

Anatomic and Pathologic Causes of Recurrent Pulmonary Infections

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Recurrent pulmonary infections are caused by an assortment of conditions that involve the lung parenchyma, pulmonary vasculature, tracheobronchial tree, esophagus, and immune system (Fig 1). Each category has its own unique pathophysiologic predisposition to recurrent lung infection, and pathophysiology can overlap among the categories. Recognition of the typical clinical and imaging manifestations of these conditions is essential to aid in the diagnosis and direct treatment of patients with multiple episodes of pneumonia.

The hallmark of esophageal disorders with regard to recurrent lung infection is spillage of gastroesophageal contents into the lungs due to alterations in esophageal structure or physiology. In achalasia, dysfunction of the esophageal myenteric plexus results in aperistalsis of the lower esophagus and defective relaxation of the lower esophageal sphincter. A Zenker diverticulum can be a reservoir for ingested contents. Systemic sclerosis causes atrophy of the esophageal smooth muscles with subsequent esophageal dysmotility, dilatation, and gastroesophageal reflux. Tracheoesophageal fistulas are serious complications that may develop from various underlying causes, including congenital disorders, iatrogenic factors, radiation, trauma, and neoplasm.

Among congenital disorders, proximal interruption of the pulmonary artery and pulmonary sequestration are both characterized by an abnormal pulmonary arterial supply. Diseases with variant pulmonary blood flow can result in poor delivery of inflammatory cells, which weakens the immune response. Congenital causes of bronchiectasis result in airway collapse and impaired airway clearance; for example, bronchial atresia (focal bronchial interruption associated with distal mucus impaction) and Williams-Campbell syndrome (deficient cartilage in the fourth- to sixth-order bronchi) (Fig 2).

The lungs' immunologic defense against infections is impaired by immunodeficiency syndromes, including common variable immunodeficiency (deficiencies in B cells), Good syndrome (associated with thymomas and B- or T-cell abnormalities), and secondary immunodeficiencies. Immunocompetent patients with asthma or cystic fibrosis are susceptible to allergic bronchopulmonary aspergillosis, caused by *Aspergillus* organisms in the bronchial mucosa causing excessive immune system activation, repeated bronchospasm, bronchial wall edema, and resultant bronchiectasis.

Tracheobronchial variants include single or multiple tracheal outpouchings (diverticuli); accessory airways, particularly

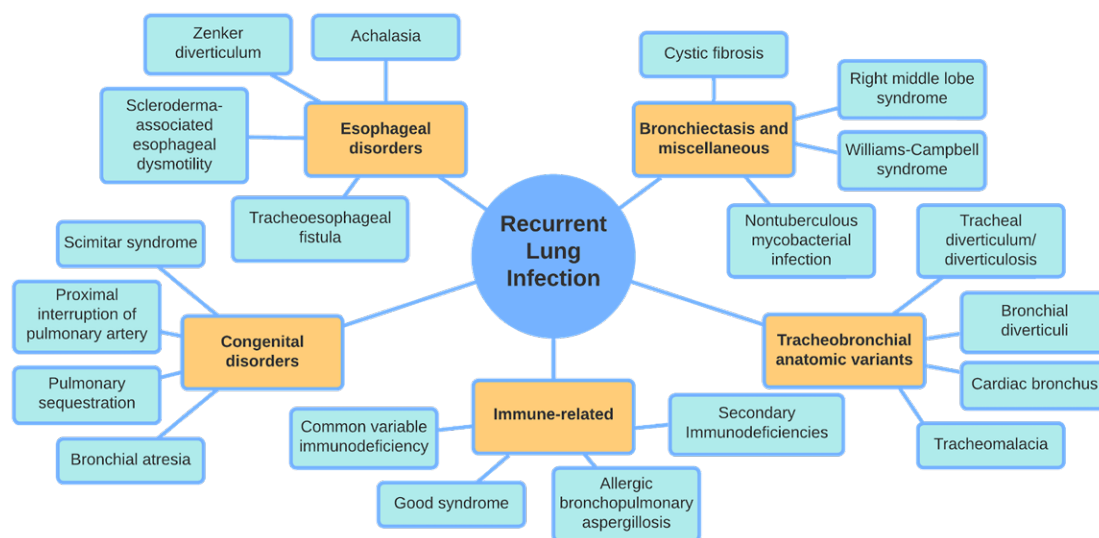


Figure 1. Overview of conditions associated with recurrent lung infection.



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TEACHING POINTS

- The hallmark of esophageal disorders with regard to recurrent lung infection is spillage of gastroesophageal contents into the lungs due to alterations in esophageal structure or physiology.
- Diseases with variant pulmonary blood flow can result in poor delivery of inflammatory cells, which weakens the immune response.
- Immune cell deficiency, impaired or excessive immune system reaction, and variations in mucociliary clearance blunt the immune response and predispose to lung infections.

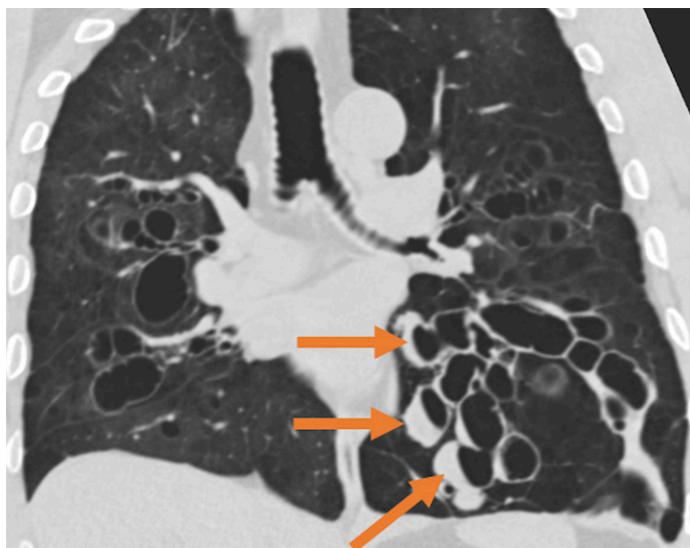


Figure 2. Coronal CT image shows marked cystic bronchiectasis and bronchial wall thickening, predominantly involving the third- to sixth-order bronchi. Left lower lobe endobronchial air-fluid levels and bronchial wall thickening suggest superimposed infection (arrows). Sputum cultures showed *Pseudomonas aeruginosa*.

cardiac bronchi; and tracheobronchomalacia. Recurrent airway injury and chronic inflammation are typical for several conditions owing to ineffective airway clearance. Thick mucus in cystic fibrosis alters bacterial killing, and repeated infections lead to bronchiectasis. Nontuberculous mycobacterial infection can have a chronic indolent or progressive course and tends to occur in the setting of structural lung disease or bronchiectasis. Right

middle lobe atelectasis due to extrinsic airway compression or nonobstructive causes results in chronic inflammation and/or infection, predisposing to bronchiectasis.

Treatment can range from watchful waiting, supportive care, and antibiotics or other medications to surgery if necessary. Prompt identification of relevant imaging abnormalities in patients who present with recurrent pulmonary infection is essential in guiding management.

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Suggested Readings

- Berrocal T, Madrid C, Novo S, Gutiérrez J, Arjonilla A, Gómez-León N. Congenital anomalies of the tracheobronchial tree, lung, and mediastinum: embryology, radiology, and pathology. *RadioGraphics* 2004;24(1):e17.
- Boiselle PM, Reynolds KF, Ernst A. Multiplanar and three-dimensional imaging of the central airways with multidetector CT. *AJR Am J Roentgenol* 2002;179(2):301–308.
- Castañer E, Gallardo X, Rimola J, et al. Congenital and acquired pulmonary artery anomalies in the adult: radiologic overview. *RadioGraphics* 2006;26(2):349–371.
- Cowman S, van Ingen J, Griffith DE, Loebinger MR. Non-tuberculous mycobacterial pulmonary disease. *Eur Respir J* 2019;54(1):1900250.
- Franquet T, Müller NL, Giménez A, Gueembe P, de La Torre J, Bague S. Spectrum of pulmonary aspergillosis: histologic, clinical, and radiologic findings. *RadioGraphics* 2001;21(4):825–837.
- Jeong YJ, Kim KI, Seo IJ, et al. Eosinophilic lung diseases: a clinical, radiologic, and pathologic overview. *RadioGraphics* 2007;27(3):617–637; discussion 637–639.
- Martinez S, Heyneman LE, McAdams HP, Rossi SE, Restrepo CS, Eraso A. Mucoid impactions: finger-in-glove sign and other CT and radiographic features. *RadioGraphics* 2008;28(5):1369–1382.
- McGuinness G, Naidich DP, Garay SM, Davis AL, Boyd AD, Mizrahi HH. Accessory cardiac bronchus: CT features and clinical significance. *Radiology* 1993;189(2):563–566.
- Pickhardt PJ, Bhalla S, Balfe DM. Acquired gastrointestinal fistulas: classification, etiologies, and imaging evaluation. *Radiology* 2002;224(1):9–23.
- Williams EA, Cox C, Chung JH, Grage RA, Rojas CA. Proximal Interruption of the Pulmonary Artery. *J Thorac Imaging* 2019;34(1):56–64.
- Woodfield CA, Levine MS, Rubesin SE, Langlotz CP, Laufer I. Diagnosis of primary versus secondary achalasia: reassessment of clinical and radiographic criteria. *AJR Am J Roentgenol* 2000;175(3):727–731.