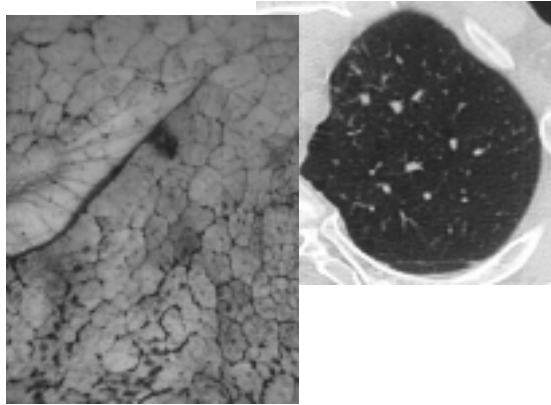


Practical Approach HRCT

Jud W Gurney MD FACR
Charles A Dobry Professor of Radiology
University of Nebraska



HRCT Technique

Maximize spatial resolution

- thin collimation

- high spatial algorithm

Advantages over chest x-

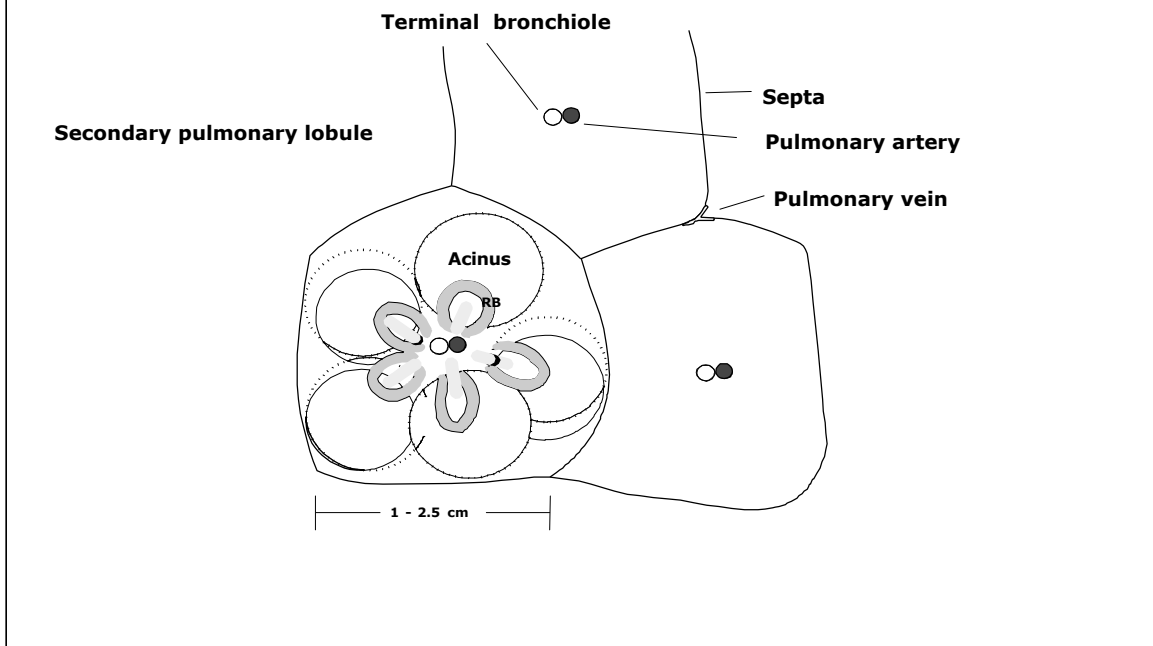
- no summation

- high contrast

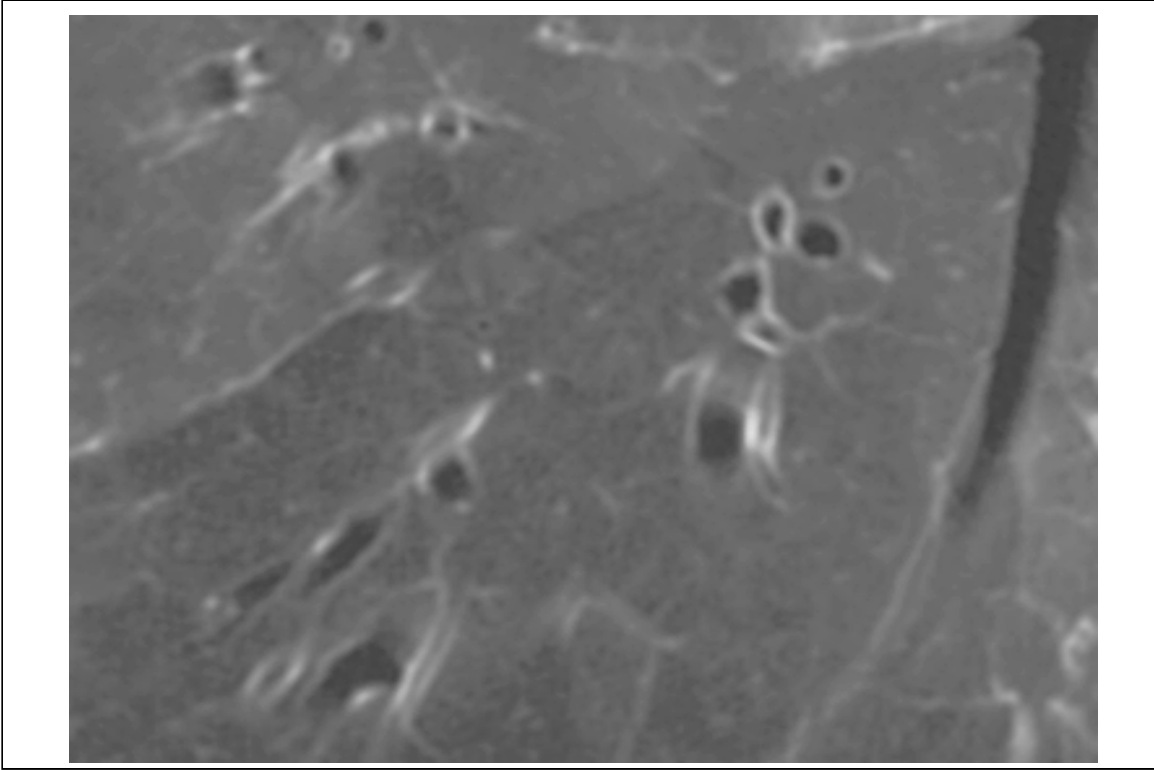


There is no magic with HRCT. The basic premise is simple, maximize spatial resolution by using the thinnest collimation and a high spatial frequency algorithm. This use to be 1.5 mm. Modern technology has cut the slice thickness in half and with multi-detector CT any scan can be a high resolution scan. Even without high resolution, CT has inherent advantages over chest radiography.

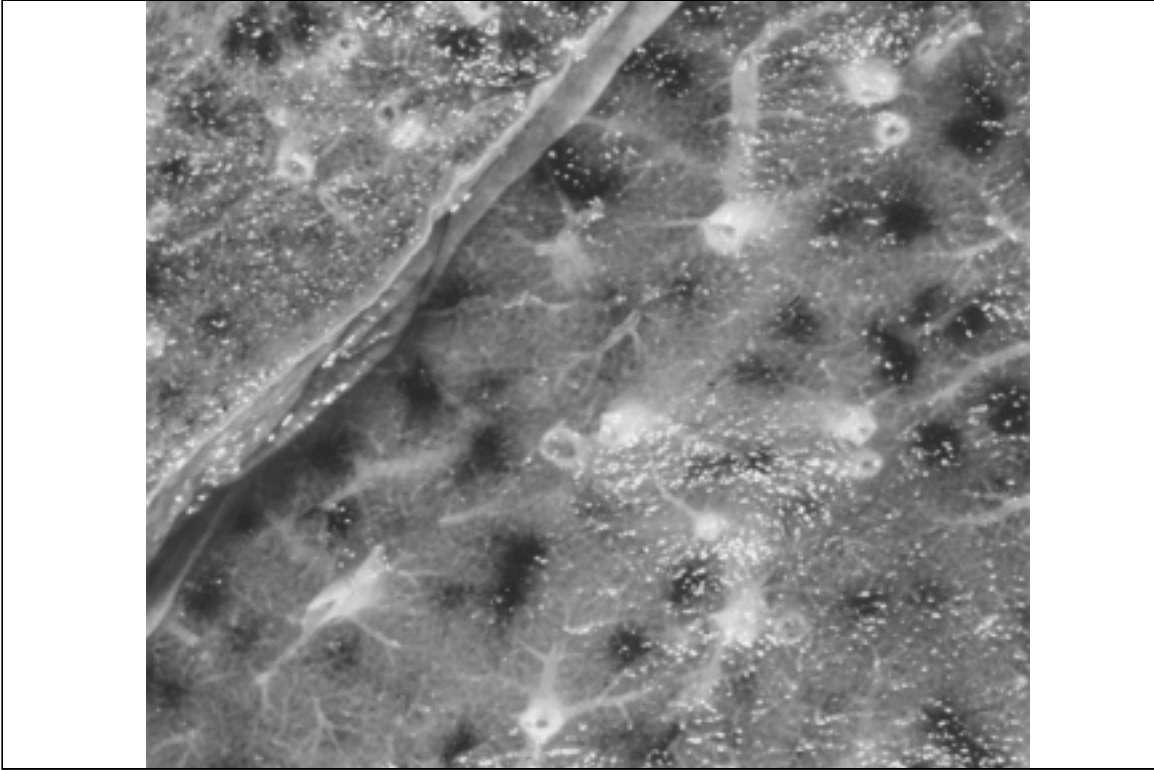
Secondary pulmonary lobule



A quick review of anatomy. One of the building blocks of the lung is the secondary pulmonary lobule. Lobules are relatively large: 1 to 2.5 cm, the size of a good thumbprint. The lobule is fed by a terminal bronchiole and artery, the periphery is partly septated. In between are the primary lobules fed by respiratory bronchioles where gas exchange takes place. Radiographically we can identify the largest of the arteries and airways and the septa. Veins run in the septa. There are 2 lymphatic systems, an axial system travels down the arteries and airways, terminating at the terminal bronchioles and a peripheral system that travels along the septa and veins. The yellow and gray rings are the centriacinar portion of the lobule.

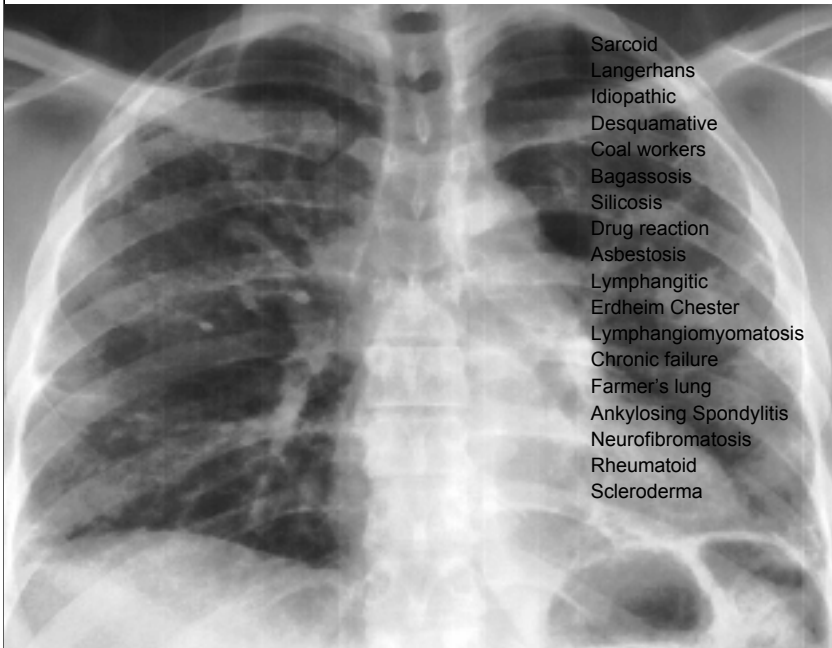


HRCT dried lung specimen. The larger terminal arteries and airways are clearly visible as are the occasional septa. However, the respiratory bronchiole and acinus are not resolvable.



In this fresh autopsy lung, the black pigmentation marks the centriacinar portion of the lobule. Anthracotic particles (environmental soot) is deposited in the respiratory bronchioles. Notice the off-axis location of the respiratory bronchioles compared to the core bronchovascular structure.

Diffuse Lung Disease



The number of insults that may give rise to pulmonary disease is large anywhere from 100-150. It might seem a hopeless task to sort out this differential. Once when I was doing a locums I came across a report that read: “the patient has diffuse interstitial lung disease. Please stop by my office to review the list of 150 possibilities”

Most common diagnoses

1. IPF
2. Farmers lung
3. Sarcoid
4. Asbestosis
5. Boop
6. Langerhans
granulomatosis

80% London
Padley

1. UIP
2. Silicosis
3. Sarcoid
4. Lymphangitic ca
5. Farmers lung

83% Vancouver
Matheson

In reality, the number of problems that we deal with are a lot less. For example, this small list of diseases accounted for over 80% of the diagnosis at top referral centers in England and North America. While the spectrum of disease differs slightly, such as asbestos in England and silicosis in British Columbia, the number of diseases one has to consider is quite small.

Round up the usual suspects

More than 100 entities manifest as diffuse lung disease. Fortunately only 10-20 of these account for about 90% of all diffuse lung disease that is assessed by open lung biopsy.

DeRemee Chest 92, 1987

Sarcoid
Langerhans granulomatosis
Idiopathic pulmonary fibrosis
Lymphangitic tumor
Edema
Asbestosis
Collagen vascular diseases
Silicosis
Farmer's lung
Drugs



Combining these 2 lists and my own practical experience, these 10 processes account for over 90% of what I see in my daily practice. The theme here comes from the famous scene in Casablanca after Ric has shot the German general. Louie, the French police captain, has to make a decision as to which side he will choose. When his lieutenants come running he utters the line; “ the general has been shot, round up the usual suspects”. This has become a running joke among the pulmonologists I work with as this is our approach. When we have a new patient with interstitial lung disease we look for the usual suspects before considering a more exotic disorder.

Sarcoidosis

Common systemic disease unknown etiology

May follow treatment lymphoma

Noncaseating granulomas

Most good prognosis, resolves within 2 years

We will start with sarcoidosis, one of the commonest of the interstitial lung diseases. We don't know what causes sarcoid, however, one curious bit of evidence is that sarcoid may follow treatment of lymphoma. It is important to recognize this possibility as the development of interstitial lung disease and adenopathy in a patient with a history of lymphoma is usually equated with recurrence and not a new disease. The pathologic finding is the noncaseating granuloma that is primarily located in the lung lymphatics. Most with sarcoid do well, only a small minority go on to develop end-stage disease.

Sarcoidosis

95% abnormal chest radiograph

Symmetric hilar adenopathy

Lung disease (<50%), often worsens with nodal regression

Most have an abnormal chest radiograph. Classic findings of symmetric hilar adenopathy may be found alone or associated with lung disease. One of the distinguishing features in sarcoid is the paradoxical response that as the lung disease worsens the nodal enlargement usually regresses.

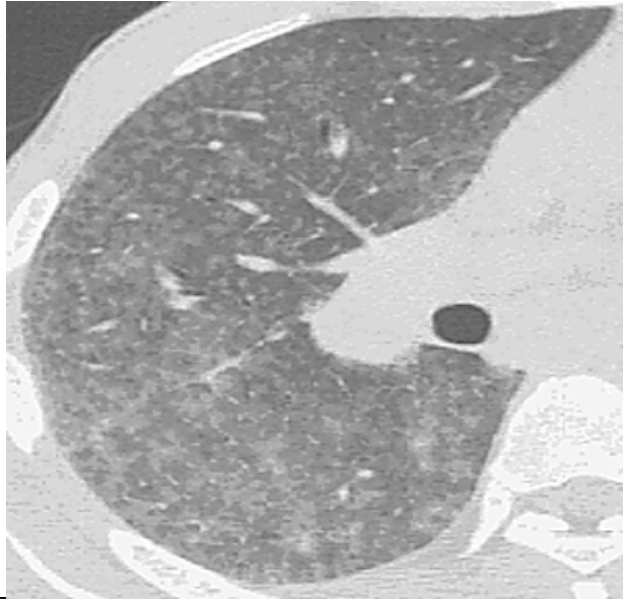
Sarcoidosis

Alveolar sarcoid
Airspace masses
Air bronchograms
Usually upper lobes
Hilar adenopathy



The least common form of lung disease is alveolar sarcoid. Typically, there are several large air space masses that contain air bronchograms. Usually the masses are larger or more extensive in the upper lung zones. Alveolar sarcoid is usually associated with adenopathy as in this case. In addition, the alveolar sarcoid pattern is at high risk for spontaneous pneumothorax.

Sarcoidosis

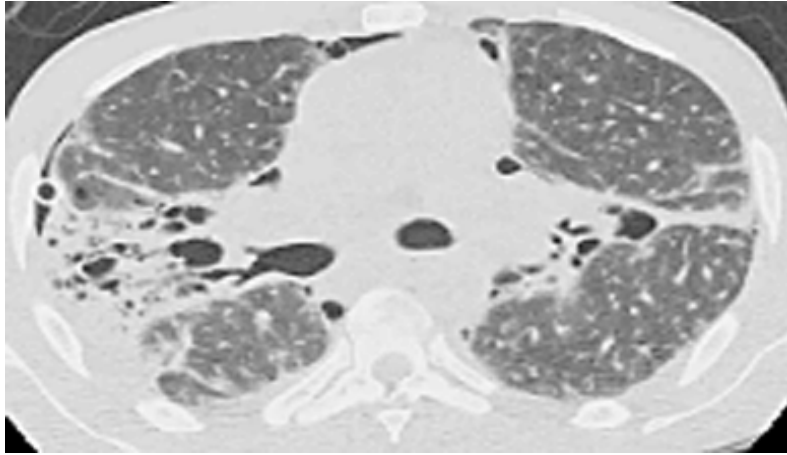


Ground glass opacities
Centriacinar nodules
Follow lymphatics (septa,
bronchi, blood vessels)

The typical features of sarcoid at HRCT vary. Micronodules are typically located along bronchovascular bundles, fissures and pleura mirroring the distribution of lymphatics in the lung.

Sarcoidosis

Fibrosis cuts a swath through lung
“Sherman’s march to the sea”



More advanced fibrosis follows the bronchi and arteries and cuts a swath through the lung from the hilum to the lung periphery. Remember, if you see something which looks like General Sherman’s march to the sea, think sarcoid.

Langerhans granulomatosis

Probable allergic reaction to cigarette smoke

Granulomas Langerhans cell

25% present spontaneous pneumothorax

2/3^{rds} spontaneously resolve

Next on our list is langerhans cell granulomatosis or histiocytosis. The etiology is thought to be an allergic reaction to some constituent of cigarette smoke. The granulomas in histiocytosis contain the langerhans cell, an antigen processing cell normally found in the lung and reticuloendothelial system. Usually affecting young adults, 25% of patients with langerhans histiocytosis present with a spontaneous pneumothorax. The natural history is unknown but most spontaneously resolve.

Langerhans granulomatosis

Mid to upper lobe lung disease

Nodules and cysts

Increased lung volumes

Burned out disease looks like emphysema

The findings of langerhans typically involve the mid to upper lung. As we will see shortly, the findings are combinations of either nodules or cysts. The earliest findings are thought to be nodules which eventually evolve into cysts. In contrast to most interstitial lung diseases, the lung volumes are either normal or increased. Burned out disease may be indistinguishable from emphysema.

Langerhans granulomatosis

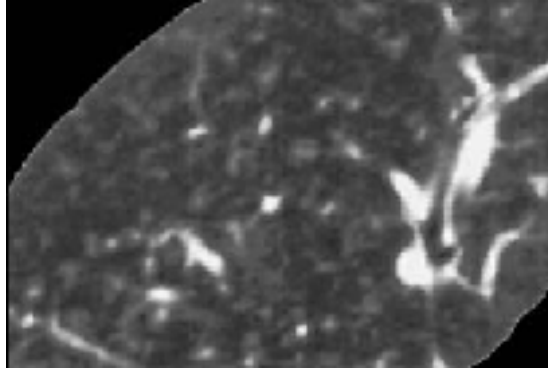
Spontaneous ptx
Biapical coarse
reticulonodular pattern
Increased lung volumes



Here is a typical example. Biapical disease with a coarse cystic pattern. The lung volumes are normal to slightly increased. A chest tube on the right was placed for a spontaneous pneumothorax.

Langerhans granulomatosis

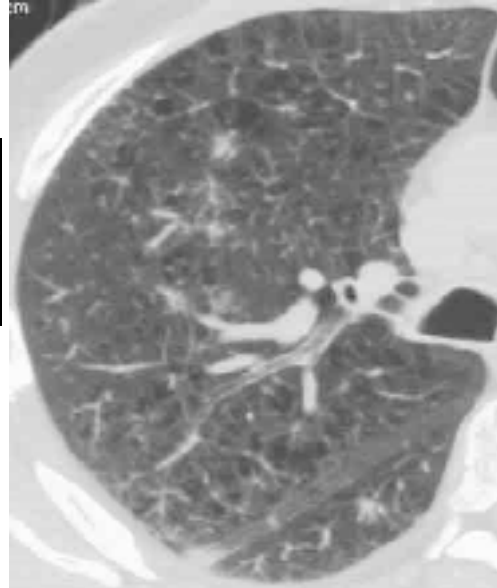
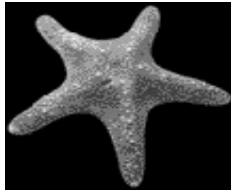
Centriacinar micronodules
Respiratory bronchioles
Lung periphery spared



Just like sarcoid, a common pattern at HRCT are centriacinar micronodules. In contrast to sarcoid, however, the periphery is spared. Judge the lung along the chest wall and fissures. If there are only a handful of subpleural nodules then consider the periphery to be spared.

Langerhans granulomatosis

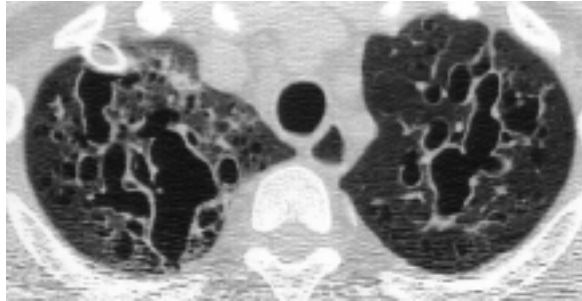
Star-fished shaped
nodules



One feature which I find useful is the shape of the larger nodules. They may have 3-5 arms or tentacles, similar to the shape of a starfish.

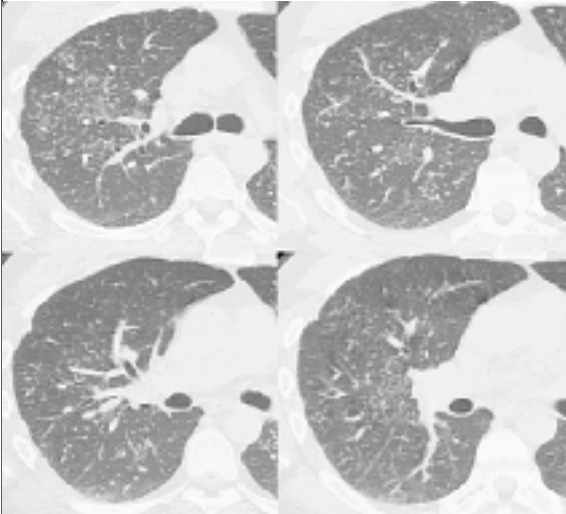
Langerhans granulomatosis

Nodules cavitate
Aggregate into bizarre
shapes

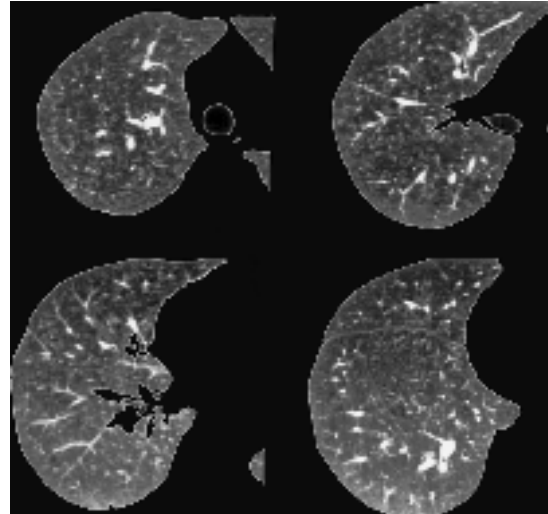


As the nodules get larger they cavitate and eventually aggregate into very bizarre shapes. This is distinctive and nearly pathognomonic for langerhans histiocytosis. This example comes from the chest x-ray that we saw earlier. Note that the cystic pattern was not as evident on the chest radiograph, the value of eliminating superimposition.

Respiratory vs lymphatic nodules



Sarcoidosis



Langerhans

In both sarcoid and langerhans cell histiocytosis, one of the earliest findings are the tiny micronodules. In sarcoid, the granulomatous nodules are located within the lymphatics and will involve both the axial and peripheral lymphatic systems. This will lead to multiple subpleural nodules along the chest wall and fissures. Langerhans cell granulomatous is a disease of the airways. Particles in cigarette smoke deposit in the respiratory bronchioles where they incite a granulomatous response. The periphery of the lobule will be spared and the number of subpleural nodules will be few. This leads to the concept of a respiratory nodule vs a lymphatic nodule. I find this concept useful in formulating a differential diagnosis.

	Distribution	Pattern
Sarcoid	Upper lobe	Nodules (lymphatic) Fibrotic swath
Langerhans	Upper lobe	Nodules (respiratory) Bizarre shaped cysts
IPF	Lower lobe (periphery)	Reticular, honeycombing
Lymphangitic tumor	Spares lobe or lung	Irregular septal
Chronic edema	Batwing, dependent lung	Smooth septal
Asbestosis	Lower lobe (periphery)	Reticular, honeycombing
Collagen vascular disease	Lower lobe (periphery)	Reticular, honeycombing
Environment dusts	Farmers lung Silicosis	Nodules, (respiratory) Nodules, (lymphatic)
Drugs	Any pattern	Any pattern

Our first two diseases share many similarities both in distribution and pattern. However, particular attention should be directed as to whether the periphery of the lung is involved. Sarcoid has a tendency to cut a swath through the lung and histiocytosis to evolve into bizarre shaped cysts.

Idiopathic pulmonary fibrosis

Unknown repetitive insult to alveolar wall

Collagen deposition and fibrosis

Gradual onset dyspnea and dry cough

Variable outcome, median survival 5 yrs

Next is IPF. The concept that IPF is an alveolitis is now discredited with the belief now that there is a repetitive insult to the alveolar wall that results in collagen deposition and fibrosis. Patients usually have the gradual onset of nonspecific symptoms. The prodromal period is unknown, once diagnosed patients have a median survival of about 5 years.

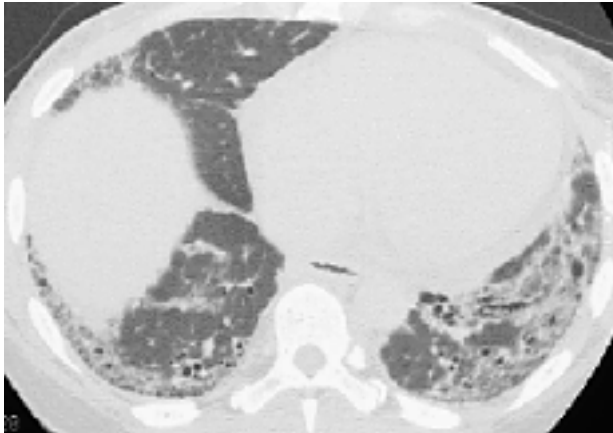
Idiopathic pulmonary fibrosis



Peripheral basilar lung
disease
Reticular lines and
honeycombing
Decreased lung volumes

In contrast to our first two disorders, the distribution of disease is predominantly in the periphery of the lower lobes. The pattern is reticular not nodular. Honeycombing or end stage lung is common. Typically the lung volumes are decreased and parallel the restrictive pulmonary function tests.

Idiopathic pulmonary fibrosis



Ground glass opacities
Irregular lines, septal
thickening
Honeycombing
Traction bronchiectasis

Ground glass opacities in IPF are nonspecific. These opacities may be due to atelectasis or fibrosis which is below the resolution of the CT scanner. The lobule may be crisscrossed by a network of fine lines. One important characteristic is the presence of traction bronchiectasis which must be present before I can confidently diagnosis IPF.

	Distribution	Pattern
Sarcoid	Upper lobe	Nodules (lymphatic) Fibrotic swath
Langerhans	Upper lobe	Nodules (respiratory) Bizarre shaped cysts
IPF	Lower lobe (periphery)	Reticular, honeycombing Traction bronchiectasis
Lymphangitic tumor	Spares lobe or lung	Irregular septal
Chronic edema	Batwing, dependent lung	Smooth septal
Asbestosis	Lower lobe (periphery)	Reticular, honeycombing
Collagen vascular disease	Lower lobe (periphery)	Reticular, honeycombing
Environment dusts	Farmers lung Silicosis	Nodules, (respiratory) Nodules, (lymphatic)
Drugs	Any pattern	Any pattern

IPF is distinctive from the previous disorders, particularly with regards to distribution and pattern.

Lymphangitic tumor

Carcinoma lung, breast, and GI tract
Permeation of lymphatics by neoplasia
Progressive dyspnea and cough
Prognosis poor

Lymphangitic spread of tumor may arise from many tumors, especially adenocarcinomas. Tumor spreads to the lymphatics either hematogenously or retrograde from hilar and mediastinal lymph nodes. Lymphangitic spread is a late finding in the natural history of tumor spread and usually portends a poor prognosis.

Lymphangitic tumor

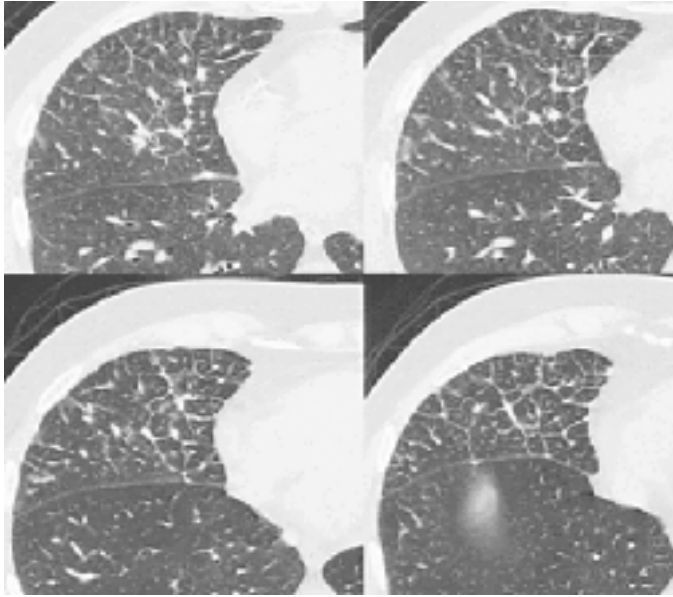
Not diffuse, usually spares
lobe or lung
Lung volumes preserved
Adenopathy or pleural
effusions



Lymphangitic tumor is usually not diffuse but tends to spare lobes or even a whole lung. In this example, the left lower lobe was completely spared. Even with extensive permeation of lymphatics, lung volumes are preserved. Patients may or may not have adenopathy or small pleural effusions.

Lymphangitic tumor

Nodular thickening axial (bronchovascular) and peripheral (septal) lymphatics



Lymphangitic spread may involve either the axial or peripheral lymphatics or both. In general, axial involvement is more common. In this example, lymphangitic spread from colon carcinoma involved the right middle lobe only. A characteristic feature of lymphangitic tumor is irregular or nodular thickening of the septa as is noted in this case.

Edema

Occult edema not uncommon

Extravascular water: ground glass opacities,
septal thickening

Orthopnea and dyspnea

Enlarged heart

It might seem surprising that edema is included in the differential of diffuse lung disease. However, it is surprising how many patients get referred to a pulmonary specialist with edema. Most specialists treat new referrals with a diuretic prior to further workup just in case that their end stage lung may disappear. The findings of edema: ground glass opacities and septal thickening are identical to those of diffuse lung disease. Patients may not give a history of orthopnea and dyspnea and while an enlarged heart is often found, some may not have recognizable cardiomegaly.

Edema

May be chronic, labeled as diffuse lung disease

Most pulmonologists treat referrals with ILD with diuretics before further investigation



Here is just such a problematic case. Moderate cardiomegaly and mild diffuse interstitial thickening had not changed over multiple films obtained over several months.

Edema

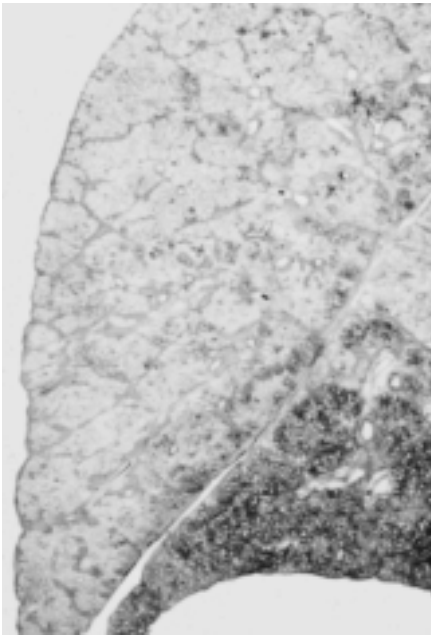
Gravitational gradient
Smooth septal thickening
Sparing lobules (mosaic perfusion)



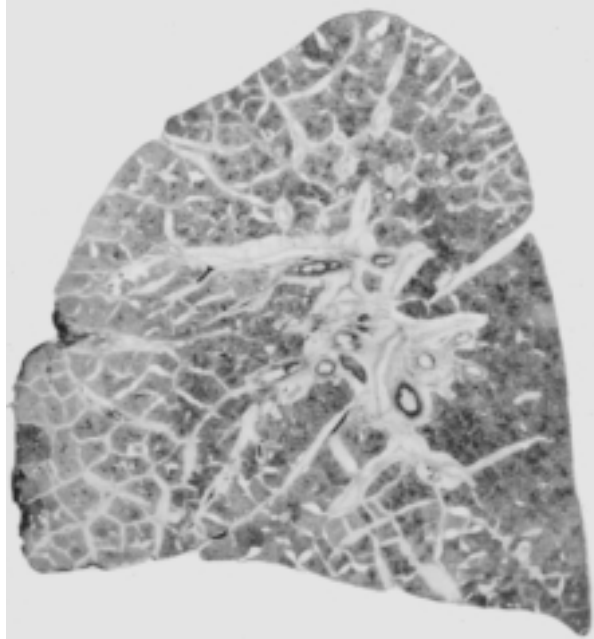
One clue to edema is the gravitational distribution. Since many of these patients are upright, ground glass opacities and septal thickening are more severe in the lung bases than in the upper lobes. Another curious unexplained findings is sparing of lobules as is seen in this patient. From animal experiments, at any given time there are some lobules that are not perfused due to a local autoregulatory mechanism. Starling forces would not affect lobules that are not perfused.

Septa

Lymphangitic tumor



Edema



Both lymphangitic tumor and edema involve the septa. In lymphangitic tumor the septa are irregularly thickened and beaded whereas in chronic edema the septa are smoothly thickened.

	Distribution	Pattern
Sarcoid	Upper lobe	Nodules (lymphatic) Fibrotic swath
Langerhans	Upper lobe	Nodules (respiratory) Bizarre shaped cysts
IPF	Lower lobe (periphery)	Reticular, honeycombing Traction bronchiectasis
Lymphangitic tumor	Spares lobe or lung	Irregular septal
Chronic edema	Batwing, dependent lung	Smooth septal
Asbestosis	Lower lobe (periphery)	Reticular, honeycombing
Collagen vascular disease	Lower lobe (periphery)	Reticular, honeycombing
Environment dusts	Farmers lung Silicosis	Nodules, (respiratory) Nodules, (lymphatic)
Drugs	Any pattern	Any pattern

The distribution of our last two entities is unique. In addition there are subtle differences in the septa, irregular and beaded in lymphangitic tumor and uniform and smooth in chronic edema.

Asbestosis

Pneumoconiosis from fibrous silicates

Latent period 20 - 30 years

Fibrosis + asbestos bodies = asbestosis

High proportion die of lung cancer

Asbestos has been widely used in industry because of its advantageous fire-retardant properties in insulation. Asbestosis is only 1 manifestation of asbestos exposure which includes pleural plaques, diffuse pleural thickening, and mesothelioma. The pathologic signature in asbestosis is the asbestos or ferruginous body. The asbestos body is an asbestos fiber surrounded by dead macrophages which stain for iron. Asbestos is carcinogenic and combined with smoking, the risk of developing lung cancer is multiplicative, that is higher than the risk of either agent alone.

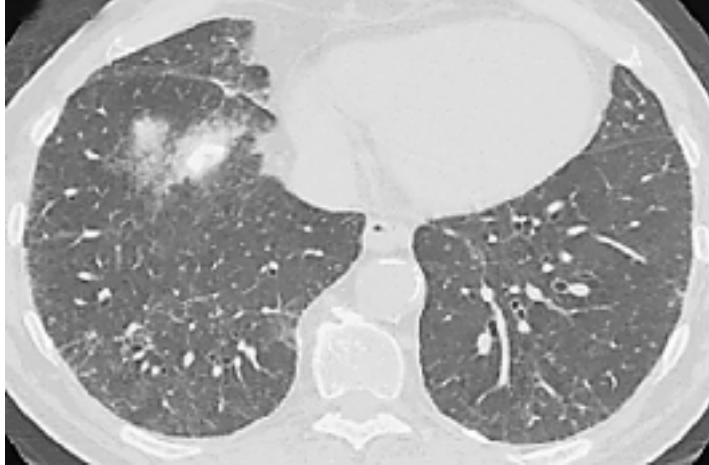
Asbestosis

Peripheral and basilar
Irregular reticular opacities
Small lung volumes
25% asbestos plaques



Identical to IPF, asbestosis primarily involves the periphery of the lower lobes. The pattern is also identical, irregular reticular opacities and honeycombing. One clue is the asbestos plaque, however, plaques are seen in only 25% of those with asbestosis.

Asbestosis



Irregular short septal lines
Long parenchymal bands
Centriacinar nodules
Honeycombing

At HRCT, short irregular lines extend from the centriacinar region to the lung periphery. Longer thicker parenchymal bands may be found in more severe cases. End-stage honeycombing when severe.

Collagen vascular disease

Generalized connective tissue disease

Cellular or fibrotic (NSIP or UIP)

Dyspnea

Variable prognosis

Diseases such as rheumatoid arthritis and scleroderma are also associated with diffuse interstitial thickening. Pathologically, there may be a UIP or NSIP pattern, indistinguishable from IPF. Dyspnea is a common complaint. Prognosis is variable and life-expectancy is longer with NSIP than UIP.

Collagen vascular disease

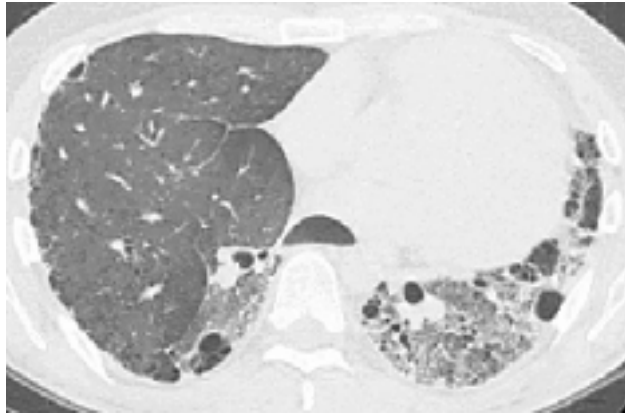


Peripheral basilar
Honeycombing
Decreased lung volumes
Dilated esophagus
(scleroderma)

In this example, the peripheral basilar interstitial thickening is identical to IPF. However, the tip-off is the dilated esophagus in this patient with scleroderma.

Collagen vascular disease

Wide spectrum: ground glass, micronodules, honeycombing
NSIP: uniform pattern



Again, at HRCT ground glass opacities are common but nonspecific. The key is the distribution and the presence of intralobular lines or honeycombing. NSIP is a newer pathologic designation, common in the collagen vascular diseases. NSIP may be either cellular or fibrotic, a wide gamut. The key in NSIP is that the pathologic fields will be the same from all sampled sites. That implies that the insult occurred at the same time. In contrast, in UIP the biopsy specimens are inhomogenous with some cellular sections and other noncellular or fibrotic.

	Distribution	Pattern
Sarcoid	Upper lobe	Nodules (lymphatic) Fibrotic swath
Langerhans	Upper lobe	Nodules (respiratory) Bizarre shaped cysts
IPF	Lower lobe (periphery)	Reticular, honeycombing Traction bronchiectasis
Lymphangitic tumor	Spares lobe or lung	Irregular septal
Chronic edema	Batwing, dependent lung	Smooth septal
Asbestosis	Lower lobe (periphery)	Reticular, honeycombing
Collagen vascular disease	Lower lobe (periphery)	Reticular, honeycombing
Environment dusts	Farmers lung Silicosis	Nodules, (respiratory) Nodules, (lymphatic)
Drugs	Any pattern	Any pattern

For our last 2 disorders, the distribution and pattern are identical to IPF. We must be attentive to additional clues: asbestos plaques or skeletal or esophageal changes in collagen vascular diseases for help.

Environmental dusts

Inorganic - organic dusts

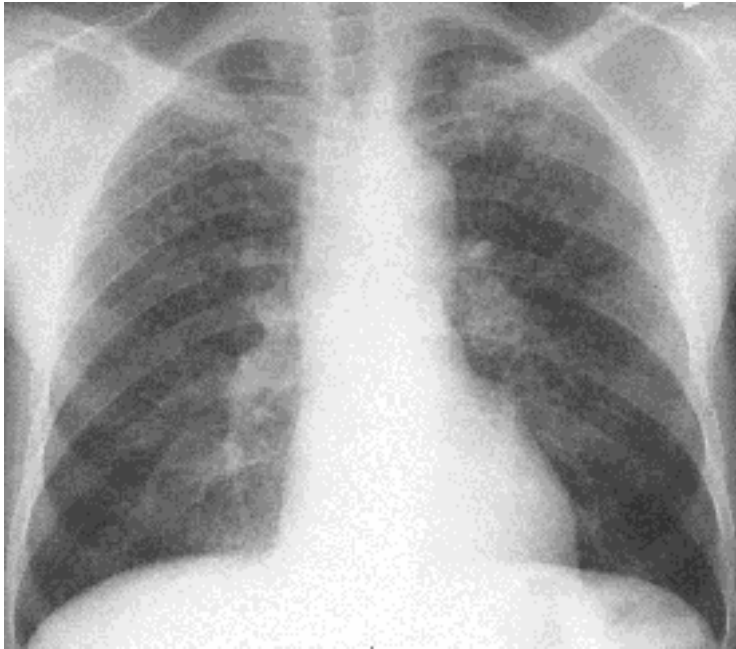
Silica, coal, farmer's lung

Granulomas, collagen nodules, dust macules

Finally let's discuss the dusts. Inorganic dusts such as coal or silica or the organic dusts such as Farmers lung. The pathologic response will vary depending on the particular dust.

Silicosis

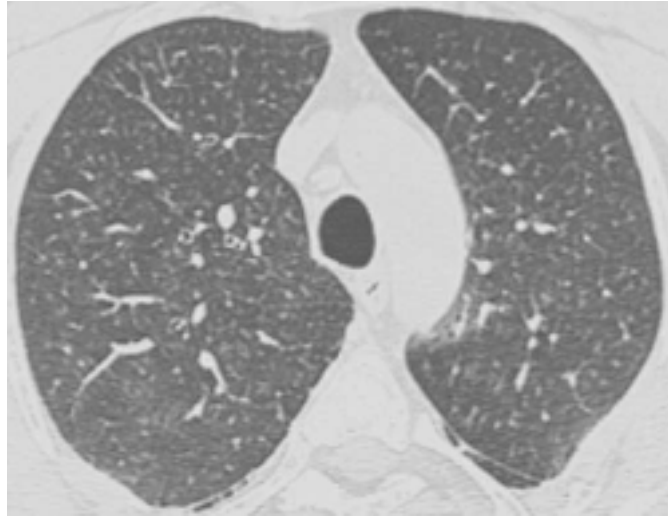
Upper lobe distribution
Nodules, PMF
Adenopathy, egg-shell
calcification



Similar to sarcoid and langerhans cell histiocytosis, silica and the other round dust particles such as coal concentrate in the upper lung zones. The pattern is nodular, and the nodules may aggregate into larger masses known as PMF. Particles are removed by lymphatics and thus the nodules are distributed in both the axial and peripheral lymphatic system. The mediastinal nodes may be enlarged. Particularly with silica, the enlarged nodes may exhibit egg-shell calcification.

Silicosis

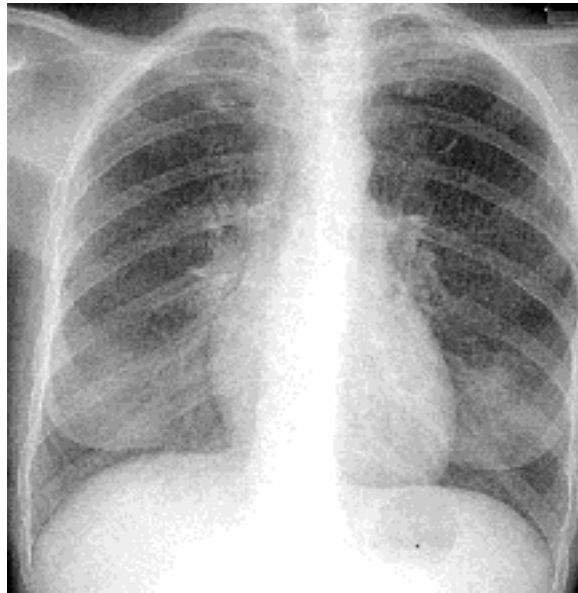
Micronodules, lymphatic
distribution
Dorsal distribution
May calcify



HRCT, the nodules are diffuse, identical to that seen in sarcoid. In some, the profusion of nodules may be greater in the dorsal aspect of the lung. Occasionally the nodules may calcify.

Farmer's lung

Often normal
Mid lung distribution
Spare costophrenic angle
Miliary nodules



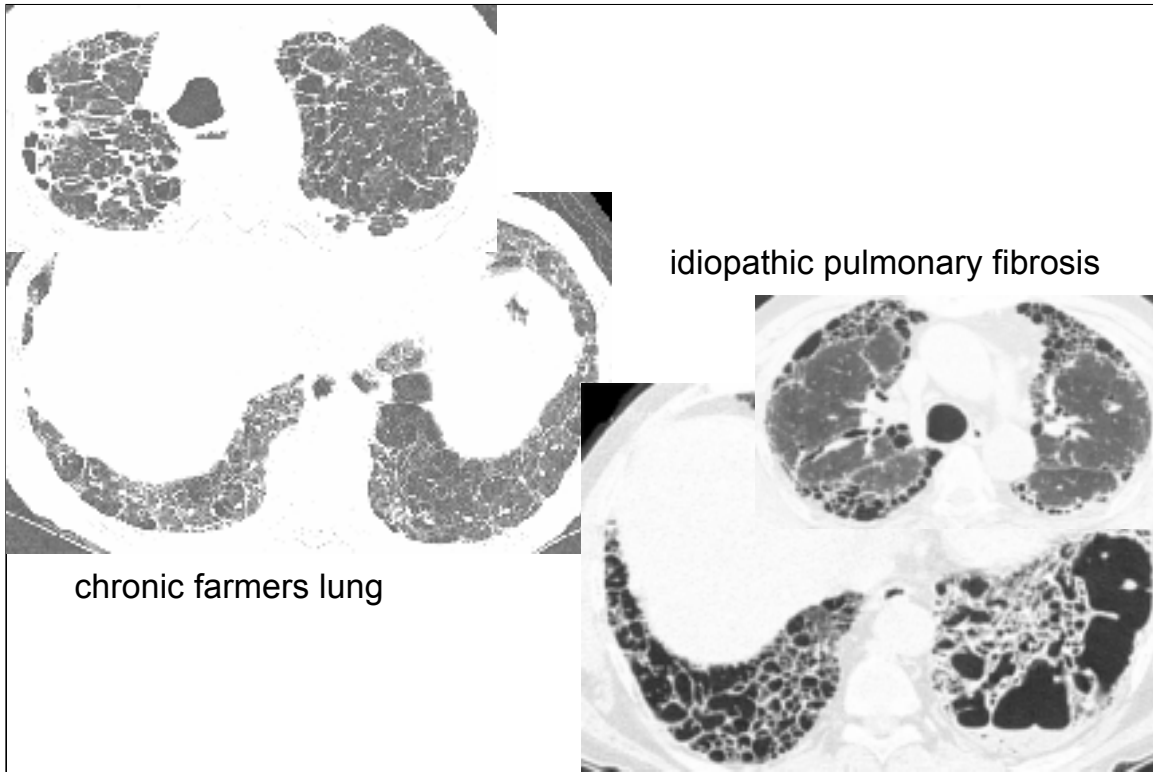
Farmers lung is our prototypical reaction to organic dusts. Numerous dusts have been shown to cause a hypersensitivity reaction, but Farmer's lung, due to a reaction to the Thermophillic actinomyces organism is the most common. Up to 90% of chest x-rays may be normal. When abnormal, a fine nodular pattern in the mid-lung spares the costophrenic angle.

Farmer's lung



Centriacinar nodules, respiratory
mid-lungs
Spare costophrenic angle

HRCT may also be normal in up to 50%.
Nonspecific ground glass opacities are common.
The most characteristic findings include centriacinar
nodules often admixed with areas of air trapping.



The main differential for chronic farmers lung is IPF. One helpful distinction is involvement of the subpleural costophrenic lung. IPF is usually most severe in the costophrenic angles, whereas this area of lung is usually less involved in chronic farmers lung.

	Distribution	Pattern
Sarcoid	Upper lobe	Nodules (lymphatic) Fibrotic swath
Langerhans	Upper lobe	Nodules (respiratory) Bizarre shaped cysts
IPF	Lower lobe (periphery)	Reticular, honeycombing Traction bronchiectasis
Lymphangitic tumor	Spare lobe or lung	Irregular septal
Chronic edema	Batwing, dependent lung	Smooth septal
Asbestosis	Lower lobe (periphery)	Reticular, honeycombing
Collagen vascular disease	Lower lobe (periphery)	Reticular, honeycombing
Environment dusts	Inorganic: upper lobe Organic: spare cp angle	Nodules, (lymphatic) Nodules, (respiratory)
Drugs	Any pattern	Any pattern

The environmental dusts are similar to sarcoid, both in pattern and distribution. Farmers lung most commonly involves the mid-lung, and spares the costophrenic angle.

Drugs

Often overlooked

Numerous pathologic responses

DAD, hypersensitivity, UIP

May lead to respiratory failure

Lastly, consider drug reaction. These are often overlooked. The number of drugs which can adversely affect the lung is immense. Nearly any pathologic response can be due to drug toxicity. If the offending drug is not removed, it may cause respiratory failure.

Drugs

Macro-dantin: UIP pattern
Add to differential for any
diffuse lung disease



In this patient with macro-dantin toxicity, the pattern is indistinguishable from IPF.

	Distribution	Pattern
Sarcoid	Upper lobe	Nodules (lymphatic) Fibrotic swath
Langerhans	Upper lobe	Nodules (respiratory) Bizarre shaped cysts
IPF	Lower lobe (periphery)	Reticular, honeycombing Traction bronchiectasis
Lymphangitic tumor	Spares lobe or lung	Irregular septal
Chronic edema	Batwing, dependent lung	Smooth septal
Asbestosis	Lower lobe (periphery)	Reticular, honeycombing
Collagen vascular disease	Lower lobe (periphery)	Reticular, honeycombing
Environment dusts	Inorganic: upper lobe Organic: spare cp angle	Nodules, (lymphatic) Nodules, (respiratory)
Drugs	Any pattern	Any pattern

Our final pattern is that of drug reaction which may produce any pattern or distribution that we've seen so far. I always add drug reaction into the mix of any differential diagnosis for diffuse lung disease.