Pneumonia in Normal and Immunocompromised Children: An Overview and Update

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KEYWORDS

• Pneumonia • Children • Imaging • Complicated pneumonia

Pneumonia is an infection of the lower respiratory tract, involving the lung parenchyma. The World Health Organization estimates that there are 150.7 million cases of pulmonary infection each year in children younger than 5 years, with as many as 20 million cases severe enough to require hospital admission.[1] In North America and Europe, the annual incidence of pneumonia in children younger than 5 years is estimated to be 34 to 40 cases per 1000, and decreases to 7 cases per 1000 in adolescents 12 to 15 years of age.[2,3] The mortality in pediatric patients caused by pneumonia in developed countries is currently low (<1 per 1000 per year).[3] However, pneumonia is still the number one cause of childhood mortality in developing countries.[1,4] The overarching goal of this article is to review cause, current role of imaging, imaging techniques, and the spectrum of acute and chronic pneumonias in children. Pneumonia in the neonate and immunocompromised host is also discussed.

CAUSE OF PNEUMONIA

Infectious agents causing pneumonia in children include viruses, bacteria, mycobacteria, mycoplasmas, fungi, protozoa, and helminths. Etiologic diagnoses of pneumonia are not so easy to determine or so accurate as is sometimes implied. In addition, proof of the cause of pneumonia is not obtained in most cases. There is a great deal of overlap in the radiographic appearance of pneumonias caused by different organisms. Imaging is usually poor at predicting the broad category (eg, bacterial vs viral) of infectious agent, let alone the specific agent. Preexisting lung disease may not only predispose to pulmonary infection but also modify the appearance of pulmonary consolidation. Furthermore, because the lungs can respond to a diverse disease processes in only a limited number of ways, it is common for the radiographic features of both acute and chronic infectious pneumonia to overlap considerably with many noninfectious lung diseases. Such noninfectious lung diseases are identified as pneumonia mimics in this article.

Viral pneumonia is rare in the neonatal period, because of conferred maternal antibody protection, whereas bacterial pneumonia is most frequently caused by pathogens acquired during labor and delivery, and is more prevalent in premature babies. With decreasing maternal antibody levels, viral pneumonia occurs at a peak between 2 months to 2 years of age. Bacterial infections become relatively more common in older children from 2 years to 18 years of age.[5] The lung response to an infective antigen seems to be more age-specific than antigen-dependent (ie, bacteria vs viral). Therefore, lobar and alveolar lung opacities

are more common in older children and are more frequently caused by bacterial infections, whereas interstitial opacities are seen in all age groups, and are relatively nonspecific as to the type of causative organism.[6,7]

ROLE OF IMAGING AND IMAGING TECHNIQUES

The role of imaging, including chest radiographs, ultrasound (US) and computed tomography (CT), is to detect the presence of pneumonia, determine its location and extent, exclude other thoracic causes of respiratory symptoms, and show complications such as parapneumonic effusion/empyema and suppurative lung complications.[5] Although magnetic resonance (MR) imaging is not routinely used for evaluating pneumonia in children, it is a promising imaging modality particularly for children with chronic lung conditions who require repeat imaging studies.

Frontal and lateral chest radiographs are the mainstay, and often the only, imaging needed in pediatric pulmonary infection. This imaging can be supplemented with other views such as lateral decubitus or other imaging modalities as the circumstances warrant. Decubitus views are not useful when an entire hemithorax is opacified because layering fluid cannot be identified without any adjacent air. The main use of US is to identify, quantify, and characterize a parapneumonic effusion/empyema, as well as provide image guidance for drainage and identify residual collections after treatment.[8,9] Operator availability and expertise are important factors in making US a useful tool for evaluating pulmonary infection. Although intrapulmonary fluid-filled cavities and even lung abscesses within consolidated lung can be identified on US, CT provides a more global view of the disease process. CT is often used to further evaluate: (1) suppurative lung complications and to differentiate these from parapneumonic effusion/empyema; (2) patients with recurrent or chronic pneumonia and concern for an underlying lesion; and (3) immunocompromised children with noncontributory or confusing chest radiographs and clinical findings that could be secondary to lung infection.[5]

Close attention to CT technique is crucial for imaging evaluation of pneumonia in pediatric patients. CT with low radiation dose technique should be carefully performed in all cases. Eighty to 120 kVp with weight-based low milliampere-seconds coupled with radiation dose modulation techniques is appropriate in most children for evaluation of pneumonia. Multiple CT image acquisitions are usually not needed and the scan field of view should be tailored to the area of interest (especially if following a specific lesion serially over time) to further decrease the overall radiation dose.[10] Occasionally, it may be useful to acquire additional expiratory scans to assess air trapping, which is an early imaging finding associated with small airway disease. In this situation, often at least 1 or both CT acquisitions can be obtained using a high-resolution CT (HRCT) gap technique. To obtain optimal CT imaging at peak inspiration and close to expiratory residual volume, controlled ventilation (CViCT) in infants and young children (≤5 years old) or spirometer-controlled CT in older children may be needed.[11] Young children have little intrinsic tissue contrast. Therefore, intravenous contrast is almost always needed for CT imaging of infection especially if mediastinal delineation is required. The exception is when HRCT is used only for evaluating lung parenchymal and airway disease. Breath-holding is usually desirable but can be adapted on a case-by-case basis depending on the needs of the study and the ability of the child to cooperate. However, for the study to be interpretable, gross patient motion should be absent. Sedation or anesthesia may be required in infants, young children, or children with intellectual disability. Delays between induction of anesthesia and scanning need to be minimized to prevent the potential for lung atelectasis with anesthesia. The anesthesiologist needs to pay close attention to techniques for preventing atelectasis or recruiting lung before the CT imaging.[12]

Peltola and colleagues[13] recently published their experience with MR imaging of lung infections in children using free-breathing T2-weighted, short tau inversion recovery, and T1-weighted with fat saturation precontrast and postcontrast sequences. Their study showed that lung parenchymal, pleural, and lymph node inflammatory abnormalities can be characterized by MR imaging in children with lung infection. Therefore, MR imaging might potentially be used to further evaluate suspected, acute complications of pneumonia.[13] Children with chronic lung conditions and recurrent infection, such as cystic fibrosis, who are often subjected to substantial radiation exposure from repeated CT studies, would benefit the most from MR imaging evaluation of the lungs instead of CT. Although MR imaging may not provide as much detail compared with CT especially with early, small or subtle changes (**Fig. 1**), there are promising indications of a role for MR imaging in pulmonary infection.[13–15]

RADIOGRAPHIC CHANGES IN RESPIRATORY INFECTION

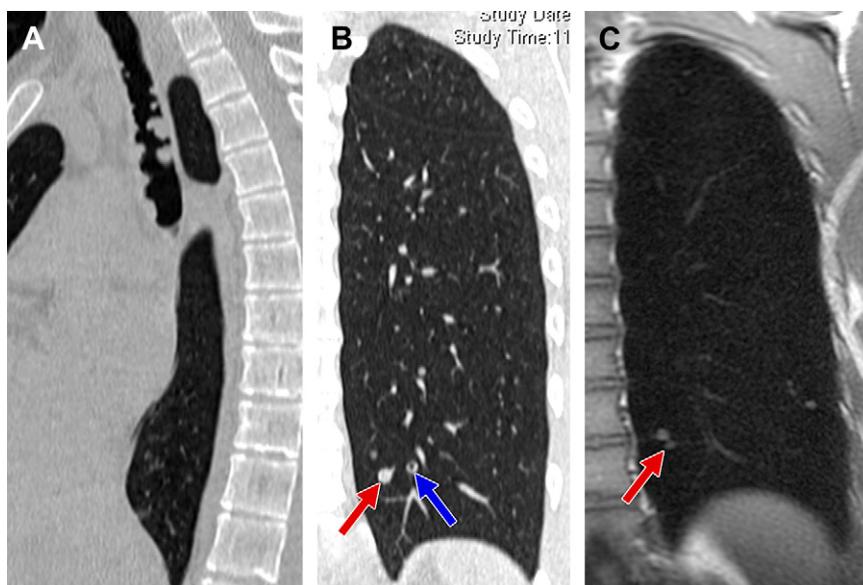There are several different descriptions of basic patterns of lung diseases on chest radiographs.

Fig. 1. Chronic lung nodules in respiratory papillomatosis in a 17-year-old male. *(A)* Sagittal contrast-enhanced (CE) CT image shows multiple intraluminal, multilobulated, nodular lesions in the trachea. *(B)* Coronal lung window CT image shows several solid parenchymal nodules (*red arrow*) in the left lower lobe. Cavitation is seen in one of the nodules (*blue arrow*). *(C)* Follow-up, coronal double-inversion recovery, MR imaging 7 weeks after CT shows the same nodules although only the larger nodule (*arrow*) seen on previous CT could be appreciated on MR imaging.

In this review article, we adopt the one described by Hansell and colleagues.[16] Almost all of these are seen as part of the spectrum of infectious lung disease (**Table 1**).

BRONCHIOLITIS VERSUS PNEUMONIA

Pneumonia and bronchiolitis are both common in infants and have overlapping clinical and imaging features. Many studies, particularly those in the developing world, use the term acute lower respiratory tract illness and make no attempt to differentiate pneumonia from bronchiolitis.[17]

Bronchiolitis occurs in children less than 2 years of age, who typically present with cough, coryza, and wheezing. Bronchiolitis is a major cause of morbidity and mortality in infants.[18] Respiratory syncytial virus (RSV) is the most common cause

Table 1	
Radiographic changes in respiratory infection	
Radiographic Pattern	**Examples of Radiographic Pattern in Specific Pneumonias**
Airspace opacities	Lobar pneumonia, round pneumonia, and bronchopneumonia
Atelectasis (collapse)	Lobar, patchy, subsegmental, linear, or discoid atelectasis may be associated with bronchiolitis and interstitial pneumonia
Linear and bandlike opacities	Bronchial/peribronchial thickening in bronchiolitis; discoid atelectasis
Cysts and bullae	Pneumatoceles, cavitary necrosis, lung abscess
Nodules and masses (solitary and multiple)	Fungal infections, septic emboli
Diffuse nodular and reticulonodular opacities	
1. Diffuse bilateral small nodular opacities	Tuberculous and nontuberculous infections
2. Diffuse bilateral reticular or reticulonodular opacities	Interstitial pneumonia
3. Diffuse ground-glass opacities	Noncardiogenic pulmonary edema (ARDS) related to sepsis or diffuse pneumonias such as pneumocystis in the immunocompromised host

of bronchiolitis, followed by rhinovirus. Other less common causes of bronchiolitis include parainfluenza virus, human metapneumovirus, adenovirus, influenza virus, coronaviruses, and human bocavirus. Chest radiographs usually show hyperinflation, perihilar opacities, peribronchial thickening, and patchy, often mobile, atelectasis (**Fig. 2**). Such imaging findings are related to diffuse airway inflammation and partial (air trapping) or complete (atelectasis) airway obstruction.[19] Similar changes are seen in older children (>2 years of age) with bronchitis although the features of diffuse small airway obstruction are less common in these older children with larger airways.

SPECTRUM OF PNEUMONIA

Pneumonia can be divided into several syndromes based on clinical presentation, imaging appearance, underlying predisposition, and cause. Pneumonia syndromes that are discussed in this article include acute focal pneumonia, atypical pneumonia, miliary or nodular pneumonia, progressive or fulminant pneumonia, aspiration pneumonias, pulmonary infiltrates with eosinophilia (PIE), and chronic or recurrent pneumonia.[20] Neonatal pneumonia is briefly highlighted separately. Pneumonia in immunosuppressed individuals is included in the general discussion of pneumonia syndromes and then specifically reviewed with regard to the different infections associated with various types of immunodeficiency. Acute and chronic complications of pneumonia are also reviewed.

Fig. 2. RSV bronchiolitis in a 6-week-old boy. Frontal chest radiograph shows perihilar streaky opacities, peribronchial thickening, hyperinflation, and patchy atelectasis.

Acute Focal Pneumonia

Characteristics that are typical for acute focal pneumonia include fever more than 38.8°C (102°F), a toxic appearance, and a focal opacity on chest radiographs. Pleuritic chest pain in lower-lobe pneumonia is sometimes referred to the abdomen and may be mistaken clinically for an acute abdominal condition. Acute focal pneumonia is most often caused by bacterial infection with streptococcus pneumonia. Other causes of acute focal pneumonia are summarized in **Box 1**. The chest radiograph of acute focal pneumonia usually shows a dense, typically more peripheral airspace opacity, which may appear segmental, lobar, or spherical (**Figs. 3** and **4**).[21–23] In a febrile child with a spherical density on a chest radiograph, the most likely diagnosis is a round pneumonia but the possibility of an underlying neoplasm may be considered. Round pneumonias tend to be solitary, have well-defined borders, and are often located in the perihilar region or posteriorly in the lungs. The radiograph should be carefully scrutinized for features of consolidation such as air bronchograms as opposed to those of a mass such as vascular/airway displacement or bony erosion. A second view such as a lateral radiograph may be helpful because a round pneumonia

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Box 1
Causes of acute focal pneumonia in children

Usual

Streptococcus pneumonia

Uncommon

Bacteria: *Hemophilus influenzae* type B, nontypable *H influenza*, *Staphylococcus aureus*, group A streptococcus, *Mycoplasma pneumoniae, Chlamydia pneumoniae*

Rare

Bacteria and mycobacteria: Francisella tularensis, Mycobacterium tuberculosis, Meningococcus, enteric bacteria

Viruses (usually lobular): RSV, parainfluenza, adenovirus, human metapneumo virus

Fungi: Histoplasma, other systemic fungi

Data from Fisher RG, Boyce TG. Pneumonia syndromes. In: Fisher RG, Boyce TG, editors. Moffet's pediatric infectious diseases: a problem-oriented approach. 4th edition. Philadelphia: Lippincott Williams & Wilkins; 2005. p. 174–221; and Brodzinski H, Ruddy RM. Review of new and newly discovered respiratory tract viruses in children. Pediatr Emerg Care 2009;25(5): 352–60.

Fig. 3. Acute focal pneumonia (ie, round pneumonia) in a 3-year-old boy. Frontal chest radiograph shows a spherical consolidation in the right upper lobe. Follow-up chest radiograph after treatment showed interval resolution of this spherical consolidation.

is often less masslike in appearance on an orthogonal view. This is one of the few scenarios in which radiologic follow-up after about 2 weeks may be useful to document interval resolution of acute pneumonia.[22] Acute respiratory distress may be secondary to an intrathoracic mass causing airway or lung compression, especially when there is complete opacification of a hemithorax on radiographs (**Fig. 5**). Intrapulmonary masses including both benign and malignant entities may present clinically with acute superinfection. In addition, other conditions or anatomic variants may be mistaken for pneumonia when a chest radiograph is obtained in a child with a fever and respiratory symptoms.
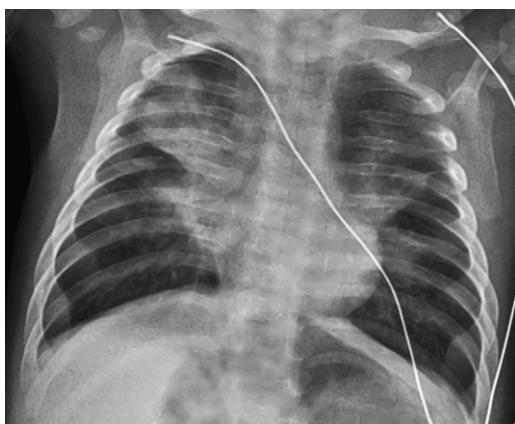

Fig. 4. Acute focal pneumonia caused by human metapneumovirus in a 3-month-old boy. Frontal chest radiograph shows multiple focal pulmonary consolidations bilaterally.

Atypical Pneumonia

Atypical features in pneumonia include prominent extrapulmonary features (eg, headache, sore throat, and pharyngeal exudates), minimal or disparate chest signs on physical examination, subacute onset, nonfocal lung opacity on chest radiographs, lack of clinical response to antibiotics, lack of substantial leukocytosis, and a slow disease course. Common infectious causes of atypical pneumonia are summarized in **Box 2**. On chest radiographs, the pulmonary opacity is seen as either airspace, reticular (linear), or bandlike opacities in a nonfocal, patchy, or mottled distribution, with various degrees of density, usually without a single dense area of consolidation (**Fig. 6**). Most patients with atypical pneumonia can be classified into one of the following subgroups or a combination of two of them based on findings on chest radiographs:[20]

Acute interstitial pneumonia
Chest radiographs show a patchy, nonfocal reticular pattern. Causes of acute interstitial pneumonia include self-limited viral infections and other pathogens.

Subacute minimal patchy pneumonia
Chest radiographs show 1 or more patches of minimal foci of airspace opacity. The most common causes of subacute minimal patchy pneumonia are *Mycoplasma pneumoniae*, *Chlamydia pneumoniae,* and adenoviruses.

Subacute dense focal pneumonia
Chest radiographs show a dense focal airspace opacity that is segmental or subsegmental. Most of the other features of acute focal pneumonia are absent. Tuberculosis needs to be excluded in these patients.

Most children exposed to *Mycobacterium tuberculosis* do not develop active disease but can have latent foci that may reactivate at a later date particularly if they become immunosuppressed or debilitated. Primary infection of *Mycobacterium tuberculosis* is more likely in infants with local spread from the initial parenchymal/lymph node complex to form larger single or multifocal parenchymal lesions, typically with prominent hilar and mediastinal lymph node involvement (**Fig. 7**) and occasional pleural or pericardial disease. The primary focus as well as involved nodes may cavitate with liquefaction of the caseous material and ultimately calcification (see **Fig. 7**). Enlarged lymph nodes may encroach on adjacent bronchi and cause bronchial narrowing with resultant air trapping or collapse in the distal lung (**Fig. 8**). Distant spread to other organs may occur either via
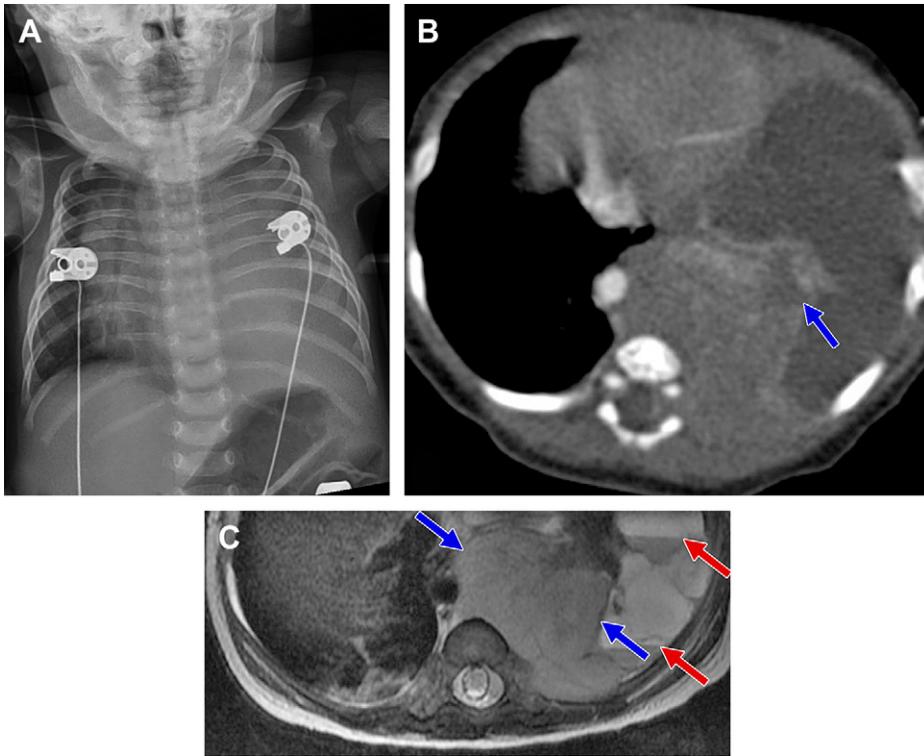
Fig. 5. Opacification of left hemithorax and respiratory distress caused by ruptured paraspinal neuroblastoma in a 5-week-old-boy. *(A)* Frontal chest radiograph shows complete opacification of the left hemithorax with contra-lateral mediastinal shift, which was initially believed to represent pneumonia with large pleural effusion. *(B)* Axial contrast-enhanced CT shows a left paraspinal mass *(blue arrow)* with intraspinal extension associated with a large pleural effusion, compressive atelectasis of the left lung, and contralateral mediastinal shift. *(C)* Axial T2-weighted MR image shows the left paraspinal mass *(blue arrows)* and a complex left pleural effusion with multiple loculations and fluid-debris levels *(red arrows)*.

lymphatics or hematogenously (including military lung involvement).[24,25]

Infections with more than 1 organism may cause the atypical pneumonia pattern, resulting in confusing persistence of the illness or prominent findings in another organ system. An example of this situation is influenza infection with superimposed typical or atypical pneumonia (see **Fig. 6**B). The more common mimics that simulate the appearance of atypical pneumonia syndromes are summarized in **Box 3**.

Miliary or Nodular Pneumonia

Miliary or nodular pneumonia is characterized by chest radiographic findings of multiple miliary or larger nodular opacities. Miliary pneumonia in pediatric patients is seen most commonly in tuberculous and fungal infections (**Fig. 9**). Nodular pneumonia (including reticular and reticulonodular patterns) in pediatric patients is seen in septic emboli, viral pneumonia, lymphocytic interstitial pneumonia associated with Epstein-Barr virus (EBV) infection with underlying human immunodeficiency virus (HIV) infection, and some fungal and bacterial infections (**Box 4**; **Figs. 10** and **11**).[25,26] Septic pulmonary emboli usually occur secondary to a focal *Staphylococcus aureus* infection (eg, right-sided bacterial endocarditis, septic thrombophlebitis, osteomyelitis, soft tissue

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Box 2
Common infectious causes of atypical pneumonia syndrome in children

Viruses: RSV (<5 years), adenoviruses, parainfluenza viruses, influenza virus (in epidemics), cytomegalovirus (CMV), varicella zoster virus (immunosuppressed)

Bacteria: *Chlamydia trachomatis* (<4 months), *Mycoplasma pneumoniae* (>5 years), *Chlamydia pneumoniae* (>5 years), *Bordatella pertussis*

Data from Fisher RG, Boyce TG. Pneumonia syndromes. In: Fisher RG, Boyce TG, editors. Moffet's pediatric infectious diseases: a problem-oriented approach. 4th edition. Philadelphia: Lippincott Williams & Wilkins; 2005. p. 174–221.
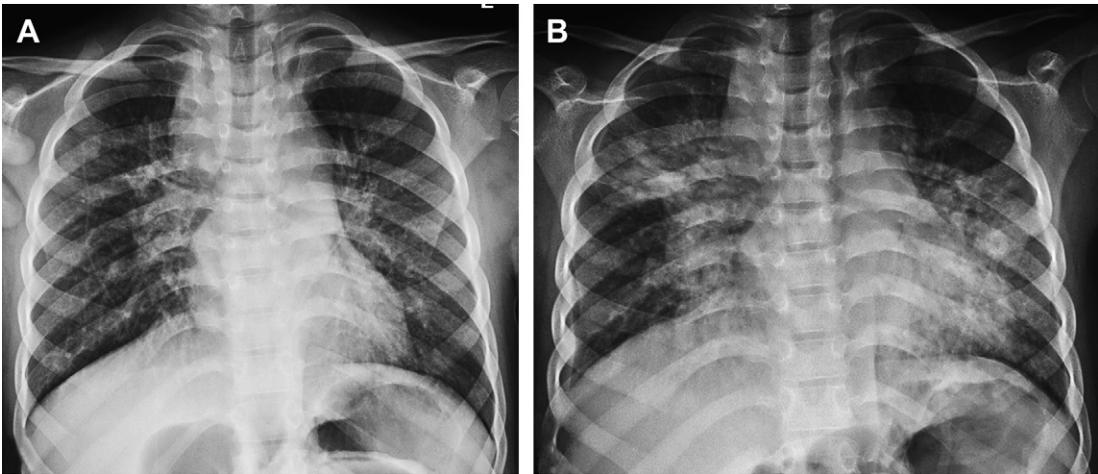
Fig. 6. Atypical pneumonia caused by influenza virus A subtype H1N1 with superimposed hospital-acquired pneumonia in an 8-year-old boy with history of renal transplant. *(A)* Frontal chest radiograph shows bilateral parahilar streaky opacities. *(B)* Patient developed respiratory distress while on Tamiflu. Frontal chest radiograph 4 days after initial chest radiograph (*A*) shows multifocal consolidations suggestive of superimposed hospital-acquired pneumonia.

infection, or urinary tract infection). The pulmonary nodules in septic emboli may cavitate (see **Fig. 11**).[27] Mimics of the pattern of miliary or nodular pneumonia are summarized in **Box 5**.

Progressive or Fulminant Pneumonia

Pneumonia is deemed progressive when it becomes radiologically and clinically worse despite antibiotic therapy that should be effective against the presumed cause. In this situation, the cause is often nonbacterial pathogens and mimics should also be carefully considered. Fulminant pneumonia is defined as a severe bilateral

Fig. 7. Chronic pneumonia with lymphadenopathy caused by disseminated *Mycobacterium tuberculosis* in a 2-year-old boy. Axial contrast-enhanced CT shows mediastinal (*blue arrows*) and hilar (*not shown*) lymphadenopathy with central low attenuation, peripheral rim enhancement, and calcification. A left upper lobe mass with cavitation and punctuate calcification is consistent with a cavitating tuberculoma (*red arrow*).

pneumonia with an unusually rapid progression clinically or radiologically, over 24 to 48 hours after initial presentation. A common cause of progressive or fulminant pneumonia is the influenza virus during an epidemic. Uncommon infectious causes of this pattern and mimics are summarized in **Boxes 6** and **7**, respectively (**Fig. 12**).[20]

Aspiration Pneumonia

Aspiration pneumonia refers to the pulmonary consequences of abnormal entry of fluid, particulate matter, or endogenous secretions into the lower airways. Aspirated material can be relatively inert, toxic, or oropharyngeal secretions. The most commonly aspirated materials in children include oropharyngeal secretions, gastric contents, water, hydrocarbon, lipid, and foreign bodies. Radiographic pulmonary opacities related to aspiration may have an upper rather than lower lobe distribution when the child aspirates in the supine position.

Bacterial aspiration pneumonia is an infectious process caused by the inhalation of oropharyngeal secretions that are colonized by pathogenic bacteria. The basic defect leading to bacterial aspiration pneumonia is failure of the normal oropharyngeal defense mechanisms. The patient typically has a depressed state of consciousness, abnormal swallowing, a neuromuscular defect that prevents adequate coughing, or an abnormal connection between the airway and esophagus (such as an H-type tracheoesophageal fistula).

Acute lung aspiration (Mendelson syndrome) is an acute chemical injury caused by inhalation of gastric contents. In neurologically normal children,
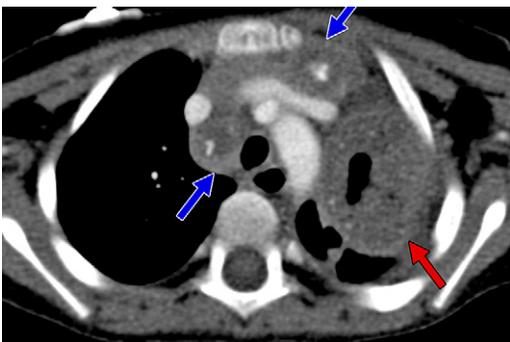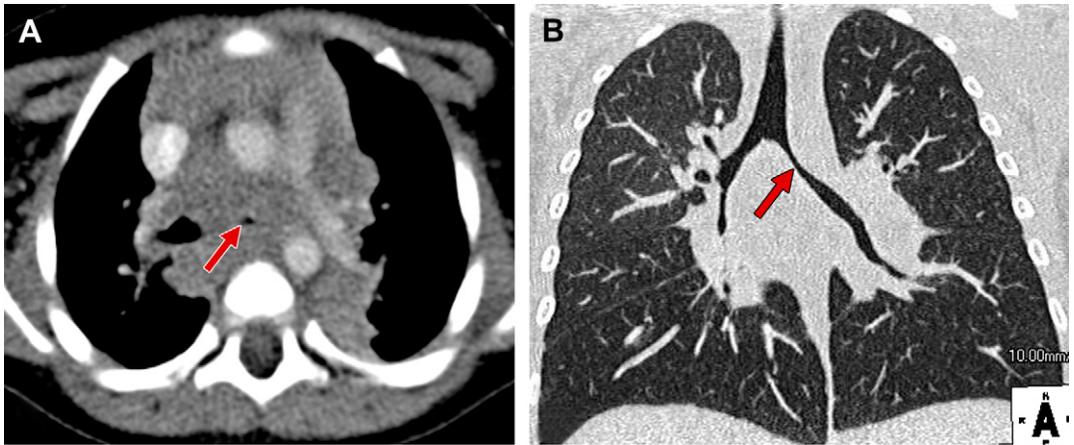
Fig. 8. *Mycobacterium tuberculosis* in an 11-month-old boy with 1.5-month history of intermittent stridor. *(A)* Axial contrast-enhanced CT shows low attenuation mediastinal and hilar lymphadenopathy with compression of the left mainstem bronchus *(arrow)*. *(B)* Curved oblique multiplanar reformation (MPR) shows the extent and degree of left mainstem bronchial narrowing *(arrow)*.

gastric aspiration usually occurs as a complication of anesthesia. The diagnosis of acute aspiration is mainly clinical and usually involves witnessed inhalation of vomitus or tracheal suctioning of gastric contents.[28,29]

Chronic lung aspiration (CLA) is repeated passage of food, gastric reflux, or saliva into the subglottic airways that causes chronic or recurrent respiratory symptoms. CLA may present with chronic cough, wheeze, noisy breathing, choking during feeding, recurrent episodes of pneumonia or bronchitis, and failure to thrive. Chronic aspiration often results in progressive lung disease, recurrent pneumonia, chronic airway inflammation, bronchiectasis, and respiratory failure. It is

a major cause of death in children with severe neurologic disorders (**Fig. 13**). Pulmonary aspiration may occur as a result of swallowing dysfunction, gastroesophageal reflux, and inability to adequately protect the airway from oral secretions or a combination of these. Anatomic conditions that predispose to aspiration lung disease include esophageal stricture or obstruction (eg, vascular ring, foreign body, achalasia), cleft palate, tracheoesophageal fistula (**Fig. 14**), laryngeal cleft, and bronchobiliary fistula.[28,29]

Aspiration related to near-drowning occurs when fluid enters the lungs without being prevented by laryngospasm. It typically manifests as pulmonary edema radiographically.[30] In a recent series of 83 children, secondary infections from aspiration related to near-drowning were rare.[31]

Box 3
Mimics of atypical pneumonia pattern

Congestive heart failure

Hypersensitivity pneumonitis

Drug hypersensitivity

Collagen vascular diseases (rheumatic fever, rheumatoid arthritis)

Pulmonary infarction or embolism (uncommon in children)

Airway and lung injury from toxic fume inhalation (silo filler diseases and other occupational inhalants)

Data from Fisher RG, Boyce TG. Pneumonia syndromes. In: Fisher RG, Boyce TG, editors. Moffet's pediatric infectious diseases: a problem-oriented approach. 4th edition. Philadelphia: Lippincott Williams & Wilkins; 2005. p. 174–221.

Fig. 9. Miliary or nodular pneumonia caused by recurrent *Mycobacterium avium-intracellulare* in an 8-year-old boy with immunodeficiency (interferon γ receptor 2 deficiency). Axial CT shows multiple, diffuse 2-mm to 3-mm nodules in bilateral lungs.

Hydrocarbon pneumonia is an acute, intense chemical pneumonitis after unintentional aspiration of volatile hydrocarbon compounds. Most cases of hydrocarbon pneumonia occur in children. Chest radiographs typically show bilateral, scattered pulmonary densities with middle and lower zone predominance. Such densities may become confluent and progress to acute respiratory distress syndrome (ARDS) and respiratory failure. They typically worsen over the first 72 hours and then clear over the next few days. However, occasionally radiographic changes may take weeks to months to be cleared. Obstructive emphysema, pneumatoceles, subsegmental, or segmental atelectasis may also be seen.[32]

Lipoid pneumonia is a rare form of pneumonia caused by inhalation or aspiration of a fatty substance. Oral administration of various oils is a common cultural practice, including mineral oil, olive oil, shark liver oil, cod liver oil, coconut oil, and ghee. Such oily materials can readily slide into the airway even in normal infants and young children without eliciting a cough reflex and are poorly removed by cilia. Lipoid pneumonias are typified by mild, subacute, or chronic clinical findings with accompanying marked radiographic changes. Chest radiographs of children with lipoid pneumonia typically show bilateral parahilar ill-defined, airspace opacities. In a series of 7 pediatric patients, CT showed dense consolidation surrounded by ground-glass opacity with a geographic lobular distribution.[33] Within the dense consolidations, areas with relatively low attenuation were identified in only 1 patient. Therefore, low-density consolidation in the posterior lungs is an infrequent CT finding in the diagnosis of lipoid pneumonia in children (**Fig. 15**). Interlobular septal thickening in areas of ground-glass opacity (ie, crazy paving pattern) has also been described in children with lipoid pneumonia.[33] Lipoid pneumonias may be complicated by superimposed infection especially with atypical mycobacteria. Slow recovery usually takes place with cessation of the oil administration. There may be residual scarring/fibrosis especially with animal rather than vegetable oils.[34,35]

Foreign body aspiration can also result in pneumonia. Accidental aspiration of both organic and nonorganic foreign bodies is a cause of childhood morbidity and mortality, requiring prompt recognition and early treatment to minimize the potentially serious and sometimes fatal consequence. Eating is the most common circumstance during which it occurs, with small food items being
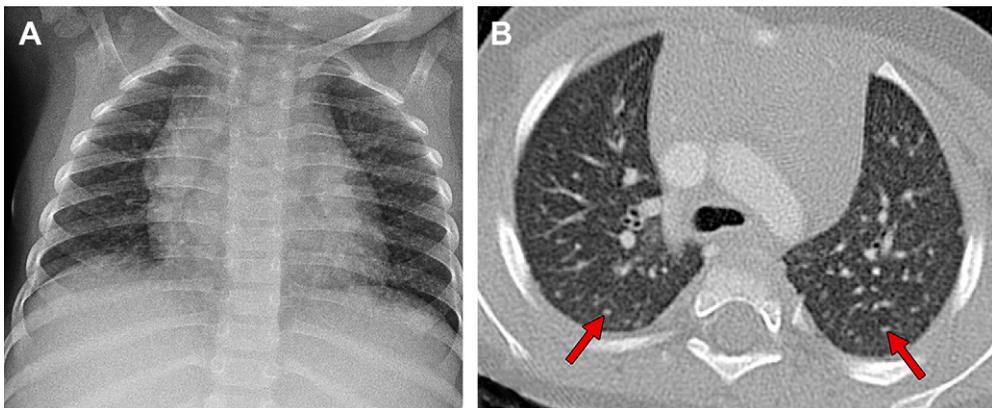

Fig. 10. Miliary or nodular pneumonia caused by lymphocytic interstitial pneumonia in a 19-month-old boy with HIV. *(A)* Frontal chest radiograph shows diffuse reticulonodular pattern. *(B)* Axial chest CT shows multiple bilateral 2-mm to 3-mm nodules *(arrows)*.
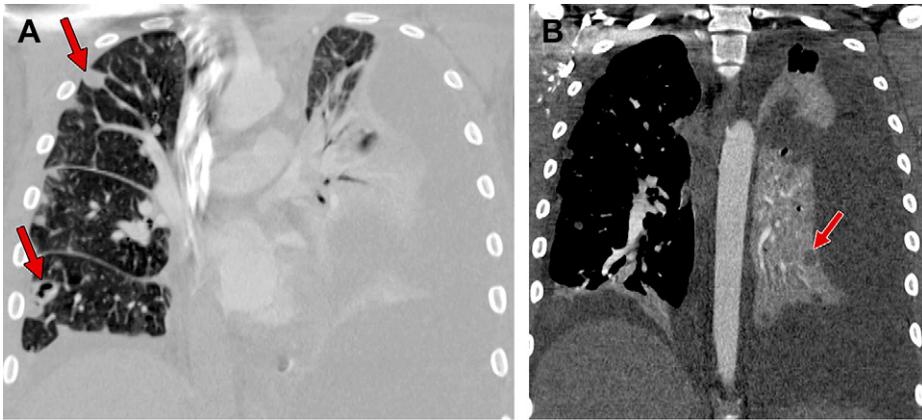
Fig. 11. Nodular pneumonia caused by septic emboli in an 18-year-old male with L3 vertebral body osteomyelitis and an epidural abscess. *(A, B)* Coronal contrast-enhanced CT reformats in lung and soft tissue windows show a large left pleural effusion and compressive atelectasis of the left lung. There are multiple, predominantly peripheral, solid and cavitating nodules in bilateral lungs *(arrows)* consistent with septic emboli. Left lung septic emboli are seen as hypoattenuating rounded lesions in the atelectatic lung; some are cavitating.

the most common foreign bodies aspirated. Coughing, choking, acute dyspnea, and sudden onset of wheezing are the most common symptoms. Clinical signs of foreign body aspiration have low positive predictive values. Chest radiographs are the initial imaging modality for patients with clinically suspected tracheobronchial aspiration of a foreign body. Chest radiographs may show air trapping, atelectasis, a radiopaque foreign body (rare), or be normal (**Fig. 16**).[36] When the routine inspiratory chest radiograph is unhelpful or confusing, inspiratory and expiratory radiographs (in a cooperative child) or bilateral decubitus views (in a younger child unable to follow breathing instruction) are useful in confirming focal or unilateral air trapping. In selected cases, CT (possibly integrated with virtual bronchoscopy) may be considered to exclude a foreign body. CT evaluation may avoid bronchoscopy or provide the exact location and postobstructive complications of the foreign body before bronchoscopy.[37] An underlying chronic unrecognized airway foreign body should be considered among other causes of recurrent or chronic pneumonia, particularly in the pediatric population (see **Fig. 16**).

Box 5

Mimics of acute miliary and nodular pattern (including reticular and reticulonodular patterns)

Miliary: pulmonary edema; airway and lung injury from toxic fume inhalation (constrictive bronchiolitis)

Nodular: Wegener granulomatosis, recurrent aspirations

Bilateral reticular or reticulonodular pattern: Langerhans cell histiocytosis, hypersensitivity pneumonitis, lipoid pneumonia (bilateral airspace opacities in most cases)

Pulmonary alveolar proteinosis produces variable patterns including airspace, nodular, miliary, and scattered linear densities

Data from Fisher RG, Boyce TG. Pneumonia syndromes. In: Fisher RG, Boyce TG, editors. Moffet's pediatric infectious diseases: a problem-oriented approach. 4th edition. Philadelphia: Lippincott Williams & Wilkins; 2005. p. 174–221.

Box 6

Uncommon infectious causes of progressive or fulminant pneumonia in children

Viruses: measles virus, varicella zoster virus, adenovirus, hantavirus, Nipah virus (exposure to pigs in Malaysia and Singapore), Hendra virus (exposure to horses in Australia), severe acute respiratory syndrome coronavirus

Bacteria: *Bordetella pertussis*, *Mycoplasma pneumonia*, *Chlamydia psittaci*, *Listeria monocytogenes* (in newborn), *Legionella pneumophila*, *Coxiella burnetti*, group A streptococcus, group C streptococcus, Rocky Mountain spotted fever, ehrlichosis

Data from Fisher RG, Boyce TG. Pneumonia syndromes. In: Fisher RG, Boyce TG, editors. Moffet's pediatric infectious diseases: a problem-oriented approach. 4th edition. Philadelphia: Lippincott Williams & Wilkins; 2005. p. 174–221.

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Box 7
Mimics of progressive or fulminant pneumonia pattern

Pulmonary vasculitis

Neoplasms and lymphoproliferative disorders (particularly Hodgkin or non-Hodgkin lymphoma)

Adult respiratory distress syndrome

Hemorrhage or infarction from a pulmonary embolus

Data from Fisher RG, Boyce TG. Pneumonia syndromes. In: Fisher RG, Boyce TG, editors. Moffet's pediatric infectious diseases: a problem-oriented approach. 4th edition. Philadelphia: Lippincott Williams & Wilkins; 2005. p. 174–221.

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Pulmonary Infiltrates with Eosinophilic Pneumonia

PIE syndrome comprises a group of heterogeneous disorders having the common findings of lung disease and eosinophilia in the peripheral blood, bronchoalveolar lavage fluid, or pulmonary interstitium. PIE syndrome is rare in children. A subclassification for the PIE syndromes in children is summarized in **Box 8**.[38,39] Infectious causes of PIE syndrome are uncommon and include *Chlamydia trachomatis* (especially in infants less than 3 months of age), allergic bronchopulmonary aspergillosis (in asthmatics and cystic fibrosis), parasitic larvae in lungs (*Toxocara*, *Ascaris*, and others), and fungi (eg, *Cryptococcus*, *Candida* species).[20]

The radiographic findings in PIE syndromes tend to be nonspecific. Chest radiographs may show interstitial, alveolar, or mixed (interstitial and alveolar) infiltrates, which tend to be bilateral and diffuse. Certain PIE syndromes may be associated with more specific findings. The classic radiographic appearance of chronic eosinophilic pneumonia is characterized by peripheral infiltrates with sparing of the central lung zones. This radiographic appearance has been described as the "photographic negative of pulmonary edema."[40] Bronchiectasis with mucoid impaction is generally present on chest radiographs or CT in patients with allergic bronchopulmonary aspergillosis (**Fig. 17**).[41] Acute eosinophilic pneumonia is frequently associated with small bilateral pleural effusions. Imaging is often helpful in determining the extent of disease, localizing the potential sites for lung biopsy, and in assessing response to therapy once treatment has begun.[38]

Chronic or Recurrent Pneumonia

Chronic pneumonia is defined as a pulmonary opacity that does not improve within 1 month. It is best classified from the anatomic pattern, as focal, interstitial, with hilar lymphadenopathy, or with cysts, cavities, or spherical masses. Spherical masses, with or without cavitations, are often features of an infectious cause. Infectious causes and mimics of this pattern are summarized in **Boxes 9** and **10** (see **Fig. 16**; **Figs. 18–20**).[42] Obstructive atelectasis may both mimic and predispose to chronic pneumonia. It may have many underlying causes, including foreign body, mucoid impaction, narrowed bronchus, and extrinsic bronchial compression by cardiovascular anomalies, lymphadenopathy, tumor, or postpneumonic inflammatory changes. Anomalies of the lung, mediastinum, and diaphragm that may mimic an acute or chronic pneumonia pattern include atypical thymus, diaphragmatic eventration and hernia, tracheal bronchus, lung hypoplasia, and congenital bronchopulmonary malformations (BPMs).[20] Several of these lesions, such as the BPMs, predispose to recurrent or chronic infection but differentiating an infected from uninfected lesion may be difficult or impossible on imaging. Sometimes having previous imaging for comparison is helpful in terms of features such as the new presence of fluid in a previously air-filled cavity or perilesional consolidation.

Recurrent pneumonia is defined as more than 1 episode within a 1-year period or more than 3 episodes in a lifetime. Many children with a chronic pulmonary lesion (especially a congenital anomaly) are believed to have recurrent pneumonias if chest radiographs are taken only during a febrile illness. Recurrent pneumonias may be either focal or interstitial (linear). Underlying abnormalities that may

Fig. 12. Acute alveolar hemorrhage secondary to idiopathic pulmonary vasculitis in a 4-year-old child mimicking progressive or fulminant pneumonia. Axial contrast-enhanced CT shows multifocal consolidation, with sparing of the peripheral bilateral lungs.
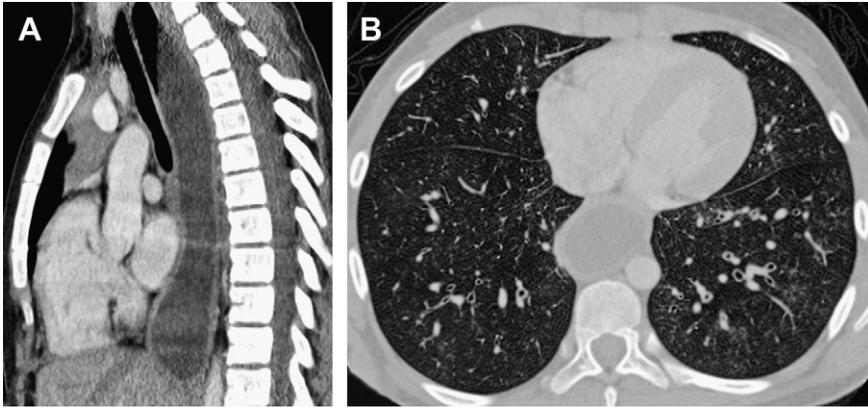
Fig. 13. Chronic lung aspiration secondary to lower esophageal stricture in an 11-year-old girl with history of gastroesophageal surgery at the age of 3 years in Mexico. *(A)* Sagittal contrast-enhanced CT reformation shows marked dilatation of the fluid-filled esophagus. *(B)* Axial contrast-enhanced CT shows bilateral diffuse centrilobular lung nodules and peripheral tree-in-bud pattern.

predispose to recurrent focal pneumonia include chronic aspiration (see section on aspiration pneumonia), congenital heart disease, bronchopulmonary foregut malformations (including BPMs with enteric-respiratory tract fistula), airway abnormalities (foreign body, stenosis, bronchiectasis, cystic fibrosis, immotile cilia disease), paralysis or eventration of the diaphragm, and congenital, acquired, and iatrogenic immune deficiencies.[43,44] Recurrent

Fig. 14. Recurrent aspiration caused by tracheoesophageal fistula without esophageal atresia in a 6-day-old girl who presented with recurrent episodes of apnea, cyanosis, and choking desaturations. Barium esophagram shows an oblique connection (*red arrow*) coursing anterosuperiorly from the esophagus to the trachea at the level of the thoracic inlet. Contrast has opacified the central tracheobronchial airways. Inferior to the fistula, there is a focal mild narrowing of the esophagus (*blue arrow*), raising concern for a congenital esophageal stricture.

interstitial pneumonia may be secondary to asthma, hypersensitivity pneumonitis, or pneumonias in children with AIDS (including *Pneumocystis jiroveci* pneumonia, lymphoid interstitial pneumonitis, and recurrent *Streptococcus pneumoniae* infection) (see **Fig. 10**).[20]

NEONATAL PNEUMONIA

Neonatal lung infections can be generally classified into 3 types depending on the initial source of neonatal infection: transplacental, perinatal, and postnatal (including nosocomial) infections.

Transplacental infections enter the fetus hematogenously via the umbilical cord. Most infants affected with transplacental infections typically

Fig. 15. Lipoid pneumonia secondary to oral administration of olive oil in a 3-month-old infant who presented with mild chronic cough and marked persistent opacities on chest radiographs. Axial contrast-enhanced CT shows bilateral airspace opacities in the posterior lungs with low density (−60 Hounsfield units).

Fig. 16. Chronic pneumonia caused by foreign body in left mainstem bronchus in a 13-year-old boy. *(A, B)* Axial contrast-enhanced CT shows 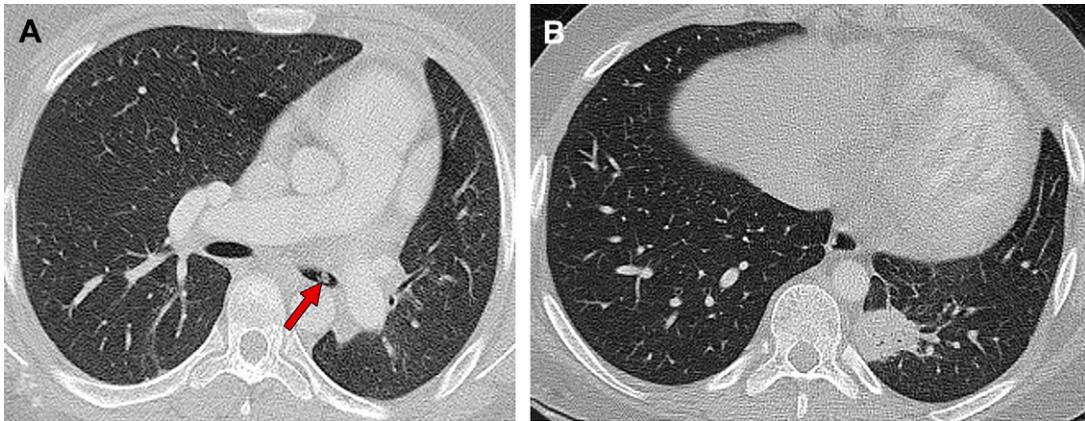the foreign body (a piece of plastic) in the left mainstem bronchus (*arrow*), mediastinal shift to the left, and focal consolidation in the left lower lobe.

manifest systemic and multiorgan disease rather than a primary lung infection. The most common transplacental infection is caused by CMV, which manifests as a diffuse reticulonodular pattern.[45] Other less common, transplacentally acquired pneumonias include rubella, syphilis, *Listeria monocytogenes*, and tuberculosis.

Perinatal infections can be acquired via ascending infection from the vaginal tract (most commonly group B *Streptococcus* or *Escherichia coli*), transvaginally during the birth process, or nosocomially in the neonatal period.[46] Radiographic findings in neonatal pneumonia are nonspecific in differentiating between various etiologic pathogens, as well as differentiating pneumonia from other causes of respiratory distress (eg, transient tachypnea of the newborn, surfactant deficiency disease, and meconium aspiration). The most common radiographic manifestation of neonatal pneumonia is bilateral coarse perihilar reticular densities with possible scattered airspace opacities (**Fig. 21**). Solitary lobar consolidations are uncommon.[47] There is an association between group B streptococcal pneumonia and an ipsilateral diaphragmatic hernia.[48] Chest radiographs in group B streptococcal sepsis can mimic the diffuse ground-glass opacity seen in surfactant deficiency disease. However, the presence of this finding in a full-term infant or the presence of cardiomegaly or pleural effusions may help to differentiate group B streptococcal infection from surfactant deficiency disease.[49]

Pneumonia caused by *Chlamydia trachomatis* occurs in about 10% of infants born to women who carry this organism in their genital tract and becomes symptomatic more than 2 to 3 weeks after birth. Chlamydia pneumonia is characterized

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Box 8
PIE syndromes: subclassification

Simple pulmonary eosinophilia (Loeffler syndrome)

Chronic eosinophilic pneumonia (rare in children)

Acute eosinophilic pneumonia

Allergic granulomatosis (Churg-Strauss syndrome)

Allergic bronchopulmonary aspergillosis (in asthmatics, cystic fibrosis)

Parasite-induced eosinophilia (*Strongyloides*, *Ascaris*, *Toxocara*, and *Ancylostoma*)

Drug reaction

Idiopathic hypereosinophilic syndrome (rare in children)

Infant pulmonary eosinophilia
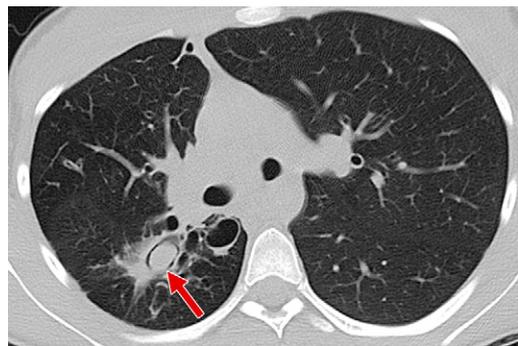
Data from Refs.[20,38,39]

Fig. 17. Pulmonary infiltrates with eosinophilia caused by allergic bronchopulmonary aspergillosis in a 12-year-old girl with cystic fibrosis. Axial contrast-enhanced CT shows central bronchiectasis with intraluminal mucoid impaction/mycetomas (*arrow*).

Box 9
Infectious causes of chronic pneumonia syndrome

1. *Chronic focal pulmonary disease*

    a. Untreated or undertreated acute pneumonia

        i. *Bacteria and Mycobacteria*: *Mycobacterium tuberculosis*, nontuberculous mycobacteria (especially in children with defective cell-mediated immunity)

        ii. *Systemic fungi*: histoplasmosis, coccidioidomycosis, blastomycosis cryptococcosis, sporotrichosis

        iii. *Parasites*: Paragonimiasis

2. *Chronic interstitial pulmonary disease*

    a. *Bacteria: Chlamydia trachomatis*

    b. *Viruses:* CMV, late-onset congenital rubella syndrome, LIP (in HIV infection)

    c. *Fungi: Pneumocystis jiroveci*

3. *Chronic pneumonia with hilar lymphadenopathy*

    a. Common

        i. *Bacteria and Mycobacteria: Mycobacterium tuberculosis, Mycoplasma pneumonia, Chlamydia pneumonia; Fungi:* histoplasmosis

    b. Uncommon

        i. *Bacteria:* nontuberculous mycobacteria, *Actinomyces*, *Francisella tularensis*, *Bacillus anthracis*

        ii. *Fungi:* coccidioidomycosis, blastomycosis

        iii. *Lung abscess*

4. *Chronic cavitary, cystic, or nodular pneumonias*

    a. Lung abscess, necrotizing bacterial pneumonia

        i. *Bacteria and Mycobacteria*: tuberculosis, actinomycosis, nocardiosis; *Nocardia* and *Rhodococcus equi* in immunocompromised patients

        ii. *Fungi*: *Aspergillus*, *Pneumocystis jiroveci* and other systemic fungi in immunocompromised patients

        iii. *Viruses*: atypical measles, papillomatosis of lung

        iv. *Protozoa and helminths*: paragonimiasis, *Entamoeba histolytica*, *Ecchinococcus*, *Dirofilaria immitis*

Data from Fisher RG, Boyce TG. Pneumonia syndromes. In: Fisher RG, Boyce TG, editors. Moffet's pediatric infectious diseases: a problem-oriented approach. 4th edition. Philadelphia: Lippincott Williams & Wilkins; 2005. p. 174–221; and Kawanami T, Bowen A. Juvenile laryngeal papillomatosis with pulmonary parenchymal spread. Case report and review of the literature. Pediatr Radiol 1985;15(2):102–4.

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by hyperinflation and bilateral diffuse reticular perihilar densities that are disparate with relatively mild clinical symptoms.[50] Concomitant conjunctivitis, which used to be a useful clue to the cause, is prevented by the routine instillation of antibacterial eye drops at birth. Neonates with chlamydia pneumonia frequently have accompanying eosinophilia.

Bordetella pertussis has recently resurfaced to produce epidemics of infection, probably related to waning community immunity. The clinical presentation of pertussis in the newborn may lack some features (characteristic whooping cough and fever) typical of the disease in older children. The clinical presentation of the most severely affected newborns may be dominated by marked respiratory distress, cyanosis, and apnea. Mortality caused by pertussis usually results from secondary pneumonia, encephalopathy, cardiac failure, or pulmonary hypertension. A suggested mechanism for pulmonary hypertension that may develop in newborns with *Bordetella pertussis* infection is formation of leukocyte thrombi in pulmonary venules secondary to hyperleukocytosis.[51,52] The classic radiographic appearance in pertussis is the shaggy heart with diffuse peribronchial cuffing related to airway inflammation. However, chest radiographic findings such as hyperaeration, atelectasis, segmental

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Box 10
Mimics of chronic pneumonia pattern

1. *Chronic focal pulmonary disease pattern*

   a. Malignancy (neuroblastoma)

   b. Obstructive atelectasis

   c. Foreign body, mucous plug, or endobronchial tumor

   d. Congenital anomalies of lung, thymus, or mediastinum

   e. Vascular rings

   f. Eventration of diaphragm

   g. Inflammatory pseudotumor of lung

2. *Chronic interstitial pulmonary disease pattern*

   a. Bronchopulmonary dysplasia

   b. Congestive heart failure

   c. Pulmonary sarcoidosis

   d. Collagen vascular diseases

   e. Idiopathic interstitial pneumonia

   f. Pulmonary hemosiderosis

   g. Constrictive bronchiolitis

   h. Histiocytosis (including Langerhans cell histiocytosis)

   i. Pulmonary alveolar proteinosis

3. *Chronic pneumonia pattern with hilar lymphadenopathy*

   a. Benign lymphoproliferative disorders and lymphoma

   b. Other neoplasms

4. *Chronic cavitary, cystic, or nodular pneumonia pattern*

   a. Congenital anomalies

   b. Malignancy (lymphoma and metastases)

   c. Traumatic pneumatocele

   d. Hyper-IgE syndrome

   e. α_1-antitrypsin deficiency

   f. Langerhans cell histiocytosis

   g. Pulmonary sarcoidosis

   h. Bronchopulmonary dysplasia

   i. Cystic bronchiectasis

   j. Wegener granulomatosis

Data from Fisher RG, Boyce TG. Pneumonia syndromes. In: Fisher RG, Boyce TG, editors. Moffet's pediatric infectious diseases: a problem-oriented approach. 4th edition. Philadelphia: Lippincott Williams & Wilkins; 2005. p. 174–221.

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consolidation, and lymphadenopathy are usually nonspecific.

Recurrent pulmonary infection, with bacterial, viral, or occasionally fungal pathogens, are frequent problems in neonates undergoing prolonged hospitalization and complex treatments, especially in premature infants with chronic lung disease. Radiographic alterations caused by infection may be subtle when superimposed on chronic lung changes.[47]

PNEUMONIA IN IMMUNOCOMPROMISED HOSTS

Pneumonia is a common disease in the immunocompromised host. Immunocompromise may be congenital (congenital immunodeficiencies), acquired (HIV/AIDS, malnutrition) or iatrogenic (during chemotherapy for cancer or after tissue transplantation). Immunodeficient states can result in: (1) humoral immunodeficiency (hypogammaglobulinemia, functional B-lymphocyte deficiency accompanying HIV infection); (2) cellular immunodeficiency (severe malnutrition, late stages of AIDS, some congenital immunodeficiencies such as DiGeorge syndrome); and (3) neutrophil dysfunction and neutropenia (chronic granulomatous disease, pure neutropenia). Iatrogenic immunodeficiencies may be a combination of neutropenia or neutrophil dysfunction, innate or drug-induced defective lymphocyte function, and drug-induced breaks in the oral and intestinal mucosal barriers.[53]

The causes of pneumonia in the immunocompromised host consist not only of the same agents that cause pneumonia in the normal host but also of several opportunistic agents depending on the type and severity of immunodeficiency as well as temporal pattern after chemotherapy or transplant.

In an immunocompromised child with a noncontributory chest radiograph and clinical findings that could be attributed to a lung infection, chest CT is often required for evaluation of a possible lung infection. In this situation, there are 4 major advantages of chest CT over chest radiographs. First, the presence, pattern, and extent of the disease process are better visualized. Second, more than 1 pattern of abnormality may be detected, suggesting dual pathologic entities. Third, invasive diagnostic procedures (eg, bronchoscopy or needle aspiration) can be more precisely planned. Fourth, CT also allows for increased sensitivity in assessment of the response to treatment.[54,55]

Although the radiographic or CT appearance might not be specific for a pathogen, knowledge of the clinical setting in combination with the
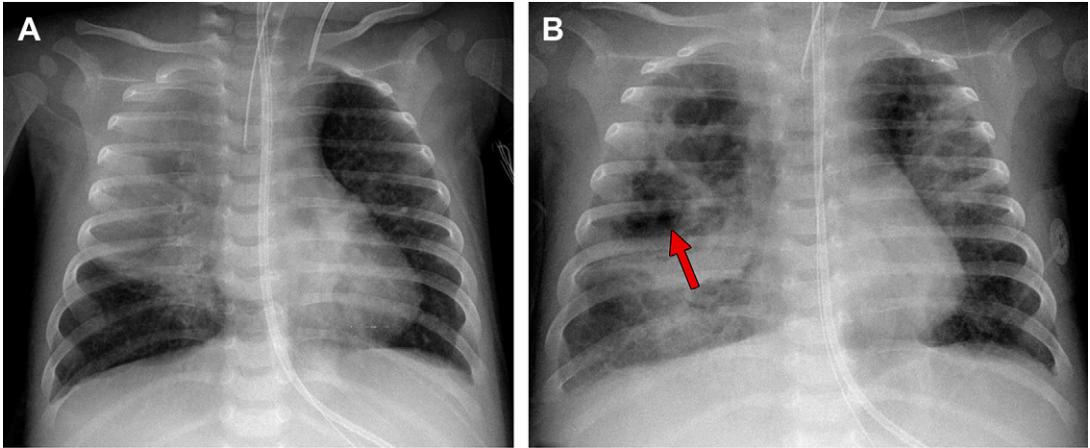
Fig. 18. Chronic focal pneumonia caused by persistent pulmonary coccidioidomycosis in a 3-month-old girl who presented with a 5-week history of pneumonia unresponsive to antibacterial agents. *(A)* Frontal chest radiograph shows consolidation of the right upper and middle lobes in addition to diffuse bilateral reticulonodular opacities. *(B)* Frontal chest radiograph obtained 8 days after initial chest radiograph *(A)*, shows development of multiple cavities *(arrow)* bilaterally superimposed on reticulonodular opacities.

type and severity of immunodeficiency and imaging pattern may narrow the differential diagnosis. A commonly encountered clinical issue is the possibility of fungal infection in immunocompromised children. The hallmark CT finding of fungal infections is the presence of pulmonary nodules. Such pulmonary nodules are often clustered peripherally and can show poorly defined margins, cavitation, or a surrounding halo of ground-glass opacity (CT halo sign) (**Fig. 22**). The CT halo sign is a nonspecific finding and represents either hemorrhage around a nodule or neoplastic or inflammatory infiltration of the lung parenchyma. In the immunocompromised host, the CT halo sign can be seen most commonly with fungal infections (eg, aspergillosis, mucormycosis, or *Candida*) but also in viral infections (eg, CMV infection, herpes infection), organizing pneumonia, and pulmonary hemorrhage.[54,56–61] Microorganisms associated with severe pneumonia in immunodeficiency states are summarized in **Box 11**.

LIP is a form of subacute pneumonia seen in several states of immunologic dysregulation, particularly in children with HIV infection. LIP is characterized by micronodules of proliferating lymphoid tissue associated with infection by EBV. Chest radiographs in LIP show a characteristic diffuse, bilateral nodular, or reticulonodular pattern

Fig. 19. Wegener granulomatosis mimicking chronic cavitary pneumonia in a 15-year-old male who presented with hemoptysis and respiratory distress. Initial chest radiograph showed bilateral confluent airspace opacities secondary to diffuse pulmonary hemorrhage *(not shown)*. contrast-enhanced CT obtained 9 days after initial presentation shows multifocal consolidation with cavitation *(arrow)*, ground-glass opacities, and a right pneumothorax.

Fig. 20. Hodgkin lymphoma in a 14-year-old boy mimicking chronic cavitary pneumonia. Axial contrast-enhanced CT shows multiple bilateral cavitating lung nodules *(arrows)*. Mediastinal and bilateral hilar lymphadenopathy was also present *(not shown)*.
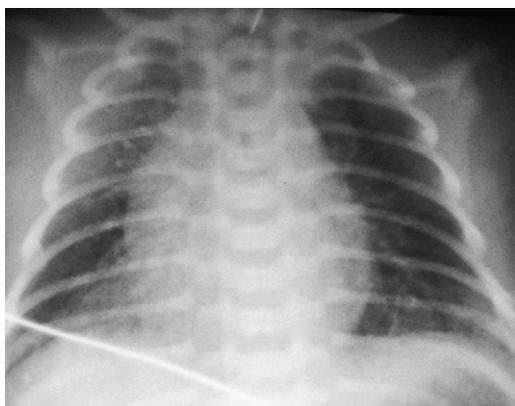
Fig. 21. Neonatal pneumonia caused by group B *Streptococcus* in a newborn infant. Frontal chest radiograph shows bilateral coarse perihilar reticular densities with scattered airspace opacities.

(see **Fig. 10**). Other recognized associated imaging findings of LIP include consolidation, mediastinal adenopathy, and bronchiectasis.[26,58,62] Radiologic features in common HIV-associated infections in pediatric patients are summarized in **Table 2**.

In pediatric patients with iatrogenic deficiencies of the immune system, pneumonia is commonly caused by opportunistic bacteria and fungi, acquired nosocomially or from resident mucosal flora (**Fig. 23**). In addition, after solid-organ transplantation although total immunoglobulin levels are normal, children and adolescents may be susceptible to encapsulated bacteria (eg, *Streptococcus pneumoniae*, *Haemophilus influenzae*). Viruses that commonly cause pneumonia in healthy hosts (eg, RSV, influenza, parainfluenza viruses, human
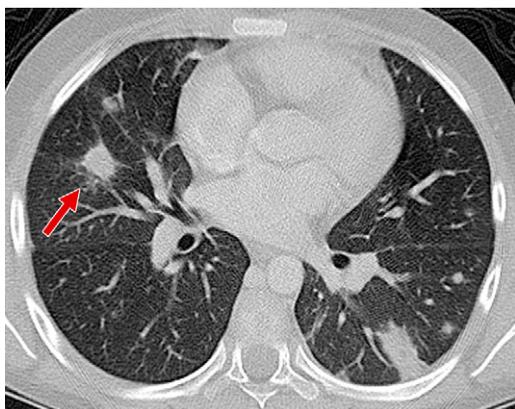

Fig. 22. Pneumonia in a febrile neutropenic patient caused by angioinvasive aspergillosis in a 7-year-old boy. Axial contrast-enhanced CT shows multiple bilateral pulmonary nodules, some have rims of ground-glass opacity (*arrow*), which is also known as the CT halo sign.

Box 11
Microorganisms associated with severe pneumonia in immunodeficiency states in pediatric patients

Humoral immunodeficiency

Viruses: enteroviruses

Pyogenic bacteria: *Streptococcus pneumonia*, *Haemophilus influenza*

Cellular immunodeficiency

Viruses: varicella zoster virus, rubeola virus, herpes simplex virus, CMV, adenovirus, RSV, parainfluenza virus, human metapneumovirus

Pyogenic bacteria: *Listeria monocytogenes*, *Pseudomonas aeruginosa*, *Stenotrophomonas* spp, *Burkholderia* spp, *Legionella* spp, *Nocardia* spp, other opportunistic bacteria

Mycobacteria: *Mycobacterium avium- intracellulare*, *Mycobacterium fortuitum*, Calmette-Guérin bacillus, other opportunistic mycobacteria

Fungi: *Pneumocystis jirovecii*, *Candida albicans*, *Cryptococcus neoformans*

Neutrophil dysfunction and neutropenia

Pyogenic bacteria: *Staphylococcus aureus, Burkholderia cepacia, Serratia marcescens, Nocardia* spp, *Pseudomonas aeruginosa*, *Stenotrophomonas* spp, *Burkholderia* spp, other opportunistic bacteria

Mycobacteria: Calmette-Guérin bacillus, nontuberculous mycobacteria

Fungi: *Aspergillus* spp, *Candida* spp, *Pseudallescheria boydii*, agents of mucormycosis

Iatrogenic immunodeficiency

Pyogenic bacteria: *Pseudomonas aeruginosa* and *Stenotrophomonas maltophilia,* α-hemolytic streptococci and other oral bacteria, *Nocardia, Streptococcus pneumoniae, Haemophilus influenza*

Mycobacteria: *Mycobacterium tuberculosis*

Viruses: CMV, varicella zoster virus, herpes simplex virus, human herpesvirus 6, RSV, influenza, parainfluenza viruses, human metapneumovirus, and adenovirus

Fungi: *Candida, Aspergillus* spp, and other fungi

Data from McIntosh K, Harper M, Murray M. Pneumonia in the immunocompromised host. In: Long SS, Pickering LK, Prober CG, editors. Principles and practice of pediatric infectious diseases revised reprint. 3rd edition. Philadelphia: Saunders; 2009. p. 265–9.

Table 2
Radiologic features in common HIV-associated infections in pediatric patients

Organism/Disease	Alveolar	Interstitial	LN	Cavities/Cysts
Bacteria	++			+
Mycobacterium tuberculosis	++	+	++	+
Atypical mycobacteria	+	++	+	+
Pneumocystis jiroveci	+	++		+(cysts)
Cryptococcus neoformans	+	+	+	
Invasive aspergillosis	+	+		++
RSV	+			
CMV	+	++	+	+
Measles	+	++		
Varicella	+	++		
Herpes simplex		+		

Abbreviations: Alveolar, focal of diffuse alveolar pattern; interstitial, focal or diffuse interstitial pattern; LN, lymphadenopathy; + and ++, refer to relative frequency of radiographic finding with each organism/disease.

Data from George R, Andronikou S, Theron S, et al. Pulmonary infections in HIV-positive children. Pediatr Radiol 2009;39(6):545–54.

metapneumovirus, and adenovirus) display greater virulence in both children and adults after solid-organ or human stem cell transplantation, particularly when cellular immunity is profoundly suppressed. In the posttransplant setting, EBV can cause progressive pulmonary disease in the form of posttransplantation lymphoproliferative disease.[53] In 2 studies of HRCT in bone marrow transplant recipients, the most useful distinguishing feature was the presence of large nodules and visualization of the halo sign, suggestive of fungal infection.[63,64]

After solid-organ transplantation, nosocomially acquired bacteria predominate as a cause of pneumonia in the first month. Later, viruses, especially CMV and adenovirus, as well as *Listeria*, *Nocardia*, and *Aspergillus*, may be the cause. After more than 6 months after solid-organ transplantation, community-associated bacterial pneumonia becomes more common.[53]

A variety of noninfectious pulmonary processes can present with acute or subacute clinical findings mimicking pulmonary infection.[54] These findings include alveolar hemorrhage, pulmonary edema, drug reaction, idiopathic interstitial pneumonia, benign and malignant lymphoproliferative disorders, constrictive bronchiolitis, bronchiolitis obliterans with organizing pneumonia, and chronic graft-versus-host disease. The CT findings of many of these entities are nonspecific.[54,57–61]

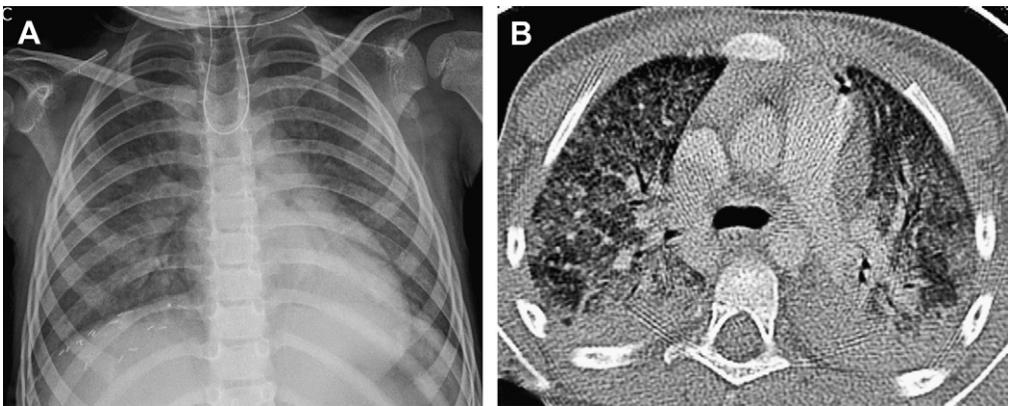**Fig. 23.** *Pneumocystis jiroveci* pneumonia in a 4-year-old girl 2 years after liver transplant with known posttransplant lymphoproliferative disorder. *(A)* Frontal chest radiograph shows diffuse bilateral ground-glass opacities and malpositioned nasogastric tube coiled in the esophagus. *(B)* Axial contrast-enhanced CT shows bilateral diffuse ground-glass opacities and dependent atelectasis.

ACUTE COMPLICATIONS OF PNEUMONIA

Acute complications of pneumonia can be categorized as suppurative lung parenchymal complications and pleural complications.

Suppurative Lung Parenchymal Complications

Suppurative lung parenchymal complications span a spectrum of abnormalities and include cavitary necrosis, lung abscess, pneumatocele, bronchopleural fistula (BPF), and pulmonary gangrene. The name given to the suppurative process depends on several factors including the severity and distribution of the process, condition of the adjacent lung parenchyma, and temporal relationship with disease resolution.[65,66]

Cavitary necrosis

Cavitary necrosis represents a dominant area of lung necrosis associated with a variable number of thin-walled cysts. Characteristic findings on CT for cavitary necrosis include loss of normal lung architecture, poor parenchymal enhancement, loss of the lung-pleural margin, and multiple thin-walled fluid-filled or air-filled cavities (Fig. 24). Cavitary necrosis is seen earlier in chest US or CT compared with chest radiography because these cavities need to be filled with air to be visible on chest radiographs. Such cavities filled with air are accomplished only after communication with bronchial airways. Most pediatric patients can be managed successfully with conservative treatment. Follow-up chest radiographs typically show complete or near-complete resolution of the cavitary necrosis (see Fig. 24).[65,66]

Lung abscess

Lung abscess is a severe complication of pneumonia in children, mostly occurring in the presence of predisposing factors, such as congenital or acquired lung abnormalities, or immunodeficiency. Lung abscess represents a dominant focus of suppuration surrounded by a well-formed fibrous wall. The predominant pathogens isolated from primary lung abscesses in children include streptococcal species, *Staphylococcus aureus* and *Klebsiella pneumoniae*. Children with a lung abscess have a significantly better prognosis than adults with the same
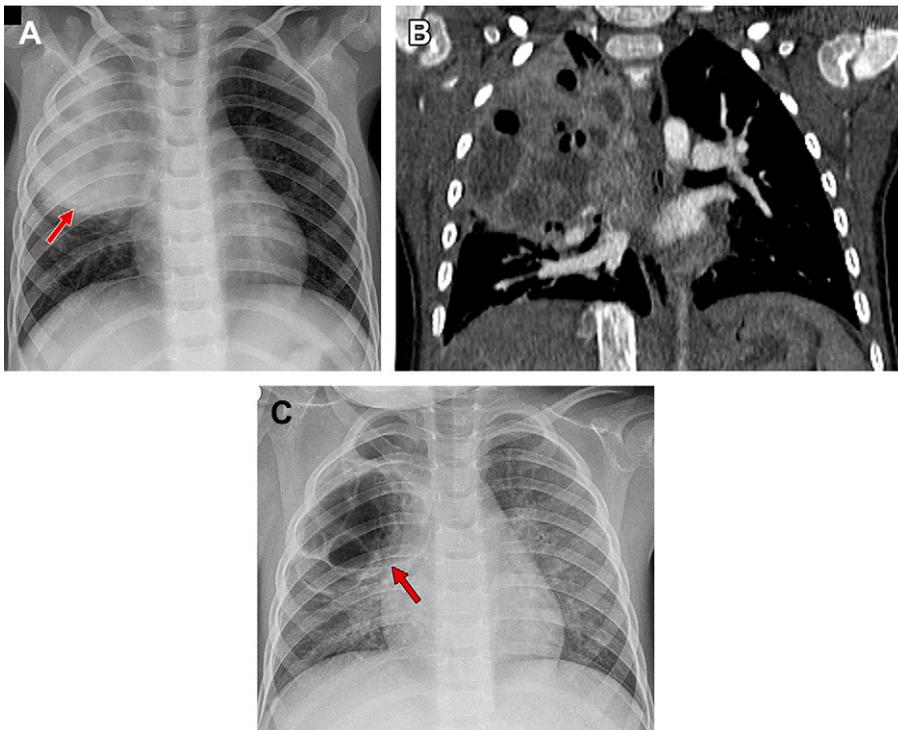

Fig. 24. Cavitary necrosis and pneumatoceles in a 2-year-old boy. *(A)* Frontal chest radiograph shows consolidation of the right upper lobe with an inferiorly bulging minor fissure *(arrow)* and multiple lucencies suggestive of underlying cavitation. *(B)* Coronal contrast-enhanced CT obtained 1 day after initial chest radiograph *(A)* shows consolidation of the right upper lobe and multiple thin-walled cavities with air-fluid levels. *(C)* Frontal chest radiograph obtained 1.5 months after initial chest radiograph *(A)* shows evolution of the cavitary necrosis into thin-walled air-filled cysts (ie, pneumatoceles; *arrow*). These cysts subsequently resolved on chest radiographs obtained at 3.5 months after presentation, with no significant lung scarring *(not shown)*.

condition.[67] Lung abscesses are uncommon in immunocompetent children. On contrast-enhanced CT, a lung abscess appears as a fluid-filled or air-filled cavity with a thick definable, enhancing wall (**Fig. 25**).[65,66,68] Although lung abscess in children has been managed successfully for many years with prolonged courses of intravenous antibiotics, the evolution of interventional radiology has seen the accelerated use of percutaneously placed pigtail drainage catheters using US and CT guidance.[67]

Pneumatocele

Pneumatocele is a term given to thin, smooth-walled air-filled cysts seen at imaging and may represent a later or less severe stage of resolving or healing lung necrosis (see **Fig. 24**). Pneumatoceles are most often caused by severe lung infection from staphylococcal pneumonia. However, they may be seen with other bacterial infections including streptococcus pneumonia and after hydrocarbon aspiration. On CT, thin-walled small or large cysts containing air with or without fluid are identified. The wall of a pneumatocele does not enhance. The surrounding lung may be opacified but does not typically show findings of lung necrosis.[65,66] Pneumatoceles usually resolve spontaneously over time although pneumatoceles may be atypically persistent in children with hyper-IGE syndrome. Large pneumatoceles containing fluid can be a source of ongoing infection and may occasionally require drainage.

Bronchopleural fistula

BPF is defined as a communication between the lung parenchyma or airways and the pleural space. Central BPFs (ie, main or lobar bronchi communicating with the pleural cavity) most often develop after traumatic injury to large airways or leak from the bronchial stump after pneumonectomy or lobectomy. The main causes for peripheral BPFs (ie, segmental or more distal airways or lung parenchyma communicating with the pleural cavity) are necrotizing pulmonary infection (ie, cavitary necrosis), trauma, lung surgery, and malignancy.[69,70] Presence of air in the pleural space before aspiration or drainage attempts is suggestive of either a peripheral BPF or infection with a gas-producing organism. Multidetector CT with thin-section axial and multiplanar reformation images may show a fistulous tract between the pleural space and peripheral airway or lung parenchyma in peripherally located BPFs (**Fig. 26**).[70]

Pulmonary gangrene

Pulmonary gangrene is a rare complication of severe lung infection with devitalization of lung parenchyma and secondary infection.[71] The primary feature that distinguishes pulmonary gangrene from necrotizing pneumonia and lung abscess is the extent of necrosis and the fact that thrombosis of large vessels plays a prominent role in the pathogenesis. Chest imaging shows lobar consolidation with bulging fissures and is followed by tissue breakdown to form many small cavities, which subsequently coalesce into a single large cavity occupying the entire lobe. Such a large cavity is filled with fluid and irregular pieces of sloughed lung parenchyma. However, these findings are not invariably present. Surgical resection of necrotic tissue is often necessary for proper management of children with pulmonary gangrene.[71–74]

The differential diagnosis of suppurative lung complications includes an underlying cystic
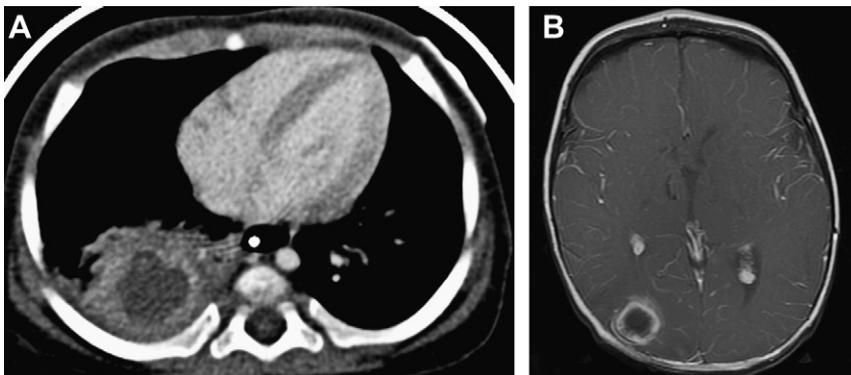
Fig. 25. Lung abscess in immunocompromised host caused by angioinvasive aspergillosis in a 6-month-old girl with acute myelogenous leukemia on induction chemotherapy. *(A)* Axial contrast-enhanced CT shows an air-filled cavity with a thick, definable enhancing wall in the right lower lobe and surrounding consolidation. *(B)* Axial contrast-enhanced T1-weighted MR image of the brain shows a rim-enhancing brain abscess in the right occipital lobe. The patient underwent right lower lobectomy and craniotomy for resection of these lesions.

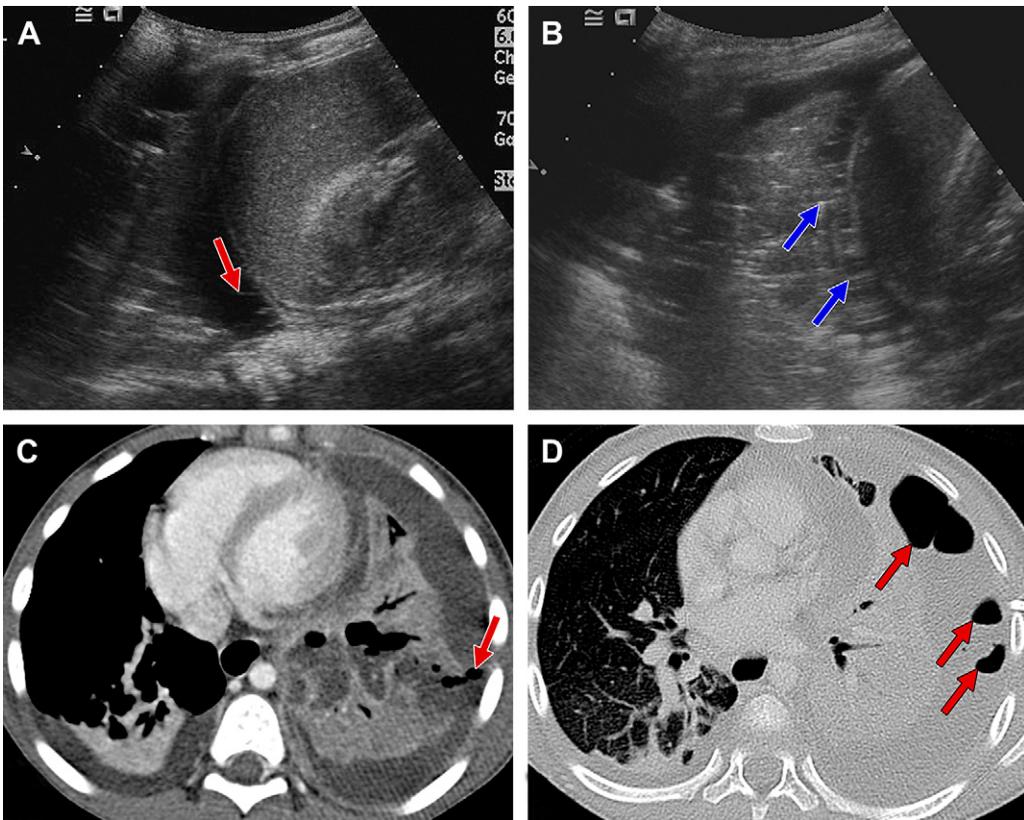**Fig. 26.** Cavitary necrosis, peripheral BPF, and empyema in a 7-year-old boy with acute complications of pneumonia. *(A, B)* US shows fibrinous strands in the left pleural effusion (*red arrow*) and multiple, small cavities in the left lower lobe (*blue arrows*). Left lung is consolidated with echogenic air bronchograms. *(C)* Axial contrast-enhanced CT soft tissue window, performed 2 days after US, shows consolidation of the left lung with multiple thin-walled cavities with air-fluid levels. There is an abnormal communication between the peripheral airways and the pleural space (*arrow*) consistent with a BPF. *(D)* Axial contrast-enhanced CT image located at a higher level to (*C*) on bone window image reconstruction shows a multiloculated left pleural fluid collection with multiple air-fluid levels (*arrows*). The drained fluid was mucopurulent consistent with an empyema.

congenital BPM that has become secondarily infected. Prior and follow-up imaging may aid in the distinction. The presence of large, well-defined cysts early in the course of the illness or a systemic arterial supply to the lung may be helpful in suggesting an underlying BPM, although a chronic inflammatory process in the lower lobe can acquire some systemic vascular supply from diaphragmatic vessels.

Pleural Complications of Acute Pneumonia

Pleural complications from acute pneumonia include parapneumonic effusion and empyema. Parapneumonic effusion is defined as a pleural effusion in the setting of a known pneumonia. It may be simple or complicated based on the absence or presence of the infecting organism within the pleural space, respectively.[75] Empyema is defined as thick purulent pleural effusion. It may

be free-flowing or loculated. Progression of a pleural effusion to empyema occurs through 3 stages: exudative, fibrinopurulent, and organization. Parapneumonic effusions complicate pneumonia in 36% to 56% of cases in pediatric patients.[76] Empyema complicates an estimated 0.6% of all childhood pneumonias.[77]

Chest radiographs can often detect a parapneumonic collection, although some fluid, especially in a subpulmonic location, may not be visible and is often seen better on a decubitus film. In cases with complete or almost complete opacification of a hemithorax with or without contralateral mediastinal shift, additional erect or decubitus views are unhelpful in defining the quantity or nature of the pleural fluid. US is most helpful in this situation because it can readily distinguish a parapneumonic collection from extensive consolidation or an underlying mass. The US determination of the echogenicity of the pleural collection (anechoic

or echogenic) and showing fibrin strands, septations, loculations, or fibrinous pleural rind is helpful in determining appropriate therapy (see **Fig. 26**). Treatment options for parapneumonic effusions/ empyemas include antibiotics alone, simple tube drainage, chest drain insertion with fibrinolytics, or surgery (eg, video-assisted thoracoscopic surgery or open thoracotomy with decortication). Although imaging techniques are used as a guideline, they do not always accurately stage empyema, predict outcome, or guide decisions regarding surgical versus medical management.[75]

CT provides a more global overview of pleural and pulmonary abnormality from acute pneumonia, but is poor at differentiating parapneumonic effusion from empyema in pediatric patients. Findings on CT, in patients with parapneumonic effusion/ empyema, include: (1) enhancement and thickening of visceral and parietal pleura; (2) thickening and increased density of extrapleural subcostal tissues; and (3) increased attenuation of extrapleural subcostal fat.[78] Loculation can be inferred by the presence of a lenticular fluid collection or nondependent air. Septations are usually not appreciated on CT (see **Fig. 26**).

Pleuropulmonary infection may occasionally spread to involve the chest wall, including soft tissues and adjacent bones. *Mycobacterium tuberculosis*, *Aspergillus*, and *Actinomyces* are the most common organisms in this scenario.

CHRONIC COMPLICATIONS OF PNEUMONIA

Chronic complications or consequences of pneumonia include parenchymal scarring, bronchial wall thickening, bronchiectasis, a predisposition to asthma, constrictive bronchiolitis, fibrothorax and a trapped lung, fibrosing mediastinitis, constrictive pericarditis, and pleural thickening. For practical purposes, bronchiectasis, constrictive bronchiolitis, fibrothorax and trapped lung, and fibrosing mediastinitis are discussed in the following sections.

Bronchiectasis

Bronchiectasis is defined by the presence of permanent and abnormal dilation of the bronchi. This condition usually occurs in the context of chronic airway infection causing inflammation. Bronchiectasis is nearly always diagnosed using HRCT. The main diagnostic features of bronchiectasis on HRCT are: (1) internal diameter of a bronchus that is wider than its adjacent pulmonary artery; (2) failure of the bronchus to taper peripherally; and (3) visualization of bronchi in the outer 1 to 2 cm of the lung zones (see **Fig. 17**; **Figs. 27** and **28**). A wide variety of factors predisposing to the development of bronchiectasis have been identified, including hereditary (cystic fibrosis, ciliary dyskinesia), infective, immunodeficiency (antibody deficiency), obstructive (intrabronchial foreign body), and systemic causes. Causes most
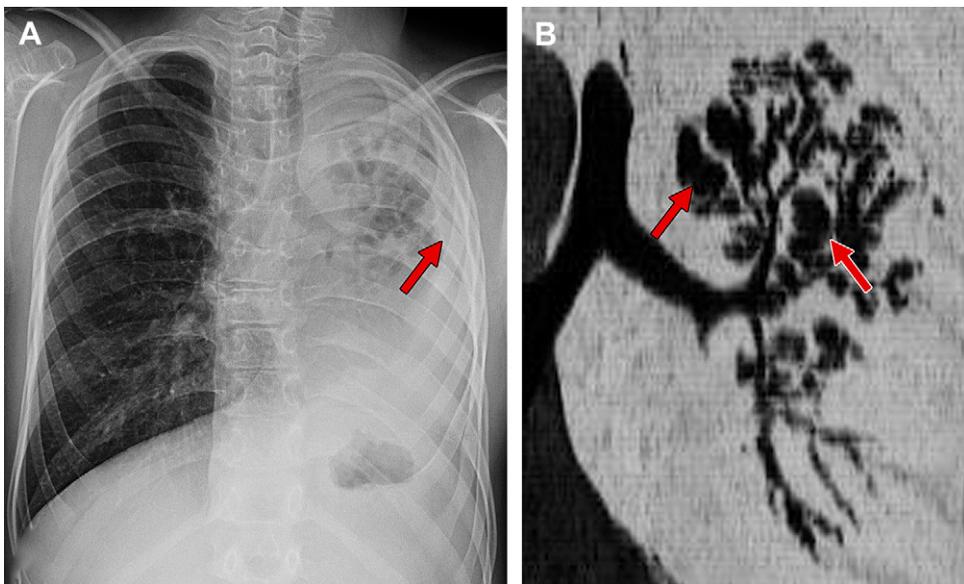
Fig. 27. Bronchiectasis secondary to recurrent infections, fibrothorax, and trapped left upper lobe in a 12-year-old boy with history of left lower lobectomy. *(A)* Frontal chest radiograph shows contraction of the left hemithorax with ipsilateral mediastinal shift and elevation of the left hemidiaphragm. Bronchiectasis of the left upper lobe and severe pleural thickening *(arrow)* are also seen. *(B)* Coronal minimum intensity projection (MinIP) image shows varicose and cystic bronchiectasis *(arrows)*.

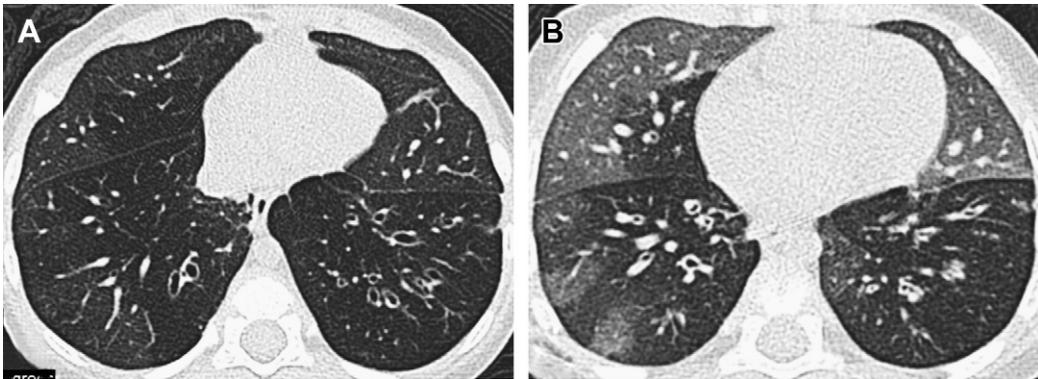**Fig. 28.** Constrictive bronchiolitis in a 1-year-old boy. Chest CViCT scans were obtained at 30 and 0 cm H_2O pressures after intubation and lung recruitment maneuvers. Inspiratory *(A)* and expiratory *(B)* axial CT images of the lower lung zone show cylindrical bronchiectasis and mosaic attenuation pattern; patchy air trapping is more apparent in expiration.

commonly associated with bronchiectasis are childhood infections, including pneumonia, pertussis, complicated measles, and tuberculosis (eg, *Mycobacterium tuberculosis* and *Mycobacterium avium* complex).[79–81]

Constrictive Bronchiolitis (Bronchiolitis Obliterans)

Constrictive bronchiolitis (bronchiolitis obliterans) is characterized by the presence of concentric narrowing or obliteration of the bronchioles caused by submucosal and peribronchiolar fibrosis. A common cause of constrictive bronchiolitis is previous childhood infection, resulting in the so-called Swyer-James syndrome, identifiable as asymmetric hyperlucent lung on chest radiographs. Whereas the process may appear unilateral on chest radiographs, there is usually bilateral but asymmetric abnormality on CT (see **Fig. 28**). Central bronchiectasis and a characteristic mosaic appearance with patchy expiratory air trapping are seen on HRCT. Causes and associations of constrictive bronchiolitis include previous infections (viral including adenovirus, RSV, influenza, parainfluenza; mycoplasma and pertussis), collagen vascular diseases, previous transplant, toxic fume exposure, ingested toxins, drugs, and cryptogenic constrictive bronchiolitis.[82]

Fibrothorax and Trapped Lung

Pleural fibrosis can result from a variety of inflammatory processes (**Box 12**). The development of pleural fibrosis follows severe pleural inflammation, which is usually associated with an exudative pleural effusion. Fibrothorax and trapped lung are 2 uncommon consequences of pleural fibrosis (see **Fig. 27**).[83]

Fibrothorax represents the most severe form of pleural fibrosis. With a fibrothorax, there is dense fibrosis of the visceral and parietal pleural surfaces, leading to fusion of these membranes, contracture of the involved hemithorax (and ipsilateral mediastinal shift), and reduced mobility of the lung and thoracic cage (see **Fig. 27**). Decortication is the only potentially effective treatment of fibrothorax in patients with severe respiratory compromise.[83,84]

A trapped lung is characterized by the inability of the lung to expand and fill the thoracic cavity because of a restrictive, fibrous, visceral pleural peel (see **Fig. 27**). Restriction of lung parenchymal expansion and subsequent negative pressure in the pleural space result in filling of the pleural space with pleural fluid (usually a transudate). The diagnosis of a trapped lung implies chronicity, stability over time, and a purely mechanical cause for the persistence of a fluid-filled pleural space. Patients with a trapped lung usually do not

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Box 12
Noninfectious causes of pleural fibrosis

Immunologic diseases such as rheumatoid pleurisy

Asbestos exposure

Malignancy

Improperly drained hemothorax

Postcoronary artery bypass graft surgery

Medications

Uremic pleurisy

Data from Jantz MA, Antony VB. Pleural fibrosis. Clin Chest Med 2006;27(2):181–91.

experience improvement in dyspnea after thoracentesis. In symptomatic patients, decortication should be considered. The underlying lung parenchyma should be assessed before decortication. If the trapped lung is severely diseased and fibrotic, decortication is unlikely to result in lung reexpansion and the procedure does not provide symptomatic benefit. In contrast, lung entrapment is the result of an active inflammatory process or malignancy in the pleural space, leading to a restricted pleural space. Pleural fluid from lung entrapment is an exudate, and symptoms in patients with lung entrapment typically improve after thoracentesis.[83,85]

Fibrosing Mediastinitis

Fibrosing mediastinitis is a rare condition characterized by proliferation of fibrous tissue within the mediastinum. Symptoms are related to compression of the central airways, superior vena cava, pulmonary veins, pulmonary arteries, and esophagus. The most common cause of this disorder is fungal infection, especially *Histoplasma capsulatum* in the United States.[86]

SUMMARY

Pneumonia is an infection of the lung parenchyma caused by a wide variety of organisms in pediatric patients. Imaging evaluation plays an important role in children with pneumonia by detecting the presence of pneumonia and determining its location and extent, excluding other thoracic causes of respiratory symptoms, and showing complications such as effusion/empyema and suppurative lung changes. Clear understanding of the underlying potential cause, current role of imaging, proper imaging techniques, and characteristic imaging appearances of acute and chronic pneumonias can guide optimal management of pediatric patients with pneumonia.

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