Chest radiology: Lung diseases by distribution, Matt Covington, MD

Listen to the associated podcast episodes from The Radiology Review Podcast, available at theradiologyreview.com or on your favorite podcast directory. Copyrighted.

Upper lobe Predominant

1) Pneumoconioses
    a) Silicosis
    b) Coal Workers Pneumoconiosis
    c) Progressive massive fibrosis
    d) Berryliosis
    e) Inhalation talc pneuomoconiosis
2) Tuberculosis
3) Sarcoidosis
4) Respiratory bronchiolitis-interstitial lung disease (RB-ILD)
5) Caplan syndrome
6) Ankylosing spondylitis
7) Allergic bronchopulmonary aspergillosis (ABPA)
8) Centrilobular emphysema
9) Cystic fibrosis
10) Langerhans Cell Histiocytosis

Lower Lobe predominant

1) Usual Interstitial Pneumonia (UIP)
2) Nonspecific Interstitial Pneumonia (NSIP)
3) Desquamative interstitial pneumonia (DIP)
4) Scleroderma
5) Rheumatoid arthritis (exception is Caplan syndrome)
6) Secondary emphysema from alpha-1 antitrypsin deficiency
7) Asbestosis (the only common inhalational disease that is lower lobe predominant)
8) Lymphocytic interstitial pneumonia (LIP)
9) Cryptogenic organizing pneumonia (COP)
10) Primary ciliary dyskinesia

Diffuse, Central, RML/lingula, and/or Randomly Distributed

1) Granulomatosis with polyangiitis (Wegener's granulomatosis)
2) Goodpasture syndrome
3) Eosinophilic granulomatosis with polyangiitis (prior Churg-Strauss)
4) Hypersensitivity pneumonitis (hard to classify but can have findings in various areas of lung)
5) Pneumocystic jiroveci pneumonia (PJP) *also hard to classify
6) Pulmonary amyloidosis
7) Lymphangioleiomyomatosis
8) Acute interstitial pneumonia/ARDS
9) Miliary tuberculosis
10) Pulmonary alveolar proteinosis
11) Pulmonary edema
12) Mycobacterium avium complex (MAC)

Listen to the associated podcast episodes from The Radiology Review Podcast, available at theradiologyreview.com or on your favorite podcast directory. Copyrighted.

Upper Lobe Predominant:

Silicosis: Upper lobe predominant perilymphatic nodules which may be calcified and hilar nodes with eggshell calcifications. Look for history of working in a mine or other industrial exposure. May progress to progressive massive fibrosis with large upper lobe masses with radiating strands. If see cavitation must rule out silicotuberculosis given higher risk of TB when silicosis is present.

Coal Workers Pneumoconiosis: Similar to appearance of silicosis including higher risk of developing TB, risk of progressing to progressive massive fibrosis.

Progressive massive fibrosis: I think of this as end stage silicosis/coal workers pneumoconiosis. Upper lobe predominant masses. May also be seen with other processes such as inhalational talc pneumoconiosis. PMF is often T2 dark on MRI vs cancer that is often T2 bright. In reality, I'm not sure how many of these patients really get an MRI or if the T2 dark finding has good enough diagnostic performance to avoid tissue sampling but you should know this for the board exam.

Berryliosis: Results from inhalation of metal particles used in aircraft and other industries. Results in upper lobe predominant granulomatous disease and fibrosis including hilar adenopathy and reticular opacities. Bronchoalveolar lavage can be helpful for diagnosis.

Inhalational talc pneumoconiosis aka talk-induced lung disease: Be aware that some consider "pulmonary talcosis" to denote the IV injection of talc (filler in tablets such as pain pills that are crushed and injected for IV drug use) which ends up embolized in the pulmonary arteries. So to be specific for lung disease use the other terms specified in bold. For inhalational talc pneumoconiosis look for hyperdense micronodules in upper lobe predominant distribution that can conglomerate into masses much like progressive massive fibrosis. This inhalation classically happens from occupational exposure rather than drug abuse, look for a history of a rubber factory worker. This is also different from talc pleurodesis but you should be familiar with that as well (intentional talc injection into pleural space to prevent/reduce recurrent pleural effusion/pneumothorax--look for pleural thickening and pleural calcification due to talc).

Tuberculosis: Any cavitation in setting of silicosis/coal workers pneumoconiosis is assumed to be TB until proven otherwise. Is upper lobe predominant in chronic form with exception of miliary TB which is bilateral and diffuse micronodularity. If you see upper lobe tree-in-bud opacities with cavitation think TB first. May also see upper lobe patchy consolidation/nodularity. Get bronchoalveolar lavage for diagnosis. If you see upper lobe fibrosis think old TB, progressive pulmonary fibrosis, sarcoidosis. Acute (primary) TB can be opacity anywhere in lung, even presenting with lobar consolidation. Cavitation is much more

common in chronic TB. When infection is walled off and forms a caseating granuloma that calcifies, that is your Ghon complex. Look for the right>left mediastinal lymphadenopathy for primary TB, especially in kids. Ranke complex=Ghon complex and mediastinal/hilar nodal calcification. Miliary TB means hematogenous spread and has a poor prognosis. TB associated with increased risk of aspergilloma.

Sarcoidosis: This can look like just about anything anywhere in the body. Almost all patients with sarcoidosis will show lung changes. If provided history is that of an African American young adult woman with lung findings be ready to look for findings of sarcoidosis. Classic findings are symmetric mediastinal and hilar lymphadenopathy, micronodularity, other lung opacities, pleural effusions, less common with masses and cavitation. Late stage sarcoidosis may show fibrosis/honeycombing/bronchiectasis. Note that nodules and fibrosis are most common in upper lung zones.

Respiratory bronchiolitis-interstitial lung disease (RB-ILD): Smoking related. On spectrum with desquamative interstitial pneumonia (DIP)--these are the same disease. RB-ILD is slightly upper lobe predominant and DIP is lower lobe predominant. RB-ILD has apical centrilobular ground glass nodules. DIP is basilar predominant ground glass opacities with small cysts often in subpleural location. RB-ILD is early change and DIP is late stage. RB-ILD is indistinguishable on imaging from acute hypersensitivity pneumonitis so during board exams look for the smoking history to confirm RB-ILD.

Caplan syndrome aka rheumatoid pneumoconiosis: Pulmonary fibrosis in setting of rheumatoid arthritis. Upper lobe predominant nodules. This is essentially pneumoconiosis from various cases in patient with rheumatoid arthritis.

Ankylosing spondylitis: Pulmonary presentation includes upper lobe fibrosis and bullae. May start unilateral and then become bilateral. Additional manifestations of ankylosing spondylitis are highly tested and are beyond the scope of this pulmonary review but should be carefully studied.

Allergic bronchopulmonary aspergillosis (ABPA): Upper lobe predominant endobronchial lesion. Look for hyperdense material (calcified mucoid impaction) within dilated upper lobe bronchi--"finger in glove sign". Look for history of asthma and eosinophilia. As per name, this is an allergic phenomenon and not an infection. Less common in patients with cystic fibrosis. Mild cases may only need corticosteroids, antifungals may also be used. If immunocompromised may develop angioinvasive aspergillosis including if on long-time high dose steroids. This is dangerous, may have associated hemoptysis, and on imaging may show a halo around a nodule due to hemorrhage or atoll/reversed halo sign which is central ground glass opacity surrounded by denser consolidation. May see wedge-like consolidation from

pulmonary infarction and direct invasion into chest wall. Note reverse halo sign is more classic for cryptogenic organizing pneumonia but may also be seen with invasive fungal infections and other entities (TB, sarcoid, etc.).

Centrilobular emphysema: Smoking related. Look for evenly distributed areas of absent lung (low attenuation) in upper lobes. Small pulmonary vessels.

Cystic fibrosis: Upper lobe bronchiectasis with recurrent infections. Really if they give you history of recurrent infection and show upper lobe bronchiectasis it is CF. Bronchiectasis in order of increasing size is: cylindrical, varicoid and cystic. CF has cystic bronchiectasis. May see air-fluid levels, asymmetric consolidation. Cause is too little chloride removal due to genetic abnormality resulting in thick secretions. May see air trapping/mosaic attenuation and finger in glove sign from mucous impaction. (Finger in glove with recurrent infection is CF, finger in glove with asthma/eosinophilia is ABPA). Highly tested and also make sure to review the other non-pulmonary manifestations of CF.

Pulmonary Langerhans Cell Histiocystosis (LCH): Nodules and irregular/bizarre cysts in upper lobe predominant distribution sparing the extreme lung bases/costophrenic sulci. Late disease may have fibrosis/honeycombing. Associated with smoking in young adults (by far most common) or a manifestation of disseminated LCH and these are distinct processes with same imaging appearance. LCH is upper lobe predominant, lymphocytic interstitial pneumonia (LIP) is lower lobe predominant, lymphangioleiomyomatosis (LAM) is all over. Treatment is smoking cessation (if smoking related) and/or corticosteroids. If severe may need lung transplant.

Listen to the associated podcast episodes from The Radiology Review Podcast, available at theradiologyreview.com or on your favorite podcast directory. Copyrighted.

Lower Lobe Predominant:

Usual Interstitial Pneumonia (UIP): This has a poor prognosis so it is unfortunate that this is the most common interstitial lung disease. I think of interstitial lung disease as either UIP or NSIP with UIP being the bad one (I think *usual interstitial pneumonia* is *usually* terrible to help me remember that fact). This presents with low lung volumes and fibrosis with reticulation/honeycombing and a smaller ground-glass component starting in the posterior costophrenic sulcus with subsequent apical to basal gradient (most severe at lung bases) as things progress. This is heterogeneous on histology and has spatial and temporary heterogeneity (NSIP is homogeneous). Classic is basal and peripheral predominant honeycombing with traction bronchiectasis from the fibrosis. Typically, no air trapping unlike chronic hypersensitivity pneumonitis which can have similar appearance to UIP but will also have air trapping and may not be basilar predominant. UIP takes typically a year or so to really progress unlike chronic ARDS which progresses with rapid fibrosis. Almost all patients with UIP will die within about 5 years. About 10% will develop cancer, often in region of fibrosis. Etiology includes idiopathic, collagen vascular disease/connective tissue disorders, asbestosis, drug toxicity, aspiration, chronic hypersensitivity pneumonitis. If classic imaging and clinical history you do not need to biopsy to confirm UIP. Note that UIP is the radiology and pathology manifestation of the clinical syndrome that is idiopathic pulmonary fibrosis. IPF has chronic dyspnea, cough, possible finger clubbing, abnormal PFTs.

Nonspecific Interstitial Pneumonia (NSIP): Two varieties: cellular and fibrotic. Both have better prognosis than UIP but cellular NSIP also has better prognosis than fibrotic NSIP. Response to steroids is often more robust for the cellular NSIP subtype. Histology shows homogeneous inflammation and fibrosis unlike the heterogeneity of UIP. NSIP: temporal and spatial homogeneity. UIP temporal and spatial heterogeneity. Posterior, peripheral, lower lobe predominant with immediate subpleural sparing in about half of cases with ground-glass opacities. Boards love the immediate subpleural sparing aspect so try to remember that. If you only see symmetric basilar ground glass opacities with subpleural sparing think cellular NSIP. If you see symmetric basilar ground glass opacities and reticulation think cellular or fibrotic NSIP. If you see symmetric ground glass, fibrosis and traction bronchiectasis (with possible minimal honeycombing) think fibrotic NSIP. If you see a lot of honeycombing and heterogeneity think UIP. Causes of NSIP are broad but include cryptogenic organizing pneumonia (COP), drug reaction, collagen vascular disease, other autoimmune disease, idiopathic.

Also, another pearl:
-Rheumatoid arthritis: UIP>NSIP.
-Systemic sclerosis: NSIP>UIP.
-Sjogren's: think LIP first

Desquamative interstitial pneumonia (DIP): The end-stage of the RB-ILD->DIP spectrum. Strongly associated with cigarette smoking. Look for diffuse symmetric ground-glass opacities in a current or prior smoker, basilar predominant. This results from long-standing bronchiolar inflammation, accumulation of pigmented macrophages in the small airways, and some fibrosis. This would not be expected to have the subpleural sparing of NSIP. You can't distinguish based on imaging alone from acute hypersensitivity pneumonitis so look for the chronic smoking history for DIP or a more acute exposure of another sort for acute HP such as massive smoke or dust inhalation, etc. Most common in middle aged males. Treatment is smoking cessation and steroids, good prognosis with successful treatment.

Scleroderma: Look for lower lobe predominant lung opacities. If see NSIP (or possibly UIP) look with basilar ground-glass opacities and a dilated, fluid filled esophagus they are probably wanting you to identify possible scleroderma as an underlying factor/disease. With scleroderma NSIP is more common than UIP. Scleroderma lung involvement is seen at least eventually in almost all patients with scleroderma. May have pulmonary cysts and mediastinal/hilar nodal calcifications as well. Increased risk of lung cancer and commonly have pulmonary arterial hypertension.

Rheumatoid arthritis: Basilar predominant with exception of Caplan syndrome which is upper lobe nodules/fibrosis in setting of RA. Associated with follicular bronchitis which has lymphoid hyperplasia looking like small centrilobular ground glass nodules with scattered dilated bronchi—a tree-in-bud appearance. Can also have larger rheumatoid nodules in the lungs that may cavitate. May see consolidative opacities as well which are basilar predominant and a form of organizing (non-infectious) pneumonia. On imaging look for the associated osseous erosions, for example erosions of the medial clavicular heads, or superior rib notching, with lung changes, to realize they are asking you about rheumatoid arthritis.

Secondary emphysema from alpha-1 antitrypsin deficiency: Lower lobe predominant pan-acinar emphysema. Pan-acinar emphysema in lower lobes think A1AT. Centrilobular emphysema in upper lobes think smoking related. Results from lack of neutrophil elastase inactivation due to genetic abnormality. If smoking and have this emphysema progresses faster. On chest radiograph expect basilar bullae and preservation of upper lobe vasculature. Association with aneurysms so could show an aneurysm on brain imaging with basilar emphysema and expect you to make the connection. VQ scan would show a matched defect at lung bases as both ventilation and perfusion are reduced. Also associated with cirrhosis, pancreatitis, and lung cancer.

Asbestosis: The only common inhalational disease that is lower lobe predominant. Asbestosis is pulmonary fibrosis related to asbestos exposure. Exposure without fibrosis is not technically asbestosis. This may look a lot like UIP but you will also see pleural thickening and have a history of occupational or environmental exposure to make you think this is a pneumoconiosis.

If they start giving you a history of inhalational exposure and then you see the lower lobes with honeycombing and pleural thickening it is a slam dunk for asbestosis. Look for the classic "shipyard" work history. Get a bronchoalveolar lavage to confirm the diagnosis. It takes about 20 years to start getting the lung cancer/mesothelioma but only about 5 years to get pleural effusions. It may take more than 20 years to get the calcified pleural plaques. Don't forget about peritoneal mesothelioma and other abdominal cancers as well. Also don't forget about the round atelectasis with associated pleural abnormalities, which may be considered an asbestosis pseudotumor. Why is asbestosis the only common lower lobe pneumoconiosis? Because the asbestos particles are relatively big and heavy, therefore deposit into the lower lung, and are too large to be removed by lymphatics.

Related point: 80% of mesothelioma patients have asbestos exposure and development of mesothelioma is thought to not be dependent on the amount (dose) of asbestos exposure. It can take 30-40 years from exposure to get mesothelioma.

Lymphocytic interstitial pneumonia (LIP): Associated with Sjogren's disease and other connective tissue/autoimmune disorders. Look for extensive ground-glass opacities with scattered thin-walled cysts in mid and lower lung zones. Cause is small lymphoid hyperplasia causing a ball and valve mechanism in the small airways causing peripheral ballooning of airway/pulmonary cysts. Therefore, mediastinal adenopathy is also common as this is a lymphoid hyperplasia process. Can also see in patients with lymphoma (which LIP/Sjogren's patients are at risk to develop) or HIV. If see this in a kid it is AIDS until proven otherwise.

To review:
Pulmonary LCH: Upper lobe predominant with bizarre cysts and nodules.
LIP: Lower lobe predominant, uniform cysts, Sjogren's disease.
Lymphangioleiomyomatosis: Diffuse distribution of cysts, often younger females.

Cryptogenic organizing pneumonia (COP): Top cause of NSIP. Expect mid and lower lung zone consolidation with peripheral clearing and absence of subpleural involvement. Consolidation may migrate over time through the lungs. Small peribronchial nodules also common. May see reverse halo (atoll) sign which is specific for COP which is central ground-glass opacity surrounding by crescentic dense consolidation in a ring-like pattern. You can also see perilobular consolidation in an arcade-like pattern which to me look like a ring like pattern of multiple spicules in a radial pattern. Etiology of COP is unknown (hence cryptogenic). Organizing pneumonia is alveolar inflammation from a known non-infectious cause (for example a drug reaction, collagen vascular disease). If etiology is unclear OP would become COP. Previously termed bronchiolitis obliterans organizing pneumonia (BOOP) which is distinct from bronchiolitis obliterans. Treat with corticosteroids.

Listen to the associated podcast episodes from The Radiology Review Podcast, available at theradiologyreview.com or on your favorite podcast directory. Copyrighted.

Primary ciliary dyskinesia aka immotile cilia syndrome: Lower lobe predominant bronchiectasis. Associated with dextrocardia/situs inversus as well as impaired fertility and chronic sinusitis. These are all frequently tested associations. Other less tested associations include biliary atresia and pectus deformity. Know that cystic fibrosis is upper lobe bronchiectasis and primary ciliary dyskinesia is lower lobe bronchiectasis. This has an autosomal recessive inheritance pattern. Kartagener syndrome is triad of bronchiectasis, chronic sinusitis, and situs inversus.

Bonus tip: Young syndrome has similar lower lobe predominant bronchiectasis and azoospermia and possible sinusitis but is of unknown etiology. The cilia are fine in Young syndrome but it has a lot of clinical similarities.

Listen to the associated podcast episodes from The Radiology Review Podcast, available at theradiologyreview.com or on your favorite podcast directory. Copyrighted.

Diffuse, Central, RML/lingula, and/or Randomly Distributed Lung Diseases:

Granulomatosis with polyangiitis (prior Wegener's granulomatosis): Triad is commonly tested although not super common and consists of upper respiratory tract, lung (95% involvement), and renal involvement. Expect random nodules with cavitation on chest CT with additional history of renal or upper respiratory tract abnormalities including hematuria and hemoptysis. Pulmonary hemorrhage around nodules may happen and would look like ground-glass surrounding the nodule. Labs show c-ANCA positivity.

Goodpasture syndrome aka antiglomerular basement membrane antibody disease: An autoimmune pulmonary renal syndrome most common in younger men with diffuse alveolar hemorrhage from the pulmonary capillary inflammation (pulmonary capillaritis. Chest imaging shows bilateral coalescent airspace opacities that may mimic pulmonary edema but actually results from pulmonary hemorrhage (pulmonary edema=fluid in alveolar spaces/Goodpasture syndrome=blood in alveolar spaces). Recurrent episodes of pulmonary hemorrhage can lead to fibrosis and hemosiderosis (iron deposition) causing appearance of multiple small nodules. More advanced disease may present with crazy paving. Need to exclude infection before you treat with steroids.

Eosinophilic granulomatosis with polyangiitis (prior Churg-Strauss): Is an eosinophilic lung disease and most patients have associated asthma and eosinophilia. Expect migratory lung opacities that are transient. If see nodules with cavitation think granulomatosis with polyangiitis (prior Wegener's) instead of this. May have additional cardiac, renal, CNS, GI involvement. Treat with steroids.

ANCA:
c-ANCA think granulomatosis with polyangiitis first
p-ANCA think Goodpasture syndrome, eosinophilic granulomatosis with polyangiitis (prior Churg-Strauss) first

Hypersensitivity pneumonitis: Results from a type 3 hypersensitivity reaction (type 1 hypersensitivity reaction is asthma) resulting from an immune reaction to an antigen with hundreds of known antigens that can cause HP. Smoking may be protective against hypersensitivity pneumonitis due to diminished antibody response (the antibodies throw in the towel due to the constant barrage of cigarette smoke), but I still do not advocate cigarette smoking. Has acute, subacute, and chronic phases. List of possible antigens is extensive and includes many farm/food preparation entities (mushroom worker's lung, cheese worker's lung, bird fancier's lung), many industrial activities (wine maker's lung, machine operator's lung), the classic hot tub lung, and some interesting ones such as saxophone lung and shower curtain disease.

Acute HP is rare, requires a fairly significant inhalational event (such as caught in dust storm without mask or rescued from house fire with lots of smoke inhalation), presents with edema and centrilobular nodules, diffuse alveolar damage/hemorrhage, and is indistinguishable from DIP but history will be very different (acute massive inhalation event is acute HP, chronic smoking is DIP).

Subacute HP presents with many ill-defined ground-glass nodules and mosaic attenuation. No or only minimal fibrosis but you start to see changes from inflamed airways (mosaic attenuation resulting from air trapping). Subacute HP is most common for the head cheese sign and this is a sign that you should associate with HP. This results from the mixture of mosaic attenuation, normal lung, and ground-glass opacities. You need to know what this looks like so look it up.

Chronic HP: Fibrosis in lungs from long-term exposure presenting with dyspnea, finger clubbing, etc. On CT expect pulmonary fibrosis and honeycombing, mosaic attenuation from airway narrowing, ground-glass opacities. This may spare the lung bases unlike UIP/NSIP.

Mosaic attenuation: lung pattern with areas of varying pulmonary parenchymal attenuation resulting from reduced ventilation (air trapping), reduced perfusion (oligemia), mixture of above, or parenchymal problems.

Pneumocystic jiroveci pneumonia (PJP): Presents primarily in HIV/AIDS patients or less commonly in otherwise immunocompromised or bone marrow transplant patients. CT shows ground-glass opacities, septal thickening (therefore a possible crazy paving pattern) and possible cysts/blebs/pneumatoceles that form in area of infection. Expect CD4 count <200. Groundglass opacities most common in perihilar/mid lung zones. There is a cystic variation that is upper lobe predominant with bilateral upper lobe cysts and pneumothorax risk. Don't forget about the diffuse gallium-67 pulmonary uptake that is classic for this and frequently tested. Lack of gallium-67 uptake = no PJP.

*If you see similar clinical history and imaging findings and they ask about CMV remember this can look very similar to PJP pneumonia but CMV does not have the cystic pattern with PJP.

Pulmonary amyloidosis: Presents with multiple calcified pulmonary nodules. There are other forms that can look like other things in the lungs but the one I would be most familiar with for the Core exam is the nodular variant with nodules that have central and/or irregular calcifications with slow growth over years. Sjogren syndrome association. Nodules can look similar to pulmonary hamartomas.

Diffuse

Lymphangioleiomyomatosis: Expect a history of a woman of child bearing age with tuberous sclerosis. Diagnosis is based on "possible", "probable" or "definite" LAM which is combination of imaging findings and other criteria such as biopsy, things like associated renal angiomyolipoma, thoracic or abdominal chylous effusion, so forth. CT findings alone without the other supporting factors is "possible" LAM. Imaging shows multiple uniformly distributed thin-walled cysts, may have associated chylous pleural effusion. Extra-thoracic findings include abdominal chylous ascites, renal AMLs, uterine fibroids, splenic cysts, and cystic hygroma. Risks include recurrent pneumothorax.

Acute interstitial pneumonia/ARDS: Results from alveolar injury with leakage of fluid into the alveolar space, ie non-cardiogenic pulmonary edema. ARDS requires both the imaging findings and respiratory failure that is of non-cardiac or volume-overload etiology. Many things can cause this including viral illness and entities like burn injuries, pancreatitis, post-traumatic, head injury, etc. Expect some involvement of groundglass opacities in all 5 lobes with some normal areas of lung as well. CT may show a gradient effect where dependent lungs show consolidation on background of groundglass in the middle and normal lung in the non-dependent lung. If unresolved but the patient survives can lead to rapid fibrosis within weeks and the minotiry of patients will recover completely. Would look like UIP pattern that developed in weeks rather than years.

Miliary tuberculosis: Tuberculosis was previously discussed. Miliary pattern is random nodules throughout both lungs. Differential considerations for a miliary pattern includes hematogenous metastases such metastatic thyroid cancer, melanoma, or renal cell carcinoma.

Pulmonary alveolar proteinosis: An intrinsic lipoid pneumonia with abnormal surfactant clearance with a highly tested, classic look that is crazy paving pattern in both lungs with batwing appearance on chest x-rays. Male smokers are at particularly high risk. Cause is the abnormal proliferation two pneumocytes that make fat like surfactant that become ingested by macrophages that don't work correctly due to autoimmune issues and can test for anti-GM-CSF antibodies. Treatment includes intubation with therapeutic lavage of each lung with large amounts (10-15 liters) of fluid. If see this in the first year of life remember association with lymphoid tissue hypoplasia presenting with low lymphocyte counts and reduced/absent thymic tissue. Classic is imaging findings worse than expected for clinical presentation. Superimposed infection is a concern.

Crazy paving: admixture of ground-glass opacities with inter- and intralobular septal thickening. For board purposes this is most classic for pulmonary alveolar proteinosis (PAP), but may also be seen with ARDS and other infectious/inflammatory processes such as bacterial pneumonia, Goodpasture syndrome, UIP, COP, etc.

Pulmonary edema: May have cardiac and non-cardiac causes. Lots of classic radiograph findings like Kerley lines, batwing opacification, pleural effusions, etc. CT expect ground-glass opacities, often central, interlobular septal thickening, possible superimposed consolidation and pleural effusions.

*Imaging appearance similar to diffuse pulmonary hemorrhage, pulmonary alveolar proteinosis (see above) and diffuse pneumonia/infection. Diffuse hemorrhage does not have a pleural effusion and typically no increase in the dependent lung.

Mycobacterium avium complex (MAC): The most common nontuberculous mycobacteria in North America. May present with a cavitary pattern, nodular bronchiectasis pattern, and a hypersensitivity pneumonitis pattern. Cavitary pattern can mimic TB and is most common in older men with COPD, presents with thick-walled cavities with adjacent consolidation and possible tree-in-bud opacities. Nodular bronchiectasis pattern is most common in older, thin women and has the classic tendency to present in the right middle lobe and lingula (Lady Windermere syndrome) which is more likely indolent. This may also present with a hypersensitivity pneumonitis pattern and MAC is likely to be the etiology of “hot tub lung”. Patients with bronchiectasis more at risk (cystic fibrosis/alpha 1 antitrypsin). Get a sputum culture to confirm the diagnosis. Treatment/clearance of disease can be difficult, similar to TB.