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Bronchiectasis: An In-Depth Review of Pathogenesis, Diagnostic Approaches, And Management Strategies

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ABSTRACT:
Bronchiectasis is a chronic and heterogeneous respiratory disorder marked by permanent dilatation of the bronchi, recurrent infections, and persistent airway inflammation. Once considered a neglected disease, its prevalence is now recognized as rising globally, posing significant challenges to patient quality of life and healthcare systems. The condition arises from a vicious cycle of impaired mucociliary clearance, microbial colonization, and neutrophil-driven inflammation, with various underlying causes ranging from post-infectious damage to immune deficiencies and comorbid conditions. High-resolution computed tomography remains the gold standard for diagnosis, offering detailed visualization of airway dilatation and structural abnormalities. Current management relies mainly on airway clearance techniques, antibiotic therapy for chronic bacterial infection, and control of exacerbations, with surgery considered only in selected cases. Despite these measures, there is no approved disease-modifying treatment for bronchiectasis. This review summarizes the current knowledge of the pathogenesis, diagnostic challenges, and therapeutic strategies for bronchiectasis
Keywords: Bronchiectasis; Diagnosis; Epidemiology; Aetiology; Treatment

INTRODUCTION:

The term *bronchiectasis* originates from the Greek words *bronkhia* (“airway”) and *ektasis* (“widening” or “dilation”), aptly describing the hallmark pathological change of the disease—irreversible dilatation of the bronchi. The historical understanding of bronchiectasis can be traced back to the early 19th century, when René Laennec, the French physician and inventor of the stethoscope, first identified the condition in 1819 using his revolutionary device. Laennec’s observations provided the earliest clinical recognition of bronchiectasis as a distinct pulmonary disorder. Later, in the late 1800s, Sir William Osler, one of the founding professors of Johns Hopkins Hospital, expanded on these findings with detailed descriptions that greatly advanced medical knowledge of the disease. Interestingly, it is speculated that Osler himself may have suffered from undiagnosed bronchiectasis, as his biographies recount a history of recurrent, severe chest infections over many years, which may have contributed to his death. Collectively, these historical milestones underscore how the combination of novel medical tools, clinical acumen, and early pathological insights shaped the foundational understanding of bronchiectasis. Bronchiectasis refers to both a clinical disease and a radiological appearance that has multiple causes and can be associated with a range of conditions. Bronchiectasis can be a feature of many diverse clinical

entities, including cystic fibrosis, chronic obstructive pulmonary disease or asthma, and traction associated with interstitial lung disease or tuberculous-associated lung destruction [1].

EPIDEMIOLOGY:

The epidemiology of bronchiectasis reflects its diverse and multifactorial causes, varying significantly across age groups, geographic regions, and healthcare settings. Historically more common in low- and middle-income countries due to post-infective causes such as childhood infections (e.g., tuberculosis, whooping cough, and measles), bronchiectasis is now increasingly recognized in high-income countries due to improved imaging and awareness. It affects both children and adults, with rising prevalence in older populations. [2]

Bronchiectasis is increasingly recognized as a significant global health concern, with a pooled prevalence of 680 per 100,000 persons based on 15 large-scale studies encompassing over 437 million individuals across the United States, Korea, China, Belgium, Germany, and Australia. The condition appears more common in Asia, particularly Korea and China, likely due to the high regional burden of tuberculosis (a key underlying cause), environmental exposures, and possible genetic predispositions. Subgroup analyses reveal a higher prevalence in females, possibly linked to conditions more frequent in women, such as connective tissue diseases and non-tuberculous mycobacterial infections. Additionally, bronchiectasis prevalence was notably higher among individuals with low BMI and those with comorbidities such as TB and inflammatory bowel disease, highlighting nutritional status and systemic inflammation as important contributing factors. Although smoking's role in bronchiectasis remains unclear, preliminary findings suggest increased prevalence among current and former smokers. The high rate of comorbidities among patients with bronchiectasis—ranging from chronic respiratory and autoimmune conditions to systemic disorders—further complicates disease management and contributes to poorer outcomes. Variations in prevalence between countries, sexes, BMI categories, and health statuses underscore the need for population-specific research, improved disease registries, and tailored management strategies. [3]

AETIOLOGY:

Bronchiectasis is a chronic pulmonary disorder with a broad and multifactorial aetiology, which includes post-infective, inflammatory, genetic, congenital, immunological, and obstructive causes. Historically, it was most commonly attributed to previous respiratory infections during childhood, such as whooping cough, measles, or tuberculosis, leading to long-term structural damage of the airways. Numerous bacterial infections contribute to its development, including *Mycobacterium tuberculosis* and atypical mycobacteria, *Haemophilus influenzae*, *Pseudomonas aeruginosa*, *Staphylococcus aureus*, and *Mycoplasma pneumoniae*, while HIV-associated infections further increase susceptibility. Viral infections, such as respiratory syncytial virus and measles, and fungal infections, notably *Aspergillus* species, also play pathogenic roles. Non-infectious airway insults, like aspiration of gastric contents, inhalation of toxic gases or smoke, or post-inflammatory pneumonitis, can initiate or worsen bronchial damage. Bronchial obstruction, whether intrinsic (e.g., tumors, mucus plugs) or extrinsic (e.g., hilar lymphadenopathy in right middle lobe syndrome), may cause localized bronchiectasis.

Genetic and congenital abnormalities significantly contribute to impaired mucociliary clearance and host defense mechanisms. These include cystic fibrosis (CF), primary ciliary dyskinesia (PCD), Young syndrome, Mounier–Kuhn syndrome, pulmonary sequestration, and common variable immune deficiency (CVID). In such disorders, ineffective clearance of pathogens leads to chronic infection and inflammation. Several autoimmune and inflammatory conditions—including rheumatoid arthritis, ulcerative colitis, and Sjögren's syndrome—have been associated with bronchiectasis due to systemic immune-mediated airway injury. Additionally, pulmonary diseases such as asthma, bronchomalacia, chronic obstructive pulmonary disease (COPD) (observed in up to 50% of patients with moderate-to-severe COPD), diffuse panbronchiolitis, and idiopathic pulmonary fibrosis (through traction bronchiectasis) also result in secondary bronchiectatic changes. Other notable contributors include chronic sinusitis, GERD, yellow nail syndrome, and hypersensitivity pneumonitis.

Moreover, bronchiectasis may stem from an exaggerated immune response, as seen in allergic bronchopulmonary aspergillosis and other hypersensitivity states. In some patients, especially those with genetic immunodeficiencies, there is an inherent impairment of the host's ability to clear infections, predisposing them to recurrent microbial insults and airway destruction. Despite exhaustive investigations, a substantial proportion of bronchiectasis cases remain idiopathic, with no identifiable cause. Nonetheless, identifying the underlying etiology has significant clinical implications. [4-5]

PATHOPHYSIOLOGY:

The pathophysiology of bronchiectasis is best understood through two evolving conceptual frameworks: the vicious cycle and the more recent vicious vortex models. Together, they encapsulate the self-perpetuating mechanisms underlying the disease, emphasizing the interplay between impaired airway clearance, microbial infection, chronic inflammation, and tissue damage.

The original vicious cycle hypothesis, proposed by Cole in 1986, suggests that the disease begins with impaired mucociliary clearance, a critical first-line defense of the respiratory tract. This impairment can be congenital, as seen in primary ciliary dyskinesia (PCD) and cystic fibrosis (CF), or acquired due to infections, inhalational injuries, or systemic diseases. When the mucociliary escalator is dysfunctional, mucus accumulates in the airways, creating an environment conducive to microbial colonization and infection. The host's immune response to these pathogens triggers a robust inflammatory reaction, primarily dominated by neutrophils. Neutrophilic inflammation leads to the release of proteolytic enzymes such as elastase and reactive oxygen species, which, while intended to eradicate pathogens, also degrade the structural components of the airway walls, causing irreversible bronchial dilatation.

The structural damage inflicted by this process exacerbates airway obstruction and further impairs mucociliary clearance, thus perpetuating the cycle. The underlying etiology of bronchiectasis often determines the entry point into this loop. For instance, in immunodeficiency syndromes, recurrent infections due to the lack of effective immune surveillance act as the initiating factor, while in inflammatory bowel disease or autoimmune conditions, systemic inflammation plays a major contributory role. However, real-world disease progression is rarely linear. To better reflect the intricate, multi-directional interactions among the various components of

disease, the vicious vortex model, as proposed by Flume and colleagues, was introduced. This model highlights the dynamic interdependence among four key factors: airway dysfunction, infection, inflammation, and structural damage. Each element not only leads to but is also influenced by the others, creating a complex, swirling pathophysiological network rather than a simple sequential loop.

Central to bronchiectasis pathophysiology is the persistent infection, often with gram-negative bacteria such as *Pseudomonas aeruginosa* and non-typeable *Haemophilus influenzae* (NTHi). These pathogens have evolved mechanisms that allow them to thrive in the mucus-laden, inflamed airways of bronchiectatic lungs. *P. aeruginosa*, for example, produces ciliotoxic substances like pyocyanin, which impair ciliary beat frequency and further hinder mucociliary clearance. It also secretes elastases and proteases that directly damage epithelial cells and the extracellular matrix, worsening structural deterioration. In addition, these microbes form biofilms, which protect them from antibiotics and immune attacks, enabling chronic colonization.

The immune response in bronchiectasis is largely neutrophil-dominated, although other immune cells play significant roles. Neutrophils, while essential in clearing infections, contribute significantly to tissue injury through the release of neutrophil extracellular traps (NETs)—web-like structures composed of DNA, histones, and antimicrobial proteins. While NETs can trap pathogens, they are also highly inflammatory and cytotoxic to host tissues. Elevated NET formation has been linked to disease severity and lung function decline in bronchiectasis patients. In addition to neutrophils, macrophages are involved in pathogen clearance and cytokine release. However, in bronchiectasis, their ability to phagocytose bacteria is often impaired, especially in the presence of biofilms or in the setting of immune dysfunction. Eosinophils, traditionally associated with asthma and allergic diseases, have been found in subsets of bronchiectasis patients, particularly those with overlapping features of eosinophilic airway inflammation, and may contribute to exacerbations and mucus hypersecretion.

Epithelial cells, which line the airways, are not passive players. They actively sense pathogens through pattern recognition receptors (PRRs) and initiate innate immune responses. In bronchiectasis, repeated infections and inflammation damage these cells, leading to loss of barrier function and dysregulated repair processes. As a result, epithelial cells may undergo phenotypic changes, including mucous cell metaplasia, which increases mucus production and thickens airway secretions, further worsening clearance.

The persistent presence of pro-inflammatory cytokines such as interleukin-1β (IL-1β), tumor necrosis factor-α (TNF-α), interleukin-8 (CXCL-8), and matrix metalloproteinases (MMPs) creates a highly inflammatory milieu. IL-8, in particular, is a potent neutrophil chemoattractant and is heavily involved in the recruitment of neutrophils to the airways. Chronic inflammation not only causes tissue damage but also drives airway remodeling, characterized by bronchial wall thickening, smooth muscle hypertrophy, goblet cell hyperplasia, and fibrosis. These changes are irreversible and represent the end-stage consequences of the ongoing inflammatory and infectious processes.

Immune deficiencies, both primary (e.g., common variable immunodeficiency) and secondary (e.g., due to HIV or immunosuppressive therapies), are recognized contributors to bronchiectasis. In such cases, the immune system fails to effectively clear pathogens, allowing infections to persist and inflammation to become chronic.

Additionally, autoimmune conditions such as rheumatoid arthritis or systemic lupus erythematosus may also cause bronchiectasis through immune-mediated tissue injury or recurrent infections due to immunosuppressive treatment.

Environmental and lifestyle factors, including exposure to noxious inhalants, smoking, and air pollution, can exacerbate bronchial injury and inflammation, further promoting disease progression. Moreover, gastroesophageal reflux disease (GERD) and aspiration are common in patients with bronchiectasis and may contribute to recurrent infections and worsening lung damage.

The pathophysiology of bronchiectasis is a multifactorial, interdependent process driven by impaired mucociliary clearance, chronic infection, persistent inflammation, and irreversible airway damage. The disease may be initiated through various entry points depending on the underlying cause, but regardless of origin, the pathogenesis ultimately converges into a vicious vortex—a self-amplifying network of interactions that sustains and worsens the disease over time. Understanding these complex mechanisms is essential for guiding effective therapeutic strategies aimed at breaking this destructive cycle, including airway clearance techniques, antibiotic management, anti-inflammatory therapies, and targeted interventions against specific pathogens or immune pathways. [6-7]

SIGNS AND SYMPTOMS:

Bronchiectasis presents primarily with a chronic cough and daily mucopurulent sputum production, often persisting for months to years. The cough is typically productive, with sputum being mucoid and odorless under stable conditions but becoming purulent, foul-smelling, and more copious during infectious exacerbations. In some patients, sputum production can exceed 150 mL daily, indicating severe disease. Hemoptysis—ranging from blood-streaked sputum to massive bleeding—occurs in 56–92% of cases and may be the first sign prompting medical evaluation. It is particularly common in dry bronchiectasis, a rare form often secondary to tuberculosis and involving minimal sputum but episodic bleeding, typically affecting the upper lobes. [8]

Less specific symptoms include dyspnea (shortness of breath), pleuritic chest pain, wheezing, fever, fatigue, weight loss, and general malaise. Dyspnea is reported in 62–72% of patients and is usually associated with extensive disease or coexisting conditions like chronic bronchitis or emphysema. Wheezing is frequent due to airflow obstruction from bronchial damage or coexisting asthma. Pleuritic chest pain occurs in up to 46% of patients and may be linked to persistent coughing or infection. Fatigue affects a majority (up to 73%) and, along with unintentional weight loss, is often a marker of advanced disease, suggesting increased energy expenditure from chronic coughing and infection.

Acute exacerbations are typically triggered by bacterial infections and are characterized by increased sputum volume and viscosity, changes in sputum color or odor, worsening dyspnea, low-grade fever, and increased constitutional symptoms. These episodes often reflect underlying colonization or infection with organisms such as *Pseudomonas aeruginosa* or *Haemophilus influenzae*. Some patients may experience repeated respiratory infections or pneumonias since childhood, leading to the development of bronchiectasis.

In children, a chronic wet cough that persists beyond 4 weeks despite antibiotics is a strong predictor of bronchiectasis. Studies show that Indigenous populations and those with a history of unresolved infections have a higher incidence.

Physical examination findings are often nonspecific but may include crackles, rhonchi, wheezing, inspiratory squeaks, and signs of chronic respiratory illness. Crackles occur in up to 73% of patients, especially during active infection or exacerbation. Wheezing, present in about one-third of cases, may be due to mucus obstruction or airway collapse. Digital clubbing is seen in approximately 2–3% of patients and is more frequent in moderate-to-severe disease. Rarely, cyanosis, plethora, and signs of cor pulmonale (right heart failure) such as peripheral edema and hepatomegaly may be observed, especially in end-stage disease. Additional signs may include nasal polyps, chronic sinusitis, and wasting.

Interestingly, urinary incontinence has also been reported more commonly in women with bronchiectasis (47% vs 12% in age-matched controls), although the exact cause remains unclear.

Although some patients exhibit dramatic daily symptoms, others may have subtle or minimal symptoms, despite significant radiological disease. This clinical-radiological dissociation emphasizes the importance of imaging—particularly high-resolution CT—in diagnosis. In cystic fibrosis (CF), which is the leading cause of bronchiectasis in children and young adults, sputum production tends to be even more profuse.

DIAGNOSIS:

Initial Evaluation: In patients with clinical features suggestive of bronchiectasis, baseline investigations are essential for diagnostic triage and ongoing monitoring. These typically include:

- Chest radiograph – useful for identifying structural lung changes but limited in sensitivity and specificity.
- Pulmonary function tests – including forced expiratory volume in 1 second (FEV_1), forced vital capacity (FVC), lung volumes, and diffusion capacity, which provide insight into functional impairment.
- Sputum culture – for bacteriological analysis, aiding in the detection of common pathogens and guiding antimicrobial therapy.

While these tests provide important clinical information, they are not definitive for confirming the diagnosis.

Confirmatory Imaging: The gold standard for confirming bronchiectasis is high-resolution computed tomography (HRCT) of the chest, ideally performed when the patient is clinically stable.

- Thin-section slices (<1 mm) reconstructed with a high-spatial frequency algorithm are recommended.
- Volumetric CT may provide superior sensitivity but exposes patients to higher radiation doses.

Identifying the Underlying Cause: Establishing the etiology is critical, as it may influence management in up to 37% of adult patients. A targeted diagnostic workup, guided by patient history and clinical features, helps to identify causes such as infections, immune deficiencies, or genetic disorders.

Assessing Infection and Disease Severity: Ongoing infection and inflammation are central to disease progression. Investigations include:

- Sputum analysis – for bacterial, mycobacterial, and fungal cultures.
- Lung function tests – to monitor severity and progression.

Persistent airway bacterial infection is common, present in 64–79% of patients even during stable disease. The most frequent pathogens include *Haemophilus influenzae* (47–55%) and *Pseudomonas aeruginosa* (12–26%), with less frequent isolation of *Moraxella catarrhalis*, *Streptococcus pneumoniae*, *Staphylococcus aureus*, and occasionally nontuberculous mycobacteria. Chronic *P. aeruginosa* colonization is strongly associated with accelerated lung function decline and reduced quality of life. Emerging studies using molecular methods have revealed a more diverse airway microbiome, correlating with clinical parameters such as FEV_1 and cough severity. However, the clinical implications of these findings remain under investigation.

Prognostic Assessment: Bronchiectasis has a highly variable natural history, with some patients experiencing infrequent infections and stable disease, while others progress rapidly to respiratory failure and increased mortality risk.

Two validated scoring systems are used to assess severity and predict outcomes:

- FACED Score – incorporates FEV_1% predicted, age, chronic *Pseudomonas* colonization, number of affected lobes, and dyspnea (Medical Research Council scale). It predicts five-year mortality.
- Bronchiectasis Severity Index (BSI) – integrates variables such as age, BMI, FEV_1, dyspnea score, extent of disease, and chronic bacterial infection. Initially validated for hospitalization and four-year mortality, it is now also used to predict exacerbations, lung function decline, and quality of life.

Risk stratification with FACED and BSI should be applied alongside clinical judgment, as their role in monitoring treatment response or disease progression over time is not fully established. [9]

DIFFERENTIAL DIAGNOSIS:

Bronchiectasis shares clinical features with several other pulmonary and systemic conditions, making differential diagnosis essential for accurate treatment. Chronic obstructive pulmonary diseases such as COPD, chronic bronchitis, and emphysema often mimic bronchiectasis due to overlapping symptoms like chronic cough, sputum production, and dyspnea. Asthma may present similarly with wheezing and airflow obstruction, but lacks radiographic evidence of bronchial dilation. Cystic fibrosis, a major cause of bronchiectasis in younger patients, must be differentiated based on genetic and sweat chloride testing. Infectious diseases such as tuberculosis, bacterial pneumonia, and aspiration pneumonitis can lead to or resemble bronchiectatic changes but differ in etiology and course. Alpha-1 antitrypsin deficiency may also present with airflow limitation and radiographic abnormalities, requiring serum level testing. Additionally, gastroesophageal reflux disease (GERD) and recurrent aspiration may mimic or contribute to bronchiectasis development. Parapneumonic effusions and empyema thoracis may complicate infections and lead to localized changes that resemble bronchiectasis. [10]

MANAGEMENT:

The management of bronchiectasis requires a comprehensive, multifaceted, and individualized approach that targets the underlying cause, improves mucus clearance, controls infection and inflammation, relieves airway

obstruction, and prevents complications. The initial step involves determining whether a modifiable underlying cause exists, such as immunoglobulin deficiency or alpha-1 antitrypsin deficiency, since identifying and correcting these can significantly improve patient outcomes. Once this evaluation is complete, treatment is directed toward ongoing symptom control and prevention of disease progression through pharmacological and non-pharmacological strategies.

Airway clearance therapy: It represents the cornerstone of bronchiectasis management, as it directly addresses the cycle of mucus retention, infection, and inflammation that perpetuates disease progression. The primary goal of airway clearance is to loosen secretions, improve breathing, relieve cough, and facilitate the expectoration of phlegm and mucus plugs. Several airway clearance techniques are available, including chest physiotherapy, high-frequency chest wall oscillation, and the active cycle of breathing technique, which can be employed with or without oscillatory flutter devices. Inhaled agents such as hypertonic saline are often used alongside these methods to hydrate airway secretions and enhance clearance. The choice of technique or device is individualized based on sputum viscosity, patient comfort, lifestyle, and cost considerations. While mucolytic agents like dornase alfa are beneficial in cystic fibrosis, they are not recommended for non-CF bronchiectasis. Similarly, mannitol, despite its hyperosmolar action, has not consistently demonstrated clinical efficacy.

Anti-inflammatory therapy: It is another critical component in managing bronchiectasis, with macrolides and corticosteroids being the most commonly employed classes. Macrolides, such as azithromycin, are unique in that they not only act as antibiotics but also possess immunomodulatory properties. They reduce mucus production, inhibit biofilm formation, and suppress the release of inflammatory mediators, thereby lowering exacerbation frequency and improving cough and dyspnea. However, long-term use must be carefully monitored due to risks of gastrointestinal intolerance, hepatotoxicity, and increased antimicrobial resistance. Inhaled corticosteroids may also help in reducing sputum production and airway hyperresponsiveness over time. Nevertheless, they are not recommended for routine use, especially in children, and their prolonged administration carries risks such as osteoporosis, cataracts, and systemic adverse effects.

Antimicrobial therapy: Antibiotics remain central to the treatment of bronchiectasis due to the frequent bacterial colonization of airways and recurrent exacerbations. They are used for eradication of pathogens such as *Pseudomonas aeruginosa* or MRSA, suppression of chronic bacterial colonization, and treatment of acute infective episodes. Oral antibiotics, particularly macrolides, are effective in reducing exacerbations, while inhaled antibiotics are increasingly used in patients with recurrent exacerbations and persistent *Pseudomonas* colonization. Options include aerosolized tobramycin, ciprofloxacin, aztreonam, and colistin. However, challenges such as achieving optimal drug concentrations in the infected lung tissue remain, and ongoing research into inhalable nanostructured lipid carriers may help overcome this limitation in the future.

Bronchodilators: They have a role in selected patients, particularly those demonstrating bronchodilator reversibility on spirometry. In these individuals, inhaled bronchodilators can improve symptoms of dyspnea and cough, enhance quality of life, and optimize lung function. However, current evidence does not support their

routine use in all bronchiectasis patients, and their administration should be individualized based on spirometric findings and clinical benefit.

Surgical intervention: The primary role of surgery in the management of bronchiectasis is in localized disease, where resection of the affected lung segments can help reduce the burden of infection and control complications such as massive hemoptysis. Surgery may also be indicated in cases of airway obstruction contributing to localized bronchiectasis that cannot be managed by other means. The goals of surgical intervention are conservative, aiming to control specific disease manifestations and improve quality of life rather than completely cure or eliminate all diseased lung tissue. Reported outcomes from surgical case series are favorable, with low operative mortality rates (less than 2%) and symptomatic improvement in the majority of carefully selected patients. For example, a study of 75 patients followed for a median of 15.3 months after resection found 84% of patients became asymptomatic and another 10.7% reported substantial improvement. Similarly, in a larger series of 277 patients, 68.5% were symptom-free 4.5 years after surgery. Despite these promising results, no randomized clinical trials have been conducted to formally evaluate the efficacy of surgical resection in bronchiectasis. Therefore, surgery remains a selective option reserved for patients with localized disease refractory to medical therapy or those suffering from recurrent or life-threatening complications. [11-13]

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