

Infectious pneumonia in the immunocompetent host: What the radiologist should know

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Abstract

Lung infections are an important cause of morbidity and mortality, particularly because of the rising antimicrobial resistance. According to the clinical setting, they can be categorized as community-acquired pneumonia and hospital-acquired pneumonia. Radiological patterns of lung infections are lobar consolidation, bronchopneumonia, interstitial pattern, and nodular pattern. In addition, typical imaging features of several infections serve as “red flag signs” in reaching a diagnosis or altering the management. It would be prudent for the radiologist to be well informed regarding these aspects of lung infections to be able to make a valuable contribution to the management.

Key words: Imaging; immunocompetent; lung infections; pneumonia; radiological signs

Introduction

The most common question put up to an Indian radiologist in the setting of pulmonary infection is “does it look tubercular?” Once tuberculosis is ruled out, most of our job is considered done. We need to, however, contribute more to the diagnostic trail. Lung infections caused by organisms other than *Mycobacterium tuberculosis*, are, nevertheless, a major cause of morbidity and mortality. In recent times, there has been an alarming increase in drug resistance strains. Recent data from the Indian intensive care unit (ICU) setting shows that most of the pathogens are resistant to common antibiotics.^[1] Empirical broad spectrum antimicrobial therapy is one of the causes responsible for this. Unusual infections are encountered more frequently now because of increased survival in acquired immunodeficiency syndrome (AIDS) patients,

incidence of organ transplants, and emergence of newer viruses with atypical imaging features. Radiologists need to be more aware about the dominant imaging patterns in different types of these infections and the “red flag” imaging signs which make a difference to the management.

Clinical Setting

The most widely referenced guidelines in pneumonia are the Infectious Disease Society of America (IDSA) and American Thoracic Society (ATS) guidelines. A consensus guideline document was issued by these two societies in 2007.^[2] Among its various recommendations was the development of locally adapted guidelines. Accordingly, Indian Chest Society and National College of Chest Physicians (India) developed national pneumonia guidelines.^[3] As per the IDSA/ATS

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guidelines, community-acquired pneumonia (CAP) is defined as “an acute infection of the pulmonary parenchyma that is associated with at least some symptom of acute infection, accompanied by the presence of an acute infiltrate on a chest radiograph or auscultatory findings consistent with pneumonia, in a patient not hospitalized or residing in a long-term care facility for more than 14 days before the onset of symptoms.^[2] The definition itself includes confirmation by chest radiograph (moderate recommendation, level III evidence). The role of the radiologist is, therefore, to confirm the presence of lung infiltrate and its severity. In simple cases, if the clinical improvement upon treatment is satisfactory, then a follow-up radiograph is not recommended (Strong recommendation, level II evidence).^[3] Computed tomography (CT) scan is not routinely indicated in the diagnosis of CAP (strong recommendation, level II evidence).^[3]

Hospital-acquired pneumonia (HAP) is defined as pneumonia occurring ≥48 hours after admission not incubating at the time of admission. Related terms are ventilator-acquired pneumonia (VAP) and healthcare associated pneumonia (HCAP). VAP refers to pneumonia that arises after more than 48 hours of endotracheal intubation. As per the IDSA/ATS consensus statement, HCAP includes any patient who was hospitalized in an acute care hospital for 2 or more days within 90 days of the infection; resided in a nursing home or long-term care facility within the past 30 days of the current infection. HCAP is included in the spectrum of HAP and VAP, and patients with HCAP need therapy for MDR pathogens.^[2,4] In the Indian scenario, similar long-term health care facilities are uncommon and the term HCAP is avoided in the ICS/

NCCP (I) guidelines.^[3] The diagnosis of HAP/VAP is made by the presence of signs and symptoms to acute infection with appearance of new or progressive infiltrate on the chest radiograph or by positive culture samples. It can be divided into early or late HAP/VAP depending upon whether the pneumonia occurs within or after 4 days of admission/mechanical ventilation, respectively.^[2] CT is not routinely indicated in the routine diagnosis of HAP/VAP (strong recommendation, level III evidence).^[3]

A comparison of the pathogens responsible for CAP and HAP is presented in Table 1. Most common organism in both CAP and early onset HAP is *Streptococcus pneumoniae*. In more severe cases of CAP requiring inpatient care, additional organisms such as *Legionella pneumophila* and mixed infection in aspiration pneumonitis can be found. More virulent pathogens such as *Staphylococcus aureus* and Gram negative bacilli (GNB) can be found in addition to the routine organisms in CAP patients requiring ICU care.^[5] However, in India, it is not uncommon to find Methicillin-Resistant *S.aureus* (MRSA), *Pseudomonas aeruginosa*, and Drug Resistant *S. pneumoniae* (DRSP) in CAP isolates.^[6] In late onset (>4 days) HAP, aerobic GNB and *S. aureus* are more common. Anaerobes, *Legionella*, *Pneumocystis*, tuberculosis, viruses, and fungi are uncommon causes and occur irrespective of the timing of acquiring HAP.

Imaging Patterns

Lobar consolidation

Lobar pattern of consolidation occurs when the alveoli are filled with inflammatory exudate as host response to the

Table 1: Comparison of pathogens causing CAP and HAP

Patient profile	CAP	Timing of infection	HAP	%
	Organism		Organism	
Outpatient	<i>Streptococcus pneumoniae</i> <i>Mycoplasma pneumonia</i> <i>Haemophilus influenza</i> <i>Chlamydomphila pneumonia</i> Respiratory viruses	Early onset bacterial <4 days	<i>S.pneumoniae</i> <i>H.influenzae</i>	5-20<5-15
Inpatient	<i>S.pneumoniae</i> <i>M.pneumoniae</i> <i>C.pneumoniae</i> <i>H.influenzae</i> <i>L.pneumophila</i> Aspiration Respiratory viruses	Late onset bacterial >4 days	a) Aerobic GNB <i>Pseudomonas aeruginosa</i> <i>Enterobacter spp</i> <i>Acinetobacter spp</i> <i>Klebsiella pneumoniae</i> <i>Serratia marcescens</i> <i>E. coli</i>	20-60
			b) Gram positive cocci <i>S.aureus</i>	20-40
ICU	<i>S.pneumoniae</i> <i>Staphylococcus aureus</i> <i>L.pneumophila</i> Gram negative bacilli (GNB) <i>H. influenzae</i>	Early and late onset pneumonia	Anaerobic bacteria <i>L.pneumophila</i> <i>Pjiroveci</i> <i>M.tuberculosis</i> Influenza A, B viruses RSV Fungal <i>Aspergillus</i> <i>Candida spp</i>	0-35 0-10<1 <1<1 <1<1 <1

infection. The exudate spreads across lung parenchyma through channels of collateral air drift, and hence the resultant lung opacity is homogenous and contiguous. There is no destruction of lung parenchyma and complete resolution occurs with treatment. The most common organism in CAP setting is *S. pneumoniae*. There is predilection for basal segments [Figure 1]. Less commonly, *L. pneumophila* may occur in hospitalized CAP patients. The infection begins as unifocal infiltrate but spreads bilaterally. The radiographic picture lags behind clinical status.^[7,8]

In HAP, the appearance can be more severe with the consolidation being bilateral and extensive. *S. pneumoniae* is the most common pathogen in early onset HAP. *Klebsiella* can also cause lobar pneumonia with typical “bulging fissure sign” due to copious exudates. *Pseudomonas* has a predilection for lung bases, often bilaterally, and a tendency for abscess formation. Extensive necrotizing pneumonia can be caused by *S. aureus*, *Pseudomonas*, *Klebsiella*, and tuberculosis. Bilateral upper lobe consolidation with coalescent lucencies and “air crescent sign” can be seen [Figure 2].

Aspiration pneumonia needs a special mention in the setting of HAP because here additional lung injury due to acidic gastric contents. Infection is mostly polymicrobial and begins in the distal dependent airways as bronchopneumonia, but can progress to lobar consolidation. Air bronchogram is mostly absent because of the aspirated content in the airways [Figure 3].

In children, *S. pneumoniae* can cause a rounded mass-like pattern of consolidation. Children are also prone to

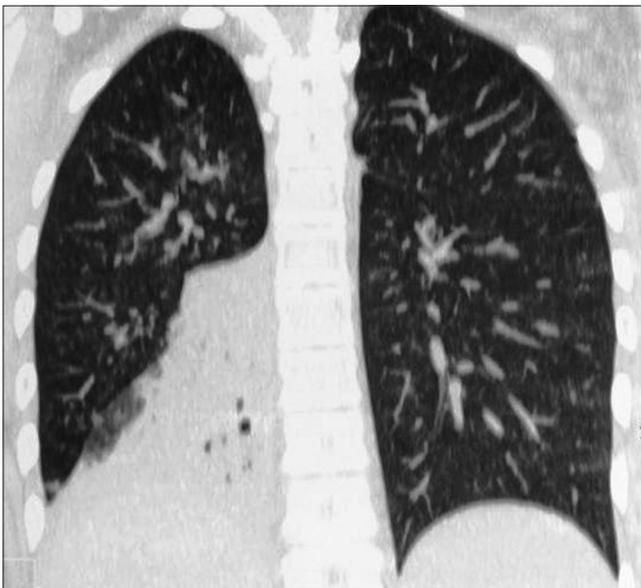


Figure 1: Coronal lung window image shows lobar pattern of consolidation in the right lower lobe. The upper margin of the consolidation is limited by oblique fissure. Air bronchogram is present

hematogenous spread from Staphylococcal pyoderma. Staphylococcal pneumonia can commonly lead to complications such as lung abscess and empyema. It is important to differentiate the two because empyema requires drainage whereas medical therapy is curative for most lung abscesses.^[7,9] Empyema typically forms an obtuse angle with the lung parenchyma and shows “split pleura sign” on CECT due to enhancement of visceral and parietal pleura [Figure 4A]. Lung abscess forms acute angle with lung parenchyma and bronchovascular structures end on the wall of abscess rather than being splayed [Figure 4B]. In the healing phase, pneumatoceles can be seen as thin-walled air filled lung cysts [Figure 4C], which can rupture to cause pneumothorax. Pneumatoceles are not specific for *S. aureus* because they can occasionally be seen in *Escherichia coli*, *S. pneumoniae*, and *Pneumocystis jiroveci* infection. A variant strain of *S. aureus* named Panton Valentine Leukocidin toxin positive (PVL+ve) was discovered in 2002 and is more common in children than adults. There is a history of rapid deterioration of flu-like illness caused by alveolar hemorrhage and extensive necrotizing pneumonia [Figure 5].^[7,10]

Bronchopneumonia

Bronchopneumonia begins in the distal airways as bronchiolitis with or without peribronchial inflammation. It is seen on chest radiograph as scattered infiltrates, which are frequently bilateral. The opacities are smaller because it is usually seen in the setting of virulent organisms and poor host response such as HAP and aspiration pneumonia. Smaller infiltrates can later form larger coalescent opacities [Figure 6]. On CT, bronchial wall thickening and centrilobular nodules suggesting infectious bronchiolitis can be seen initially. Apart from bacteria such as *S. aureus*, *GNB*, *Mycoplasma*, *Chlamydia*, fungal infections such as *Aspergillus* can also give rise to bronchopneumonic pattern.^[7,8]

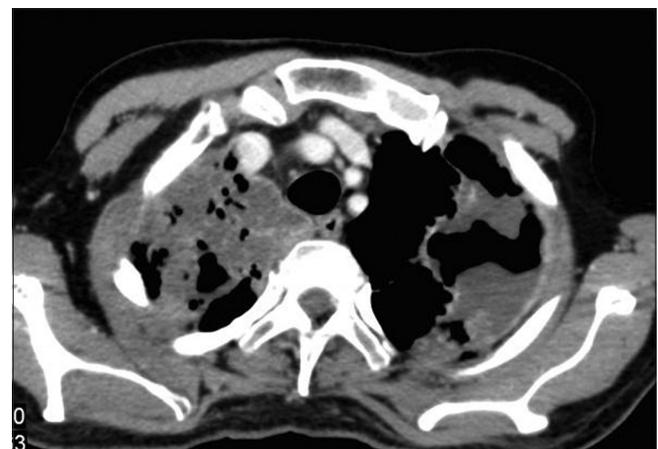


Figure 2: Bilateral upper lobe consolidation with extensive necrosis and areas of cavitation. Blood culture in this patient was positive for *S aureus*

Interstitial pattern

Interstitial pattern results when there is cellular infiltrate in the alveolar septae and peribronchovascular interstitium. Ground glass opacity (GGO) is caused by incomplete alveolar filling. In the immunocompetent patient, interstitial pattern of pneumonia is caused by viruses and *Mycoplasma pneumoniae*. Uncommon rickettsial infections such as scrub typhus should also be considered in the differential in appropriate endemic setting [Figure 7A and B].

M. pneumoniae is a bacterial pathogen causing interstitial CAP. Bronchiolitis is the most distinct finding on CT, seen as poorly-defined centrilobular nodules. Areas of patchy air-space consolidation or GGO in lobular distribution, bronchopneumonia, thickening of the axial interstitium, and interlobular septa can be commonly seen.^[11]



Figure 3: Lobar consolidation in right lower lobe due to aspiration. Air bronchogram is absent. Centrilobular nodules are seen in dependent location in left lower lobe also

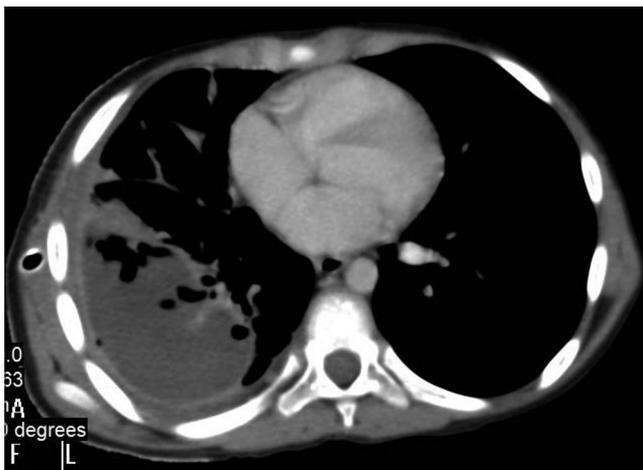


Figure 5: A 7-year-old child with 15 days history of cough, cold, and flu-like symptoms with rapid clinical deterioration requiring hospitalization. Large area of liquefied necrotic consolidation with cavitation in right lower lobe. PVL positive *S. aureus* should be considered in such patients

Viral pathogens are more commonly reported than previously estimated in CAP. Respiratory viruses were detected as a pathogen in 37.8% adult outpatients with LRTI in a recent study.^[12] The range of viruses producing pneumonia in adults includes common agents, such as varicella-zoster virus and influenza virus, as well as respiratory syncytial virus, human metapneumovirus,

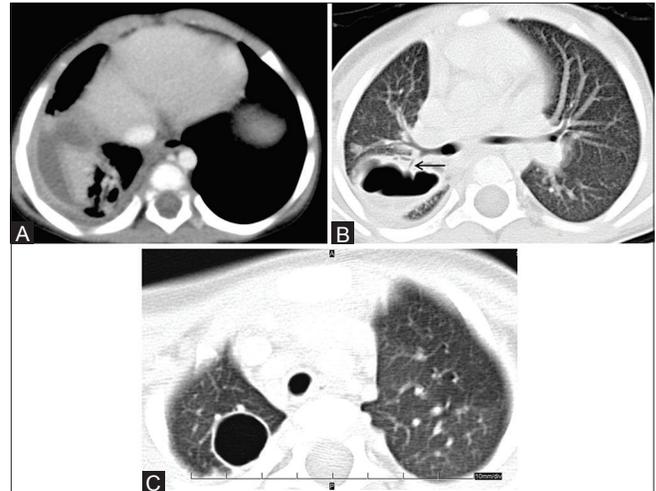


Figure 4 (A-C): Right lobe Staphylococcal pneumonia. (A) lentiform fluid collection in right pleural cavity with thickening and enhancement of visceral and parietal pleura (split pleura sign) suggestive of empyema. (B) Cavity with air-fluid level in apical segment of right lower lobe. Bronchi are seen to end at the margin of the cavity (arrow) rather than being splayed, indicating intraparenchymal abscess. (C) Pneumatocele seen as a thin-walled air filled cyst

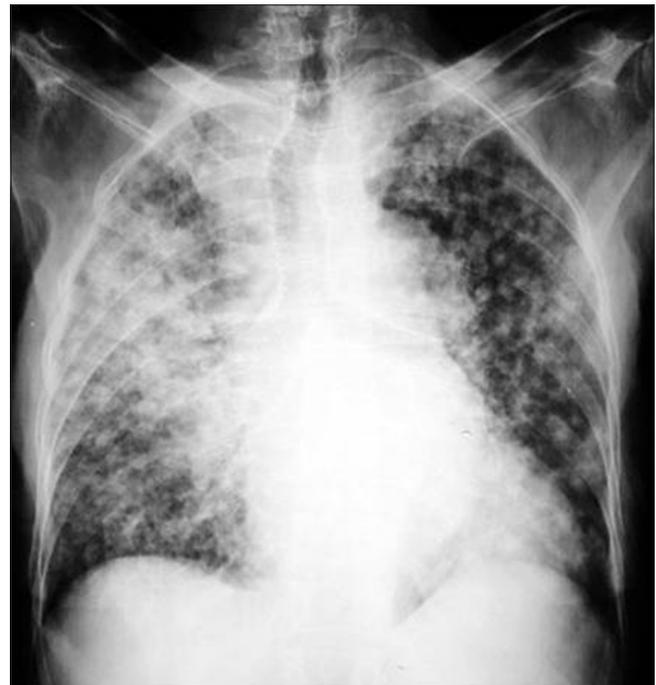


Figure 6: Chest radiograph showing extensive and coalescent larger acinar opacities in bronchopneumonia

adenoviruses, picornaviruses, and coronaviruses. Adenoviruses more commonly cause severe multifocal pneumonia in institutionalized elderly patients.^[13]

There is a considerable overlap in CT features of various types of viral pneumonias which are broadly described as (a) parenchymal attenuation disturbances, (b) GGO and consolidation, (c) nodules, micronodules and "tree-in-bud" opacities, (d) interlobular septal thickening, (e) nodules with halo sign, (f) pleural effusions, and (g) lymphadenopathy. GGO is the most common feature seen in almost all of the viral pneumonias, but is not a typical feature of adenoviruses.^[14] The distribution

of GGO is also important because subpleural and peribronchovascular predominance is a feature of H1N1 [Figure 8].^[15,16] The Patterns of pulmonary infiltration caused by Influenza viruses are most variable and do not significantly differ between immunocompetent and immunocompromised patients or between different types and subtypes of Influenza virus.^[12,17] Nodules and micronodules are seen in common viral infections such as influenza and parainfluenza viruses, measles, RSV, varicella, and CMV, but are not common in H1N1, H5N1, enterovirus, and adenovirus. Septal thickening is a predominant feature in measles, Hantavirus, and SARS, but is not a typical feature of other more common viruses.

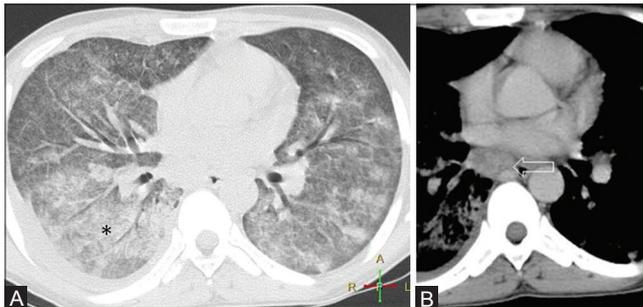


Figure 7 (A and B): A 40-year-old patient with acute onset of fever with dyspnea, not resolving with antibiotics. (A) Interstitial pneumonia in the form of well-defined consolidation, ill-defined scattered acinar infiltrates with air bronchograms (asterisk), GGO, and diffuse micronodular pattern. (B) Adenopathy (open arrow) seen on mediastinal window. There were microabscesses in the liver and spleen and ascites. The serology was positive for scrub typhus. Significant reduction in the lung lesions was seen after tetracycline therapy



Figure 8: A 40-year-old patient with fever, dry cough, headache, and dyspnea. Bilateral consolidation and GGO with subpleural and peribronchovascular predominance is seen. Throat swab was positive for H1N1



Figure 9: Chest radiograph in an infant with fever and respiratory distress shows features of bronchiolitis as hyperinflated lung fields and fine linear opacities due to atelectasis



Figure 10: A 12-year-old child with septicemia. Multiple well-defined nodules, more in subpleural region and most of them show a "feeding vessel sign" (arrow), suggesting septic emboli. Cavitation is seen in many nodules

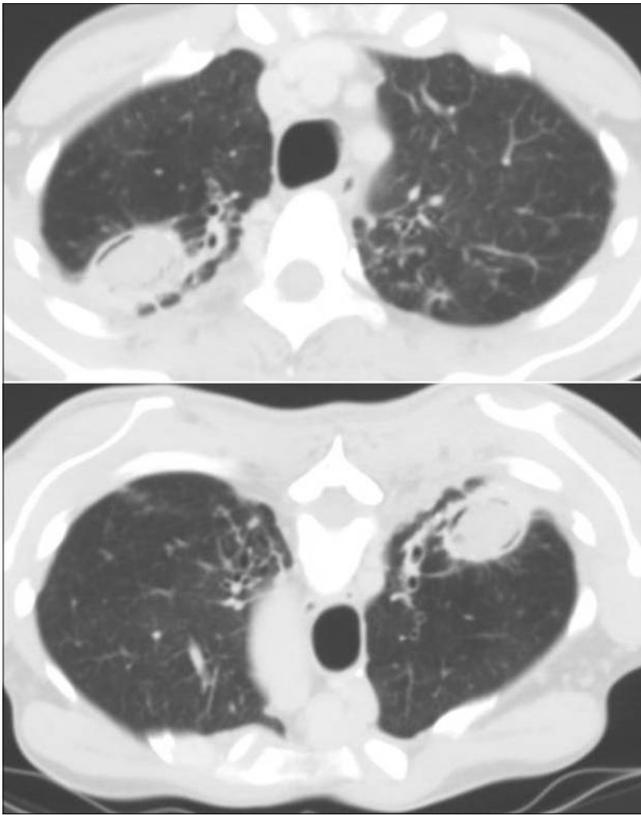


Figure 11: Supine and prone axial CT scans showing “air crescent” sign of a dependent fungal ball in old lung cavity



Figure 13: Serpentine old calcified guinea worm in muscles of the chest wall in a 60-year-old female overlying the right lung shadow

Bronchial or bronchiolar wall thickening is common in RSV, measles, and adenovirus but not common in others. Pleural effusion and lymphadenopathy is an additional feature of measles virus not encountered with others. Nodules with “*peripheral halo sign*” are more typical of DNA viruses.^[14]

RSV is the most frequent viral cause of respiratory tract infection in infants. Acute bronchiolitis is the main presenting feature and lung hyperinflation is the predominant radiographic finding [Figure 9].

Nodular pattern

Macronodular pattern can be seen in hematogenous infections such as septic emboli [Figure 10] or by granulomatous and fungal infections. Parasitic infections such as Paragonimiasis are not uncommon in the



Figure 12: Coronal reconstructed CT image showing fluid filled branching bronchoceles or “gloved finger sign” in ABPA

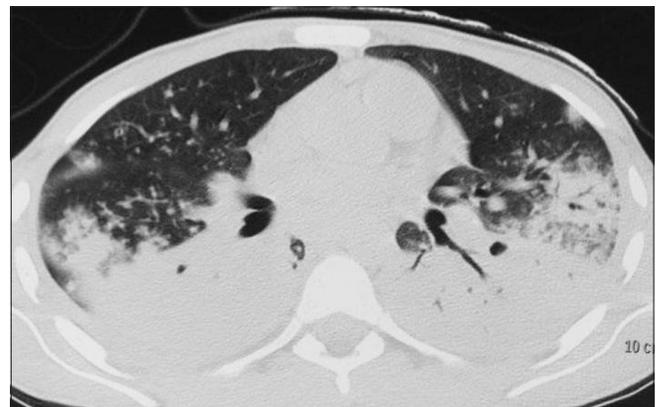


Figure 14: Bilateral dependent consolidation with air bronchogram. There is an anteroposterior gradient from normal lung to GGO to consolidation suggesting ARDS in the appropriate clinical setting



Figure 15: Wedge-shaped subpleural GGO in both lung apices. CT pulmonary angiogram in the revealed right pulmonary artery thrombosis



Figure 16: "CT angiogram sign." Lobar consolidation with homogenous low attenuation and visibility of pulmonary vasculature through it

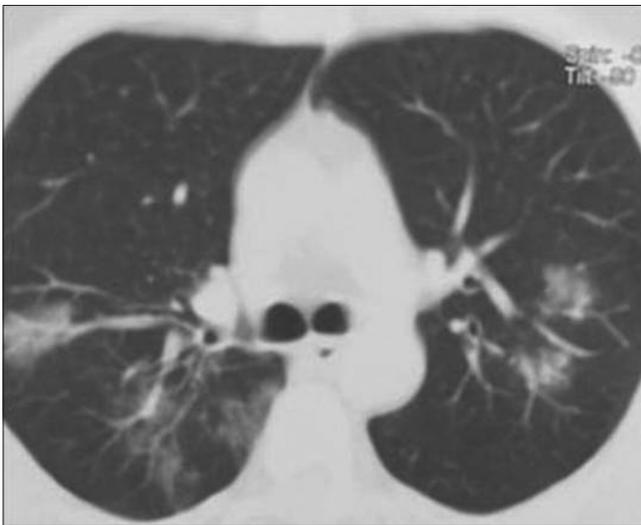


Figure 17: A 50-year-old male patient with dry cough for 6 months. Patchy wedge-shaped GGO which tend to be subpleural in location. The S.IgE levels in this patient were markedly raised upto 1818 kU/L, suggestive of chronic eosinophilic pneumonia

northeastern parts of India. The worm cysts are seen as nodules once the initial exudative or hemorrhagic consolidation has cleared.^[18]

Associated abnormalities

Mediastinal adenopathy

Most nonnecrotic enlarged nodes associated with lung infections are reactive in nature. Necrotic nodes make a case for tuberculosis and in immunodeficiency state, fungal infections such as candidiasis.^[19]

Pleural fluid

Parapneumonic effusions are common in *S. pneumoniae*. They resolve along with the pneumonia with antibiotic therapy and need drainage only if large causing lung collapse or loculated. Empyema develops when infection spreads to pleural cavity. Management of empyema is more aggressive

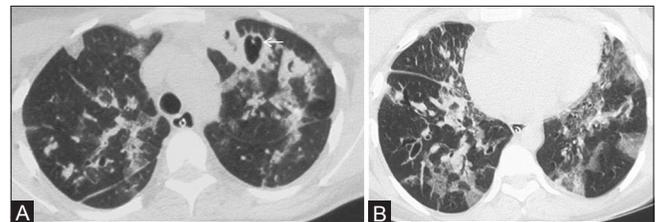


Figure 18 (A and B): A 40-year-old male patient with cough and hemoptysis. (A, B) Combination of peripheral wedge-shaped GGO, consolidation, and cavity in left upper lobe (arrow), indicating a pulmonary vasculitis. ANCA was positive suggestive of granulomatous polyangitis

Table 2: Imaging signs in lung infections

Imaging Sign	Condition
Air bronchogram sign	Alveolar consolidation
Bulging fissure sign	Klebsiella
Air crescent sign	Necrotising pneumonia: <i>S.aureus, Pseudomonas, Klebsiella</i> and tuberculosis
	Aspergilloma
Split pleura sign	Empyema
Tree-in-bud sign	Bronchiolitis
Feeding vessel sign	Septic emboli
Gloved finger sign	ABPA
Water lily sign	Hydatid cyst
Serpentine calcification sign	Guinea worm
CT angiogram sign	Mucinous adenocarcinoma lung

with percutaneous or surgical drainage. Left untreated, pleural space is obliterated with enhancing thick visceral and parietal pleura with no intervening fluid. This can be treated only by surgical removal of the thick pleural peel.^[7,19]

Lung infections with pathognomic radiological appearances

Although all patterns of lung infection have a differential diagnosis, there are certain conditions with pathognomic

radiological appearances.^[19-22] Examples are illustrated in Figures 11–13.

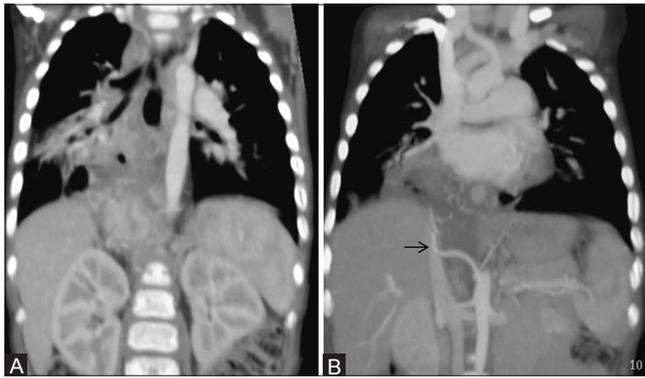


Figure 19 (A and B): A 5-year-old male child with recurrent right lower lobe pneumonia and episode of massive hemoptysis. (A) Mass-like consolidation with cavitation in right lower lobe, with caudal extension into the retroperitoneum. (B) CT angiography shows an arterial feeder from the celiac artery (arrow). Radionuclide examination was positive for ectopic gastric mucosa indicating an infected transdiaphragmatic duplication cyst

The characteristic imaging signs seen in lung infections are summarised in Table 2.

Lung infection mimics and pitfalls

Various other conditions may mimic lung infections. A summary of the conditions with imaging pearls and red flag signs which help in differentiation are given in Table 3 [Figure 14–19].^[8,10,19,23-26]

An algorithm summarizing the imaging approach to differential diagnosis is presented in Figure 20.

Conclusion

In conclusion, role of the radiologist in lung infections extends much beyond confirming the diagnosis of pneumonia and ruling out tuberculosis. International as well as Indian consensus guidelines on pneumonia have been laid down. Initial chest radiograph is sufficient to confirm the diagnosis. Ultrasound is a useful tool in pleural complications. CT is indicated in

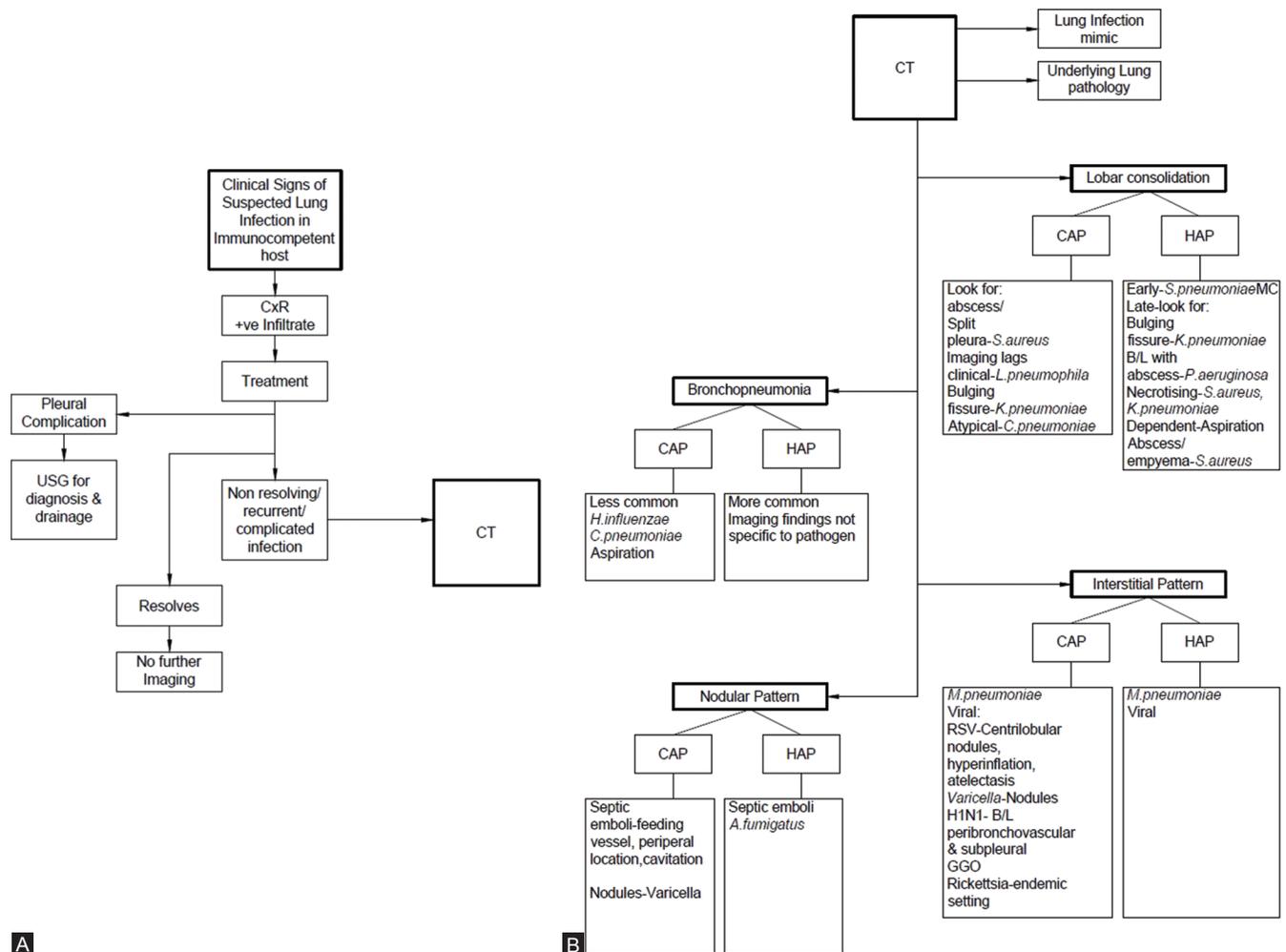


Figure 20 (A and B): (A, B) Imaging algorithm for approach to differential diagnosis of infectious pneumonia in immunocompetent host

Table 3: Conditions mimicking lung infections with imaging pearls and red flag signs

Pitfalls	Conditions	Imaging pearls and red flag signs to differentiate from infection
Air space opacification	Atelectasis	Direct and indirect features of volume loss Lack of air bronchogram in obstructive collapse
	Acute Respiratory distress syndrome	Anteroposterior gradient ranging from normal lung attenuation to ground glass to consolidation
	Pulmonary infarction	The peripheral location, wedge shape and direct visualisation of thrombus on CT pulmonary angiography
	Mucinous adenocarcinoma of lung	"CT angiogram sign"
GGO	Interstitial lung diseases: Granulomatous polyangitis Cryptogenic organising pneumonia, Eosinophilic pneumonia	Additional features like thin walled upper lobe cavities, peripheral and wedge shaped location of the opacities in pulmonary vasculitic diseases
Cavitation	Primary lung malignancy Metastasis	Wall of malignant cavity is thick (- mm) with irregularity of inner wall, Invasion of surrounding structures
	Granulomatous polyangitis Congenital anomalies	Usually inner wall of abscess is smooth and surrounding consolidation is present Associated features of primary disease Serial imaging, high index of suspicion in pediatric age group
Nodules		
Micronodules	Miliary metastasis	Associated features of primary disease
Tree-in-bud nodules	Aspiration	Dependent lower lobe
	Cystic fibrosis Diffuse panbronchiolitis	Upper lobe bronchiectasis, mosaic attenuation Diffuse and uniform
Macronodules	Metastasis Granulomatous polyangitis Lymphoma	Associated features of primary disease

nonresolving, recurrent, or complicated infections, and for guided aspiration for sampling or drainage in selected nonresponders.^[2,3,8] The dominant imaging pattern setting in which pneumonia is acquired and immune status are useful navigators in differential diagnosis. Although the imaging findings in various infections overlap, a practical approach in sync with management goals can be achieved.

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Conflicts of interest

There are no conflicts of interest.

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