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Navneet Singh  
Dept. of Pulmonary Medicine  
PGIMER, Chd.

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Ritesh Agarwal  
Dept. of Pulmonary Medicine  
PGIMER, Chd.

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## BRONCHIECTASIS

### Introduction and Epidemiology

Bronchiectasis (BE) (ectasia meaning dilation and bronkos meaning of the airways) is defined as an abnormal, irreversible dilatation of the bronchi. Strictly speaking, it is not a disease per se but the end stage of a variety of pathologic processes. Since, the primary clinical manifestations of BE are recurrent, chronic and sometimes refractory infections, it is appropriate to discuss this entity along with other suppurative diseases of the lung.

### Classifications

#### Etiological

#### Postinfectious bronchial damage

Postinfectious bronchial damage

Bacterial

Staphylococcus aureus, Pseudomonas aeruginosa,  
Haemophilus influenza, Anaerobes

Mycobacteria

Mycobacterium tuberculosis

Atypical mycobacteria (Mycobacterium avium-complex)

Fungi

Aspergillus sp. [including allergic bronchopulmonary  
aspergillosis (ABPA)]

Histoplasma capsulatum

Coccidioides immitis

Viral

Measles

Pertussis

Adenovirus

Congenital

Ciliary dyskinesia

Immotile cilia syndrome

Kartagener's syndrome

Young's syndrome

Secondary ciliary dyskinesia

Cystic fibrosis (CF) and variants

Alpha-1 anti-trypsin deficiency

Swyer-James (MacLeod's) syndrome

Tracheobronchomegaly (Mounier-Kuhn syndrome)
Congenital cartilage deficiency (Williams-Campbell syndrome)
Pulmonary sequestration
Abnormal host defense
Immune deficiency
Primary
Hypogammaglobulinemia
Immunoglobulin G subclass deficiency
Complement deficiency
Secondary
Malignancy
Chemotherapy
Post-transplant
HIV infection
Mechanical bronchial obstruction
Foreign body
Tumor
Lymphadenopathy
Sequelae of aspiration or inhalation of toxic gases
Systemic diseases
Collagen vascular diseases
Rheumatoid Arthritis
Sjogren's syndrome
Ankylosing Spondylitis
Relapsing Polychondritis
Marfan's syndrome
Inflammatory bowel disease
Miscellaneous
Diffuse panbronchiolitis
Yellow nail syndrome

**Table 1: Etiological Classification of Bronchiectasis Pathological / Radiological**

Three main types are described. Tubular or cylindrical bronchiectasis is characterized by dilated bronchi only. Varicose bronchiectasis (named so in view of the similarity of its appearance with that of varicose veins) is characterized by presence of defects in the bronchial wall that lead to focal areas of constriction along the dilated bronchi. The third and most severe type is saccular (sacculae meaning grape like cluster) or cystic bronchiectasis results from progressive dilatation of bronchi that ultimately leads to the formation of large cysts.

## Extent

Bronchiectasis can be either focal or diffuse. Focal or localized disease (typically seen when bronchiectasis results from mechanical bronchial obstruction) is that which affects a lobe or a segment of a lung while diffuse disease (e.g. bronchiectasis occurring in ABPA, ciliary dyskinesias) is one that involves major portion of both lungs in 0.5%.

## Pathophysiology

Chronic inflammation involving the medium sized bronchi and bronchioles is the hallmark of bronchiectasis. This is evidenced by the findings (on histopathological examination of biopsy specimens) of recruitment of neutrophils into the lumina of affected airways as well as infiltration of the bronchial/bronchiolar wall by neutrophils, mononuclear cells and CD4+ T lymphocytes. Recruitment of neutrophils is mediated by proinflammatory cytokines [like interleukin-1 $\alpha$  (IL-1 $\beta$ ), interleukin-8 (IL-8), tumour necrosis factor-alpha (TNF- $\alpha$ ) and leukotriene B4 (LT B4)] while their interaction with the vascular endothelium is mediated by the up regulation of adhesion molecules. Activation of neutrophils (and the subsequent presence of neutrophil elastase in the airways) leads to damage to the respiratory mucosa as well as stimulation of mucous secreting glands in the airways. This is followed by a reduction in ciliary beating, impaired mucociliary clearance and ultimately retention of inflammatory secretions. A consequence of damage to the airways is that local immune response to inhaled pathogens becomes deficient and this results in colonization of the bronchial tree by bacteria like *Pseudomonas aeruginosa*. Infact, the two key pathophysiological processes in bronchiectasis – recurrent/chronic infections and airway obstruction – lead to a vicious cycle that induces and perpetuates the inflammatory process (see Figure 1). The inflammatory response mounted by the host in an attempt to eliminate the abnormal microbial flora is often unable to eliminate the microorganisms and the latter's persistence in the airways is associated with a state of chronic inflammation that leads to secondary bronchial damage which further compromises the local defence mechanisms and enhances susceptibility to bacterial invasion.

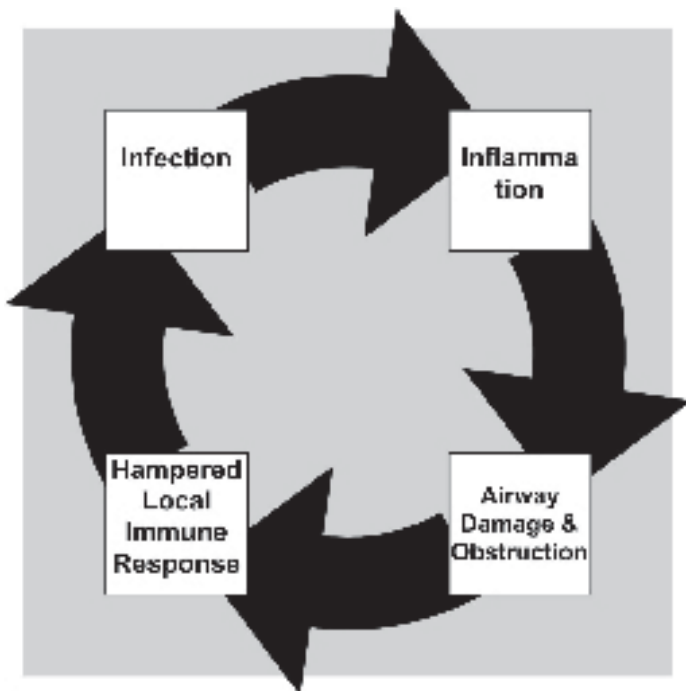


Figure 1: Pathophysiological in bronchiectasis

## Clinical Features

The characteristic symptoms of bronchiectasis are chronic cough with expectoration, the sputum being typically copious, thick and purulent. However, the sputum may also be mucoid, mucopurulent or tenacious. This symptom is present in majority (as high as 95%) of the patients – in fact, the quantity of sputum correlates with the quality of life.

In a recent review that summarized the clinical features of 670 patients, the mean age of presentation was 56 years with a predominance of females (female to male ratio being approximately 2:1). Associated rhino-sinusitis was present in two-thirds of cases and ranged in severity from a mild postnasal drip to fulminant pansinusitis. Dyspnea and chest pain were present in 75% and 30% of patients respectively. Hemoptysis, seen in 30% of patients, ranged from mild blood streaking of sputum to massive hemoptysis. The most common findings on physical examination were crackles (78%) and wheezing (20%). Digital clubbing, a frequent finding in the past, was seen in less than 5 percent of patients.

## Investigations

### 1. Radiology

#### Chest Radiograph

Chest radiography is a relatively insensitive tool for diagnosing bronchiectasis although symptomatic patients are more likely to have abnormal chest radiographs. Moreover, the findings may be nonspecific and include atelectasis (linear, plate-like, segmental or even lobar), focal area of consolidation, increased lung markings and scattered irregular opacities. More specific findings include ring-like shadows (airways parallel to the x-ray beam), tram lines (airways perpendicular to the x-ray beam) [both indicating dilated and thickened airways] and cystic lesions (saccular bronchiectasis).

#### HRCT Thorax

High Resolution Computed Tomography (HRCT) scanning is the investigation of choice for diagnosing bronchiectasis with the characteristic signs being listed in Table 2. This is usually done as a non-contrast study using thin sections (1.0 to 1.5 mm window every 10 mm) and a fast scan time (image acquisition time typically of 1 second or less) during full inspiration to reduce artifacts induced by respiration and cardiac pulsation.

#### Major Signs

- Bronchial dilatation (abnormal widening of bronchi)
  - Horizontal bronchi – internal diameter of bronchus greater than diameter of its adjacent pulmonary artery (typically 1.5 times or more) – **Signet Ring Sign**
  - Vertical bronchi – Non-tapering or flaring of bronchi towards the periphery – Tramlines
- Presence of bronchi in the outer lung fields (within 1–2 cm of pleura)

#### Minor Signs

- Bronchial wall thickening
- Mucus impaction in dilated bronchi (with/without plugging of centrilobular bronchioles)
- Crowding of bronchi (volume loss of affected lobe)
- Areas of reduced attenuation (mosaic pattern)

Table 2: Diagnostic Signs of Bronchiectasis on HRCT Scans

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Varicose constrictions of bronchi giving a beaded appearance and cysts (often with air-fluid levels) at the end of dilated bronchi are some other HRCT findings that help to diagnose bronchiectasis.

## 2. Spirometry

Spirometry with bronchodilator reversibility testing and flow–volume loops should be done in all patients with suspected or proven bronchiectasis. Spirometry often shows an obstructive pattern (i.e. a reduced ratio of forced expiratory volume in one second ( $FEV_1$ ) to forced vital capacity (FVC) – typically less than 70%). Pooled data from different studies shows that the  $FEV_1$  and FVC values are approximately 64% and 78% of predicted respectively. Significantly, almost one third of patients also demonstrate presence of bronchodilator reversibility (defined as an improvement in either  $FEV_1$  or FVC values by more than 12% (relative) and 200 ml (absolute) after 200 µg of inhaled salbutamol. Presence of a restrictive defect ( $FEV_1$ :FVC ratio  $\geq$  70% along with a reduction in FVC  $<$  80% of predicted values) indicates presence of significant atelectasis (due to mucus plugging), fibrosis (scarring resulting from chronic inflammation) or consolidation (commonly due to infection).

## 3. Sputum analysis

Patients with stable bronchiectasis commonly (usually 60-80%) have colonization with microorganisms in their airways. Onset of bronchiectasis at an early age, radiological presence of varicose and cystic lesions and  $FEV_1$   $<$ 80% predicted are proven risk factors for colonization. Haemophilus influenzae and Pseudomonas aeruginosa are the two most commonly isolated microorganisms from the sputum of patients with bronchiectasis (with the former being nearly twice as common as the latter). The frequency as well as type of microorganism isolated may, however, vary significantly between different geographical regions. Even the two commonly listed bacteria above have many different strains and subtypes, more than one of which may often be isolated from the sputum of a single patient. It is important to understand that 30–40% of sputum samples will show no growth and this may reflect suboptimal specimen collection, prior/current use of antimicrobial drugs or presence of microorganisms [Nocardia sp., atypical mycobacteria like Mycobacterium avium-intracellulare (MAC) and

fungi like Aspergillus sp.] which do not grow readily on conventional bacterial culture media. The frequency of isolation of the microorganisms probably correlates with the frequency with which they cause periodic acute exacerbations thus implying that these microorganisms are not merely colonizers but also potentially pathogenic. Patients who have colonization with Pseudomonas aeruginosa tend to have more severe disease (both in terms of structural involvement as well as functional impairment).

## 4. Specific tests directed at ascertaining etiology

These are listed in Table 3 although a detailed discussion about them is beyond the scope of this chapter.

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- Paranasal sinus disease (X ray or CT paranasal sinuses)
  - Gastro-esophageal Reflux (barium swallow, 24 hour esophageal pH monitoring, esophageal manometry)
  - Immune Deficiency [Total levels of IgG (and subclasses), IgM, IgE and IgA]
  - ABPA [Aspergillus skin test (detection of type 1 and/or 3 hypersensitivity reactions against Aspergillus antigens), serum precipitins against Aspergillus fumigatus and sputum for Aspergillus fumigatus]
  - Connective Tissue Disorders (antinuclear antibodies and rheumatoid factor)
  - $\alpha$ 1-antitrypsin Deficiency (serum  $\alpha$ 1-antitrypsin levels)
  - Cystic Fibrosis (sweat chloride analysis, nasal potential difference, genotyping)
  - Endobronchial abnormalities (Fibre-optic bronchoscopy)
  - Ciliary Dyskinesias (semen analysis, nasal mucociliary clearance – saccharine test, respiratory cilia sampling followed by microscopy (light and electron microscopy)
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**Table 3: Tests for detecting specific etiologies of bronchiectasis**

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## Treatment

### Antibiotics

In general, most infective exacerbations are caused by bacteria that are also common etiological agents for acute exacerbations of COPD namely *Haemophilus influenzae*, *Streptococcus pneumoniae* and *Moraxella catarrhalis* and thus can be treated by conventional  $\beta$ -lactam antibiotics (penicillins and cephalosporins). These should remain the first-line therapy unless  $\beta$ -lactamase production is suspected or proven. Patients with *Pseudomonas aeruginosa* infection may need to be treated differently (including use of parenteral anti-pseudomonal drugs). Antimicrobial therapy should be guided by sputum cultures which are preferably taken before initiation of treatment since patients may have colonization with multiple pathogens including intracellular bacteria. The latter respond poorly to  $\beta$ -lactam antibiotics and hence patients with severe structural or functional impairment should be treated empirically by a combination of a  $\beta$ -lactam antibiotic with an agent from another class (tetracycline, macrolide or quinolone) till the causative organism(s) is identified.

Patients with bronchiectasis may benefit from the use of macrolides (especially azithromycin) and this is related to the potential immunomodulatory properties possessed by this class of drugs.

Itraconazole may be useful in treating cases of ABPA that have not responded appropriately to corticosteroid therapy (not responded initially or relapsed after/during tapering of corticosteroid therapy).

Antibiotic therapy in bronchiectasis is a complex issue and has been discussed in detail in the section CURRENT UNCERTAINTIES AND AREAS OF FUTURE RESEARCH (vide infra).

### Bronchopulmonary Hygiene

Bronchopulmonary hygiene consists of pharmacological and non-pharmacological measures to assist the patient in removal of secretions from his/her respiratory tract.

### Physiotherapy

This involves techniques that utilize physical therapy to clear excessive secretions that are not amenable to removal by normal mucociliary clearance

mechanisms. It includes postural drainage, chest percussion, forced exhalation and controlled cough. Mechanical device that cause chest vibration are being increasingly used in developed countries. Although lung functions may not be affected much, symptoms and quality of life (QOL) tend to improve.

### Mucolytics

These are not recommended for routine use in non-CF bronchiectasis. Nebulized saline, human recombinant DNase and acetyl cysteine all have been shown to have beneficial only in patients with CF.

### Surgery

Localized bronchiectasis that is associated with intractable symptoms despite optimal medical management may benefit from surgical resection of the affected segment or lobe especially bronchiectasis resulting from mechanical obstruction.

### Bronchodilators

Data on use of inhaled drugs is sparse. Inhaled corticosteroids (ICS), when used in high doses, have been shown (in small randomized trials) to have beneficial effects on symptom related parameters (sputum volume, sputum purulence, cough, dyspnoea) and thus QOL but without any changes in pulmonary function, number or severity of exacerbations, or microbiological profile of sputum. The benefit may be more marked in those with *Pseudomonas aeruginosa* infection. Therefore, at present their use may be restricted to patients showing significant reversibility of airway obstruction. There is little data on the usefulness of other inhaled bronchodilators (short and long acting  $\beta_2$  agonists, anti-cholinergics). Similarly, except in ABPA, oral corticosteroids have no proven role in bronchiectasis.

### Complications

Important complications can be of infectious [recurrent pneumonia (typically in the same anatomical location if the disease is localized), empyema and lung abscess] or non infectious etiologies [pneumothorax, cor pulmonale and respiratory failure]. Hence, patients with severe and/or long standing disease may need to be investigated for the presence of the latter [ECG (with/without echocardiography) and arterial blood gas analysis respectively].

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## Current uncertainties and areas of future research

### Antibiotic Therapy

Controversies exist regarding the optimal dose, route and duration of antibiotic therapy.

#### Dose

Proponents for use of a higher (than usual) dose of antibiotics in bronchiectasis argue that the penetration of antibiotics into the bronchial mucosa and secretions is suboptimal and thus, micro-organisms that are shielded by them (formation of biofilms) do not get eradicated.

#### Duration

In general, it is prudent to use antibiotics only during an acute infective exacerbation (generally for a period of 10-14 days). Hence, if clinical worsening is not associated with symptoms suggestive of an infective etiology (increase in sputum quantity or purulence or fever), it is preferable to avoid using antibiotics. Even during stable disease, antibiotics have been used regularly (continuously or on a monthly basis) in an attempt to control inflammation (related to chronic bacterial colonization) and thus improve symptoms, reduce microbial load and improve lung function. However, this has the potential drawback of promoting drug resistance and till conclusive evidence is available to support its role, it cannot be routinely advocated for management of bronchiectasis.

#### Route

Nebulized antibiotics have shown promise when used in patients with CF who are colonized by potentially pathogenic micro-organisms like *Pseudomonas aeruginosa* although recent studies have reported similar benefits even among patients with non-CF bronchiectasis. Aminoglycosides (especially tobramycin) are the drugs that have been most commonly used through the inhalational route. Advantages of this route include preferential delivery of the drug to the airways (site of inflammation) with reduced systemic effects.

#### Indian Scenario

Systematically analyzed data on bronchiectasis from India is lacking. The largest series of allergic bronchopulmonary aspergillosis (ABPA) patients in world literature was however, recently published from the author's centre. 27.2% of 564 asthmatics presenting to the chest clinic over a 2 year period were diagnosed to have ABPA. At the time of presentation to the authors' institute, the vast majority of patients had developed bronchiectasis while almost half (46.8%) had received

prior antitubercular therapy (misdiagnosed as pulmonary tuberculosis). High-attenuation mucous impaction, a characteristic (although not pathognomonic) finding for ABPA was seen in one-sixth of patients. There was no significant difference between the stages of ABPA and the duration of illness, the severity of asthma, and the serologic findings (absolute eosinophil count and IgE levels). All patients were treated with oral prednisolone for a total duration of 6 to 12 months of which 86.5% experienced complete remission. This study has reinforced the need for screening (with an *Aspergillus* skin test) asthmatic patients (especially those with poorly controlled asthma, history of hemoptysis or infiltrates on chest radiograph) for the presence of ABPA since the latter remains an under-recognized disease entity in India.

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**Dr. Navneet Singh M.D., D.M**

Research Officer

Department of Pulmonary Medicine

Postgraduate Institute of Medical Education and

Research Sector-12

Chandigarh-160012. (India)

Email; navneetchd@yahoo.com

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## JOURNAL CLUB CRITIQUE

The Canadian Critical Care Trials Group. A randomized trial of diagnostic techniques for ventilator-associated pneumonia. *N Engl J Med* 2006; 355: 2619-30

**Background:** Critically ill patients who require mechanical ventilation are at risk for ventilator-associated pneumonia (VAP). Current data are conflicting as to the optimal diagnostic approach in patients who have suspected VAP.

**Methods:** In a multicenter trial, we randomly assigned immunocompetent adults who were receiving mechanical ventilation and who had suspected VAP after 4 days in the intensive care unit (ICU) to undergo either bronchoalveolar lavage (BAL) with quantitative culture of the bronchoalveolar-lavage fluid (BALF) or endotracheal aspiration (ETA) with non-quantitative culture of the aspirate. Patients known to be colonized or infected with *Pseudomonas* species or methicillin-resistant *Staphylococcus aureus* were excluded. Empirical antibiotic therapy was initiated in all patients until culture results were available, at which point a protocol of targeted therapy was used for discontinuing or reducing the dose or number of antibiotics, or for resuming antibiotic therapy to treat a pre-enrollment condition if the culture was negative.

**Results:** We enrolled 740 patients in 28 ICUs in Canada and the United States. There was no significant difference in the primary outcome (28-day mortality rate) between the BAL group and the ETA group (18.9% and 18.4%, respectively;  $P = 0.94$ ). The BAL group and the ETA group also had similar rates of targeted therapy (74.2% and 74.6%, respectively;  $P = 0.90$ ), days alive without antibiotics ( $10.4 \pm 7.5$  and  $10.6 \pm 7.9$ ,  $P = 0.86$ ), and maximum organ-dysfunction scores (mean [ $\pm$ SD],  $8.3 \pm 3.6$  and  $8.6 \pm 4.0$ ;  $P = 0.26$ ). The two groups did not differ significantly in the length of stay in the ICU or hospital.

**Conclusions:** Two diagnostic strategies for VAP - BAL with quantitative culture of the BAL fluid and ETA with non-quantitative culture of the aspirate - are associated with similar clinical outcomes and similar overall use of antibiotics.

### Main Findings of the study

The use of BAL with quantitative culture of the BALF, and ETA with non-quantitative culture of the

aspirate were associated with similar clinical outcomes (maximum organ-dysfunction scores, length of stay in the ICU or hospital and 28-day mortality) and similar overall use of antibiotics.

### Limitations of the study

The study had numerous exclusion criteria which included patients who were immunocompromised; considered unsuitable for bronchoscopy; infected or colonized with *Pseudomonas* species or methicillin-resistant *Staphylococcus aureus*; expected to die within 72 hours after enrollment; unlikely to leave the ICU within 3 weeks. Unfortunately, these subgroups probably represent the majority of patients who require ICU care or investigation for VAP.

The main purpose of invasive investigations in VAP is to allow de-escalation of unnecessary antimicrobial therapy on the basis of microbiologic findings. The exclusion of patients with MRSA, *P. aeruginosa*, and other multi-drug resistant pathogens diminishes the usefulness of the results of this study for clinical decision making. There is less concern about administering inappropriate initial antimicrobial therapy when the risk of infection with resistant pathogens is low, thus allowing for the initial use of more narrow-spectrum antimicrobial agents. Also, the culture of bronchoalveolar-lavage fluid is more likely to result in modification of prescribed broad-spectrum regimens than is the culture of an endotracheal aspirate. In fact, a recent meta-analysis comparing invasive diagnostic techniques versus the clinical criteria and quantitative culture of endotracheal aspirate found that the likelihood of changing the antibiotics was almost three times in the invasive group.

### Implications for day-to-day practice

In patient populations with a low prevalence of infection or colonization with antibiotic-resistant bacteria, the use of endotracheal aspiration may suffice, since initial empirical treatment with broad-spectrum antimicrobial agents is not required. However the results of this study are not valid for ICUs with high rates of infection with multi-drug resistant pathogens.

Another situation is the use of surveillance cultures of endotracheal aspirates in all intubated patients as a means of assisting in the choice of antibiotic therapies when the presence of VAP is subsequently suspected.

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**Further Reading**

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**Dr Ritesh Agarwal**

Assistant Professor

Department of Pulmonary Medicine

Postgraduate Institute of Medical Education and Research

Sector-12, Chandigarh-160012. (India)

E-mail: riteshpgi@gmail.com

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Phone: (0172) 2756823, 2756821

Fax: (0172) 2745959, 2744401

E-mail: dheeraj@indiachest.org

Websites: www.napcon2007.com, www.indiachest.org