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Pathology and Pathophysiology of Bronchiectasis

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Opinion Article

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ABOUT THE STUDY

Bronchiectasis is a chronic infective and inflammatory disease of the tracheobronchial tree and affected patients suffer from recurrent sputum production, hemoptysis and exacerbations. The pathogenesis of bronchiectasis is poorly understood. Although many known causes of bronchiectasis have been identified between 60%-80% of cases are regarded as idiopathic. Despite the disappearance of the original causative assault to the respiratory tract such as pertussis these patients continue to produce significant amounts of sputum which indicates an underlying active tracheobronchial inflammation. However, studies in the last two decades have identified infective, inflammatory and enzymatic elements which are distinct and yet inter related pathogenic components in the pathogenesis of bronchiectasis. All these symptoms act together to perpetuate continued airway damage and clinical deterioration in bronchiectasis.

While most clinicians recognize the presence of the infective component in the pathogenesis of bronchiectasis many do not appreciate the need and also address the inflammatory and enzymatic activities. Our recent studies have strongly suggested that the latter two pathogenic components also play very important role in the pathogenesis of bronchiectasis.

The most frequent isolated respiratory pathogen in early bronchiectasis is *Haemophilus influenza*. However, more severely affected patients such as those with copius sputum production and very impaired spirometry. The predominant pathogen is *Pseudomonas aeruginosa*. It is a versatile bacterium, which is virtually impossible to eradicate despite intensive antibiotic therapy. *Pseudomonas aeruginosa* infection causes considerable morbidity and leads to recurrent exacerbations among patients with bronchiectasis among patients with bronchiectasis. Both of these pathogens produce exotoxins which are harmful to the respiratory mucosa and perturn mucociliary clearance. Recent studies on the immunopathology of bronchial mucosa in bronchiectasis have revealed an increase inactivated $CD8^+$ T-lymphocytes neutrophils and macrophages in the epithelial, lamina propria and submucosal layers. This suggests an important role for cell mediated immune response in the progressive airway destruction in bronchiectasis. Intense neutrophil infiltration into the tracheobronchial tree occurs in bronchiectasis which also aggravates the underlying tracheobronchial damage. Neutrophil derived toxic products such as elastase, cause ultrastructural and functional damage and release of pro-inflammatory mediators in the tracheobronchial tree. These elastase and matrix metallo proteinases are likely to further damage the airways by direct lytic actions on collagen and other connective tissue components and provoke more inflammatory activities thus perpetuating this vicious circle of events. There is ample evidence to suggest that this neutrophil influx into the bronchiectatic airways is mediated by pro-inflammatory mediators. For instance leukotriene B4 promotes neutrophil migration and degranulation interleukin-1 beta mediates airway inflammation and fibrosis. Tumour necrosis factor mediates elastolytic degradation of lung proteoglycans and interacts synergistically with interleukin-1 in prostaglandin infection and interleukin-8 is one of the most potent chemoattractants which also degranulates neutrophils in bronchiectatic airways.

Neutrophil mediated degradation of bronchial matrix is an important pathogenetic factor in disease progression in bronchiectasis and patients with bronchiectasis ultimately die from progressive lung function impairment and respiratory failure. Neutrophil elastase in the airways would not only degrade lung elastin and collagen, but also proteoglycans which normally maintain the integrity of the lung extracellular matrix. Recent work also demonstrates that neutrophil elastase in bronchiectatic sputum may complex with heparin sulphate/syndecan, proteoglycans present in airway secretions and this could compromise the inhibitory efficiency of prevailing anti-elastases.