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Occupational Lung Disease

An APCCMPD Ambulatory Care Curriculum Teaching Script

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Occupational Lung Disease

Scenario 1:

Mr. G is a 58-year-old African-American man with a history of hypertension (HTN) who presents to your clinic for a second opinion on an abnormal chest x-ray. He has a mild cough but is otherwise asymptomatic from a respiratory standpoint. He has a distant 10 pack-year smoking history and is currently only taking amlodipine for his blood pressure. His physical exam is normal as are his vital signs, including his resting pulse oximetry on room air. He is originally from Pittsburgh and denies any recent travel. He has worked in a foundry for the last 25 years. Prior to that time, he spent some time working in coal mines for approximately 3 years. He also briefly worked as an auto mechanic.

Question 1: Before you dive into the case further, you decide to take a more thorough social history. What are the components of a thorough occupational exposure history?

Scenario 1 (Continued):

Upon further exploration of Mr. G's social history, you learn that he held multiple jobs throughout his life. As a teenager he worked at a local grocery store and briefly at a pet store. In his 20s, he worked as a welder and auto-mechanic. Beginning at age 35, he worked at the local foundry and unfortunately did not routinely wear personal protective equipment until 5 years ago.

Question 2: Mr. G read the report from his other doctor's office that mentioned multiple lung nodules and is worried he might have lung cancer. What do you think is the most likely diagnosis? What are the features of the imaging and his history that support this diagnosis?

Question 3: Although you are reasonably confident of the diagnosis, should you do any additional testing at this time to confirm?

Question 4: Mr. G is distressed to hear that he has lung disease related to his work in the past and wants to know about his options for treatment. What are the guidelines for continuing to survey his disease?

Scenario 1 (Continued):

You continue to follow Mr. G for several years and, unfortunately, have noted progression of his imaging findings over the years with the presence of PMF. He presents today for his routine follow up visit, and he now reports worsened productive cough, malaise, and night sweats. CXR is shown below in Figure 4. Is this a manifestation of his silicosis? Are there any additional tests that you should perform?

Figure 4. Interval Chest X-ray of the Patient Described Above



Image courtesy of Radiopaedia.org¹⁸

Question 5: Are there any other possible manifestations of lung disease besides silicosis for which Mr. G should be concerned?

Scenario 2:

Mr. S is a 79-year-old male with a history of diabetes and asthma who comes to see you in clinic for a second opinion. He notes that over the last 2 years he has had increased difficulty with a non-productive cough and dyspnea on exertion. He used to be quite active but now leads a most sedentary lifestyle and feels winded after climbing a flight of stairs. He has also been losing weight unintentionally (about 10 lbs. in the last year). He is a former 30 pack-year smoker who quit about 10 years ago. He currently lives at home with his wife of 45 years. He held many jobs throughout his life including working as a desk clerk and manufacturing dental appliances. He had a CT scan performed (Figure 5).

Figure 5. Representative CT Scan for Mr. S

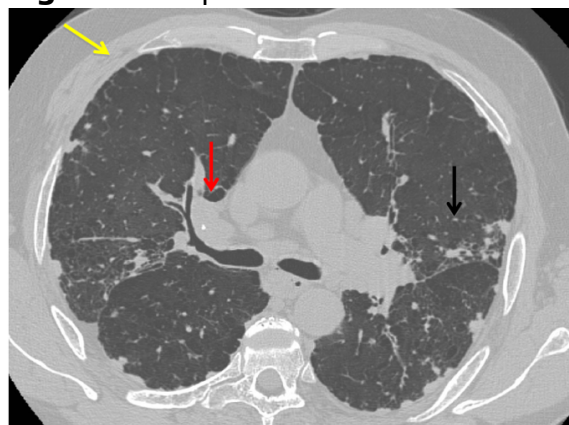


Image reproduced from *ATS Review for the Pulmonary Boards*⁴

Question 6: What do you think is the most likely diagnosis? What testing would you order to confirm?

Question 7: How does berylliosis differ from sarcoidosis?

Question 8: What are Mr. S's options for treatment?

Scenario 3:

Mr. JD is a 44-year-old man who presents to your practice for a second opinion regarding worsening shortness of breath with exertion, as well as persistent productive coughing. He works at Hamm's Blue Sky Coal Company in McDowell County WV. He has worked underground for the past 25 years at various positions inside the mine. He started as a face miner and for the past ten years, he has been the chief roof bolt operator. Exam reveals a robust plethoric middle-aged man in no distress while sitting. Pulmonary exam is clear though he does cough with deep breathing. Chest imaging shows mass-like opacities in bilateral upper lobes with volume loss. PFTs revealed a reduced FEV1 1.9L (55% predicted), reduced FVC 3.2L (75% predicted), FEV1/FVC 60%. His oxygen saturation dropped to 85% on a six-minute walk test (305 meters).

Question 9: What is the underlying mechanism on JD's respiratory findings?

Question 10: What are the clinical phenotypes of CMDLD?

Scenario 3 (Continued):

Upon further questioning, JD reports regularly trouble sleeping and mood lability. He has trouble concentrating at work and is very irritable when he comes home. A further work history finds that JD over his time in the mine has witnessed five friends die in mining accidents. On one occasion, he and his co-workers worked 30 straight hours to free a friend who had been covered in a mining collapse. He had his left hand crushed in a rock fall and has chronic pain from that accident. JD had been a promising student who had obtained an academic scholarship to attend a state university in chemical engineering. He ultimately had to forgo this scholarship when his father became disabled working in the mines in order to support his family. His close friends have very similar stories.

Question 11: What additional health outcomes should you inquire about when meeting with JD? What social determinants of health are at play in this case?

Question 12: JD asks you about the process for obtaining disability through the mining company. How would you advise him?

Occupational Lung Disease

Educational Objectives:

1. Review necessary components of a thorough social and environmental exposure history
2. Discuss the diagnostic and treatment options for the most common pneumoconiosis: silicosis, coal workers pneumoconiosis, and berylliosis
3. Review common characteristic radiographic findings of the pneumoconiosis

Authors Note: Asbestos-related lung disease, hypersensitivity pneumonitis, occupational asthma and Deployment Related Respiratory Disease are covered elsewhere in the curriculum.

- [Asbestos-Related Pleuropulmonary Disease](https://apccmpd.memberclicks.net/assets/AmbulatoryCareWorkgroup/Year_2_Scripts/06%20Asbestos-Related%202026%20Final.pdf)
https://apccmpd.memberclicks.net/assets/AmbulatoryCareWorkgroup/Year_2_Scripts/06%20Asbestos-Related%202026%20Final.pdf
- [Initial Outpatient Evaluation of Interstitial Lung Disease](https://apccmpd.memberclicks.net/assets/AmbulatoryCareWorkgroup/Year_1_Scripts/2025/06_The_Initial_Evaluation_ILD_2025.pdf)
https://apccmpd.memberclicks.net/assets/AmbulatoryCareWorkgroup/Year_1_Scripts/2025/06_The_Initial_Evaluation_ILD_2025.pdf
- [Diagnosis and Management of RADS \(Reactive Airways Dysfunction Syndrome\)](https://apccmpd.memberclicks.net/assets/AmbulatoryCareWorkgroup/Year_2_Scripts/09%20Diagnosis%20RADS%202026%20Final.pdf)
https://apccmpd.memberclicks.net/assets/AmbulatoryCareWorkgroup/Year_2_Scripts/09%20Diagnosis%20RADS%202026%20Final.pdf
- [Deployment Related Respiratory Disease \(DRRD\)](https://apccmpd.memberclicks.net/assets/AmbulatoryCareWorkgroup/Year_1_Scripts/2025/21_DRRD_Deployment_Related_Respiratory_Disease_2025.pdf)
https://apccmpd.memberclicks.net/assets/AmbulatoryCareWorkgroup/Year_1_Scripts/2025/21_DRRD_Deployment_Related_Respiratory_Disease_2025.pdf

Scenario 1:

Mr. G is a 58-year-old African-American man with a history of hypertension (HTN) who presents to your clinic for a second opinion on an abnormal chest x-ray. He has a mild cough but is otherwise asymptomatic from a respiratory standpoint. He has a distant 10 pack-year smoking history and is currently only taking amlodipine for his blood pressure. His physical exam is normal as are his vital signs, including his resting pulse oximetry on room air. He is originally from Pittsburgh and denies any recent travel. He has worked in a foundry for the last 25 years. Prior to that time, he spent some time working in coal mines for approximately 3 years. He also briefly worked as an auto mechanic. His chest radiograph (CXR) is shown below in Figure 1.

Figure 1. Representative Chest X-ray of the Patient Described Above



Figure reproduced from Rajavel S et al.²

Question 1: Before you dive into the case further, you decide to take a more thorough social history. What are the components of a thorough occupational exposure history?

It is important to review all jobs that a patient has held. Detailed work history is essential, as many exposures can have a long latency period. One method of doing this is to have the patient start by describing their current or most recent employment and then work backwards to describe each preceding job, as in Table 1.

Table 1. Taking an Occupational History to Assess Symptoms and Exposure

Respiratory Symptoms
• What are the relevant symptoms and signs? (e.g., cough, wheeze, dyspnea, chest tightness, and reduced exercise tolerance)
• Do symptoms worsen when at work and improve when away from work?
• Are other workers experiencing similar symptoms?
• Are your symptoms associated with any particular activity?
Occupational Details
• <u>What is your current job?</u>
• <u>What industry is that in?</u>
• <u>What tasks do you do?</u>
• <u>What materials do you work with?</u>
• <u>Are there exposure protections in place?</u> (eg, dust suppression, wet cutting, extraction, ventilation, personal protective equipment)
• <u>How long have you been in this job? What hours do you work and how frequently?</u>
• <u>What job did you do before this? (for each previous job, ask about the industry, tasks, exposure protections, etc.).</u>
• <u>Is there visible dust on surfaces or in your sputum at the end of a shift?</u>
• <u>Is the worksite cleaned during or after your shift?</u>
• <u>Are you exposed to exposure to a family member's dust-laden clothing, any personnel responsible for hazardous cleaning?</u>
• <u>What job did you do before this? (for each previous job, ask about the industry, tasks, exposure protections, etc.).</u>

Table adapted from Barnes H & Glaspole I.³

Scenario 1 (Continued):

Upon further exploration of Mr. G's social history, you learn that he held multiple jobs throughout his life. As a teenager he worked at a local grocery store and briefly at a pet store. In his 20s, he worked as a welder and auto-mechanic. Beginning at age 35, he worked at the local foundry and unfortunately did not routinely wear personal protective equipment until 5 years ago.

Question 2: Mr. G read the report from his other doctor's office that mentioned multiple lung nodules and is worried he might have lung cancer. What do you think is the most likely diagnosis? What are the features of the imaging and his history that support this diagnosis?

His clinical history and radiographic imaging are most consistent with a diagnosis of chronic silicosis. Silicosis is an extremely common pneumoconiosis in hard rock miners with one-third demonstrating radiographic evidence of silicosis.

Table 2. High-risk Industries and Occupations Associated with Silica Exposure

Industries	Occupations
Mining, Tunneling and Excavating	
Underground: gold, copper, iron, tin	Miner, driller, tunneler
Surface: coal, iron, foundation excavation	Drill operator
Quarrying: granite, slate, sandstone	Digger, driller, hammerer
Stonework: granite sheds, monument masonry	Cutter, dresser, polisher, grinder
Foundries: iron and noniron metals	Molder, caster, knockout man
Abrasives: metal polish, paint fillers, sandblasting, oil rigs, tombstones	Crusher, mixer, abrasive work, denim jean sandblasting
Ceramics: pottery, stoneware, oven bricks	Oven-brick maker
Others: glassmaking, boiler scaling, gemstone working, dental	Dental technicians

Table adapted from American College of Chest Physicians.⁴

Despite efforts to reduce the rate of occupational silicosis, globally the number of cases has started to rise again due to increasing industrial exposure, driven by the use of silica in the production of artificial stone used as countertops and denim sandblasting among other industries.^{5,6} The United States is also seeing increased numbers of cases related to the fabrication of engineered stone countertops.⁷

Silicosis can present in one of three forms depending on the duration, intensity, and particle size of silica dust exposure:

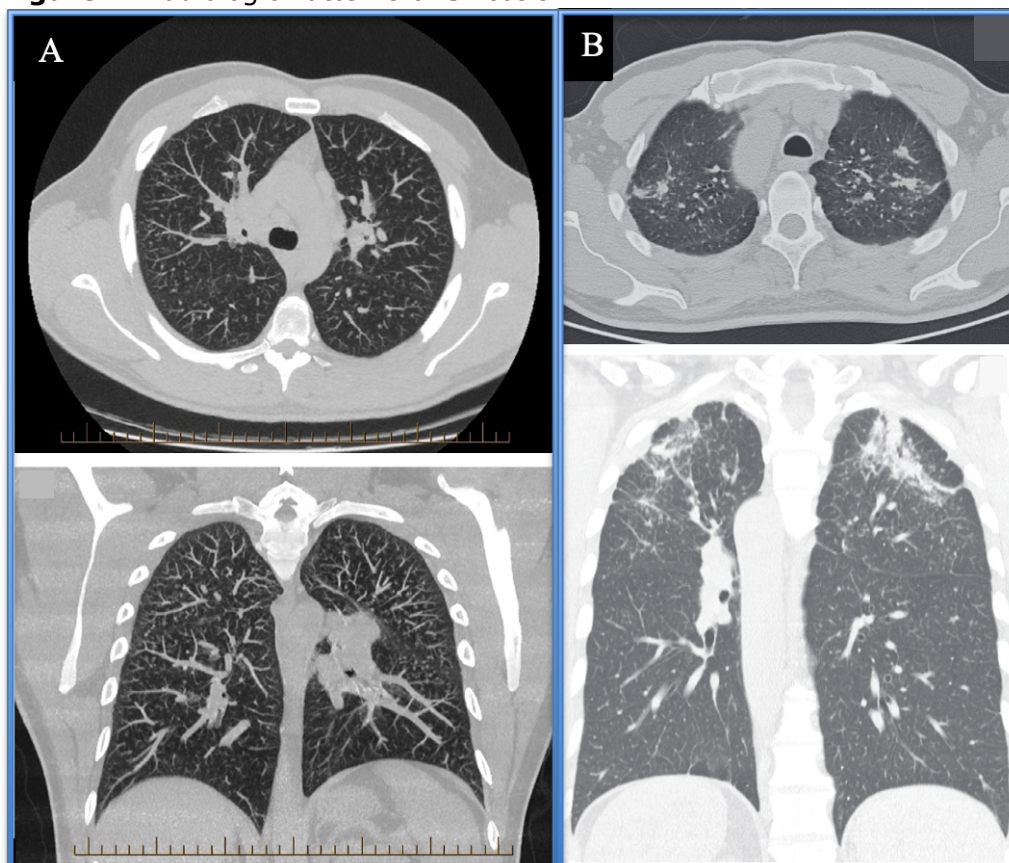
1. **Chronic (classic) silicosis** occurs after ~15-20 years of exposure to silica dust and is the most common form.
2. **Accelerated silicosis** occurs after a much shorter exposure duration of ~5-10 years, but the exposure is typically of greater intensity than what is seen in chronic silicosis.
3. **Acute silicosis** is much more rapid and can be identified as early as a few months after exposure, and is typically identified less than two years from the initial exposure. The Hawks Nest Tunnel disaster is a memorable and important historical event that "was created by ethnological prejudice and employer indifference to workers dying" of acute silicosis.⁸

While some of the differences in presentation can be explained by the type or duration of exposure, various presentations can be seen in individuals working in the same environment, so individual risk factors are likely also contributory to the development of disease. As an example, black workers have 2-7 times higher rate of developing silicosis compared with white workers with the same dust exposure.⁹

In all these cases, the diagnosis is made based on the exposure history and patterns seen on imaging. Radiographically, silicosis manifests in the following ways:

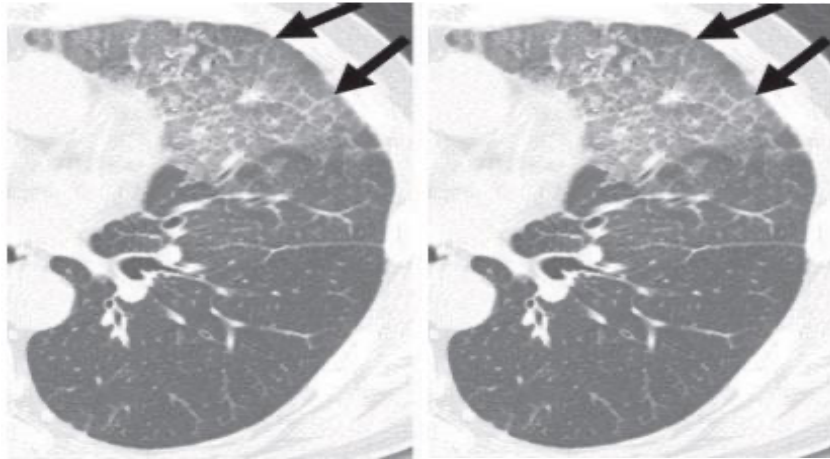
- Most commonly, simple silicosis as multiple nodules ~2-5mm in size with an upper lobe predominance (Figure 2A). This is the most common pattern seen in chronic silicosis.
- Progressive massive fibrosis (PMF) (Figure 2B) is seen when the small nodules begin to coalesce together.
- Accelerated silicosis often has a simple silicosis pattern, but progresses more rapidly and may also demonstrate the focal ground glass opacities, and patchy areas of consolidation seen in acute silicosis (Figure 3).
- Enlargement of hilar and mediastinal lymph nodes with calcification at the periphery of the node is common (eggshell calcification).
- Although pleural effusions are unusual, pleural thickening is common, especially among patients with more severe disease. Pulmonary function testing is indicated to assess functional impairment, but the presence of obstruction or restriction and degree of abnormality does not consistently correlate with imaging findings.¹⁰

Figure 2. Radiologic Patterns of Silicosis



2A. Multiple nodules visualized in the upper lung zones in axial (top) and coronal (bottom) planes.
2B. Coalescing nodules observed in the upper lung zones in axial (top) and coronal (bottom) planes.
Images adapted from Jones CM et al.¹¹

Figure 3. CT Image of Acute Silicosis



Arrows indicate areas of ground glass opacity in a patient with acute silicosis. Images courtesy of Chong S et al.¹²

Question 3: Although you are reasonably confident of the diagnosis, should you do any additional testing at this time to confirm?

The diagnosis of silicosis requires a history of sufficient respirable crystalline silica exposure, compatible radiological features, and exclusion of alternate diagnoses, such as sarcoidosis, hypersensitivity pneumonitis, tuberculosis, and lung cancer.¹³ There is no laboratory testing that can confirm the diagnosis. Workup is typically focused on excluding other causes or assessing for latent mycobacterial infection, for which these patients are at increased risk. A PET CT can be positive in the presence of PMF, so this is not a good test to exclude malignancy. Lung biopsy is rarely necessary if an adequate history of exposure is obtained, but biopsy confirmation (usually via bronchoscopy) should be obtained if the diagnosis is suspected but exposure history is not present.⁷

PFTs are used to assess the degree of respiratory impairment. Spirometry usually shows a mixed picture of obstructive and restrictive ventilatory impairment with decreased FEV₁ and FEV₁/forced vital capacity (FVC) ratio.¹⁴ Decrement in FEV₁ is more severe in patients with radiologic evidence of disease. In one study of 1028 foundry workers with normal chest imaging there was a 1.1 mL per year decline in FEV₁ for each mg/m³ of mean silica exposure.¹⁵ PMF is associated with the worst pulmonary function abnormalities, including decreased compliance, decreased FEV₁ and FEV₁/FVC ratio, and decreased DLCO.¹⁴

Question 4: Mr. G is distressed to hear that he has lung disease related to his work in the past and wants to know about his options for treatment. What are the guidelines for continuing to survey his disease?

There are no proven targeted treatments for silicosis other than avoiding additional exposure and supportive care. Supportive care includes smoking cessation (if needed), treatment of airflow limitation with bronchodilators, routine vaccinations, and use of supplemental oxygen as needed to prevent complications of chronic hypoxemia.

Nintedanib, an intracellular inhibitor of tyrosine kinases, demonstrated reduction in the rate of FVC decline in the INBUILD trial, which included a small number of patients with

occupational pulmonary fibrosis and is a treatment option in these patients.¹⁶ Lung transplantation in these patients has increased over the last decade with outcomes comparable to transplants in patients who have idiopathic pulmonary fibrosis.¹⁷ There are strict guidelines in place regarding the frequency of chest imaging surveillance in these patients according to OSHA, WHO, and ACOEM. See Table 3 below.

Table 3. Screening Guidelines for Individuals at Risk for Pneumoconiosis

Particulate	Agency	Frequency
Silica	OSHA	• Pre-placement • Every 5 years if <20 years of exposure • Every 2 years if >20 years of exposure • At employment termination
	WHO	• Pre-placement • After 2-3 years of silica exposure • And then every 2-5 years of silica exposure and thereafter
	ACOEM	• Pre-placement • Every 3 years if <10 years of exposure • Every 2 years if >10 years of exposure • At termination

Table adapted from ACCP Pulmonary Board Review 26th Edition.⁴

Scenario 1 (Continued):

You continue to follow Mr. G for several years and, unfortunately, have noted progression of his imaging findings over the years with the presence of PMF. He presents today for his routine follow up visit, and he now reports worsened productive cough, malaise, and night sweats. His CXR is shown below in Figure 4. Is this a manifestation of his silicosis? Are there any additional tests that you should perform?

Figure 4. Interval Chest X-ray of the Patient Described Above



Image courtesy of Radiopaedia.org¹⁸

There is an increased risk of mycobacterial infection (both tuberculous and non-tuberculous), chronic necrotizing aspergillosis, autoimmune disease (specifically, rheumatoid arthritis and systemic sclerosis), chronic renal disease, and lung cancer in patients with silicosis. A cohort study concluded that silica exposure is associated with a

significant increase in lung cancer risk and that a joint effect greater than a merely additive one was detected between silicosis and smoking in the development of lung cancer.¹⁹

In this case, given symptoms, it would be reasonable to evaluate for tuberculosis by collecting sputum for AFB smear and culture. One could also consider a CT scan of the chest to further evaluate the cavitation of the PMF lesion which is concerning for infection, as well.¹⁹

Question 5: Are there any other possible manifestations of lung disease besides silicosis for which Mr. G should be concerned?

Mr. G has a history of foundry work, putting him at risk for silicosis. In addition, he also worked in a coal mine, putting him at risk for Coal Workers Pneumoconiosis. His work as an auto mechanic could raise concern for asbestos exposure. See Table 4 for a list of various occupational exposures and their potentially associated clinical entities.

Table 4: Occupational Exposures and Their Associated Clinical, Radiographic, and Pathologic Entities^{4,20}

Type/Agent	Occupational Exposure	Presentation (if specific)	Clinical Entity/Radiology	Pathologic Description
Asbestosis Fibrogenic type of pneumoconiosis	Pipefitters, steamfitters, electricians, insulation workers, boilermakers, wilders, construction workers and shipbuilders; plastic and rubber manufacturing workers; tam, thread or fabric millworkers; truckers and railway workers; work with brake lining or cement	10-40 year latency period Pleural disease: benign asbestos pleural effusion. Plaques with calcification Risk of Bronchogenic carcinoma	CXR: lower-lobe predominant reticular or multi-nodular opacities CT: subpleural lines; parenchymal and interlobular septal fibrosis (may appear similar to UIP pattern); honeycombing pleural plaques	Ferruginous bodies (asbestos bodies): large asbestos fibers coated with iron from hemosiderin. Asbestos fibers: small uncoated asbestos fibers of different forms within macrophages
Berylliosis Granulomatous type of pneumoconiosis	Beryllium miner, aerospace/aircraft, alloy production; dental and prosthetics, electronics, jewelry, automatic, ceramics, nuclear recycling of electronics and computers, telecommunications	Acute: rare; pneumonitis, tracheobronchitis, nasopharyngitis Chronic: latency period of 3 mo - 40 years (average 10 years), dyspnea, cough, fever, night sweats, fatigue, weight loss	CXR: upper lobe predominant reticulonodular changes, hilar and mediastinal LAD. Multiple small rounded opacities that can calcify; upper lobe scarring, volume loss and bullae CT: parenchymal nodules, septal thickening, ggo, hilar and mediastinal LAD; ground-glass opacities early; small nodules in perilymphatic distribution in the interlobular septa and in subpleural location; upper lobe scarring with volume	Sarcoid-like noncaseating granulomas

			loss and bullae as late manifestations	
Coal Worker's Pneumoconiosis (CWP) Fibrogenic type of pneumoconiosis	Coal dust and coal mining- inhalation and deposition of coal dust particles	Chronic bronchitis is most common Accelerated/complicated: <10 years after exposure Simple: >20 years after exposure	Simple: Upper lobe predominant, small, rounded, nodular opacities; less than 1 cm in diameter Complicated: coalescence of nodules into large masses with distortion and volume loss creating progressive massive fibrosis (PMF) which is similar to silicosis though with lower lobe emphysema; and with cavitation when admixed with silica An atypical idiopathic pulmonary fibrosis pattern has also been described with honeycombing and ggo	Coal macule; focal collection of coal dust in pigment-laden macrophages Centrilobular emphysema Caplan nodule: focal lesion with necrotic center surrounded by lymphocytes and plasma cells
Silicosis Fibrogenic type of pneumoconiosis	Sandblasting, quarrying, drilling, mining, tunneling, drill operation, digging, glass manufacturing, hard rock mining, oven-brick making, stone cutting, masonry, foundry work, natural gas extraction via hydraulic fracturing, sandy and dry soil agriculture	Acute: weeks to 4-5 years after exposure to high concentrations of fine respirable crystalline silica Chronic: 10-30 years after exposure Accelerated: <10 years after exposure PAP (at high level of exposure)	Simple silicosis: innumerable, sharply margined, small rounded opacities with upper lung zone predominance; mid-upper lung zone nodules (<1 cm) CT: multiple small nodules that are sub-pleural and perilymphatic with upper lobe predominance and sharp margins. Nodules can calcify Complicated: conglomerate masses that may have "angel wings" appearance; CT: coalescence of nodules leads to progressive massive fibrosis or conglomerate masses > 1 cm; paracatricial emphysema; punctate or eggshell calcifications in hilar and mediastial lymph nodes	Early: peribronchovascular paraseptal, subpleural dust-laden macrophages Silicotic nodule: concentric collagen fibers around hyaline center Acute: alveolar filling with proteinaceous material (phospholipids and surfactant). Periodic acid-Schiff positive

			Progressive Massive Fibrosis: coalescence of lung nodules >1 cm); egg shell calcification may be seen; upward hilar retraction; lower lobe hyperinflation. Acute: mid-lower lung zone ggo with consolidation (batwing distribution); diffuse ground glass and consolidative opacities with appearance similar to pulmonary alveolar proteinosis CT: diffuse ground glass opacities, consolidation, and intralobular septal thickening	
Cobalt	Metal hardener for tungsten carbide (tool grinder, metal polisher, dental drills); diamond polishing, cobalt mining <i>Hard metal is different from heavy metal. It is produced by sintering=compacting powdered tungsten carbide with cobalt</i>	Asthma/Obstructive airways	Obliterative bronchiolitis Diffuse interstitial fibrosis CT: diffuse ggo, centrilobular ground-glass nodules, consolidation, reticular opacities, and traction bronchiectasis are common; basal predominant small nodular and reticular opacities and small cystic spaces-> can progress to diffuse involvement	Giant-cell pneumonia (multinucleated giant cells), sarcoid-like noncaseating granuloma

Table derived from sources 4 and 20.

There are a number of important emerging occupational exposures associated with various interstitial lung diseases. They are summarized in Table 5 below.

Table 5. Emerging Occupational Exposures and Their Clinical Manifestations

Exposure	Clinical Association/Findings
Denim sandblasting	Silicosis
Inhaled nanoparticles (polyacrylate)	Interstitial pneumonia
Organic chemicals: Isocyanates Polyhexamethylene guanidine phosphate and oligo (2-(2-ethoxy) ethoxyethyl guanidinium chloride compounds Cross-linked acrylic acid-based polymer	Hypersensitivity pneumonitis Interstitial pneumonia Interstitial pneumonia; pulmonary fibrosis

Military deployment (desert dust, burn pit emission products, vehicular diesel exhaust, jet fuel exhaust, oil well fires, debris from detonation, industrial fires, exposures to chemicals, fumes, gases and dusts)	Bronchiolitis obliterans; acute eosinophilic pneumonia; granulomatous pneumonitis; rapidly progressive pulmonary fibrosis
Flavoring materials (diacetyl end parenthesis is missing (2,3-butanedione) and acetylpropionyl (2,3-pentanedione))	Bronchiolitis obliterans
Nylon flock	Nonspecific interstitial pneumonia/fibrosis with bronchiolocentric lymphoid nodules and lymphocytic bronchiolitis; diffuse alveolar damage; organizing pneumonia
Indium tin oxide	Interstitial pneumonia, pulmonary alveolar proteinosis
Man-made disasters (World Trade Center-related lung disease)	Sarcoidosis; interstitial pneumonia; pulmonary fibrosis
Welder's lung (welding fumes from electrode, filler wire and fluxes;; base metal, shielding gases; paint or surface coating; metal oxides)	Respiratory bronchiolitis (with risk of progression to desquamative interstitial pneumonia with ongoing exposure)

Table adapted from Spagnolo et al.⁶

Scenario 2:

Mr. S is a 79-year-old male with a history of diabetes and asthma who comes to see you in clinic for a second opinion. He notes that over the last 2 years he has had increased difficulty with a non-productive cough and dyspnea on exertion. He used to be quite active but now leads a most sedentary lifestyle and feels winded after climbing a flight of stairs. His ROS is otherwise negative. He has also been losing weight unintentionally (about 10 lbs. in the last year). He is a former 30 pack-year smoker who quit about 10 years ago. He currently lives at home with his wife of 45 years. He held many jobs throughout his life including working as a desk clerk and manufacturing dental appliances. He had a CT scan performed (Figure 5).

Figure 5. Representative CT Scan for Mr. S

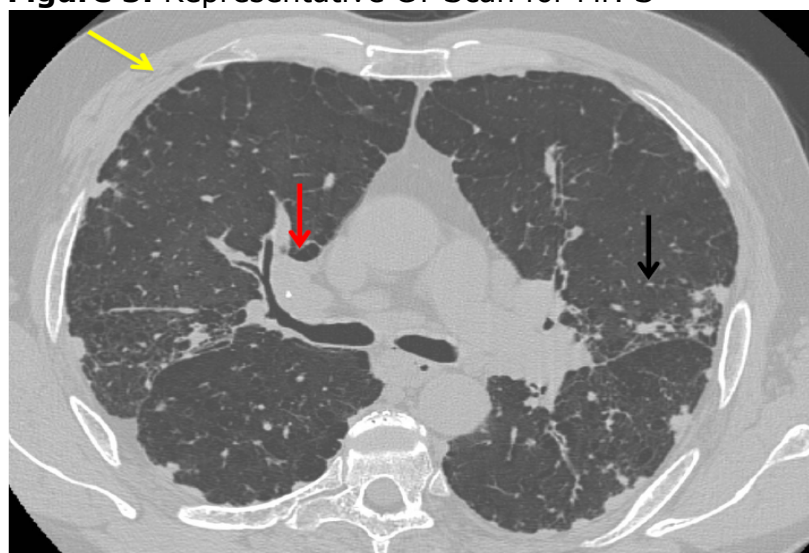


Image reproduced from *ATS Review for the Pulmonary Boards*⁴

Question 6: What do you think is the most likely diagnosis? What testing would you order to confirm?

The occupation of dental appliance manufacturing, clinical history and imaging findings are concerning for possible chronic berylliosis. Exposure to beryllium is most common among workers in metal and metal alloy machine shops, electronics, defense and beryllium extraction, and other industries where workers are exposed to automotive, ceramic, computer, aerospace, jewelry making and dental alloy/appliances as with our patient Mr. S.

The CT scan in Figure 5 shows hilar and mediastinal lymphadenopathy (red arrow), reticulation (yellow arrow) and nodularity along the bronchovascular bundles (black arrow), which are characteristic findings of chronic berylliosis. Typical clinical manifestations of this disease are non-specific and include dry cough, dyspnea, fever, weight loss and asthma-like symptoms if there is bronchial involvement. The latency period between initial exposure and symptoms can range from 3 months to 30 years.

Diagnosis requires the following:

1. Confirmed history of beryllium sensitivity as evidenced by:
 - At least two positive abnormal Beryllium lymphocyte proliferation tests (BeLPT)
 - One abnormal and one borderline BeLPT
 - Three borderline BeLPT
2. Additionally:
 - The presence of noncaseating granulomas on lung biopsy, see Figure 6
 - A positive BAL BeLPT **and** lymphocytic alveolitis >15%
 - The presence of other compatible disease²¹

A clinical diagnosis can be made if histopathology is unavailable based on radiographic imaging and history of exposure with a positive BAL BeLPT, but most patients will require biopsy.

Figure 6. Representative Histopathology of Berylliosis, Showing Compact Granulomas Without Necrosis

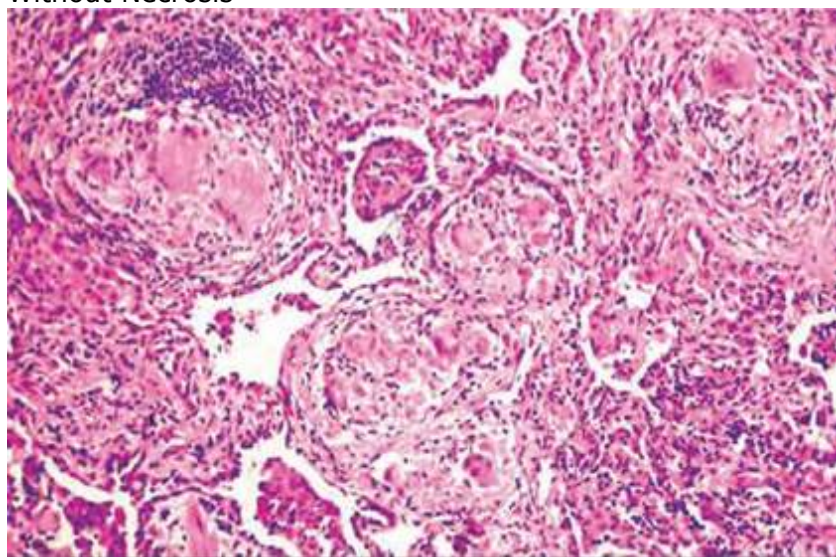


Image courtesy of Roggli VL and Shelburne JD.²²

Question 7: How does berylliosis differ from sarcoidosis?

The differences between berylliosis and sarcoidosis are summarized in Table 6.

Table 6. Clinical Differences Between Chronic Beryllium Disease and Sarcoidosis

Clinical Finding	Chronic Beryllium Disease (CBD)	Sarcoidosis
Beryllium lymphocyte proliferation test	Abnormal	Normal
Ophthalmologic	Conjunctivitis only	Conjunctivitis, uveitis, retinal involvement
Erythema nodosum	No	Yes
Lupus pernio	No	Yes
Onset	Insidious	Acute or insidious
Neurologic involvement	None	Can involve the central or peripheral nervous system
Cardiac involvement	Rare	Occasional
Hepatic involvement	Occasional	Common
Isolated hilar adenopathy	Very rare	Common
Extrapulmonary manifestations without pulmonary involvement	No	Yes

Adapted from Balmes.²³

Question 8: What are Mr. S's options for treatment?

Treatment of Berylliosis is based on severity of disease. The primary focus surrounds avoidance of further exposure to beryllium along with supportive management including vaccination (influenza, pneumococcal) and smoking cessation.

Glucocorticoids are the mainstay of treatment in symptomatic patients or those with greater than 10% decline in lung volumes or DLCO. There is a lack of randomized controlled trial data to confirm the use of steroids, but it is considered standard of care based on case reports and case series.²⁴⁻²⁷

The initial starting dose of prednisone ranges between 20-40 mg daily or 40 mg every other day with a slow taper over 6-12 weeks using a combination of PFTs and symptomatology to guide dose adjustment and taper. If there is a lack of improvement after six months, consideration is made for the addition of steroid-sparing agents such as methotrexate or azathioprine.^{21,28}

The relationship between disease progression and exposure does not appear to be linear although increasing exposure is associated with increased risk of CBD.²⁹ Exact rates of refractory disease progression are not well established. Lung transplantation has been considered in refractory disease.²³

Scenario 3:

Mr. JD is a 44-year-old man who presents to your practice for a second opinion regarding worsening shortness of breath with exertion, as well as persistent productive coughing. He works at Hamm's Blue Sky Coal Company in McDowell County WV. He has worked underground for the past 25 years at various positions inside the mine. He started as a face miner and for the past ten years, he has been the chief roof bolt operator.

Exam reveals a robust plethoric middle-aged man in no distress while sitting. Pulmonary exam is clear though he does cough with deep breathing.

Chest imaging shows mass-like opacities in bilateral upper lobes with volume loss.

PFTs revealed a reduced FEV1 1.9L (55% predicted), reduced FVC 3.2L (75% predicted), FEV1/FVC 60%. His oxygen saturation dropped to 85% on a six-minute walk test (305 meters).

Question 9: What is the underlying mechanism on JD's respiratory findings?

Coal Mine Dust Lung Disease (CMDLD) is the umbrella term used to describe a spectrum of chronic respiratory diseases that are the result of prolonged excessive exposure to coal mine dust. Coal is not pure. It is heterogeneous with a high carbon content along with several other deleterious substances, most notably silica. Respirable crystalline silica (RCS) and diesel exhaust are examples of important drivers of lung injury in coal dust.³⁰

Coal is found in seams within rock from a few feet to hundreds of feet below the ground. Although there are surface mining methods, the majority of coal miners working in the United States today work deep underground as the larger more accessible coal seams have been depleted. This requires tighter working spaces with limited ventilation and advanced technology in the form of pulverizing machines capable of reaching these seams by drilling through high quantities of rock containing silica.³¹ This change in mining practice combined with inadequate workplace protections led to a rise in the prevalence of CMDLD over the past 20 years after reaching a nadir at the end of the 20th century. Alarmingly, there has also been a dramatic rise in more severe disease, namely progressive massive fibrosis (PMF) at younger ages than seen in the past.

The pathological lesion of coal worker pneumoconiosis (CWP) is the coal macule. As coal dust reaches the alveoli, it is phagocytized by macrophages and transported to the walls of the respiratory bronchioles.³⁰ These macules are often surrounded by focal (e.g. centrilobular) emphysema. With repetitive dust exposure and subsequent inflammation more collagen is deposited forming nodules no longer confined to the respiratory bronchioles. With more exposure, multiple nearby nodules begin to coalesce into larger more conspicuous nodules, and when they become larger than 1 cm, PMF is present.

Question 10: What are the clinical phenotypes of CMDLD?

What follows is an alphabet soup of clinical and radiographic manifestations of CMDLD.

Symptoms of CMDLD are nonspecific and mainly involve exertional shortness of breath and productive cough of escalating severity, the distinguishing phenotypic features focus on radiographic and physiologic findings.

By far the most common phenotype encountered is CWP. With an affirming exposure history, typical findings are rounded upper lobe predominant nodules less than 1cm. Once a nodule reaches the arbitrary threshold of 1 cm, complicated CWP is diagnosed which is synonymous with PMF.

A patient can be described as having rapidly progressive pneumoconiosis (RPP) if radiographic progression occurs rapidly (less than 5 years). PMF and RPP appear to be on the rise in the modern (and younger) miners and calls for enactment of modern regulations to protect coal miners. In another recent article, it is estimated that “the mean CWP-attributable years of potential life lost per decedent has increased from 8.1 to 12.6 years” from 1999 to 2016.³²

There are symptomatic patients with coal dust exposure who have radiographic findings of lower lobe predominant subpleural reticulations with and without honeycombing indistinguishable from usual interstitial pneumonia (UIP). This UIP pattern may or may not be accompanied by a background pattern of nodular opacities. This is called dust-related diffuse fibrosis (DDF). Patients with DDF typically have a more indolent clinical course than those with idiopathic pulmonary fibrosis (IPF) with similar radiographic changes.³⁰ To reach a diagnosis of IPF, one has to first exclude occupational exposures that can cause a UIP pattern fibrosis. Since many different conditions can cause the UIP pattern, failure to take an occupational history can miss a diagnosis of DDF.

Coal mine dust is an independent risk factor for emphysema and chronic bronchitis. Studies have shown that emphysema is more frequent in miners than non-miners, even after controlling for the effects of smoking. A recent study shows that more than 1 in 10 former coal miners who never smoked have COPD (defined by airflow obstruction on PFT), including many without radiographic CWP.⁵ Emphysema severity has been shown to be related to cumulative lifetime exposure to coal mine dust.

Rheumatoid pneumoconiosis, also known as Caplan syndrome, is an exceedingly uncommon diagnosis characterized by nodules with or without inflammation and scarring that develops when rheumatoid arthritis and CWP or another occupational dust exposure (such as silica) meet.

Question 11: What additional health outcomes should you inquire about when meeting with JD? What social determinants of health are at play in this case?

Scenario 3 (continued):

Upon further questioning, JD reports regularly trouble sleeping and mood lability. He has trouble concentrating at work and is very irritable when he comes home. A further work history finds that JD over his time in the mine has witnessed five friends die in mining accidents. On one occasion, he and his co-workers worked 30 straight hours to free a friend who had been covered in a mining collapse.

He had his left hand crushed in a rock fall and has chronic pain from that accident. JD had been a promising student who had obtained an academic scholarship to attend a state university in chemical engineering. He ultimately had to forgo this scholarship when his father became disabled working in the mines in order to support his family. His close friends have very similar stories.

If our goal is to have your patients achieve long and healthy lives, we must consider social determinants of health.

Differences in social circumstances and opportunity are often the result of policies and practices at many levels that have created deep-rooted barriers to good health, such as unfair bank lending practices, school funding based on local property taxes, and discriminatory policing and prison sentencing. The collective effect is that a fair and just opportunity to live a long and healthy life does not exist for everyone.

JD lives in a community and county with overall higher mortality rates than nationally. Table 7 shows the stark prevalence of mental health disorders amongst Central Appalachia miners. His county also has increased mortality for COPD, heart disease, stroke, diabetes, and cancer. Smoking rates are double the national average. There is more alcohol and opiate consumption, less primary and secondary health care and screening, lower high school completions rates and higher overall mortality.^{5,32}

Table 7. Mental Health in Central Appalachia³³⁻³⁵

	Central Appalachia Coal Miners	National Comparison
Depression (PHQ9 $\geq$ 10)	883 (37%)	5%
Suicidal ideation	295 (11%)	2.5%
Anxiety (GAD-2 $\geq$ 3)	1005 (39%)	1.9%
PTSD (PC-PTSD4 $\geq$ 2)	639 (26.2%)	5%

Table derived from references 33, 34 and 35.

Question 12: JD asks you about the process for obtaining disability through the mining company. How would you advise him?

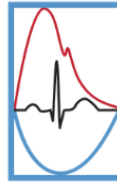
Coal miners (and their survivors) may be eligible for state and federal compensation programs if they are deemed to have disabling pulmonary disease (e.g. COPD, DDF, CWP, PMF) that is attributed in part to their occupational dust exposures. The benefits that coal miners can receive through this program can be life-changing – including supplemental health insurance as well as an indefinite monthly stipend. Determining whether a miner qualifies for these benefits is challenging and often requires a specialized evaluation at a clinic certified by the Department of Labor (or by individual states for the state compensation program). The exam may include PFT, CXR, and a treadmill stress test.

All future pulmonologists should familiarize themselves with clinics in their region that they can refer their coal miner patients to for further evaluation. Here is a good place to start to help identify a local clinic: <https://www.hrsa.gov/rural-health/grants/black-lung>.

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