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Bronchiectasis: Treatment of Breathing Difficulties

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Abstract

Bronchiectasis is a lung condition that causes cough, sputum production, and recurrent respiratory infections. Because bronchiectasis is a condition that develops over many years and worsens with repeated infections, the main treatment goal is to reduce stagnant secretions in the airways and germs contained in those secretions.

Keywords: COPD; Lung function; Resource; Glucocorticoids; Cross-study interpretation; Reported outcomes

Introduction

Bronchiectasis is a device that contain a substance that enlarge bronchi and bronchioles, decreasing resistance in the respiratory airway and increasing airflow to the lungs to make breathing easier. Bronchiectasis may be originated naturally within the body or they may be used for the treatment of breathing difficulties. They are useful in obstructive lung disease such as asthma and in chronic obstructive pulmonary disease. The impact on healthcare systems is substantial [1]. A recent multicentre European study of patients with bronchiectasis identified an annual exacerbation frequency per patient per year, with a hospitalisation rate over few years follow-up. Bronchiectasis has a clear attributable mortality. In the largest cohort study reported to date, half of the patients died from respiratory causes, with around one-quarter dying from cardiovascular diseases. Loebinger provided long-term data on mortality by following up a cohort of patients first recruited for the validation of the St. Georges Respiratory Questionnaire. These patients were followed up for years. In a prospective cohort analysis of patients in secondary care in Belgium, Goeminne found that deaths were respiratory related and remaining were cardiovascular. Therefore, it is clear, at least in secondary care bronchiectasis cohorts, that patients experience a high rate of exacerbations, hospital admissions and attributable mortality, emphasising the need for high-quality specialised care for these patients. The pathophysiology of bronchiectasis and the goals of treatment our understanding of the pathophysiology of bronchiectasis is limited, in part because of the lack of representative experimental models. Airway inflammation in bronchiectasis is dominated by neutrophils, driven by high concentrations of neutrophil chemo-attractants such as interleukin and leukotriene [2]. Airway bacterial colonisation occurs because of impaired mucociliary clearance and because of failure of neutrophil opsonophagocytic killing. Since neutrophils from bronchiectasis patients are believed to be normal prior to their arrival in the airway, it is likely that the airway inflammatory milieu itself impairs bacterial clearance. Work over several decades has implicated neutrophil elastase in this process. The effects of elastase on airway epithelial cells includes slowing of ciliary beat frequency and promotion of mucus hypersecretion while impairment of opsonophagocytosis occurs at multiple levels, through cleavage of opsonins from the bacterial surface and cleavage of the neutrophil surface receptors FcγRIIIb and CD. Alpha defensins released from neutrophil granules also suppress phagocytic responses. Other mechanisms of immune dysfunction include failure of clearance of apoptotic cells and T cell infiltration, with recent evidence pointing to an important role of Th cells.

Discussion

Nevertheless, much more work is needed to unravel the complexities of the host response in bronchiectasis. Significant recent advances in our understanding of bronchiectasis have arisen through rRNA sequencing technologies which allow a comprehensive analysis of polymicrobial bacterial communities in the lung. Such technologies have clearly disproven the previous teaching that the healthy airway is sterile [3]. Studies in bronchiectasis reveal colonisation with familiar pathogens such as Haemophilus sp., Pseudomonas aeruginosa and Moraxella sp., but also organisms previously not recognised by culture-based studies like Veilonella. Clinical translation to date suggests that loss of diversity, with dominance of one or a few species, is associated with worse lung function and more exacerbations, and that loss of diversity may occur during exacerbations. Overall these studies are consistent with data from culture based studies, with Pseudomonas aeruginosa dominance being associated with worse lung function and more exacerbations whether by molecular- or culture-based means and high bacterial loads of classical bronchiectasis pathogens being associated with higher neutrophilic inflammation and more exacerbations. Bacteria have their own methods of evading airway clearance. An important recent study identified that P. aeruginosa can induce the formation of O-antigen specific immunoglobulin antibodies which then protect the bacteria from complement-mediated killing. A significant proportion of patients with severe bronchiectasis and P. aeruginosa colonisation had these antibodies and they correlated with worse lung function and disease severity [4]. Successful stabilisation of a patient with plasma exchange demonstrated the potential of this finding to change clinical practice. Since such responses are not necessarily unique to P. aeruginosa, this finding could have even broader implications, and requires further study. Additional defects in the complement system, particularly mannose-binding lectin deficiency have now been associated with more severe bronchiectasis in CF, common variable immunodeficiency, primary ciliary dyskinesia and in a general population of patients with bronchiectasis. Despite these advances, the pathophysiology of bronchiectasis is still best understood in terms of the vicious cycle hypothesis. Since progression of the disease is linked to failed mucus clearance, airway bacterial colonisation, airway inflammation and airway structural damage, the goals of therapy should be to halt or reverse these processes and thereby

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break the cycle. As with other respiratory diseases, patients with bronchiectasis should be encouraged to stop smoking. Vaccination against influenza and pneumococcal disease is also recommended as for other chronic respiratory disorders although there are no specific data in bronchiectasis about its impact. Bronchiectasis represents the final common pathway of a number of diseases, many of which require specific treatment. Host-infectious bronchiectasis is often used as a diagnostic label for patients with a history of severe or childhood respiratory infections, affecting patients. There is little evidence so far that they represent a distinct phenotype from idiopathic bronchiectasis and some cases may represent recall bias [5]. Less data on aetiology is available outside the UK, but data from Italy and Belgium suggested a spectrum similar to the UK with perhaps fewer patients with allergic broncho-pulmonary aspergillosis and more with chronic obstructive pulmonary disease. Data from the USA clearly demonstrate more bronchiectasis due to non-tuberculous Mycobacteria in some centres, and a report by patients identified aetiology in few of cases. The BTS guidelines recommend testing for underlying causes including measurement of immunoglobulin, testing to exclude ABPA and specific antibody responses to pneumococcal and Haemophilia vaccination. Sputum culture to exclude NTM and measurement of autoantibodies are also suggested. Testing for CF is recommended for patients with recurrent P. aeruginosa and Staphylococcus aureus isolation, or upper lobe predominant disease irrespective of age. Additional testing is recommended in specific circumstances. COPD appears to be a very common aetiology, with bronchiectasis reported in up to patients with moderate-to-severe COPD. Bronchiectasis also appears relatively common in patients meeting the diagnostic criteria for asthma. Focal bronchiectasis may be associated with bronchial obstruction. Gastro-oesophageal reflux frequently co-exists with bronchiectasis and has been suggested as an aetiological factor in some cases. Immunoglobulin replacement, steroids and anti-fungal for ABPA, treatment for NTM and of CF all represent opportunities to specifically treat the underlying cause and so systematic testing of all patients is recommended in consensus guidelines [6]. Bronchiectasis not due to cystic fibrosis is characterised radiological by permanent dilation of the bronchi, and clinically by a syndrome of cough, sputum production and recurrent respiratory infections. Having been previous regarded as a neglected orphan disease, recent years have seen renewed interest in the disease, resulting in more clinical research and the development of new treatments [7]. The purpose of this article is to provide a state-of-the-art review on the rapidly developing field of bronchiectasis, focussing on existing and developing therapies. Formerly regarded as a rare disease, bronchiectasis is now increasingly recognised and a renewed interest in the condition is stimulating drug development and clinical research. Bronchiectasis represents the final common pathway of a number of infectious, genetic, autoimmune, developmental and allergic disorders and is highly heterogeneous in its aetiology, impact and prognosis [8]. The goals of therapy should be: to improve airway mucus clearance through physiotherapy with or without adjunctive therapies; to suppress, eradicate and prevent airway bacterial colonisation; to reduce airway inflammation; and to improve physical functioning and quality of life. Fortunately, an increasing body of evidence supports interventions in bronchiectasis. The field has benefited greatly from the introduction of evidence-based guidelines in some European countries and randomised controlled trials have now demonstrated the benefit of long-term macrolide therapy, with accumulating evidence for inhaled therapies, physiotherapy and pulmonary rehabilitation [9]. Medicines may be inhaled to help open the airways and loosen mucus. A bronchodilator such as albuterol or levalbuterol can help relieve or prevent spasm of the airway muscles. Hypertonic saline is a concentrated salt water solution that can help loosen secretions in your airways. Often inhaled medicines are used before or during airway clearance to help bring mucus up [10]. While bronchiectasis is a long-term condition, you may occasionally become more ill. This is called an acute exacerbation. Often this is due to a new respiratory infection or overgrowth of bacteria that are chronic. We can increase your airway clearance to help get the extra mucus up.

Conclusion

We may need antibiotics to treat the infection. Remember that repeated exacerbations can cause bronchiectasis to worsen over time. All forms of airway clearance depend on good coughs to move loose mucus out. We can learn techniques such as huffing to improve your cough strength and effort.

Acknowledgement

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Conflict of Interest

None

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