde, glutaraldehyde, ethylene oxide, pyrethrin, polyvinyl chloride vapor)	Laboratory workers, textile workers, paint sprayers, health professionals
High molecular weight organic materials	
Animal proteins (eg, domestic and laboratory animals, fish and seafood)	Farmers, veterinarians, poultry processors, fish and seafood processors
Flours and cereals	Bakers, food processors, dock workers
Enzymes (eg, pancreatic extracts, papain, trypsin, Bacillus subtilis, bromelain, pectinase, amylase, lipase)	Bakers, food processors, pharmaceutical workers, plastic workers, detergent manufacturers
Plant proteins (eg, wheat, grain dust, coffee beans, tobacco dust, cotton, tea, latex, psyllium, various flours)	Bakers, farmers, food and plant processors, health professionals, textile workers

- Pre and post shift monitoring of lung function
- Spirometry
- PEF at and off work for period of several weeks - 4 times daily, preferable every 2 hours



rx

- Primary treatment: removal of exposure
- If not removed?
- Can exposure be controlled to control disease
- Personal protective measures
- Keep in position with medication control
- Desensitization?

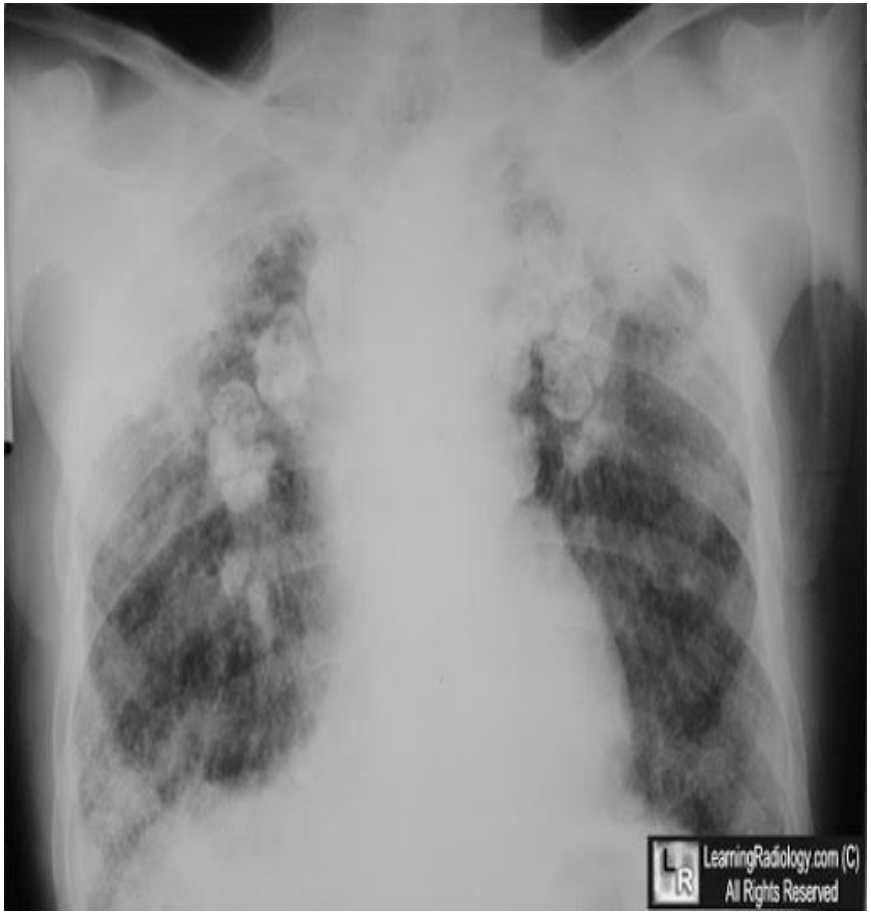
Silicosis

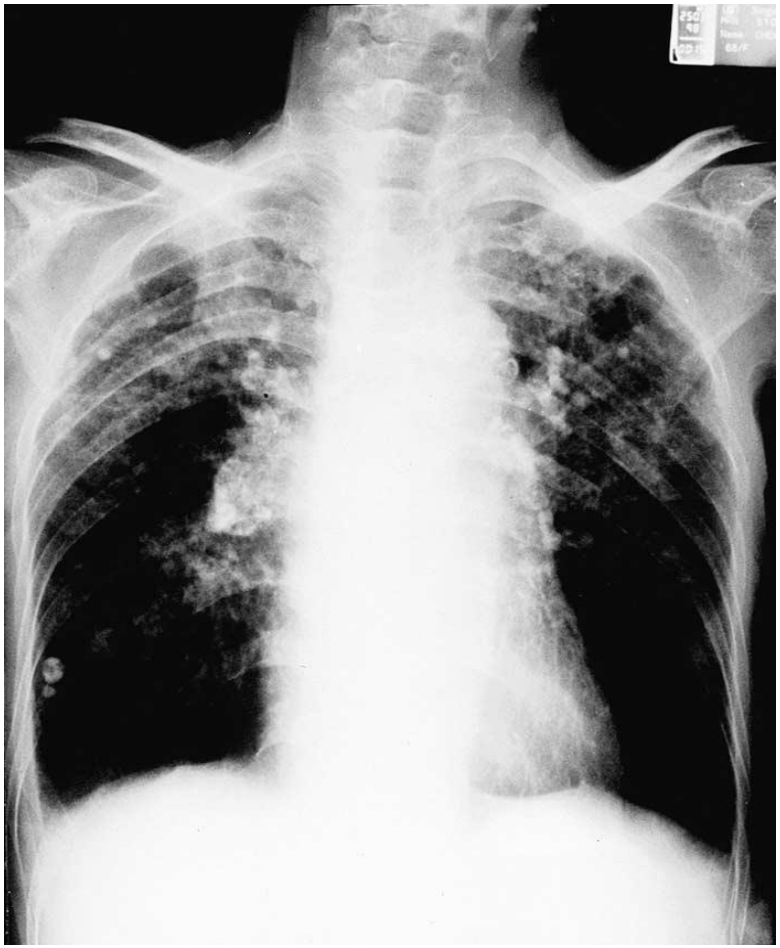
Silicosis refers to pulmonary diseases caused by inhalation of free crystalline silica (silicon dioxide)

<u>Occupations associated with silicosis</u>

1. coal mining
2. Hard rock mining
3. Tunneling
4. Quarrying and stone cutting
5. Steelworks
6. Sand blasting
7. Construction

egg-shell calcification plus the upper lobe nodules are typical of silicosis





- Chronic silicosis — simple slicosis slowly, 10 to 30 years after first exposure. to become radiographically apparent
- May many years after cessation of employment in a job associated with exposure .
- Acute silicosis is characterized by rapid onset of cough, weight loss, fatigue, and pleuritic pain.
- On physical examination, crackles are usually present.
- The prognosis is very poor. Patients rapidly develop cyanosis, cor pulmonale, and respiratory failure.

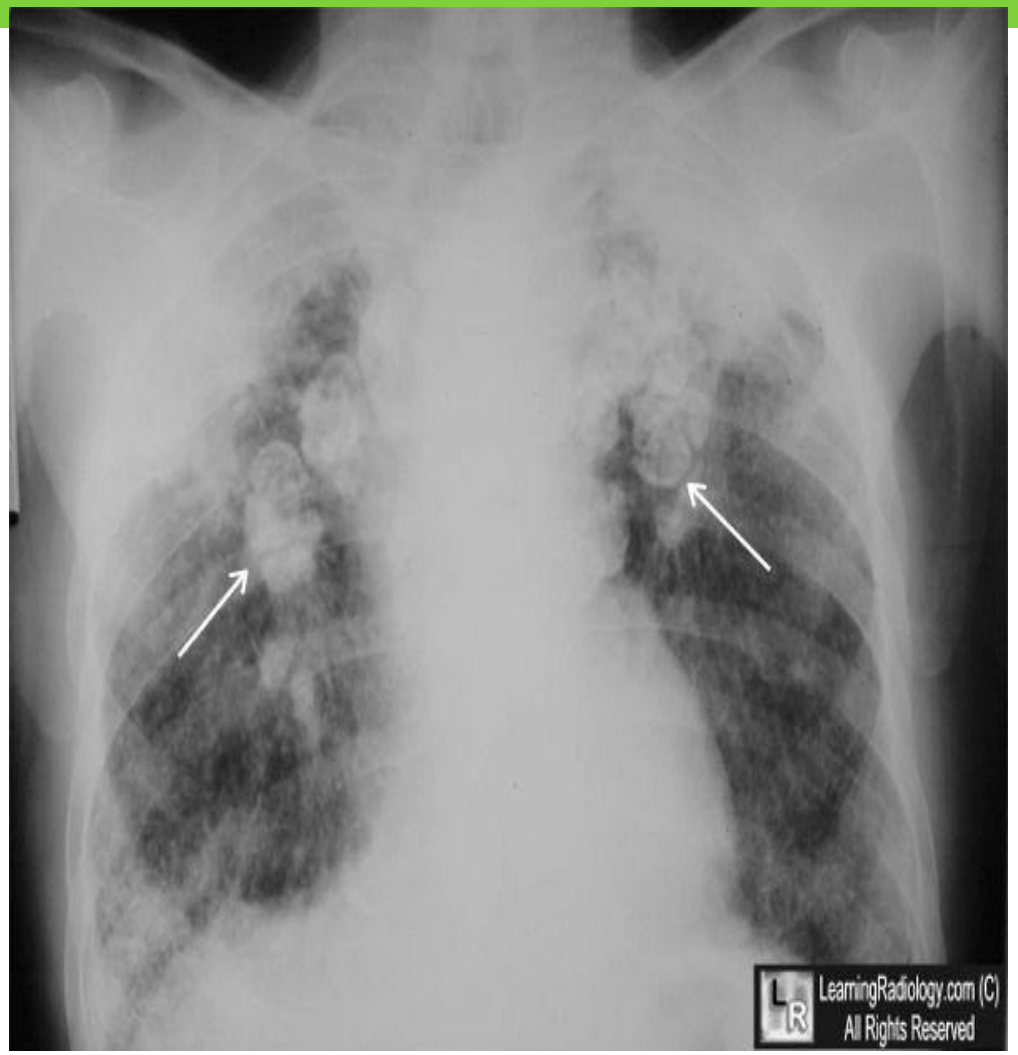
- In a minority of those with chronic disease, nodules coalesce resulting in progressive massive fibrosis (PMF).
- (PMF) is associated with more severe symptoms than simple silicosis. Physical examination frequently demonstrates decreased or other abnormal breath sounds. Signs of chronic respiratory failure and cor pulmonale..
- Patients with silicosis have increased risk of TB

  And lung cancer.

treatment

- Non specfic symptomatic therapy should include treatment of airflow limitation with bronchodilators.
- Rx of respiratory tract infection and use of supplemental oxygen (if indicated) to prevent complications of chronic hypoxemia.

- This 70-year-old male used to work in a gravel quarry. (محجرhas shortness of breath with activity that has been gradually getting worse, and a chronic cough. What is the diagnosis?



Chronic beryllium disease (berylliosis)

- occur in metal , ceramics, and nuclear weapons manufacturing .
- A patient is considered to have CBD if he or she has all of the following:
  - A history of any beryllium exposure
  - A positive blood or bronchoalveolar lavage (BAL) beryllium lymphocyte proliferation test.
  - Non caseating pulmonary granulomas on lung biopsy

Cl/ f

- dry cough, shortness of breath, night sweats, fatigue, and weight loss. .
- Pulmonary examination typically reveals bibasilar crepitations.
- advanced disease lead to cor pulmonale and digital clubbing.

- Chest radiographs may be normal or show hilar adenopathy with reticulonodular opacification
- computed tomography is more sensitive in identifying the presence of parenchymal nodules, ground glass opacities and hilar or mediastinal adenopathy
- Rx Steroids and Oxygen

Coal workers pneumoconiosis

- Prolonged inhalation of coal dust
- Simple pneumoconiosis- does not progress if miner leaves the industry.
- Progressive massive fibrosis.
- Caplan syndrome- massive fibrotic nodules in patients with RA.

silicosis

- bilateral infiltrates and egg-shell calcification of the hilar lymph nodes. The egg-shell calcification plus the upper lobe nodules are typical of silicosis.
- Differential diagnoses of upper lobe infiltrates include tuberculosis, ankylosing spondylitis and silicosis, .
- Differential diagnoses of egg-shell calcification include
- sarcoidosis, lymphoma following radiotherapy, and coal-worker's pneumoconiosis.

Thank u