TB or Not TB: Differential Diagnosis and Imaging Findings of Pulmonary Cavities

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Cavitary lesions are often encountered during radiographic evaluation of the chest. During early radiology training, residents are introduced to the mnemonic "CAVITY" for the differential diagnosis of pulmonary cavitary lesions: cancer (bronchogenic carcinoma, especially squamous cell carcinoma), autoimmune (granulomatosis with polyangiitis or rheumatoid arthritis), vascular (pulmonary emboli – septic or bland), infection (tuberculosis, fungal, *staphylococcus aureus*), trauma (pneumatocele or laceration), and youth (congenital pulmonary airway malformation, pulmonary sequestration, bronchogenic cyst).[1] Although this mnemonic is an efficient way of expanding the differential diagnosis for novice radiologists, a deeper understanding of each condition is necessary to make the correct diagnosis in practice. In addition, these differentials must be given in the appropriate clinical context. This article will discuss imaging findings of common cavitary lesions, which along with clinical history, can lead to the correct diagnosis and expedite appropriate management.

Cavity or Pulmonary Cyst

To arrive at the correct diagnosis, the difference between pulmonary cysts and cavities must be defined. According to the Fleischner Society, a pulmonary cyst is "any round circumscribed space surrounded by an epithelial or fibrous wall." The wall thickness of the cyst is usually < 2 mm.[2]

Meanwhile, a pulmonary cavity is defined as "a gas-filled space that is seen as a lucency or low-attenuation area within a pulmonary consolidation, mass, or a nodule" (**Figure 1**).[2] Wall thickness of a cavitary lesion is usually > 2 to 4 mm. Wall thickness also helps to predict malignancy of a lesion. Cysts or cavities with wall thickness < 4 mm are likely benign, while wall thickness > 15 mm suggests malignancy.[3]

Table 1 includes a broad differential diagnosis of both pulmonary cysts and cavities.

Bleb/Bulla

Blebs and bullae are often incidentally found in asymptomatic patients, mostly in thin younger males or patients with an extensive smoking history. Blebs are formed as a result of spontaneous rupture of subpleural alveoli. Both are usually located in the periphery of lungs with thin walls < 1 mm. The main distinction between blebs and bullae is size (**Figure 2**). Blebs are defined as a "small gas-containing space within the visceral pleura or in the subpleural lung, not larger than 1 cm in diameter."[2] Bullae are defined as "airspace measuring > 1 cm in diameter, sharply demarcated by a thin wall that is no greater than 1 mm in thickness."[2] Reporting large bullae is

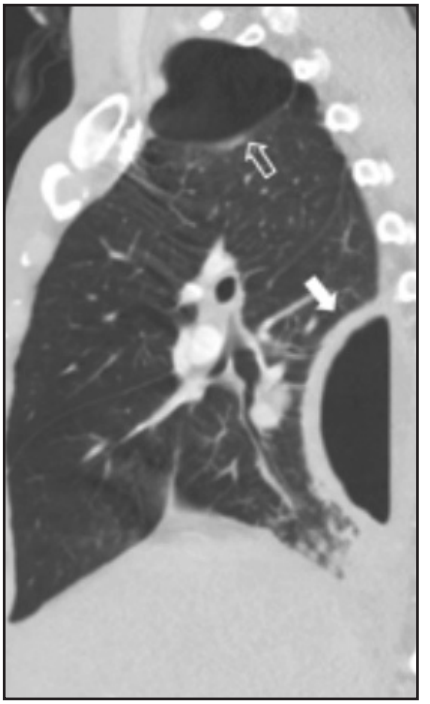

FIGURE 1. *Thin-wall cyst vs a thick-wall cavity. Sagittal contrast-enhanced CT of the chest demonstrating a thin-walled apical bulla (hollow arrow) and thick-walled posterior pleural cavity (solid arrow).*

Table 1. Differential Diagnosis of Pulmonary Cysts and Cavities

Cyst	Cavity
Bleb/Bulla	Infection
Trauma	Tuberculosis/Mycobacterium Avium Complex (MAC)
Pulmonary Laceration	Bacterial Pneumonia/Abscess
Post Traumatic Pneumatocele●	Aspergillosis
Congenital	Neoplasm
Congenital Pulmonary Airway Malformation (CPAM)	Bronchogenic Carcinoma
Sequestration	Metastasis
Bronchogenic Cyst	Vascular
Autoimmune	Septic Emboli
Lymphangioleiomyomatosis (LAM)	Autoimmune
Interstitial Lung Disease	Granulomatosis with Polyangiitis (GPA)
Progressive Systemic Sclerosis	Rheumatoid
Langerhan Cell Histiocytosis	Pyoderma Gangrenosum●
	Sarcoid

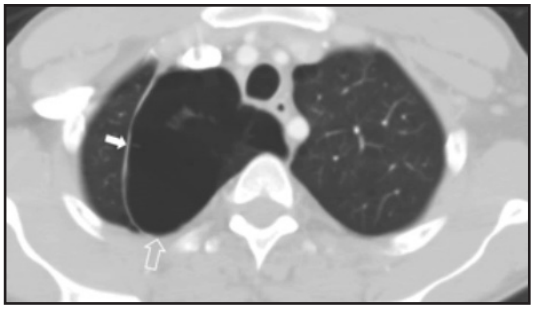

FIGURE 2. *Bulla. Axial contrast-enhanced CT image demonstrating a large bulla (hollow arrow) occupying the azygos lobe (solid arrow).*

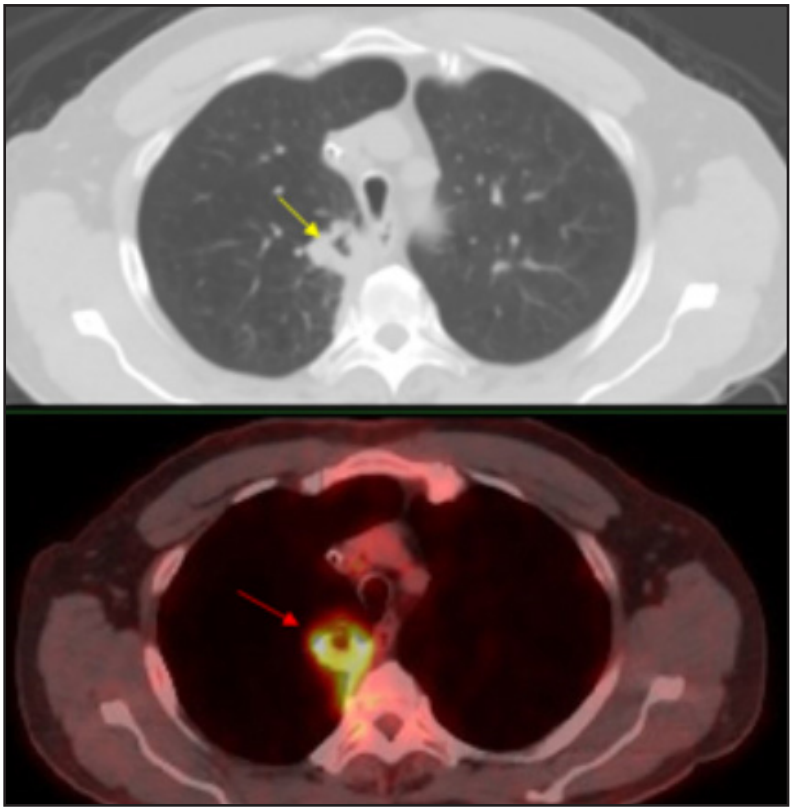

FIGURE 3. *Metastasis. Axial unenhanced CT and fused F-18 fluorodeoxyglucose (FDG) PET/CT images demonstrate a cavitary right upper lobe mass (yellow arrow) with FDG avidity (red arrow) and history of head and neck squamous cell carcinoma.*

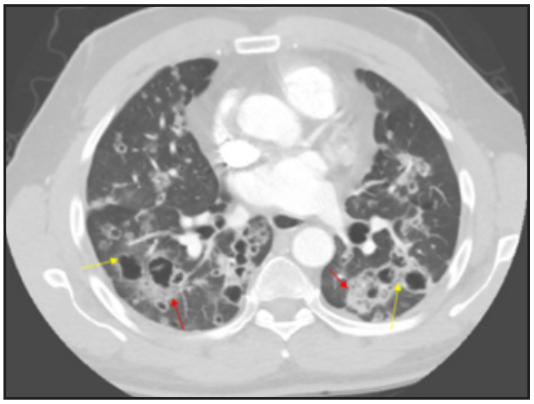

FIGURE 4. *Metastatic colon cancer. Axial-enhanced CT image demonstrating multiple bilateral cavitary lesions (yellow arrows), which were biopsied and consistent with metastatic colon cancer. There is associated surrounding hemorrhage presenting as ground-glass density (red arrows).*

important as they can rupture, resulting in a pneumothorax.

Cancer

Bronchogenic Carcinoma

There are 3 proposed mechanisms of how primary lung cancer presents as a cystic mass. The first mechanism is by having a rapid growth of lung cancer during which the blood supply cannot meet the demand of the neoplastic growth and causes central necrosis of the tumor.[3] The second mechanism is due to mass effect causing bronchial obstruction, scarring, or bronchiectasis resulting in infection distal to the obstructive mass; infection later leads to the breakdown of the lung parenchyma and forms the cystic cavity. The third mechanism is by "spillover abscess," which describes spillage of the primary infection to the

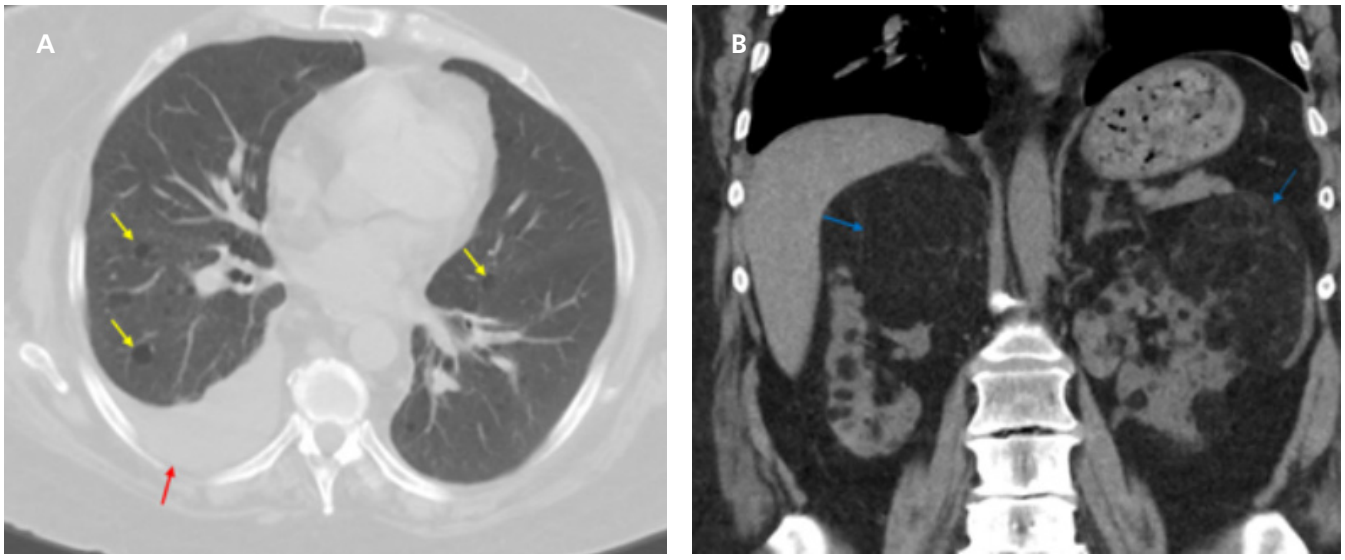

FIGURE 5. *Lymphangioleiomyomatosis. (A) Axial unenhanced CT image in a patient with a history of tuberous sclerosis demonstrates multiple bilateral thin-walled pulmonary cysts (yellow arrows) and a right pleural effusion (red arrow). These patients can present with chylous pleural effusions. Renal angiomyolipoma. (B) Coronal noncontrast CT image in the same patient demonstrating bilateral fat containing renal masses (blue arrows) compatible with angiomyolipomas.*

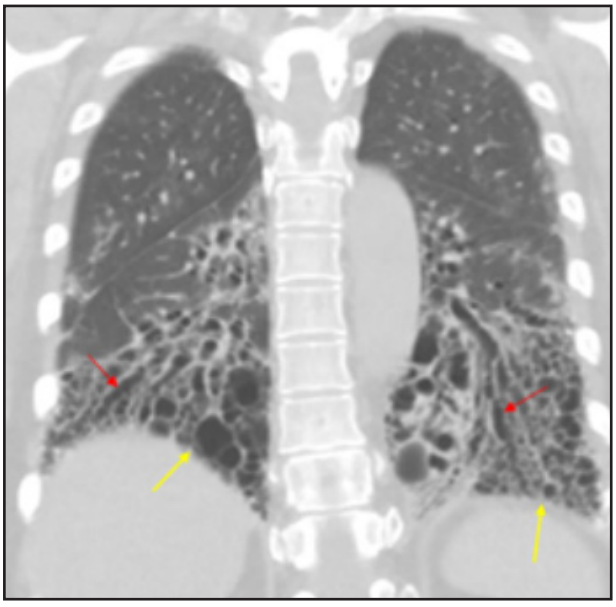

FIGURE 6. *Idiopathic pulmonary fibrosis. Coronal contrast-enhanced CT image demonstrating a UIP pattern of subpleural and basilar predominant honeycombing (yellow arrows) with traction bronchiectasis (red arrows).*

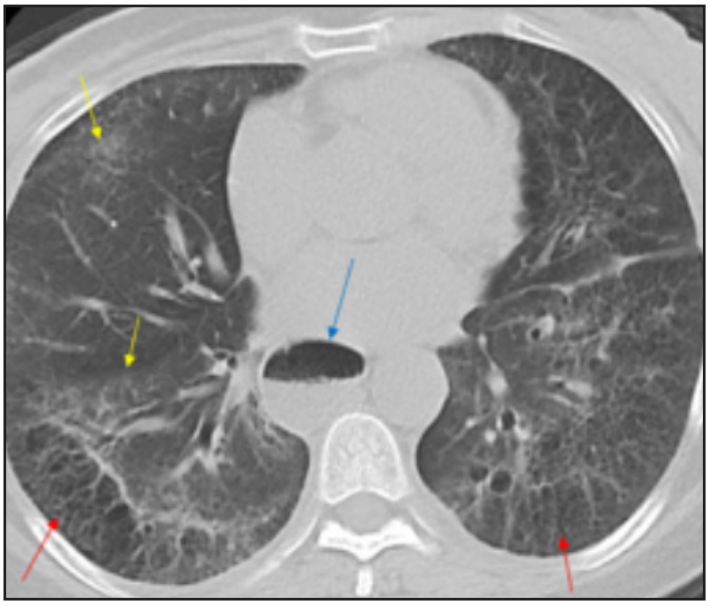

FIGURE 7. *Progressive systemic sclerosis (scleroderma). Axial unenhanced CT image demonstrating common findings of scleroderma including ground-glass opacities (yellow arrows), subpleural and basilar predominant cystic changes (red arrows), as well as esophageal dilation (blue arrow).*

distant site of cavitation, even involving different lobes of the lung.[4]

Cavitating lung cancer is most often seen in the sixth to seventh decades of life in patients with significant smoking history. Unfortunately, cavitation in primary lung cancer is associated with a poor prognosis. Out of all the types of primary lung carcinoma, squamous cell carcinoma is most commonly associated with cavitary lesions.[4] Cavitary lesions are rarely associated with small cell carcinoma. Findings associated with primary lung cancer include thick and irregular inner walls. Cavity size varies from 1 to 10 cm. According to a study by Woodring et al, most cavitary lesions with wall thickness < 4 mm were benign, >15 mm were malignant, and between 4 and 15 mm had mixed results.[3,5]

Metastasis

Cavitation of lung metastases from extrapulmonary primary cancer is uncommon, occurring in only 4% of cases.[5] The average age for presentation with pulmonary metastasis is 60 to 70 years. The most common primary origin of pulmonary metastatic disease is squamous cell cancer of the head and neck. Other primary sites include large

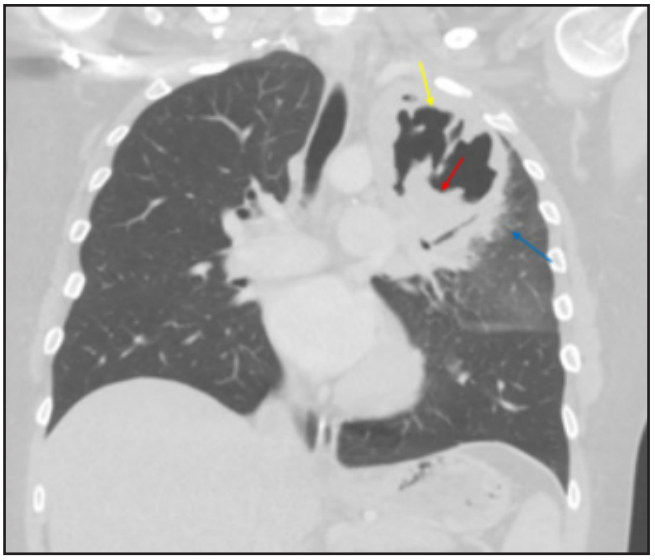

FIGURE 8. *Granulomatosis with polyangiitis. Coronal contrast-enhanced CT in a patient with history of granulomatosis with polyangiitis demonstrating a thick-walled cavitary lesion (yellow arrow) with internal soft-tissue density representing superimposed aspergillosis (red arrow) and surrounding ground-glass hemorrhage (blue arrow).*

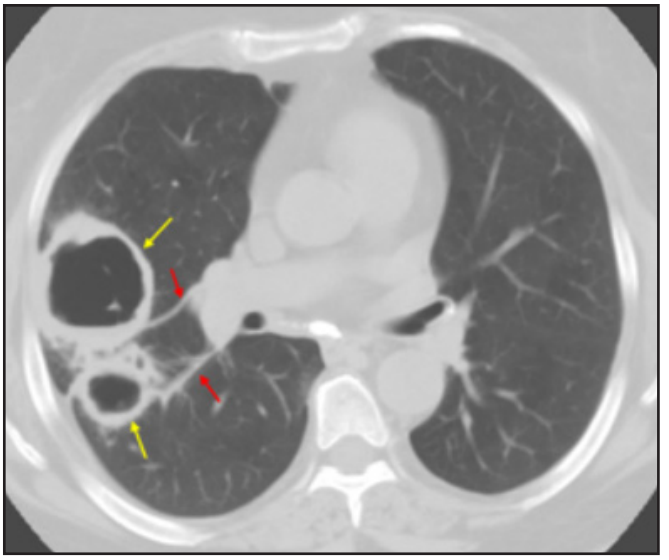

FIGURE 9. *Granulomatosis with polyangiitis. Axial contrast-enhanced CT image in a patient with history of granulomatosis with polyangiitis. This image demonstrates multiple pulmonary cavities (yellow arrows) with feeding vessels (red arrows).*

intestine, cervix, stomach, esophagus, pancreas, and kidney. Cavitation size varies from 1 to 6 cm, and the wall thickness also varies from 0.3 to 2.5 cm.[5] Similar to primary lung cancer, thick and irregular walls are the most common imaging findings, and metastasis often presents with multiple cavities, mostly seen in the periphery of lungs (**Figures 3 and 4**). The diagnosis is often made by biopsy of the lesions due to their indeterminate imaging characteristics.

Autoimmune (Cyst)

Lymphangioleiomyomatosis

Lymphangioleiomyomatosis (LAM) is defined as progressive growth of smooth muscle cells in the pulmonary parenchyma, vasculature, lymphatics, and pleurae. LAM exclusively affects females 20 to 40 years old. The etiology of LAM is not well defined, but the close relationship between tuberous sclerosis and LAM suggests that somatic mosaicism on the TSC-2 gene may have a role.[6] Another theory is that LAM is associated with a metastatic neoplasm originating from the uterus, hence the reason for involving only the female population.[7] Most patients present with cough, hemoptysis, and chest pain. The symptoms may be exacerbated during pregnancy as estrogen level increases. Diagnosis can be made with CT, which demonstrates diffuse bilateral thin-walled cysts, measuring up to 5 mm in diameter, with associated hemorrhagic ground-glass opacities (**Figure 5A**).[8] When CT is not diagnostic, histopathologic diagnosis of smooth muscle cells can confirm the diagnosis. Other imaging manifestations of tuberous sclerosis include renal angiomyolipomas (**Figure 5B**) and cardiac rhabdomyomas.[9] LAM carries a poor prognosis as it can lead to progressive respiratory failure. Current treatment includes mTOR inhibitor (eg, Sirolimus) or lung transplant.[6]

Interstitial Lung Disease

Interstitial lung disease (ILD) is a broad category encompassing many different idiopathic interstitial pneumonias. ILD affects the interstitium that surrounds alveoli. The most common and concerning condition with a poor prognosis is idiopathic pulmonary fibrosis (IPF). IPF most often occurs between ages 40 and 70 years. However, in patients who have more than 2 first-degree relatives with pulmonary fibrosis, this may present before their fifth decade.[10] As with all other types of interstitial pneumonia, IPF assessment is best performed with thin-section high-resolution CT (HRCT). Imaging findings of IPF are a definite or probable UIP pattern including general parenchymal volume loss, basilar and subpleural predominant fibrotic change, reticular abnormality, and honeycombing with or without traction bronchiectasis (**Figure 6**). The most defining characteristic of UIP/IPF on HRCT is the honeycombing fibrotic appearance, described as multilayered cystic changes ranging from 2 to 10 mm most commonly distributed in the lung bases. With the recent introduction of Pirfenidone treatment in these patients, the survival time among IPF patients improved by 52 weeks, from 2 to 3 years.[11] Five-year survival rate of IPF ranges from 30% to 50%. Pirfenidone and Nintedanib can be used in cases of mild to moderate disease to delay lung transplantation.[10]

Progressive Systemic Sclerosis (Scleroderma)

Systemic sclerosis (SSc) is a connective tissue disorder characterized by progressive fibrosis of multiple organs, including the lungs, skin, vessels, and visceral organs. This condition is seen 4 times more often in women

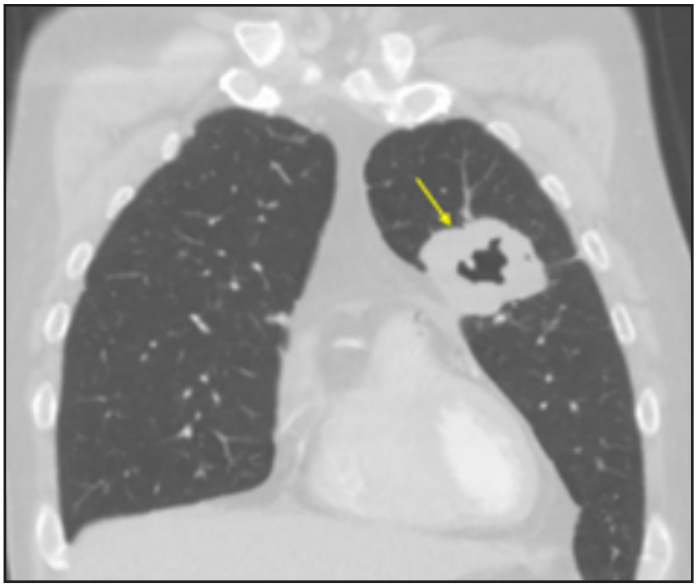

FIGURE 10. *Pyoderma gangrenosum. Coronal contrast-enhanced CT of a patient with pyoderma gangrenosum demonstrates a thick-walled cavitary lesion (yellow arrow). Extracutaneous involvement was suspected considering the lesion was sterile and negative for malignancy on biopsy.*

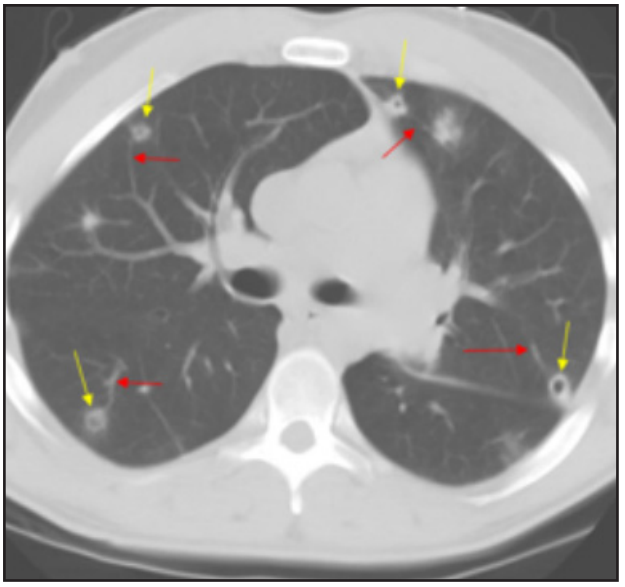

FIGURE 11. *Septic emboli. Axial contrast-enhanced CT image demonstrating multiple peripheral cavities (yellow arrows) in various stages of evolution with feeding vessels (red arrows).*

and commonly between ages 20 and 50. Patients with pulmonary involvement present with a restrictive lung disease pattern of low lung volumes, preserved flow rate, and low diffusion capacity. Interstitial lung disease can also develop, which is seen in about two-thirds of patients. On HRCT, either usual interstitial pneumonia (UIP) or nonspecific interstitial pneumonia (NSIP) pattern may be observed with variable presentation from early ground-glass opacities to late fibrosis.[12] Lung bases and subpleural spaces are most commonly affected. In some cases, cystic changes may occur with each cyst ranging from 1 to 5 cm in diameter (**Figure 7**). Dilation of the esophagus is a crucial finding and unique to SSc.[13] Once the lung disease has progressed to fibrosis/UIP, chances of disease reversal are poor. As of now, cyclophosphamide, glucocorticoids, or N-acetylcysteine can be attempted to halt disease progression. However, the efficacy of these treatments is better in earlier phases of the disease.[12]

Pulmonary Langerhans Cell Histiocytosis

Langerhans cell histiocytosis (LCH) is a rare pediatric disease with male predilection that is mainly diagnosed between the ages of 1 and 3. As the name implies, LCH is caused by uncontrolled monoclonal proliferation of langerhan dendritic cells of the skin and other tissues contacting the external surface. The overactivation of the "langerhans-like" histiocytes begin to release large amount of oxidants, proteases, and fibronectin causing destruction of the lung parenchyma.[14] In addition to the wide-spread disseminated form, a pulmonary manifestation of this disease, also known as pulmonary langerhans cell histiocytosis, can be seen in young males between 20 to 40-years-old and is highly associated with smoking. The most common presenting symptoms are dyspnea and dry cough. Patients can also present with pleuritic chest pain, weight loss, or spontaneous pneumothorax. Classic imaging findings are thin-walled, small, irregular shaped cysts (usually less than 10mm in diameter) which are upper lobe predominant and associated small nodules.[15] Fibrotic changes can be observed in the later stages of disease. Prognosis is good with 50% of patients showing spontaneous resolution after smoking cessation. Corticosteroids are often used as a treatment option with good results.

Autoimmune (Cavitary)

Granulomatosis with Polyangiitis

Granulomatosis with Polyangiitis (GPA) is a multisystem necrotizing granulomatous vasculitis that affects primarily the small to medium-sized vessels and was previously termed "Wegener granulomatosis."[16] The lungs are most commonly involved in this disease and the patients present with cough, hemoptysis, and dyspnea from ages 40 to 60.

Common imaging findings of the lungs include multiple, bilateral pulmonary masses with cavitation in more than 50% of the lesions. Cavitations are more common in the larger lesions and are thick with irregular, "shaggy" cavity walls (**Figure 8**).[17] This pulmonary disease can also present with alveolar hemorrhage in approximately 10% of patients. There can be a pulmonary vessel coursing directly to the mass in approximately 88% of cases (**Figure 9**), coined as the "feeding vessel sign." However, this is a nonspecific sign that can be seen in other entities of cavitary disease.[18] Other signs on imaging include a "reversed halo sign," which has consolidation surrounding a central ground-glass density.

This disease is an autoimmune disease of uncertain etiology and is treated

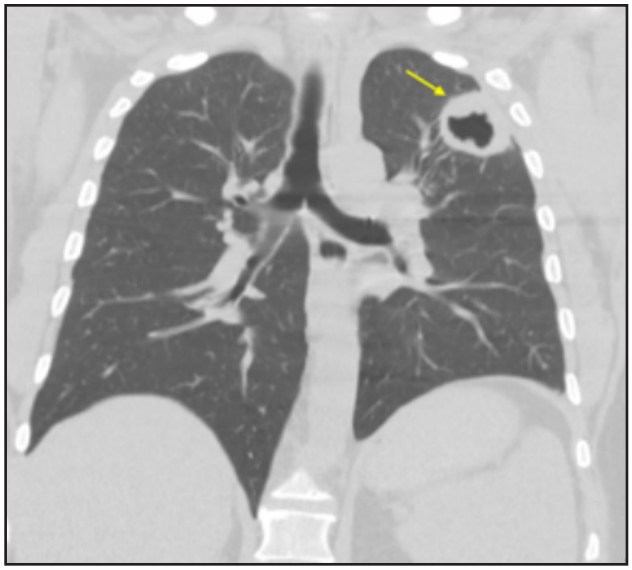

FIGURE 12. *Postprimary pattern tuberculosis. Coronal noncontrast CT image demonstrating cavitation with a thick nodular wall (yellow arrow) involving the left upper lobe apicoposterior segment.*

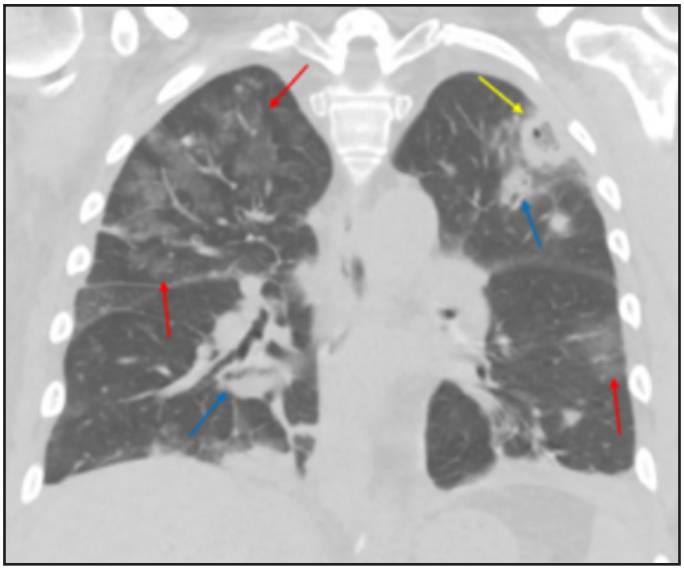

FIGURE 13. *Multifocal MSSA pneumonia. Coronal noncontrast CT demonstrating thick-walled left upper lobe abscess (yellow arrow), multilobar consolidations (blue arrows), and ground-glass opacities (red arrows).*

with immunosuppressive drugs. The nodules and pulmonary disease increase in size and number with progression; however, remission rate is approximately 90% with appropriate treatment.[19] Renal failure is the most common cause of death in this patient population.

Rheumatoid Arthritis

Rheumatoid arthritis (RA) is a progressive, systemic autoimmune disorder that is commonly an articular disease, although does present with extra-articular symptoms. The lung is a common site of extra-articular disease and can manifest as airway, parenchymal, or pleural disease.

Necrobiotic rheumatoid nodules of the lung are uncommon and are usually seen in conjunction with a high rheumatoid factor. These nodules are usually large, discrete subpleural nodules, which may develop cavitation. This cavitation can lead to hemoptysis, spontaneous pneumothorax, and the development of a bronchopleural fistula.[20]

RA commonly presents with interstitial lung disease, most commonly as UIP and NSIP. There are multiple risk factors for developing RA interstitial lung disease including smoking history, advanced age, high-titer rheumatoid factor, and a family history of RA.[21]

Pyoderma Gangrenosum (PG)

Pyoderma Gangrenosum is a rare neutrophilic dermatologic disease that occasionally can present with extracutaneous manifestations, which include pulmonary involvement. Etiology is unknown; however, more than 50% of these patients are associated with an underlying systemic disorder such as inflammatory bowel disease, rheumatoid arthritis, or hematological disorders. Pulmonary disease manifestations are rare and are usually diagnosed simultaneously or weeks to years after a cutaneous disease diagnoses.[22]

Chest imaging is nonspecific in these patients; however, they can present with pulmonary cystic change and large cavitating nodular lesions or consolidation. The cavitating masses are secondary to nodular lesions that develop central caseation (**Figure 10**) and commonly require histopathological assessment for diagnosis.[23]

Sarcoidosis

Sarcoidosis is a systemic chronic granulomatous disease characterized by unique noncaseating granulomas in multiple organs. The lungs are involved in more than 90% of these patients.[24] Peak age for presentation is 20 to 30 years and patients generally present with mild cough, dyspnea, or fatigue. This disease should be considered in patients younger than 40 with mild clinical symptoms and bilateral hilar and mediastinal lymphadenopathy on chest x-ray.[24]

The greatest morbidity and mortality in this patient population is from thoracic involvement and approximately 20% of these patients will progress to chronic interstitial lung disease. Patients display CT findings of cavitary nodules when central cavitation occurs from ischemic necrosis or angiitis of the nodules.[25] However, the classic pulmonary presentation of sarcoidosis is bilateral hilar and right paratracheal lymphadenopathy with perilymphatic micronodular disease.

Vascular

Septic Emboli

Infected embolic material can seed the lung parenchyma from an extrapulmonary source through the pulmonary vasculature. This occurs most commonly through infected foreign body material or by less likely etiologies such as infective endocarditis or Lemierre syndrome. *Staphylococcus aureus* is the most common organism related to foreign body infection and IV drug abuse. This can occur with infected venous catheters, pacemaker wires, or other

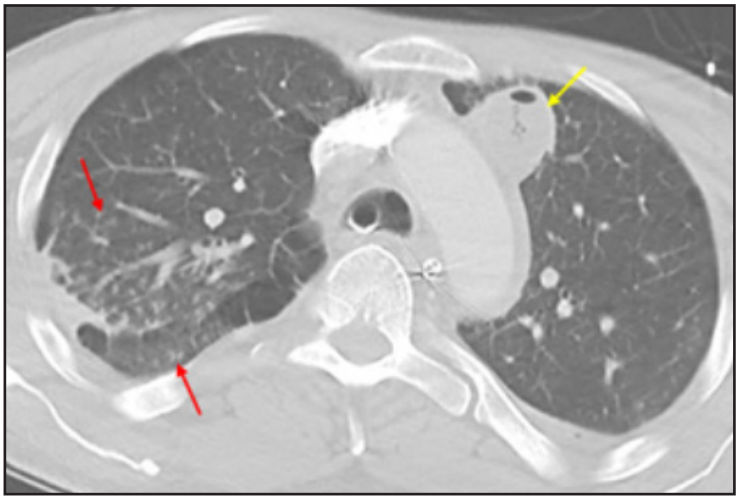

FIGURE 14. *Aspergillosis. Axial contrast-enhanced CT image of an immunocompromised patient with disseminated aspergillosis demonstrating a left upper lobe cavitary lesion (yellow arrow) and right upper lobe tree-in-bud opacities (red arrows).*

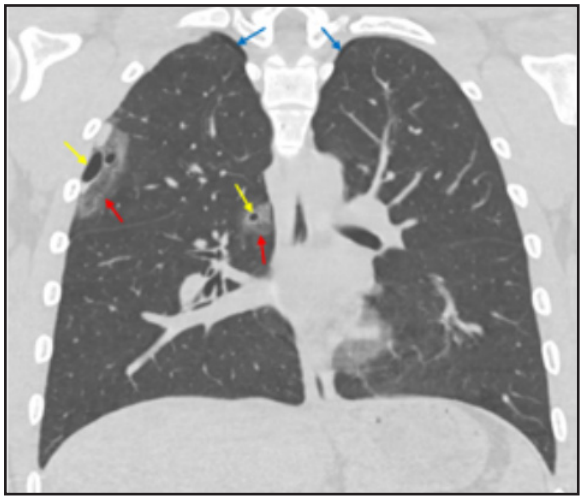

FIGURE 15. *Pulmonary laceration. Coronal unenhanced CT image in a patient after blunt chest trauma demonstrating pulmonary lacerations (yellow arrows) with surrounding ground-glass opacities representing hemorrhage (red arrows). Small biapical pneumothoraces (blue arrows) are also visualized.*

indwelling catheters.[26] Infective endocarditis of the tricuspid valve is the most commonly affected valve leading to septic embolic disease. Also, Lemierre syndrome is a possible etiology that occurs when acute pharyngotonsillitis leads to jugular vein septic thrombosis, possibly resulting in septic emboli.[27]

Imaging findings of pulmonary septic emboli include multiple discrete nodules ranging from 0.5 - 3.5 cm. The nodules are usually bilateral and peripheral with central cavitation (**Figure 11**). Other imaging features seen in this entity include a ground-glass halo surrounding the nodules as well as a "feeding vessel sign." Vessels can be seen leading directly to the nodule and are found in 60% to 70% of patients.[27,28]

These patients are treated with broad spectrum antibiotics, sometimes up to 6 to 8 weeks in cases of infective endocarditis. Removing the source of infection is imperative for improvement.

Infection

Tuberculosis and Mycobacterium Avium Complex

A myriad of bacterial, fungal, and parasitic organisms can lead to cavitary lung disease. Among them, the most familiar causative organism is *Mycobacterium tuberculosis* (TB). The incidence of TB has been decreasing in the US but remains a daunting threat among the immunocompromised population. In addition, immigrants from endemic regions (Asia, Africa, Russia, Eastern Europe and Latin America), those with low incomes and limited access to health care, intravenous drug users, people who live or work in high-risk residential centers (nursing homes, correctional facilities and homeless shelters), and health care workers are still vulnerable to opportunistic organisms.[29]

TB is divided into primary vs postprimary tuberculosis. Imaging findings for primary tuberculosis include pulmonary consolidation, effusion, and lymphadenopathy. In postprimary tuberculosis, the most common imaging findings include cavitary lesions in which patients present with fever, night sweats, weight loss, and cough. Cavitary lesions in postprimary TB tend to be in the apical regions with thick irregular walls. Surrounding airspace opacity can also be observed. In cases of superinfection, cavities may present with internal air-fluid levels. Detection of cavitary lesions on imaging do prolong the overall length of treatment (**Figure 12**).[30]

Bacterial Pneumonia/Abscess

Pulmonary abscesses are most often caused by organisms in the oral cavity, with *staphylococci* and *streptococci* the most common. Lung abscesses are divided into acute (< 6 weeks) vs chronic (> 6 weeks). Patients often present with symptoms of fever, chills, fatigue, night sweats, productive cough, and weight loss. Unlike TB, abscesses > 2 cm are almost always found with internal air-fluid levels (**Figure 13**).[31] Lung abscesses are commonly treated with antibiotics for at least 3 to 6 weeks. Moreover, abscesses > 6 cm should be considered for surgical resection or percutaneous transthoracic tube drainage.[32]

Aspergillosis

Pulmonary aspergillosis is almost exclusively seen in patients with immunodeficiency or with chronic lung disease such as chronic obstructive pulmonary disease (COPD). Many people in the general population are exposed to aspiration of aspergillosis, but only immunodeficient patients will develop clinical symptoms. Types of aspergillosis include aspergilloma, invasive aspergillosis, and semi-invasive aspergillosis. An aspergilloma is when a fungal collection, or "fungal ball," develops in a pre-existing cavity and is commonly seen in immunocompetent patients. Semi-invasive aspergillosis is a chronic necrotizing pulmonary aspergillosis, which can develop cavitary

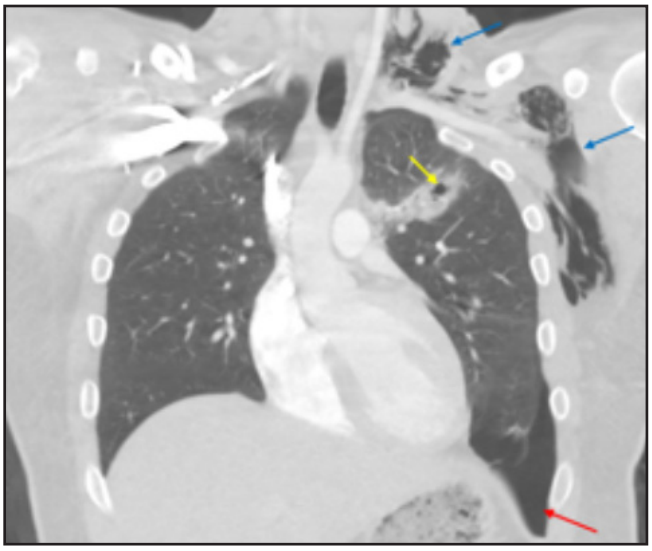

FIGURE 16. *Pulmonary laceration and associated hemorrhage. Axial and coronal contrast-enhanced CT images in a patient with a gunshot wound demonstrating a pulmonary laceration and hemorrhage (yellow arrow) with a pneumothorax (red arrow). Left supraclavicular and lateral chest wall subcutaneous emphysema (blue arrows) is also present.*

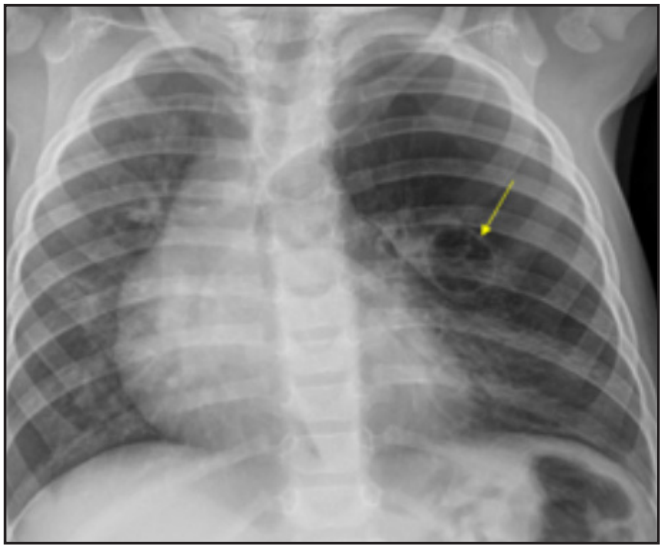

FIGURE 17. *Congenital pulmonary airway malformation (CPAM). Frontal radiograph in a pediatric patient demonstrating a left central thin-walled lesion (yellow arrow) consistent with a CPAM.*

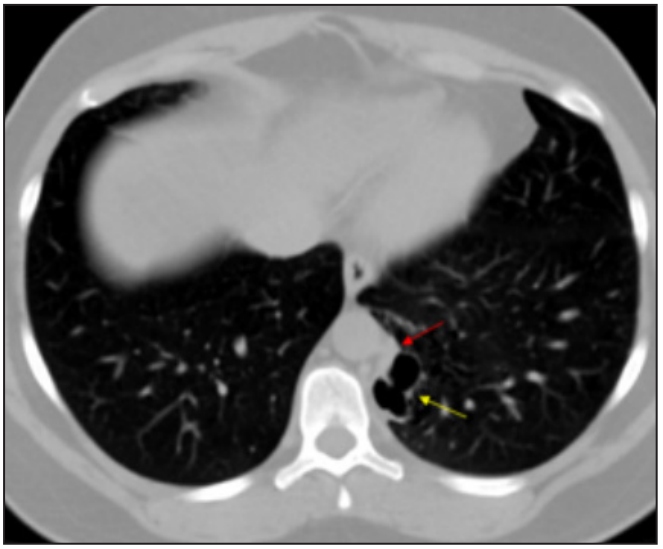

FIGURE 18. *Bronchogenic sequestration. Unenhanced CT image demonstrating a thin-walled septate cystic lesion (yellow arrow) with a feeding systemic vessel (red arrow).*

lesions (**Figure 14**).[33] Lastly, invasive aspergillosis is seen in severely immunocompromised patients with a rapidly progressive angioinvasive fungal infection and often presents with a pulmonary cavitary lesion and "air crescent sign." Typical patient presentation of chronic pulmonary aspergillosis is a middle-aged male with symptoms of weight loss, loss of appetite, productive cough, pleuritic chest pain, and often hemoptysis. Sometimes the cavity may contain an aspergilloma, which is a conglomerate of aspergillosis hyphae, fibrin and cell debris. Treatment options for aspergillosis include azoles followed by inhaled amphotericin B for a prolonged period of 6 to 12 months.

Trauma

Pulmonary Laceration

Pulmonary laceration, the tearing of lung parenchyma, occurs secondary to traumatic compression, shearing forces, direct injury from rib fractures, or at the site of previously formed pleural adhesions.

Having a round or oval shape with varying number of lesions and sizes, laceration has a highly variable appearance on imaging. Pulmonary laceration is often obscured on chest radiography the first 48 to 72 hours because of surrounding associated pulmonary contusion (**Figures 15 and 16**). CT is more sensitive in detecting pulmonary lacerations offering a more comprehensive assessment of the extent of pulmonary injury and laceration. Air and/or blood may fill in the laceration creating a thin pseudomembrane. Occasionally, active bleeding into a pulmonary laceration can be seen on contrast-enhanced CT. It presents as a linear density, similar to that of the blood pool, forming in or along the periphery of the laceration.[34] Uncommon complications include bronchopleural fistula and abscess, with the former occurring more often in the setting of peripheral lacerations.[34] Healing times vary from weeks to several months for laceration, depending on the severity and associated injury. Having an accurate history during the interpretation of subsequent radiographs or CT scans is critical to prevent mistaking a blood-filled cystic laceration for a neoplasm.[33]

Pneumatocele

Pneumatoceles are thin-walled cystic spaces in the parenchyma of the lung.[33] It is not uncommon for pneumatoceles to contain fluid, creating an air-fluid level observed on imaging. Infection is the major cause of pneumatoceles, with other

etiologies including positive pressure ventilation, hydrocarbon ingestion, and blunt trauma.[34] In the setting of antecedent trauma, pneumatoceles are most often seen as cystic spaces with peripheral ground-glass attenuation. As with pulmonary lacerations, healing may take several weeks, and will often completely resolve. Rare complications include spontaneous rupture with pneumothorax and secondary infection.[35] Rarely, surgical resection of a pneumatocele may be required if the patient has recurrent infections, mass effect, or recurrent rupture with resultant pneumothorax.[34]

Congenital ("Youth")

Congenital Pulmonary Airway Malformation

Congenital pulmonary airway malformation (CPAM) includes a spectrum of pulmonary disease that affects varying aspects of the tracheobronchial tree and distal airways. The pathophysiology of CPAM development is controversial, but the entity involves an abnormal mass of pulmonary tissue with differing levels of cystic components that communicate with the tracheobronchial tree. This lesion has normal vascular supply and drainage, which differentiates it from pulmonary sequestrations.[36] The abnormality is usually identified during routine obstetric care, due to increased use of ultrasound, or in children presenting with cough and abnormal chest radiography. It can rarely present in adulthood with recurrent pulmonary infections.

These lesions vary in histological and imaging presentation and have been classified into type 0 through IV. Type 0 is acinar dysplasia or agenesis and is incompatible with survival. Type I is the "large-cyst type" on imaging and typically affects a single lobe with cyst size 1-10 cm (**Figure 17**). Type II is the "small cyst type" on imaging, which has small lesions < 2.0 cm. Type III is a solid-appearing lesion with microcysts that coalesce to form a solid-appearing mass on imaging. Type IV disease typically affects a single lobe and has large thin-walled cysts and cannot be readily discernible from type 1 by imaging. Type II and III lesions have a poor prognosis while type I and IV lesions have a much better prognosis.[37]

Bronchopulmonary Sequestration

A sequestration is a congenital region of abnormal lung tissue that does not connect to the bronchial tree or pulmonary arteries and can present as a cystic lesion on imaging (**Figure 18**). The abnormality commonly presents in the lower lobes, most often the left lower lobe. Often the abnormality presents on chest radiography as a persistent lower-lobe opacity in a patient with recurrent pneumonia. Sequestrations often have a systemic feeding artery arising from the descending aorta, which can be visualized on CT or MR imaging. Sequestrations can also be seen with elements of other congenital pulmonary malformations, including CPAM, with "hybrid" lesions sometimes visualized.[38] Bronchopulmonary sequestrations can be divided into intralobar and extralobar types.

Intralobar sequestration is more common (approximately 75%), is intrapleural in location, and commonly has pulmonary venous drainage. It usually presents as an isolated anomaly and is often present in older children or adults who present with recurrent pneumonia. Extralobar sequestration is less common (25%) and is extrapleural in location with a separate pleural lining from the normal lung parenchyma. The extralobar sequestration usually has systemic venous drainage.[39] In symptomatic cases, patients often require surgical resection of the abnormal lung tissue.[40]

Bronchogenic Cyst

Bronchogenic cysts are ventral foregut cysts that occur with abnormal foregut budding between the 26th and 40th day of gestation. They can occur in the mediastinum and pulmonary parenchyma. The mediastinal cysts predominantly occur in the middle or posterior mediastinum and are typically subcarinal extending toward the right hilum. The pulmonary bronchogenic cysts are commonly lower lobe, and more often in the medial aspect. These cysts are well-marginated with thin walls, spherical, and often are simple cysts with no internal debris. They can have internal high attenuation on CT with intraluminal mucoid, hemorrhagic, or viscous contents. They rarely contain internal air or air-fluid levels. CT is often diagnostic of these lesions documenting their fluid-attenuation; however MR, can be useful in patients with indeterminate lesions.[41] These cysts are often asymptomatic, but can rarely present with chest pain, cough or infection. For symptomatic lesions, aspiration or ablation is common, or surgical resection can be performed if they are recurrent and symptomatic.[42]

Conclusion

When radiologists encounter pulmonary cavitary lesions the differential diagnosis is broad. Pertinent clinical history and imaging findings can help distinguish between the multitude of entities and allow the clinician to expedite appropriate patient management, ultimately improving clinical outcomes.

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