

# Occupational Lung Diseases

Occupational lung diseases are a broad group of pulmonary disorders that develop from inhalation of specific particles.

Historically, they have been a major cause of morbidity and mortality before workplace safety guidelines were rigorously established and enforced.

Although each disease has a slightly unique presentation, they all lead to progressive deterioration in lung function that can cause severe respiratory compromise if appropriate measures are not undertaken.

The chest radiograph shown is from a patient with asbestosis, an occupational lung disease caused by inhalation of asbestos fibers, and shows extensive pleural plaques and diffuse interstitial fibrotic disease of the lungs.



*diagnosis please*



asbestosis

The major classifications of occupational lung diseases are hypersensitivity pneumonitis (HP) and pneumoconiosis.

**hypersensitivity pneumonitis ( HP), or extrinsic allergic alveolitis**, refers to lung diseases caused by inhalation of organic materials.

**pneumoconiosis** is the term for lung diseases caused by inhalation of mineral dust.

Occupational Lung Diseases	
<b>Hypersensitivity Pneumonitis</b>	<b>Pneumoconiosis</b>
<ul style="list-style-type: none"><li>• farmer's lung</li><li>• <b>bagassosis</b></li><li>• humidifier/air conditioner lung</li><li>• bird breeder's lung</li><li>• cheese worker's lung</li><li>• malt worker's lung</li><li>• paprika splitter's lung</li><li>• mollusk shell hypersensitivity</li><li>• chemical worker's lung</li></ul>	<ul style="list-style-type: none"><li>• asbestos</li><li>• berylliosis</li><li>• silicosis</li><li>• coal worker's lung</li><li>• <b>byssinosis</b></li><li>• baritosis</li><li>• <b>chalcosis</b></li></ul>
canne à sucre charançon du blé	coton et textiles tailleurs de pierre

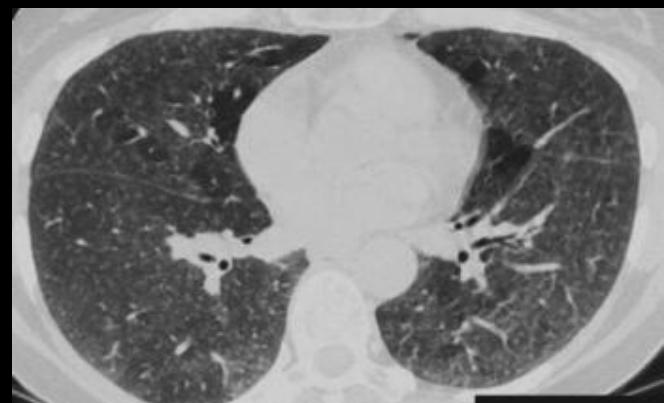
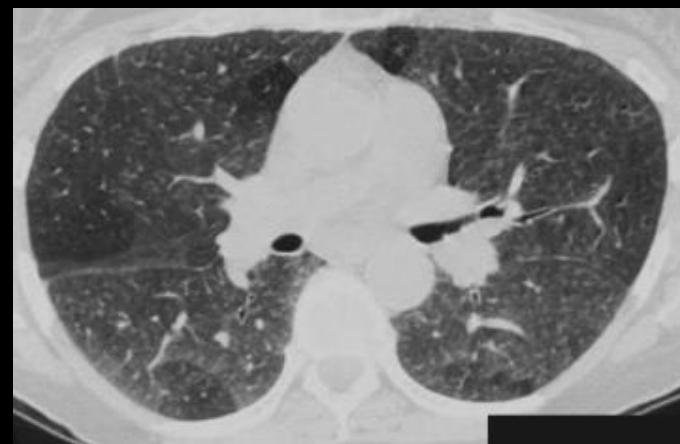
**Hypersensitivity pneumonitis (HP) is an inflammatory disease of the lung parenchyma caused by inhalation of organic dusts.**

It is characterized by diffuse inflammation of the lung parenchyma in **previously sensitized patients**.

Organic dusts can be from a variety of sources such as dairy and grain products, animal dander and protein, bark, and water reservoir vaporizers.

The most common antigens are thermophilic actinomycetes species and avian proteins.

The most common diseases are **farmer's lung** and **bird fancier's lung**. A table listing various hypersensitivity pneumonitis types, sources of exposure, and major antigens is shown.

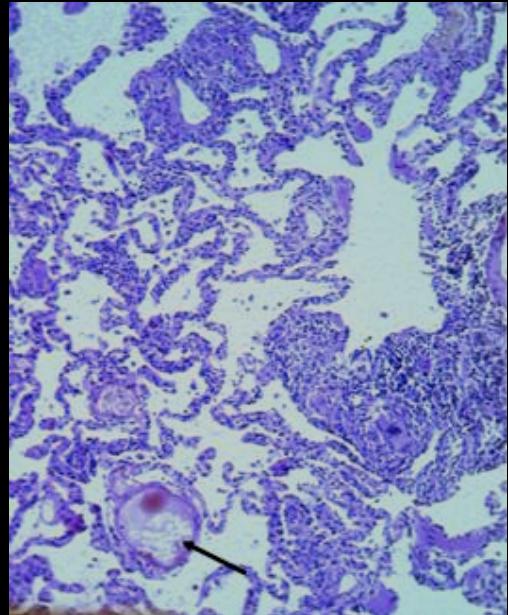


Disease	Source of Exposure	Major Antigen
Farmer's lung	Moldy hay	<i>Saccharopolyspora rectivirgula</i>
Bagassosis	Moldy sugar cane fiber	<i>Thermoactinomyces sacchari</i>
Grain handler's lung	Moldy grain	<i>S rectivirgula, T vulgaris</i>
Humidifier/air-conditioner lung	Contaminated forced-air systems, heated water reservoirs	<i>S rectivirgula, T vulgaris</i>
Bird breeder's lung	Pigeons, parakeets, fowl, rodents	Avian or animal proteins
Cheese worker's lung	Cheese mold	<i>Penicillium casei</i>
Malt worker's lung	Moldy malt	<i>Aspergillus clavatus</i>
Paprika splitter's lung	Paprika dust	<i>Mucor stolonifer</i>
Wheat weevil	Infested wheat	<i>Sitophilus granarius</i>
Mollusk shell hypersensitivity	Shell dust	Sea snail shells

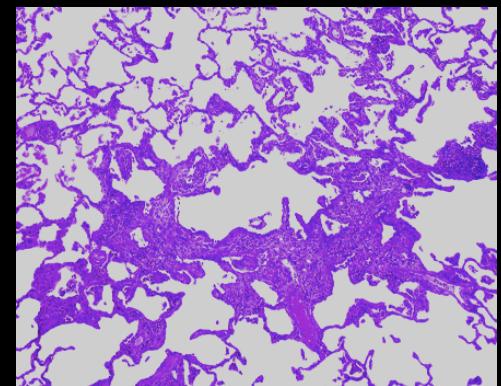
The pathophysiology of HP is based on **immunoglobulin G antibodies** to offending inhaled antigens inducing a **predominately cell-mediated immune response** that causes **extensive inflammation and fibrosis.**

There are **3 distinct pathologic forms** of hypersensitivity pneumonitis : acute, subacute , and chronic.:

-the acute form is characterized by poorly formed non caseating peribronchial granulomas (black arrow in the light microscopy image shown) and mononuclear cell infiltration with many giant cells.

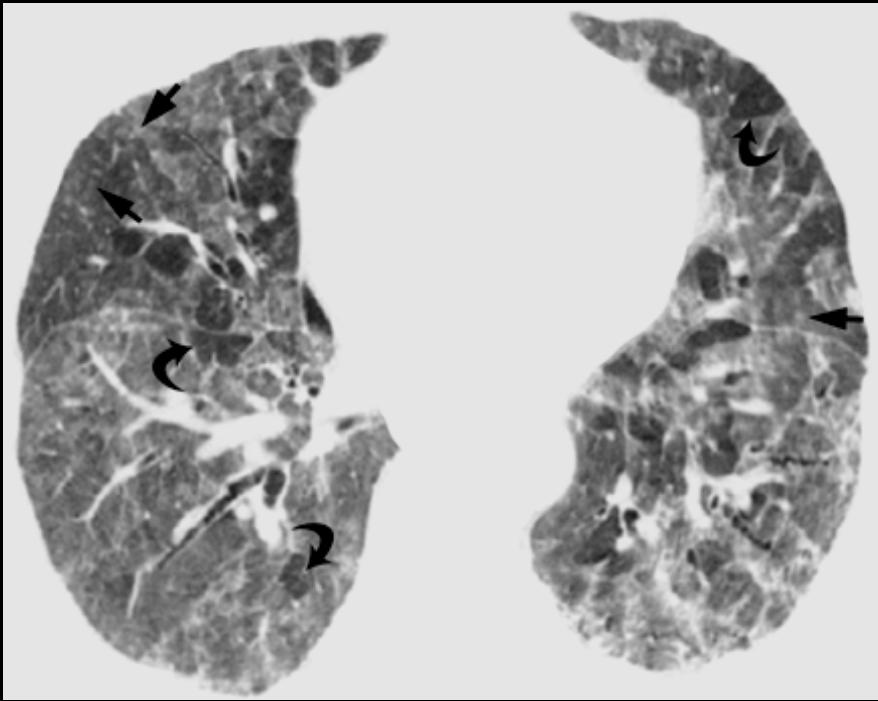


-in the subacute form, the granulomas become well-formed and there is bronchiolitis, interstitial fibrosis, and possibly organizing pneumonia.

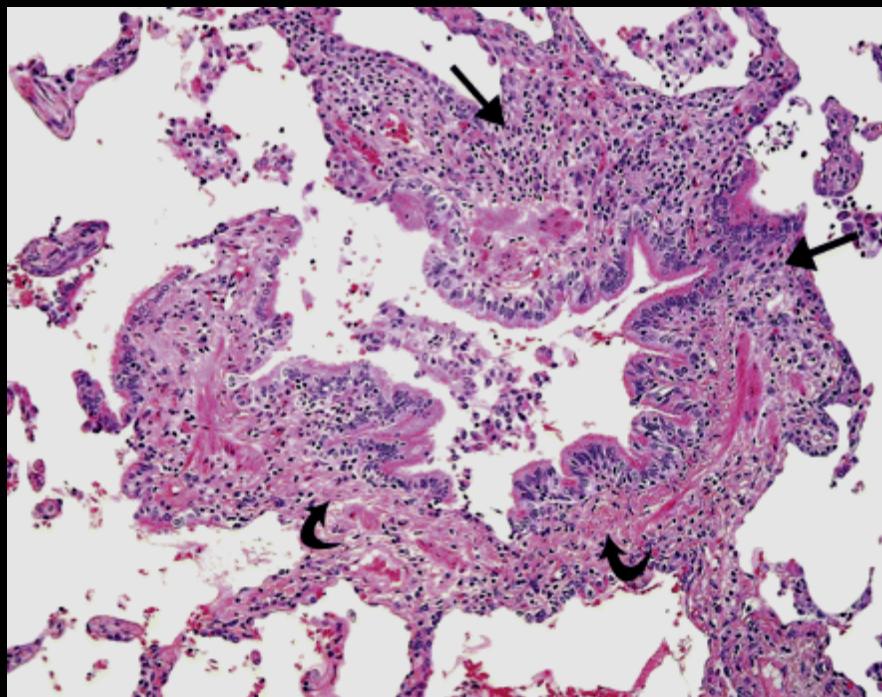


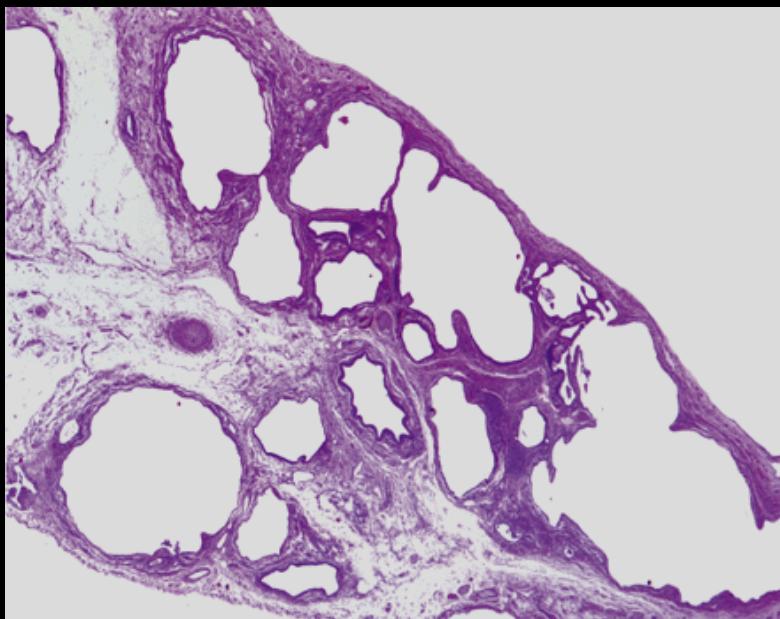
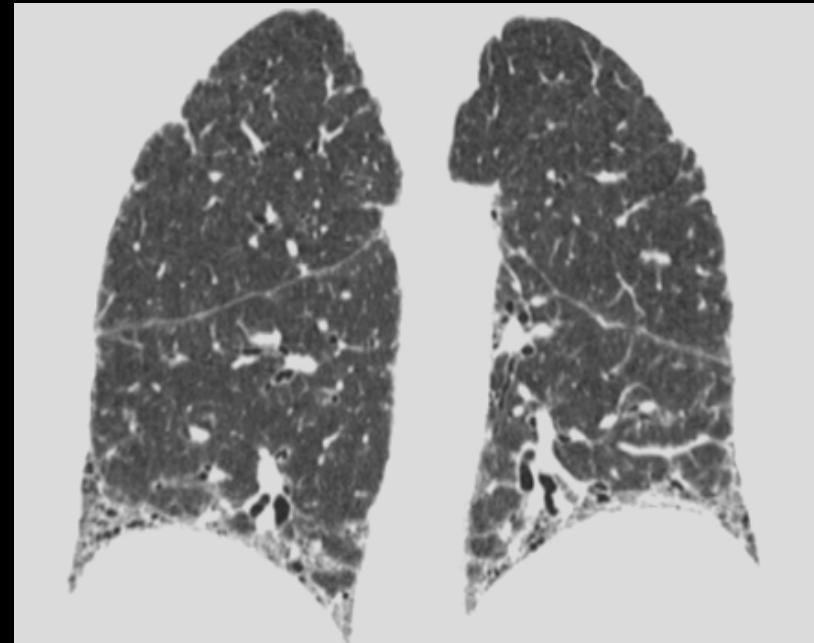
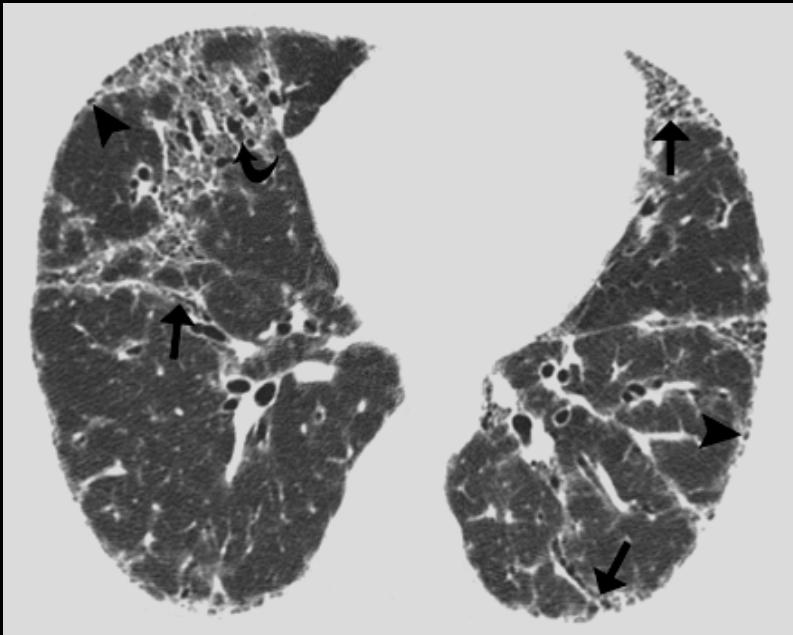
-in the chronic form, there is progressive interstitial fibrosis and honeycombing. Chronic hypersensitivity pneumonitis may have 1 of 3 patterns of fibrosis: patchy peripheral with architectural distortion, homogenous linear fibrosis, and irregular peribronchial fibrosis. The similarity in fibrotic changes to other interstitial lung diseases, such as usual interstitial pneumonitis and nonspecific interstitial pneumonia, can be misleading.





74-year-old man with chronic and subacute hypersensitivity pneumonitis (bird fancier's lung). Surgical lung biopsy specimen shows cellular bronchiolitis with infiltrate of chronic inflammatory cells (*straight arrows*), thickening wall (*curved arrows*), and narrowing lumen. This type of bronchiolitis presumably accounts for lobular areas of decreased attenuation and vascularity seen on high-resolution CT. (H and E, x160)





56-year-old man with chronic hypersensitivity pneumonitis due to occupational exposure to isocyanate compounds in paint. High-resolution CT scan shows bilateral reticulation, traction bronchiectasis (*curved arrow*), and traction bronchiolectasis (*straight arrows*). Also evident are subpleural cysts consistent with mild honeycombing (*arrowheads*). Area of ground-glass opacity with superimposed reticulation is present in right middle lobe. These high-resolution CT findings resemble those of nonspecific interstitial pneumonia. Coronal reformatted image shows predominance of abnormalities in subpleural and basal regions.

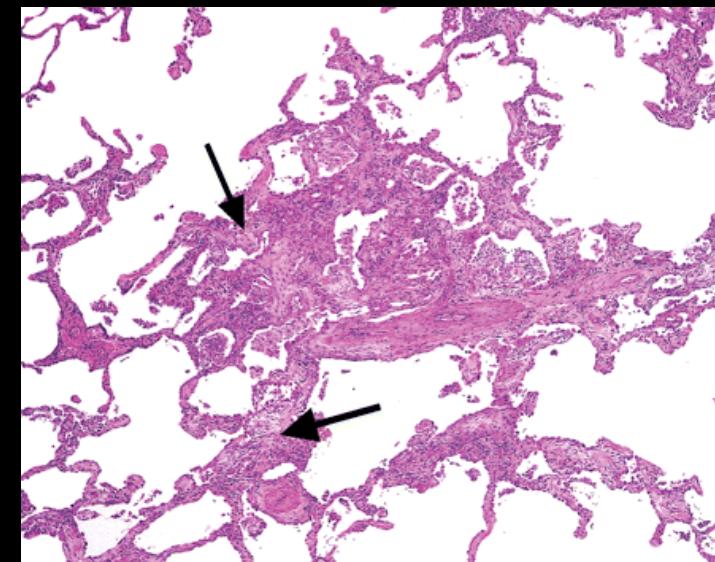
**Bird fancier's lung** is the most common type of HP in children. It has been associated with a wide variety of different birds including pigeons, pheasants, turkeys, geese, and parakeets.



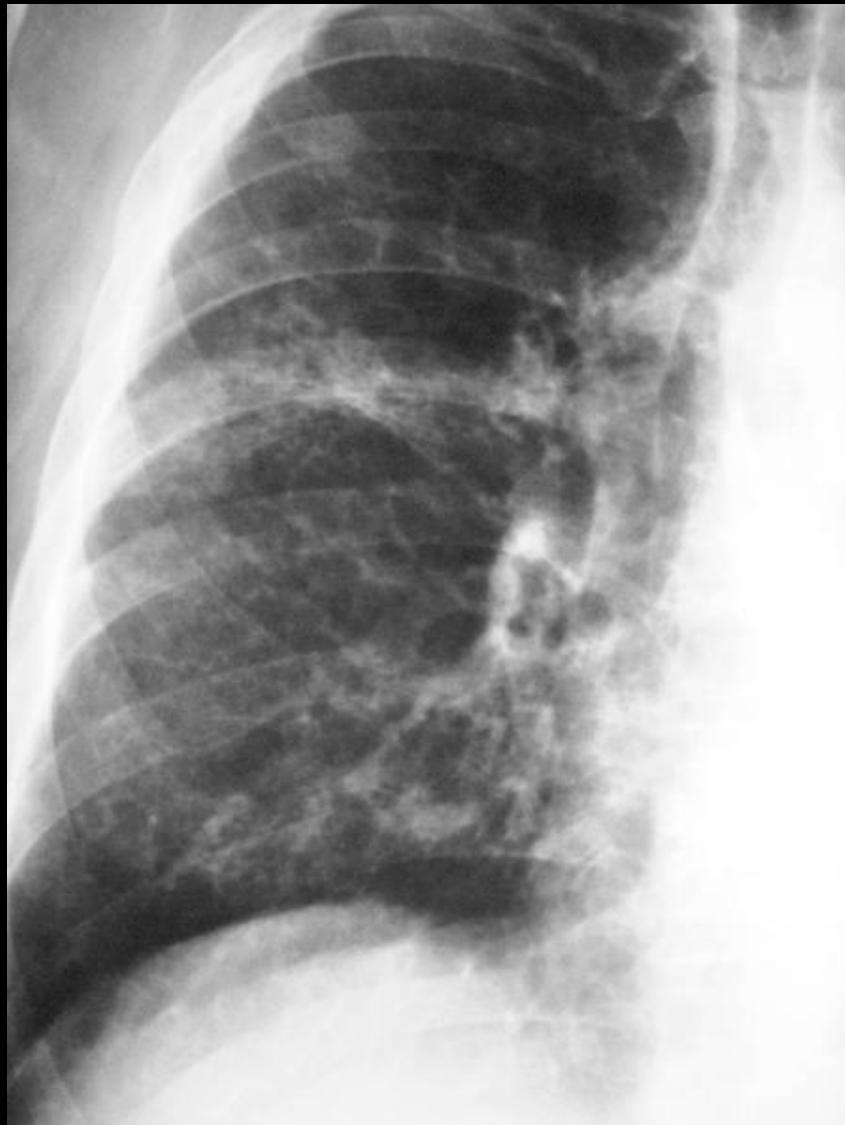
Although the classic presentation is in individuals who have birds as pets or who raise birds for consumption, more insidious cases have been identified.

Bird nests near air-conditioning intake systems, feather duvets, and bird congregations in backyard pools have all been identified as sources of HP.

A high degree of suspicion is recommended for patients who present signs and symptoms of HP but lack any obvious history of bird exposure.



65-year-old man with hypersensitivity pneumonitis (bird fancier's lung). Photomicrograph of surgical lung biopsy specimen shows chronic inflammatory infiltrate with focal area (*arrows*) of organizing pneumonia. (H and E, x60)



**Posteroanterior (PA) chest radiograph in a patient with chronic hypersensitivity pneumonitis (HP)—a pigeon fancier—shows reticular-nodular opacification**

**Farmer's lung** is caused by the inhalation of thermophilic *Actinomyces* or *Aspergillus* species in decomposing compost, hay, or sugar cane.

Exposure to large quantities of contaminated hay is the most common source. This most commonly occurs **during the winter months** due to the cold, damp climate.

The thermophilic actinomycetes include *Saccharopolyspora rectivirgula* (formerly *Micropolyspora faeni*), *Thermoactinomyces vulgaris*, *Thermoactinomyces viridis*, and *Thermoactinomyces sacchari*, among others.

Incidence is highly variable, depending on the local climate, type of farming, and exposure history, but **HP is estimated to affect 0.4%-7% of the farming population**



44-year-old man with chronic hypersensitivity pneumonitis (farmer's lung). High-resolution CT image shows bilateral ground-glass opacities and centrilobular emphysema. Patient was lifelong nonsmoker

The diagnosis of HP is based on history, physical examination, and radiographic findings.

Patients may report fever, chills, malaise, cough, dyspnea, and headaches **4-6 hours after heavy exposure to an inciting agent in acute HP.**

Subacute and chronic forms are characterized by **cough, progressive dyspnea, fatigue, anorexia, and weight loss**. On examination, patients may present with fever, tachypnea, and diffuse **fine basilar crackles**; with muscle wasting, clubbing, and respiratory distress in severe cases.

Chest radiographs may show micronodular or reticular opacities (shown) in acute or subacute HP and progressive fibrosis with lung volume loss in chronic HP.



Chest radiograph in a 60-year-old dairy farmer who had an 8-year history of intermittent dyspnea shows bilateral reticulonodular interstitial infiltration secondary to subacute hypersensitivity pneumonitis



High-resolution CT (HRCT) scan of the lungs shows ground-glass and mosaic attenuation opacification in the **acute phase** of hypersensitivity pneumonitis



Image obtained during the **chronic phase** of hypersensitivity pneumonitis shows honeycombing in the right upper lobe (RUL) and traction bronchiectasis.

High-resolution computed tomography scans will show **ground-glass opacities** in acute disease, focal air-trapping with diffuse **micronodules** in subacute HP, and **extensive fibrosis with honeycombing and air-trapping** in chronic HP (shown).

Inhalation challenges, bronchoalveolar lavage, pulmonary function testing, and lung biopsy are adjunctive tests that may facilitate a diagnosis or identify a specific etiologic agent.

Treatment is focused on antigen avoidance. **corticosteroids** may be helpful in severe disease to accelerate the initial phase of recovery, but the long-term prognosis is not affected

. Most patients will recover the majority of lung function, but it may take several years. Patients with **significant fibrosis** have a poorer prognosis.



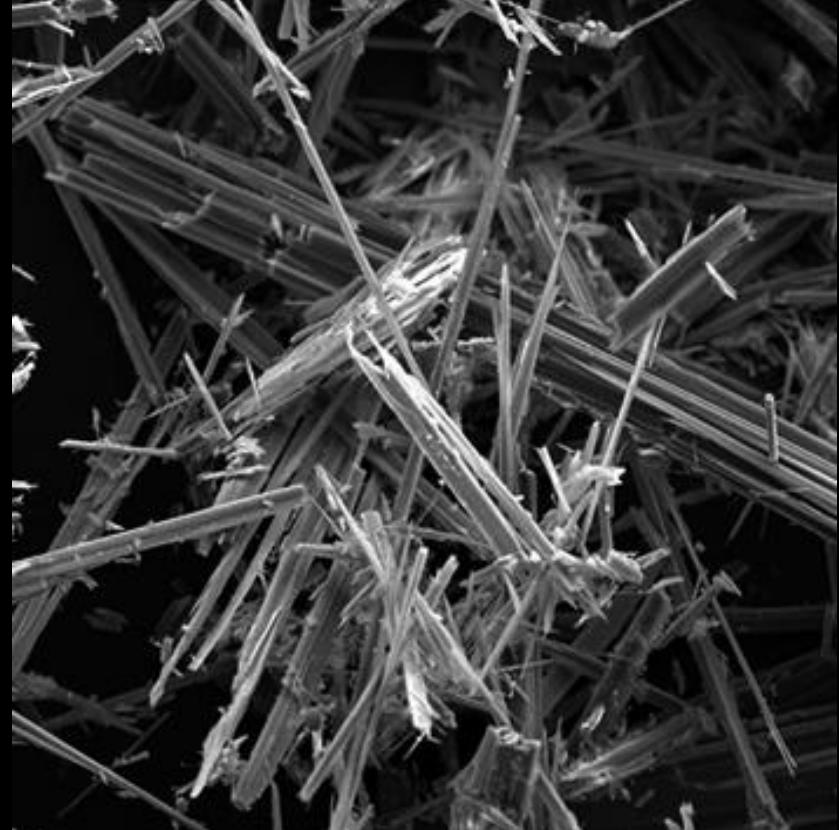
Posteroanterior (PA) chest radiograph shows bilateral reticulonodular shadowing in a patient with hypersensitivity reaction to moldy hay

**Asbestosis** is a pneumoconiosis **fibrotic** lung disease caused by inhalation of asbestos fibers.

Asbestos fibers are naturally occurring long, thin fibers of silicon that come in 1 of 2 major varieties.

The curved fibers are **serpentine** and the straight fibers are **amphiboles** (scanning electron micrograph shown).

Due to their heat-resistant properties they were extensively used to insulate heating and cooling units in World War II ships and buildings.



The **carcinogenicity** and **fibrinogenicity** of asbestos fibers are related to the type of fiber, duration of exposure, and associated host risk factors. Although both types of asbestos fibers are **fibrogenic**, **amphiboles are markedly more carcinogenic** because their straight shape allows for better penetration of cell membranes. Smaller fibers penetrate cells inducing fibrosis, while long fibers are incompletely phagocytosed and stay in the lungs inducing cycles of inflammation and cytokine release. Damage to the lung parenchyma is the result of chronic, repetitive release of oxygen free radicals, plasminogen activators, and growth factors by the macrophages.



Individuals who smoke have an increased risk for asbestosis progression **because of their impaired mucociliary clearance mechanisms**. The Prussian blue stained micrograph shows an asbestos body (arrow) within the lung parenchyma surrounded by macrophages; this is called a **ferruginous body**.

Asbestos-related diseases may develop **after a latency of up to 20 years.**

The most common presenting symptom is a dry nonproductive cough or nonspecific chest discomfort in advanced cases.

On physical examination, persistent and **dry inspiratory rales** are the most important finding. Clubbing and reduced chest expansion may also be present.

Formal diagnosis requires an exposure history, evidence of fibrosis on imaging, physical examination, and pulmonary function findings particularly **low forced vital capacity** and **a low carbon monoxide diffusing capacity** consistent with fibrosis, and the absence of another cause of interstitial fibrosis.

Chest radiographs are the most commonly used radiographic test. Classic findings include reticulonodular infiltrates at the lung bases (shown), shaggy heart borders (shown), and calcified diaphragmatic pleural plaques that are commonly called "holly leaf."



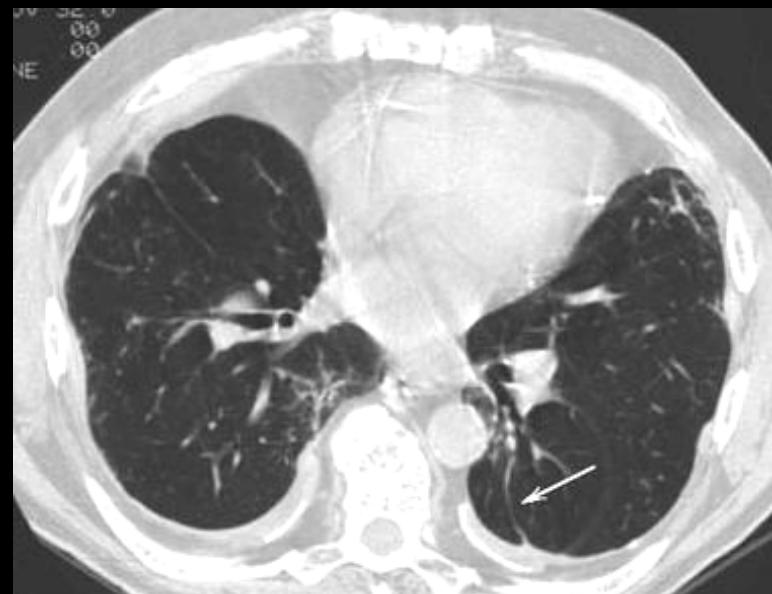
Asbestosis. Posteroanterior chest radiograph in a 54-year-old man with asbestosis demonstrates coarse linear opacities at the bases more prominent on the left, obscuring the cardiac borders and diaphragm (**shaggy heart border sign**).

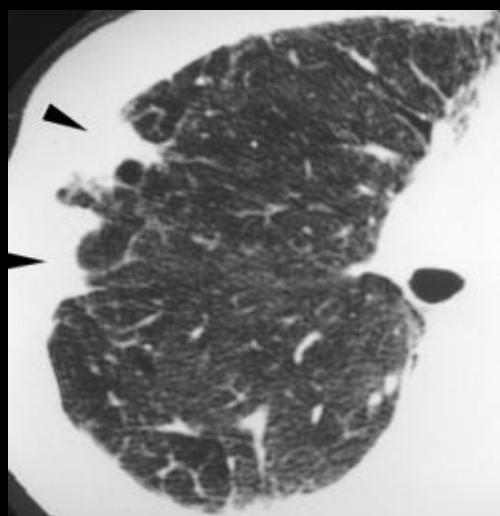
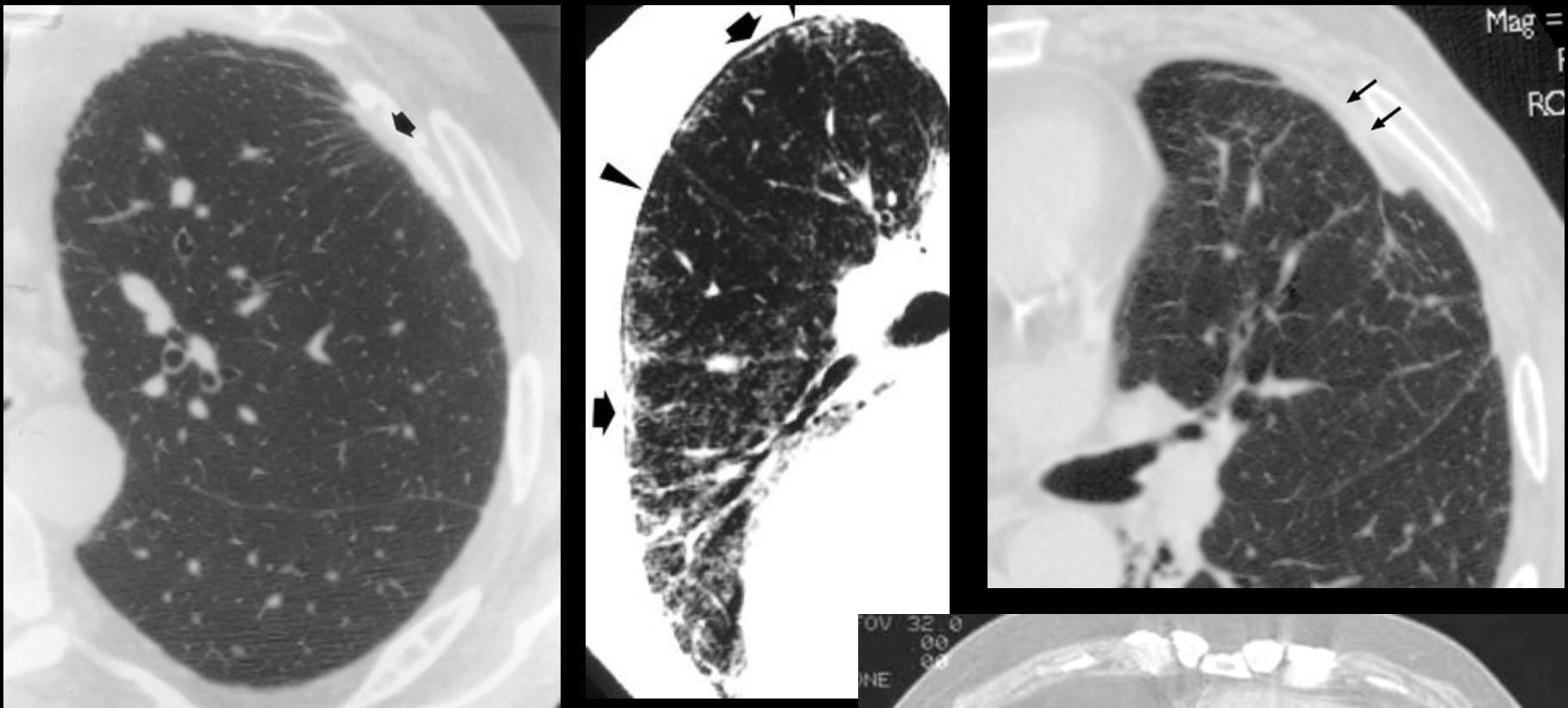
High-resolution computed tomography may show **intralobular opacities** (black arrow), **thickened septa** (white arrows), **subpleural curvilinear lines**, parenchymal bands, peribronchial fibrosis, **honeycombing**, and **calcified diaphragmatic pleural plaques** (red arrow), depending on the extent of disease.

Treatment focuses on removal of any ongoing asbestos exposure. Patients are advised to quit smoking. Prompt treatment of any subsequent respiratory infections and timely immunizations against influenza and pneumococcal pneumonia are warranted.

Some patients may require supplemental home oxygen therapy.

Corticosteroids and immunosuppressive drugs do not alter the course of the disease.





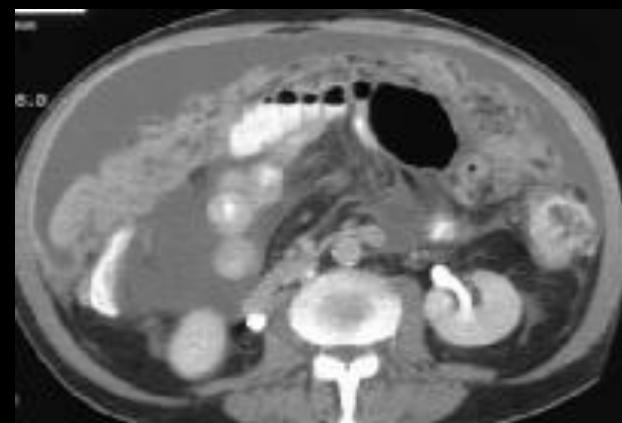
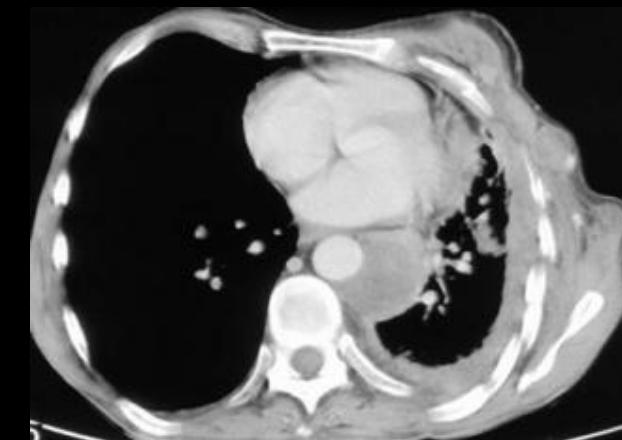
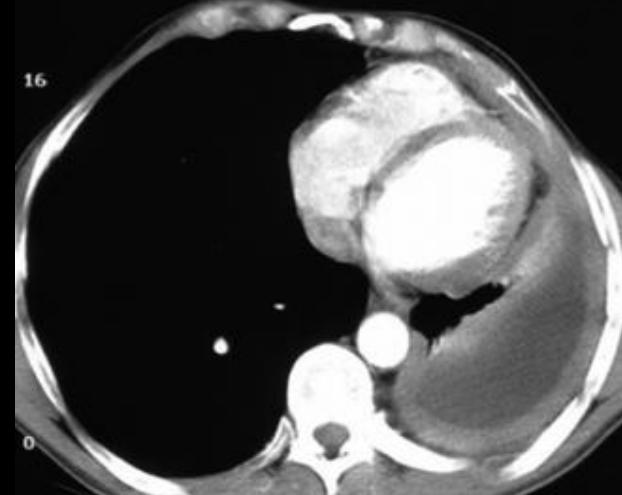
asbestosis



Patients with asbestosis are at increased risk of developing **bronchogenic carcinoma** and **malignant mesothelioma**.

The majority of mesothelioma cases are preceded by asbestos exposure, but **the latency period may be up to 50 years**. Tumor growth usually occurs along the lower part of the chest. With disease progression, the tumor often extends into the pulmonary parenchyma, brachial plexus, and superior vena cava.

Treatment options have limited success and are based on the extent of disease progression. Combinations of chemotherapy, radiation therapy, and surgery are utilized. Median survival is only 11 months and the disease is almost always fatal. The computed tomography scan shown is of a 58-year-old patient who presented with shortness of breath and was found to have mesothelioma with extensive pleural thickening, effusion, and **lung volume reduction in the affected hemithorax**.



**Silicosis** is a **fibronodular lung disease** caused by inhalation of dust containing crystalline silica. It is perhaps the oldest known occupational lung disease, referred to by both Hippocrates and Pliny.

Crystalline silica, most commonly quartz, is found in granite, slate, sandstone, and other common building materials. Individuals who work in mining, sandblasting, cement manufacturing, masonry, construction, or grinding are at risk of developing disease without appropriate protection. The historical image from 1939 depicts workers shoveling finely ground quartz for the creation of clay without respiratory protection before the dangers of silicon inhalation were known.

Small silicon particles are inhaled into the distal alveoli where they generate silicon-based radicals that lead to the production of oxygen and hydrogen-based free radicals that damage cell membranes. Alveolar macrophages ingest the particles and release their own inflammatory mediators. The end result is inflammation that damages cells and the extracellular matrix leading to fibrosis. Silica particles outlive the alveolar macrophages, thus continuing the cycle of injury

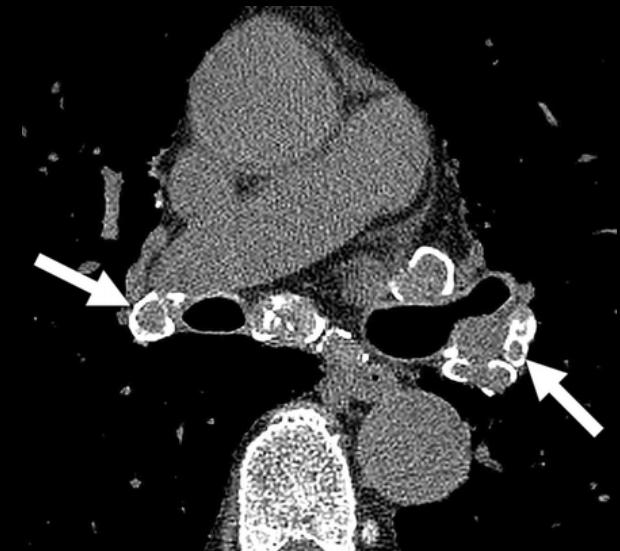
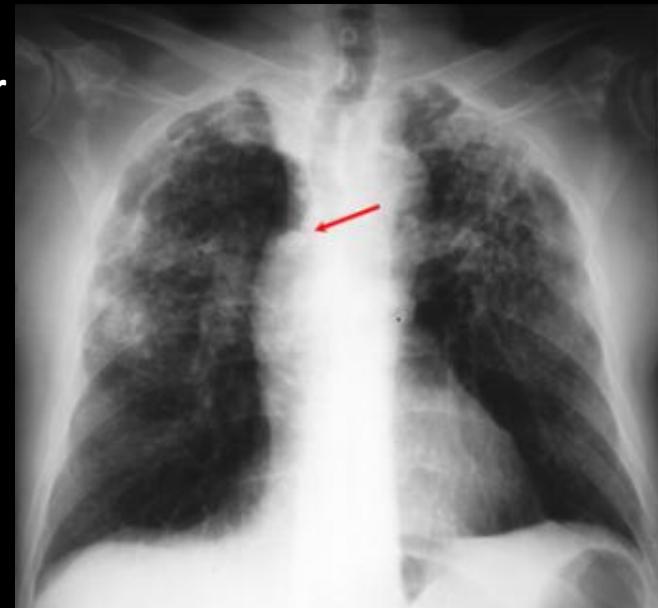


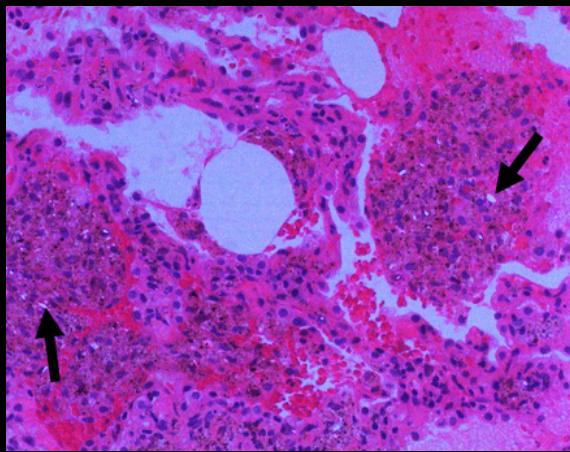
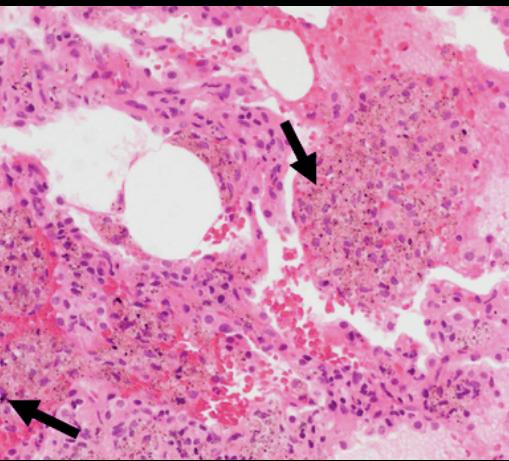
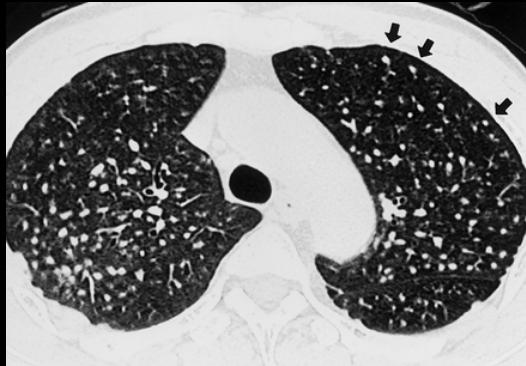
**Acute silicosis** occurs following a brief massive exposure to silica while **chronic silicosis** develops after years of exposure. In both forms, physical examination findings are typically benign with respiratory difficulty and signs of hypoxemia only in severe cases.

Diagnosis is based on a history of exposure, clinical symptoms, physical examination findings, and chest radiographic appearance.

Chest radiographs are essential to the diagnosis of silicosis. Typical findings include bilateral alveolar filing, multiple diffuse small nodules predominately in the upper lung findings, calcification of hilar lymph nodes (red arrow), and rarely cavitation of coalescing nodules.

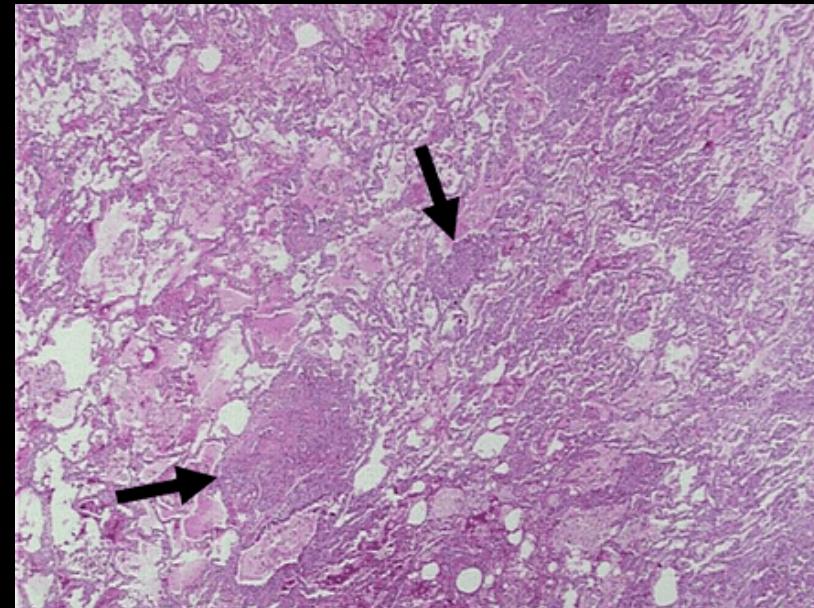
Treatment focuses on exposure avoidance, smoking cessation, immunization, and prompt treatment of respiratory infections. Corticosteroids may benefit individuals with acute silicosis. Infection of cavities by fungal organisms is a potential complication. Overall mortality is quite low, with reports of only 2 per million people in 1991 in the United States.





**Simple silicosis** in a 59-year-old man who worked in hard-rock mining for 10 years. **(a)** Chest radiograph shows diffuse nodular opacities with relative sparing of the basal lung zones. **(b)** High-resolution CT scan shows numerous micronodules in both upper lungs with posterior zonal predominance. Nodules are more profuse in the right upper lung zone than in the left. Some nodules are centrilobular in location (arrows). Note also the multiple subpleural nodules and the “pseudoplaques,” which represent the aggregate of subpleural nodules.

Classic silicosis in a 53-year-old man who worked for 12 years in sandblasting. Photomicrographs (original magnification,  $\times 100$ ; hematoxylin-eosin stain) show a transbronchial lung biopsy specimen. **(a)** Image obtained with visible light shows intraalveolar aggregation of pigmented macrophages (arrows). **(b)** Image obtained with polarized light shows scattered interstitial silica particles (arrows).



**Acute silicosis** in a 52-year-old man who worked for 10 years as a crystal craftsman. **(a, b)** Axial thin-section CT scans (1.0-mm-thick sections) obtained at the levels of the ventricles **(a)** and the left basal truncal bronchus **(b)** show ground-glass opacities and mild interlobular septal thickening (arrows) in the middle and lower lobes of the right lung and in the lingular division of the upper lobe of the left lung, respectively. **(c)** Photomicrograph (original magnification,  $\times 40$ ; hematoxylin-eosin stain) of a specimen obtained with video-assisted thoracoscopic biopsy in the lingula shows a fine granular eosinophilic material that fills the alveolar space. Note the pigment-laden perivascular and interstitial macrophages and giant cells (arrows).



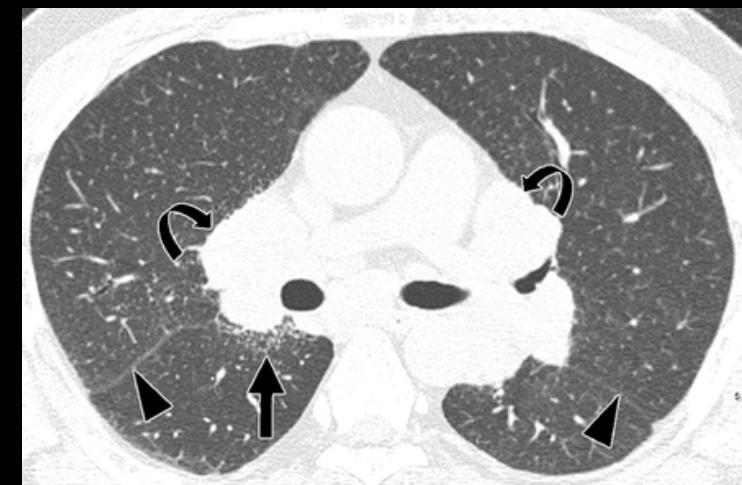
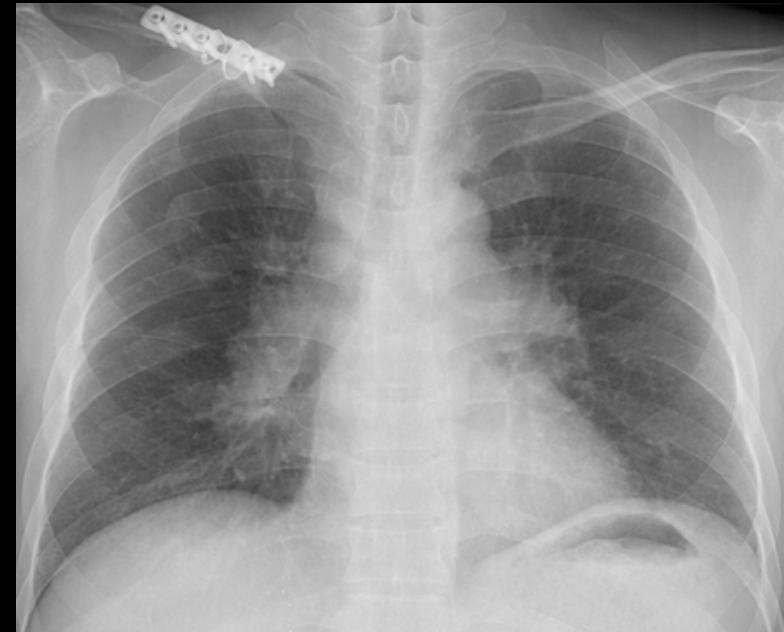
**Silicoproteinosis** 52-year-old quarry worker. Chest radiography showed bilateral ground-glass opacity and airspace consolidation, predominantly in the lower lung zones. High-resolution CT scan of the right lung shows patchy areas of ground-glass attenuation with fine intralobular reticulation ("crazy paving" pattern) (arrowheads), findings that are common in alveolar proteinosis. No silicotic nodules are seen. Bronchoalveolar lavage and transbronchial lung biopsy confirmed the presence of alveolar proteinosis and silica particles.

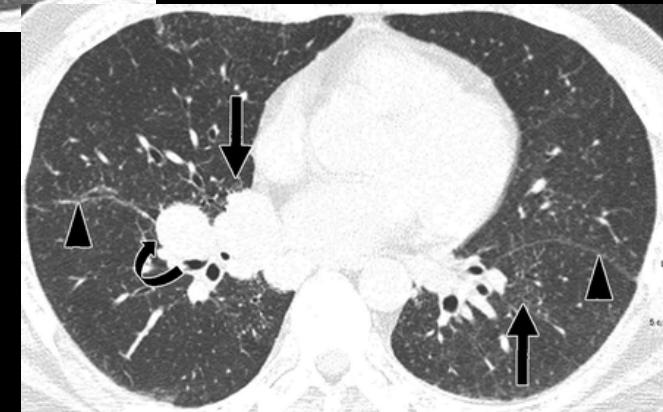
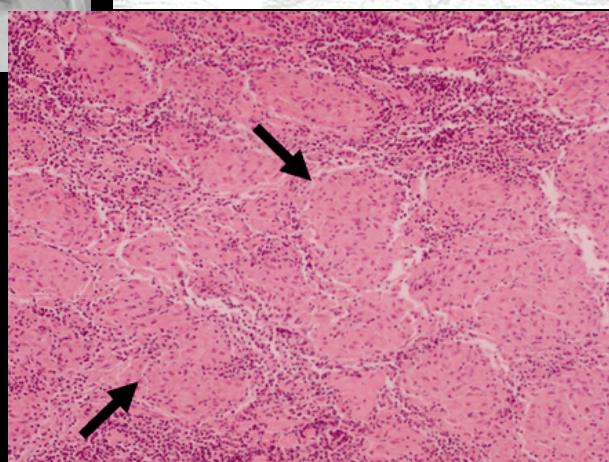
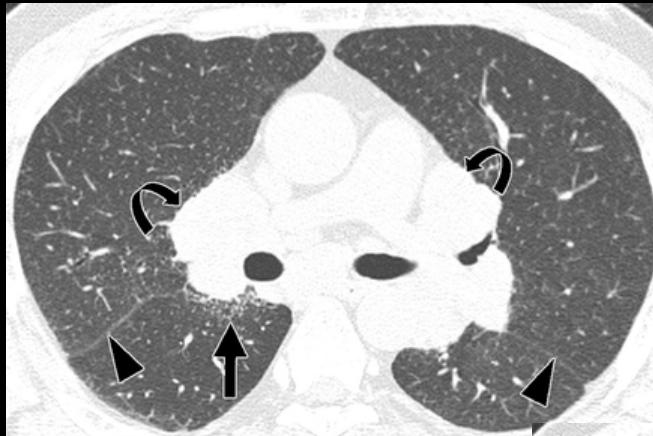
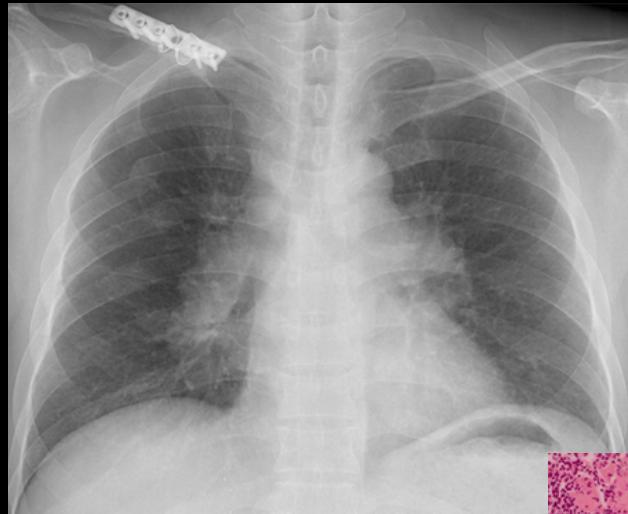
Inhalation of beryllium causes 2 distinct pulmonary syndromes: an **acute chemical pneumonitis** and a **chronic granulomatous lung disease known as berylliosis**. It is often confused with sarcoidosis as they have very similar chest radiographs.

-In acute beryllium disease, the metal acts as a direct **chemical irritant** causing an inflammatory reaction. Due to improved industrial hygiene measures, the incidence in the United States has virtually disappeared.

-Berylliosis occurs after exposure to beryllium dust or fumes, which can be found in numerous manufactured products: computer or automotive electronics, nuclear reactors, aircraft components, and nuclear reactors.

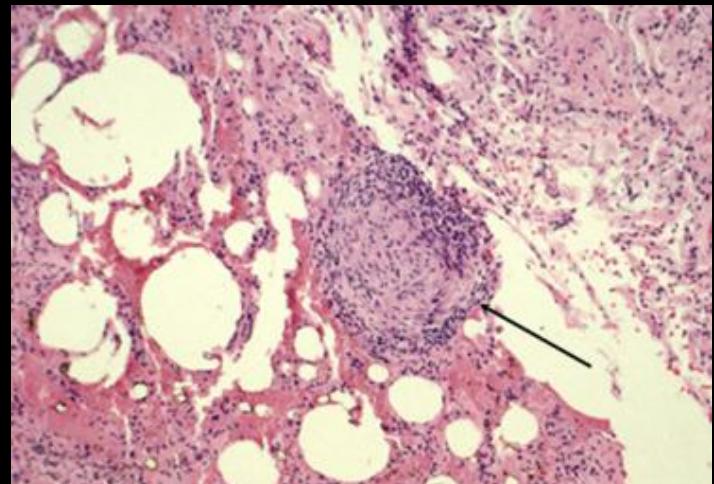
The pathogenesis is a result of **delayed-type hypersensitivity reaction** stimulating proliferation of T cells leading to inflammatory, fibrosis, and granuloma formation. Patients may report cough, chest pain, arthralgias, fatigue, and weight loss. Other than inspiratory crackles, physical examination findings are uncommon.





Berylliosis in a 49-year-old man who worked for 7 years in metal polishing. **(a)** Chest radiograph shows bilateral mediastinal and hilar lymph node enlargement, as well as internal fixation of a right clavicular fracture. **(b, c)** Axial thin-section CT scans (1.0-mm-thick-sections) obtained at the levels of the bronchus intermedius **(b)** and the basal segmental bronchus **(c)** show multiple small nodules along the bronchovascular bundles (straight arrows) and in subfissural regions (arrowheads) and enlarged hilar lymph nodes (curved arrows). **(d)** Photomicrograph (original magnification,  $\times 100$ ; hematoxylin-eosin stain) of a pathologic specimen obtained with mediastinoscopic lymph node biopsy shows multiple noncaseating granulomas (arrows).

. Diagnosis is made by a beryllium lymphocyte proliferation test from blood or **bronchoalveolar lavage** and **non-necrotizing granulomas on lung biopsy** (black arrow on hematoxylin-and-eosin histopathology).



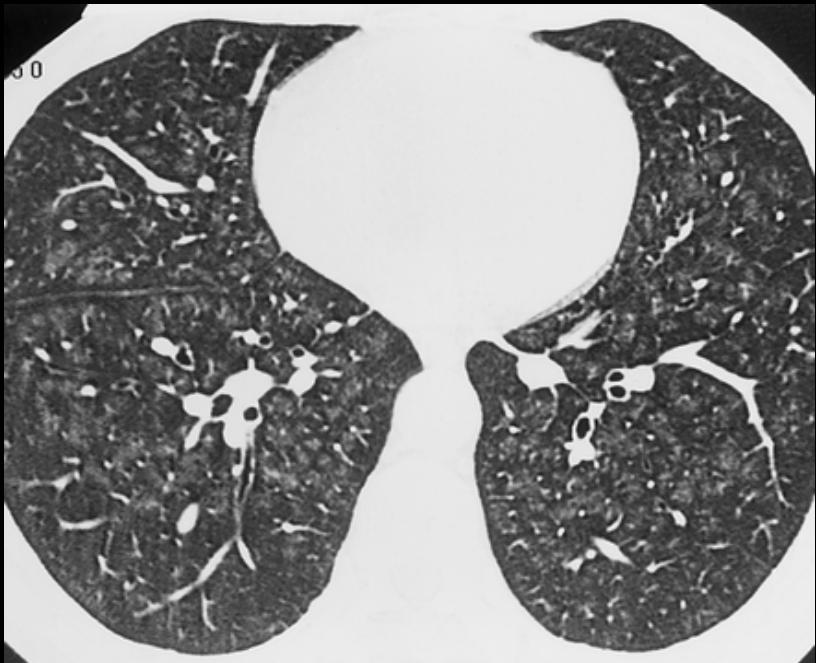
Treatment focuses on exposure avoidance and corticosteroid therapy for 4-6 weeks. Prognosis is highly variable and ranges from complete recovery to lung transplantation.



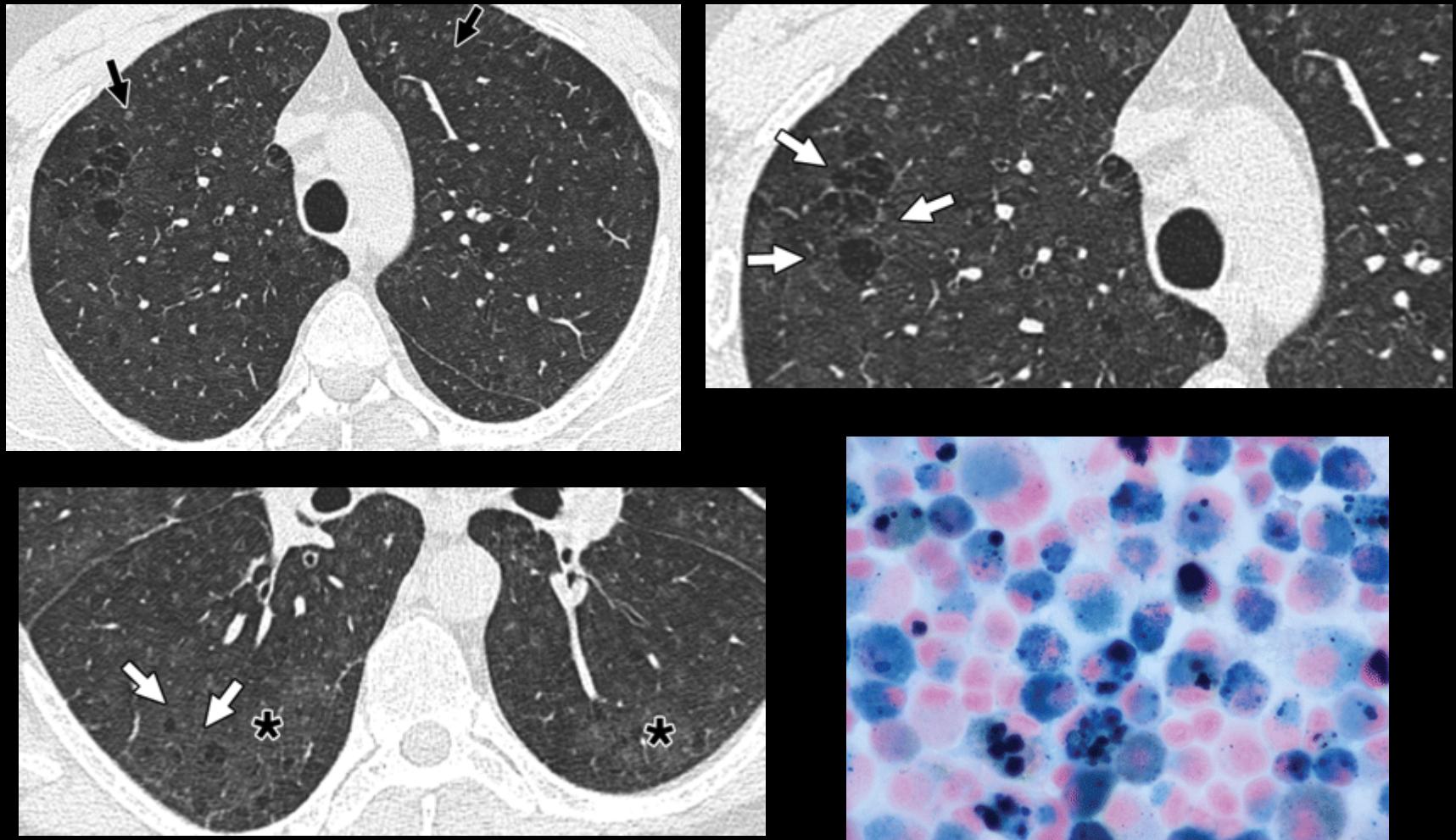
high-resolution CT scan of the chest showing the typical ground glass appearance in a patient with chronic beryllium disease, or berylliosis



**Arc welder pneumoconiosis** in a 46-year-old nonsmoker with a 15-year history of employment as a shipyard welder. High-resolution CT scan shows numerous small nodules and branching areas of hyperattenuation that are poorly defined and centrilobular. The diagnosis of **siderosis** was proved at transbronchial lung biopsy.



Arc welder pneumoconiosis in a 57-year-old former smoker with a 13-year history of work in shipyards. The patient was asymptomatic, and the results of pulmonary function tests were normal. High-resolution CT scan shows ground-glass attenuation that is diffuse and mainly centrilobular. Follow-up high-resolution CT performed 1 year later showed no change in the parenchymal disease



**Siderosis** in 39-year-old man with 30 pack-year smoking history and 20 years of exposure to arc-welding who presented complaining of cough. High-resolution CT images (1-mm-thick sections) show multiple small and poorly defined centrilobular nodules in upper lobe of both lungs (*black arrows, A and B*). Centrilobular emphysema (*white arrows, C and D*) and areas of ground-glass attenuation (*asterisks, D*) in dependent zones are seen. Results of pulmonary function test were normal

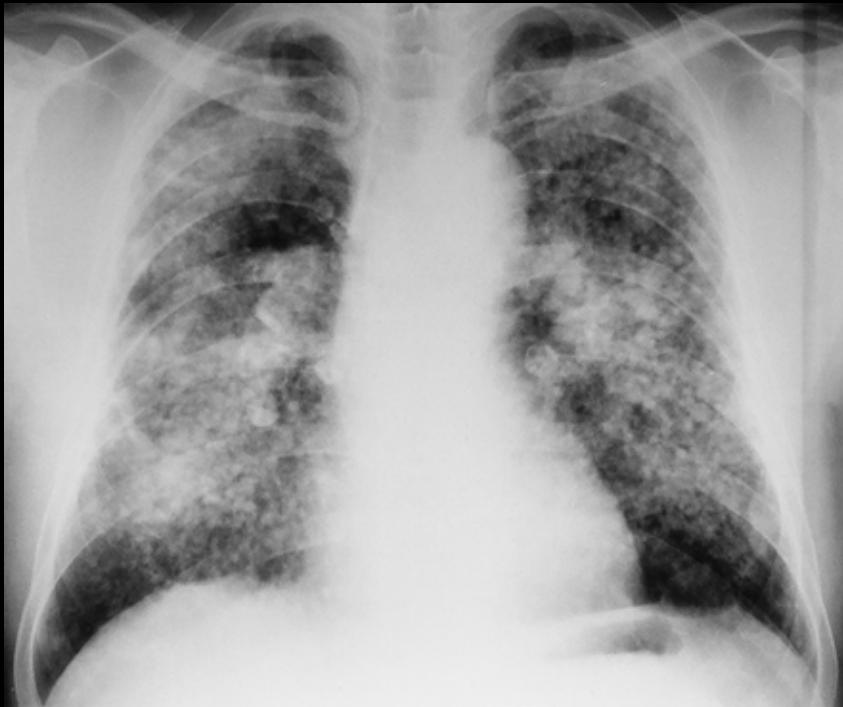
**Coal worker's lung** is a **fibrotic** pulmonary condition caused by the accumulation of coal dust in the lungs.

**Anthracosis** in comparison is the asymptomatic accumulation of coal pigment without cellular reaction found in urban dwellers and tobacco smokers.

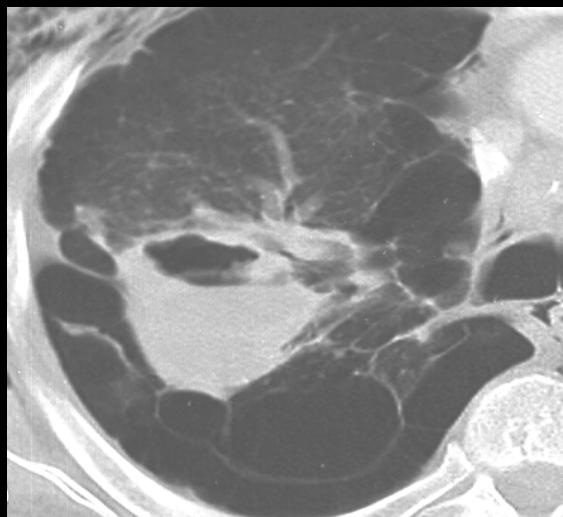
Coal dust enters the alveoli and is ingested by macrophages which expel the particles through mucus or via the lymphatic system. When the system is overwhelmed, the accumulation of macrophages triggers an immune response leading to inflammation and fibrosis).

**Periarterial fibrosis** can lead to strangulated vessels and ischemic necrosis. Focal areas of coal deposition produce **coal macules**, the hallmark of coal worker's pneumoconiosis, which can extend into one another leading to focal emphysematous changes. The degree of fibrosis is related to the duration of exposure, age at first exposure, and the quantity of inhaled silica within the coal dust.





CT of a 71-year-old patient suffering from coal worker's pneumoconiosis and melanoptysis. Notice the liquefied progressive massive fibrosis of 3.5 cm diameter in the right upper lobe (posterior segment) demonstrating a fluid level of silicotic material and mucus. A smaller massive fibrosis is seen in the posterior segment of the left upper lobe.

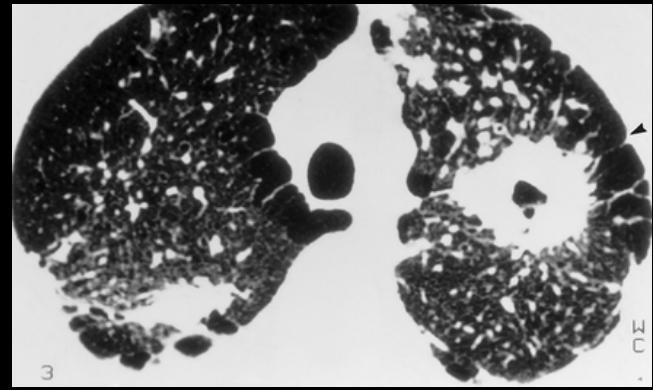
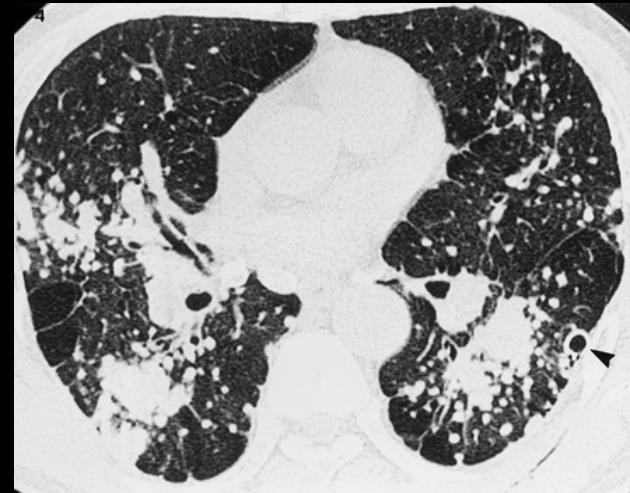


CT of a 69-year-old patient suffering from melanoptysis and severe bullous emphysema owing to coal worker's pneumoconiosis. Notice the air–fluid level within liquefied progressive massive fibrosis of 6 cm diameter. Parenchymal bands associated with local pleural thickening are visible around the lesion. Subcutaneous and mediastinal emphysema are secondary to bronchoscopy

**Patients with early disease are typically asymptomatic but may eventually report productive cough and dyspnea. Diagnosis is typically made based on history, physical examination findings, and evidence of fibrosis on radiographic imaging.**

**Treatment is largely supportive and preventative. Mortality is related to the degree of fibrosis and oxygen requirement**

The **Occupational Safety and Health Administration**, a branch of the US Department of Labor, has extensive information regarding occupational lung diseases. Epidemiologic data, workplace safety guidelines, and minimum exposure standards are all published for specific exposure types



# LA GASTRO REMPLACE LA GRIPPE A

