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Short Communication Open Access

Bronchiectasis: A Correct Diagnosis Needed for Proper Management

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Bronchiectasis is an airway disease caused by irreversible bronchial dilatation secondary to chronic bacterial infection and inflammation [1,2]. The pathogenic mechanism is an exaggerated and uncontrolled neutrophilic response in relation to bacterial load.

In about half of the cases of bronchiectasis, the etiology is unproven. However there are a number of causes that merit attention. Diffuse bronchiectasis may be due to post-infectious, immunodeficiency and congenital causes. Bacteria (pertussis), virus (adenovirus, measles) and fungi (Aspergillusfumigatus, Histoplasmacapsulatum) are post-infectious causes. Tuberculosis is an important etiology especially in developing countries. Cystic fibrosis and primary ciliary dyskinesia are two main congenital condition associated with bronchiectasis. Individuals with humoral deficiencies of IgG, IgM and IgG are at risk for recurrent sinusitis and bronchiectasis. Foreign body aspirations and benign tumours (fibroma, lipoma) are causes of focal bronchiectasis.

Bronchiectasis may affect a lobe, a segment or sub segment of a lung. It may be a diffuse process involving both lungs often in association with systemic disease such as cystic fibrosis (CF). Focal lesion may be due to poorly treated pneumonia and in developing countries it is often secondary to tuberculosis infection.

Inflamed airways, airway obstruction and impaired clearance of secretions lead to its clinical manifestations. The classic clinical manifestations of bronchiectasis are cough and daily mucopurulent sputum production. Blood-streaked sputum or haemoptysis may result from airway damage associated with acute infection. Less specific symptoms include dyspnoea, pleuritic chest pain, wheezing, fever, weakness, and weight loss. Since the lesion may only involve a segment or sub segment of a lung, some patients may be asymptomatic. These patients may produce sputum only if they have acute upper respiratory tract infection.

The diagnosis should be considered in children and adults with recurrent cough and respiratory symptoms suggestive of chest infection, particularly purulent sputum production. History of pneumonia in infancy or childhood is of particular important. Wheezing, shortness of breath and other manifestations of respiratory insufficiency may occur during exacerbation and mimics other respiratory conditions like asthma exacerbation and acute bronchitis. Other features include blood streak sputum, chest pain and poor nutritional status and failure to response to inhaled steroids and inhaled bronchodilators in suspected asthmatics.

Chest imaging is required to confirm the diagnosis. Specific findings on chest radiograph may include dilated bronchi or clustered cystic changes. However these may not be evident in all cases. HRCT is the current working gold standard for diagnosing bronchiectasis with sensitivity and specificity of 98% and 99% respectively [3,4].Immunoglobulin (IgG, IgM, IgA) quantitation should be performed because of therapeutic implications. If cystic fibrosis is suspected sweat chloride and genetic testing should be performed. Sputum should be collected and cultured at diagnosis to facilitate subsequent antibiotic choices. The most commonly found microorganism is Haemophilusinfluenzae (55%), followed by Pseudomonas species (26%) and Streptococcus pneumoniae (12%) [5].

Acute exacerbation of bronchiectasis occurs following bacterial infection. There will be an increased in sputum production from the baseline as well as change in sputum viscosity. Low grade fever may occur. Patients may have worsening of generalized constitutional symptoms such as fatigue and malaise. They may also have increased dyspnoea and wheezing.

It is very important to identify exacerbation of bronchiectasis because the symptoms mimic a few other respiratory diseases. Other differential diagnoses during acute exacerbations of bronchiectasis include acute exacerbation of bronchial asthma, acute exacerbation of COPD, acute bronchitis and community acquired pneumonia. Proper history, physical examination and chest radiograph are important to distinguish bronchiectasis from other diagnosis.

During infective exacerbations, antibiotics are effective in reducing sputum volume and purulence leading to clinical improvement [6]. The choice of antibiotics may be difficult because some patients may not have organism isolated in their sputum during exacerbations. Empirical use of antibiotics depends on knowledge of the likely causative organism most likely to be Haemophilusinflunzae or Psedomonasaeruginosa. Patients with less sputum and good lung function may require non-pseudomonal antibiotics such as third generationcephalosporins and B-Lactam with lactamase inhibitors. Severely affected patients will require pseudomonal antibiotics which include ceftazidime and levofloxacin.

Attention to bronchial hygiene is important due to viscous secretions. Physician may consider a few strategies which include systemic and airway hydration, mucolytic agent administration, chest physiotherapy and bronchodilator administration. Airway hydration may be achieved by oral fluid. However administration of hypertonic saline (7%) will reduce the viscosity of secretions. If saline nebulization is not effective, acetylcystine administered as a 20% solution by nebulizer may be tried.

If misdiagnosed, patients with bronchiectasis will be treated as 'difficult to treat asthma' or COPD.Those patients might be given high dose inhaled steroids and bronchodilators. They do not receive physiotherapy, mucolytics or appropriate antibiotics.

The objectives of good management in bronchiectasis are to reduce symptoms and exacerbations, improve quality of life and maintain

lung function and thereby prolonging survival [7]. General measures include good nutrition, no smoking strategy, good exercise and exposure to fresh air. Patients should undertake chest physiotherapy which would improve pulmonary clearance. Vaccination against pneumococcal infection is highly recommended in bronchiectasis [8]. Pneumococcal vaccine is efficacious in preventing complicated pneumococcal disease. Besides, influenza vaccination is also recommended [9].

Inhaled corticosteroids are useful for long term therapy particularly on patients with severe bronchiectasis. Use of inhaled fluticasone has been shown to improve 24-hour sputum volume, although it does not reduce the frequency of exacerbation and improvement in FEV1 or FVC. However the study did show bronchiectasis patients with Psedomonasaeruginosa infection would have improvement in sputum volume and exacerbation frequency [10].

Immunomodulatory effect of macrolide on the immune response on bronchiectasis patients has been of particular interest among researches. Low dose Erythromycin administered over 8 weeks significantly reduces sputum volume and improves lung function in stable bronchiectasis [11].The mechanism of action of macrolides in bronchiectasis is not precisely known although it is unlikely to be bactericidal in view of low dosage and poor penetration into tracheobronchial tree.

Anti-inflammatory properties of Azithromycin its and long-term use has been studied in patients with both CF and non-CF bronchiectasis. In non-CF patients, azithromycin has been shown to decrease exacerbations and there is an improvementin spirometric measurement [12]. In CF patients a meta-analysis suggests that it improves lung function, especially in those patients colonized with Pseudomonas [13].

Bronchodilators, including beta-agonists and anticholinergics, may help some patients with bronchiectasis. Bronchodilators may be useful in reversing bronchospasm in bronchiectasis patients associated with airway hyper responsiveness [14-16]. A large randomized clinical trial of bronchodilator treatment in bronchiectasis is needed to justify its use.

References

1. Martínez-García MA, Perpiñá-Tordera M, Román-Sánchez P, Soler-Cataluña JJ (2005) Quality-of-life determinants in patients with clinically stable bronchiectasis. Chest 128: 739-745.
2. King P, Holdsworth S, Freezer N, Holmes P (2006) Bronchiectasis. Intern Med J 36: 729-737.
3. Grenier P, Maurice F, Musset D, Menu Y, Nahum H (1986) Bronchiectasis: assessment by thin-section CT. Radiology 161: 95-99.
4. Giron J, Skaff F, Maubon A, Garnier T, Serres-Cousiné O, et al. (1988) [Millimetric-section x-ray computed tomography in the diagnosis and evaluation of bronchiectasis. Comparison with bronchography apropos of 54 patients]. Ann Radiol (Paris) 31: 25-33.
5. Angrill J, Agustí C, De Celis R, Filella X, Rañó A, et al. (2001) Bronchial inflammation and colonization in patients with clinically stable bronchiectasis. Am J Respir Crit Care Med 164: 1628-1632.
6. Evans DJ, Greenstone M (2003) Long-term antibiotics in the management of non-CF bronchiectasis--do they improve outcome? Respir Med 97: 851-858.
7. Pasteur MC, Bilton D, Hill AT; British Thoracic Society Bronchiectasis non-CF Guideline Group (2010) British Thoracic Society guideline for non-CF bronchiectasis. Thorax 65 Suppl 1: i1-58.
8. 1997, CDC. Prevention of Pneumococcal Disease.Recomendations of the Advisory Committee on Immunization Practices (ACIP). MMWR Morb. Mortal. Wkly Rep 46: 1-24.
9. World Health Organisation (2002) Influenza vaccines. Wkly Epidemiol Rec 77: 230-239.
10. Tsang KW, Tan KC, Ho PL, Ooi GC, Ho JC, et al. (2005) Inhaled fluticasone in bronchiectasis: a 12 month study. Thorax 60: 239-243.
11. Tsang KW, Ho PI, Chan KN, Ip MS, Lam WK, et al. (1999) A pilot study of low-dose erythromycin in bronchiectasis. Eur Respir J 13: 361-364.
12. Wong C, Jayaram L, Karalus N, Eaton T, Tong C, et al. (2012) Azithromycin for prevention of exacerbations in non-cystic fibrosis bronchiectasis (EMBRACE): a randomised, double-blind, placebo-controlled trial. Lancet 380: 660-667.
13. Florescu DF, Murphy PJ, Kalil AC (2009) Effects of prolonged use of azithromycin in patients with cystic fibrosis: a meta-analysis. Pulm Pharmacol Ther 22: 467-472.
14. Franco F, Sheikh A, Greenstone M (2003) Short acting beta-2 agonists for bronchiectasis. Cochrane Database Syst Rev : CD003572.
15. Sheikh A, Nolan D, Greenstone M (2001) Long-acting beta-2-agonists for bronchiectasis. Cochrane Database Syst Rev : CD002155.
16. Lasserson T, Holt K, Evans D, Greenstone M (2001) Anticholinergic therapy for bronchiectasis. Cochrane Database Syst Rev : CD002163.