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BRONCHIECTASIS

Developed by Lauren Wilkinson and Dr. Mahmoud Sakran for PedsCases.com. October 22, 2025

Introduction:

Hi everyone, and welcome to today's episode of *PedsCases*. I'm Lauren Wilkinson, a second-year medical student at Queen's University, and today I'm going to be talking about bronchiectasis in children. This podcast was co-developed with Dr. Mahmoud Sakran, a pediatrician at Lakeridge Health in Ontario.

By the end of this episode, listeners will be able to:

- 1. Define bronchiectasis and explain the pathophysiology
- 2. Identify common causes and risk factors for pediatric bronchiectasis
- 3. Describe the common presenting features of bronchiectasis
- 4. Describe the diagnostic approach, including imaging findings on high-resolution CT and key laboratory investigations
- 5. Outline the principles of management

Case:

Let's start with a case:

You're seeing a 9-year-old boy named Henry from a rural community in your outpatient clinic. He presents with a 2-day history of fever and worsening productive cough. You ask about the onset of his symptoms, and his parents explain that he's been coughing for over five weeks and producing foul-smelling, yellow sputum. He was prescribed oral amoxicillin 4 weeks ago with no improvement. They also noticed specks of blood in his sputum today.

Naturally, you start digging more into his history. You ask about previous hospitalizations, and his parents explain that Henry developed a chronic cough and pneumonia following a severe measles infection at 10 months. Since then, he's had multiple hospitalizations for recurrent lower respiratory tract infections, and a chronic cough off and on for the past few years. They also note that Henry has not been vaccinated.



On exam, Henry is febrile at 39°C (102°F). He has digital clubbing and rales over the right lower lung zone.

You start to think about a differential for Henry's chronic respiratory symptoms. Could this be bronchiectasis? What would you do next?

Objective 1: Define bronchiectasis and explain the pathophysiology

While we think about this case, let's define what bronchiectasis is.

Bronchiectasis is the permanent and abnormal dilation of the bronchi and bronchioles, often caused by repeated cycles of infection, inflammation, and impaired mucociliary clearance that damages the airway walls.^{1–4} It involves a decrease in function of the cartilage and elastin in conducting airways.

Think of bronchiectasis as the end result of a vicious cycle: something damages the airway, infections move in, inflammation builds up, and the bronchial walls start to break down and dilate. It usually begins with an initial airway insult, such as a severe infection or obstruction, that triggers chronic inflammation.¹

Once the airway is inflamed, mucociliary clearance is impaired. Mucus builds up, creating an environment where bacteria can linger longer, colonize the lung and in some cases form biofilms. This colonization can lead to repeated infections, and the inflammation continues. Over time, this cycle causes structural damage to the bronchial walls and progressive dilation of the airways, which reduces oxygenation and gas exchange.⁴

A key player in this process is the neutrophil, a type of white blood cell that responds to infection. When activated, neutrophils produce enzymes elastase and matrix metalloproteinases that break down airway tissue. Inflammatory cytokines like IL-8, IL-6, and TNF-alpha are also elevated in the airways, especially IL-8, which acts as a signal to recruit more neutrophils. This further amplifies the damage. Eosinophils and T-cells also play a role in this process.¹ Eosinophils are thought to further promote airway injury through neutrophil recruitment, goblet cell hyperplasia, and excess mucus production, and cytotoxic T cells are thought to contribute to the immune response.¹

While these mechanisms ultimately lead to irreversible bronchial dilation, early stages may still be reversible.

Objective 2: Identify common causes and risk factors for bronchiectasis in children

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Let's talk about what causes bronchiectasis in children.

As we previously mentioned, bronchiectasis develops through a self-perpetuating cycle of infection, inflammation, and airway damage. So, what starts this cycle?

One of the most common causes of bronchiectasis around the world is post-infectious disease, particularly following severe or untreated respiratory infections like adenovirus, pertussis, measles, and tuberculosis. Infections like Mycoplasma or Non-Tuberculous Mycobacterium can also lead to bronchiectasis, particularly in immunocompromised children or those with preexisting lung conditions.⁴ One important example of post-infectious disease leading to bronchiectasis is protracted bacterial bronchitis (PBB). This is an acute condition characterized by a chronic wet cough due to persistent bacterial infection. If left untreated, it can progress to bronchiectasis, but if treated early with antibiotics and airway clearance, the cycle can be interrupted before permanent damage occurs.^{3,4}

While post-infectious disease is the most common cause around the world, in industrialized countries, like here in Canada, the #1 cause of bronchiectasis is cystic fibrosis. In cystic fibrosis, the mucus is more viscous and therefore harder to clear, leading to bacterial colonization and the cycle of infection and inflammation.

Another cause of bronchiectasis is impaired mucociliary clearance, often caused by primary ciliary dyskinesia, a genetic disorder that causes defects in the action of cilia lining the respiratory tract. Primary immunodeficiencies that predispose children to recurrent infections also play a role. Similarly, airway obstruction from congenital malformations, foreign body aspiration, or external compression, can also lead to bronchiectasis. Children with neuromuscular disorders, gastroesophageal reflux, or swallowing dysfunction are also at risk due to chronic aspiration.⁴

Finally, in rare cases, autoimmune conditions, environmental exposures, or genetic syndromes like Williams-Campbell or Mounier-Kuhn syndrome may underlie the disease. Other diseases associated with bronchiectasis are yellow nail syndrome (which is characterized by a triad of pleural effusion, lymphedema, discolored nails) and right middle lobe syndrome.¹

Another really important thing to know is that bronchiectasis is more common is equity deserving populations, especially among Indigenous communities, where the prevalence is estimated to be around 200 per 100,000 in Inuit children.⁵ This increased burden is likely driven by environmental and social risk factors that contribute to frequent respiratory infections in early childhood, such as air pollution, overcrowding, poor sanitation, low-socioeconomic status, and limited access to healthcare. In regions where tuberculosis is endemic, prevalence is also higher.⁴

Objective 3: Describe the common presenting features of bronchiectasis



Let's move on to the clinical features of bronchiectasis.

In children, a hallmark clinical feature of bronchiectasis is a chronic wet or productive cough that persists for more than 4 weeks despite oral antibiotic therapy. This cough is often associated with purulent sputum production.^{1,3,4} Additional clinical features may include recurrent airway infections, especially recurrent pneumonia or bronchitis, hemoptysis, fever, anorexia, poor weight gain, and failure to thrive. In advanced disease, digital clubbing, chest wall deformities, dyspnea and hypoxemia can also be seen.^{1–3} On auscultation, localized crackles are often heard over affected lung regions, as well as focal or diffuse rales or rhonchi. Wheezing is a less common finding.^{2,3}

Bronchiectasis significantly impacts quality of life, especially during symptom exacerbations.^{2,3} Acute exacerbations of bronchiectasis are characterized by increase in the frequency of cough with an increase in sputum volume and purulence, as well as crepititions and wheeze.⁶ Severe exacerbations can present with dyspnea and/or hypoxia.⁷

Objective 4: Describe the diagnostic approach to bronchiectasis

Now that we know the clinical features to look for, let's discuss how to diagnose bronchiectasis.

If you have a patient with symptoms suggestive of bronchiectasis, the first thing you would do is get a chest X-ray. Chest X-rays should be performed in all children with a chronic wet cough. However, it is important to remember that they are non-specific for bronchiectasis and should not be used to rule this out.^{2,3}

The gold standard for diagnosis of bronchiectasis is a high-resolution chest CT scan.^{1–3} Classic findings include bronchial wall thickening, mucus plugging, dilated peripheral bronchi, lack of bronchial tapering to the lung periphery (so less space between the bronchi and the pleura, called "tramlines"), and the signet ring sign. The signet sign is where the bronchus appears wider than that of the adjacent pulmonary artery, when they should be the same diameter.^{1–3}

In addition to CT scans, pulmonary function testing is also part of the diagnostic workup. Spirometry should be performed at diagnosis with serial measures in follow-up. Most children show an obstructive pattern, although some display restrictive or mixed symptoms.²

Identifying the underlying etiology is critical to guide treatment, and so a thorough clinical history is essential. Further evaluations should also include a sweat chloride test, complete blood count and immunologic tests, tuberculin test, bacterial cultures, and additional tests specific to each patient.^{2,3}

Let's apply what we learned to the case.

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For Henry, you do as we discussed and order a chest X-ray, which shows peribronchial thickening and volume loss in the middle and right lobes. To confirm bronchiectasis, you do a CT, which shows tramlines (thickened bronchi without tapering), signet-sign appearance (increased bronchoarterial ratio), and bronchial wall thickening. This confirms bronchiectasis, especially in the right middle and lower lobes. Now that you know he has bronchiectasis, your next step is to figure out etiology. You order pulmonary function tests, which show a severe obstruction with mild-to-moderate reduction in lung volumes, which is consistent with chronic airway damage. His sputum culture is positive for streptococcus pneumoniae.

To rule out cystic fibrosis, you order a sweat test and genetic panel, which are negative for CF. His immunodeficiency work-up is normal, and his CBC shows anemia and an elevated white blood cell count. His parents deny any events of chocking on food or foreign body aspiration, which lowers airway obstruction on your differential. There are also no red flags in his history suggestive of chronic aspiration or primary ciliary dyskinesia. His TB skin test is negative.

You review his medical records again and remember that he had measles followed by pneumonia at 10 months old, as well as recurrent lower respiratory tract infections and hospitalizations for pneumonia. This supports the diagnosis of bronchiectasis due to post-infectious disease.

Now, how do we treat bronchiectasis?

Objective 5: Outline the principles of management

The goal of treatment is to limit infection and decrease airway obstruction and should be managed both preventatively and symptomatically. Children should receive routine immunizations against typical respiratory pathogens.^{1,3} Airway clearance therapies may also be beneficially, especially for bronchiectasis caused by cystic fibrosis.³ Airway clearance techniques includes gravity-assisted drainage, the active cycle of breathing technique, and positive expiratory pressure to clear out secretions from the airways to slow disease progression and minimize exacerbations.⁸ In some cases, bronchodilators can also help with airway clearance.^{1,3}

During exacerbations, antibiotics are used. The choice of antibiotic is dictated by the identification and sensitivity of organisms found on deep throat, sputum, or bronchoalveolar lavage fluid cultures. A 14-day cycle of amoxicillin/clavulanic acid has proven to be successful at treating most exacerbations, however, selection should still rely on culture results.¹

In severe cases, segmental or lobar resections may be warranted. Lung transplantation is also an option is severe cases.^{1,3}



In Henry's case, you admit him for intravenous antibiotics, in this case penicillin G based on his sputum culture and local resistance patterns. You initiate airway clearance therapy, such as positive expiratory pressure (PEP), to manage secretions and reduce exacerbations. You also do a trial of bronchodilators.

Once he is stable, you have a conversation with the parents about vaccination and the importance of keeping him up to date with routine vaccinations to prevent further infections.

Long-term, he will need routine follow-up with respirology where they can monitor lung function and guide antibiotic use via spirometry and sputum cultures. He should also be referred to a respiratory physiotherapist and infectious diseases to support ongoing airway clearance and manage chronic infection and antibiotic stewardship.

Summary

Let's summarize the key points.

- 1. Bronchiectasis is the permanent and abnormal dilation of the bronchi and bronchioles caused by repeated cycles of infection, inflammation, and impaired mucociliary clearance that damages the airway walls.
- 2. Bronchiectasis is caused by post-infectious disease (e.g. measles, TB, pertussis, etc.), cystic fibrosis, impaired mucociliary clearance due to primary ciliary dyskinesia, primary immunodeficiencies, airway obstruction, and various genetic syndromes. In North America, the most common cause is cystic fibrosis, while globally, the most common cause is post-infectious disease.
- 3. Bronchiectasis is diagnosed using high resolution chest CT, and etiology is determined using pulmonary function testing, sweat chloride testing, bloodwork, microbiologic cultures, and other patient specific tests.
- 4. Finally, management includes airway clearance therapies, antibiotics, bronchodilators, and in severe cases, surgical resection or lung transplantation. While the damage is often irreversible, the goal is to limit infection and decrease airway obstruction.

Outro

That brings us to the end of today's podcast on bronchiectasis. I'm Lauren Wilkinson, and this is *PedsCases*.

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