

Gastro esophageal reflux disease with bronchiectasis and aspergillosis

Sriram P, Alok Gaurav, Arun GK, Suresh R, Prakash V

Department of Pediatrics, JIPMER, Puducherry, India

Abstract

Gastro esophageal reflux disease is one of the rare causes of bronchiectasis in children. However, to the best of knowledge its association with pulmonary aspergillosis has not been reported till date in the medical literature [1]. We report a child with gastro esophageal reflux disease with bronchiectasis and pulmonary aspergillosis.

Keywords: Gastro esophageal reflux disease, bronchiectasis, aspergillosis.

Accepted November 17 2009

Introduction

Gastro esophageal reflux disease (GERD) has a wide spectrum of clinical manifestations which includes both esophageal and extra esophageal symptoms. It may present with feeding difficulties, failure to thrive, recurrent vomiting, persistent cough, recurrent pneumonia, and reactive airway disease. It is a rare cause of non- cystic fibrosis bronchiectasis in children [2,3].

Bacterial infections with common respiratory organisms such as Staph aureus, H.influenza, Pneumococcus and Pseudomonas have been commonly reported. However its association with aspergillosis is very rare. We report a case of GERD with bronchiectasis with aspergillosis. Early recognition and institution of treatment will prevent progression of the disease.

Case report

A 6 month old female infant presented to our hospital with history of fever, cough, respiratory distress and poor feeding of fifteen days duration with past history of recurrent episodes of cough with rapid breathing on and off from second month of life. Child had been admitted thrice in the hospital for severe pneumonia during the preceding four months and the last admission was for a period of twenty days. She had been treated with several antibiotics, antitussives and bronchodilators during the last four months without any clinical improvement. The child was born to second gravida mother by spontaneous vaginal delivery with uneventful perinatal period, was exclusively on breast feeds and was developmentally normal. The first child was normal and there was no family history of asthma or allergy.

On examination child was active and afebrile. Weight of the child was 5kg and length was 60cm and appropriate for age. There was mild Pallor without cyanosis or lymphadenopathy. Respiratory rate was 50/minute with wheezing and on auscultation there were bilateral coarse crepitations with ronchi. On examination of abdomen liver was 4cm firm non tender, Spleen was 3cm and firm. The cardiovascular and neurological examination was normal.

Investigations revealed Haemoglobin 12.3gm%, Total leukocyte count 26900, DC P38 L59 E2 M1, Platelet 425000 and peripheral smear showing leucocytosis. Chest x ray showed bilateral non homogenous opacities. Blood culture was sterile; HIV and Mantoux test were negative. ECHO study showed Mild to moderate TR, Pulmonary arterial pressure 38 mm Hg with RA/RV mildly dilated, normal LV/RV function. Gastric aspirate for AFB was negative and serum electrolytes and liver function tests were normal. Sweat chloride test was normal. CT Thorax done showed cylindrical bronchiectasis in the right middle lobe with evidence of bilateral lung consolidation. Cultures from tracheal aspirate and throat swab grew *Aspergillus flavus* sensitive to amphotericin B. Barium swallow showed features of Gastro oesophageal reflux disease.

A diagnosis of Gastroesophageal reflux disease with bronchiectasis and aspergillosis was made in view of recurrent pneumonia unresponsive to prolonged antibiotic therapy. Pantoprazole, domperidone and amphotericin B were started and chest physiotherapy was periodically done. Child developed pallor with haemoglobin of 8 gm% and was given packed cell transfusion of 5ml/kg and was empirically put on ceftazidime to give coverage for pseudomonas. However the child's condition worsened and expired after twenty eight days of hospital stay.

Discussion

Gastro oesophageal reflux disease (GERD) is reported to be associated with several respiratory disorders including asthma, chronic cough, recurrent pneumonias and bronchiectasis [4,5]. Aspiration into the tracheobronchial tree can be silent clinically and present as insidious onset bronchiectasis, presumably via gastric acid induced chronic inflammation of the airways and there are very few published reports from the developing countries. Chronic aspiration either from the cricopharyngeal discordination or the gastroesophageal reflux is a well recognized condition that predisposes to recurrent pneumonias which can lead to bronchiectasis in children [6]. The diagnosis of Gastroesophageal reflux disease is based on Barium meal study, upper gastrointestinal endoscopy, oesophageal biopsy, gastroesophageal scintiscan and 24h ambulatory oesophageal pH monitoring [7].

Karakoc from Turkey described 23 children with bronchiectasis and found that factors other than infections have contributed to the condition such as gastroesophageal reflux, immunodeficiency and asthma [8]. Dawson and Bakalnova from Abu Dhabi region described 32 children with bronchiectasis from a population of 3,00,000[9]. He found out that congenital anomalies of the respiratory system, prematurity, gastro oesophageal reflux disease and immunodeficiency were some of the factors that contributed to the cause of the disease in addition to the viral and bacterial infections. In their report, bronchiectasis was found in one in four cases presented with persistent pneumonia and makes it a common association in that part of the world. Bacterial infections with common respiratory organisms were found in 51% of cases and the common organisms were *Staphylococcal aureus*, *Haemophilus influenza*, *Pneumococcus* and *Pseudomonas*. Gastroesophageal reflux and recurrent aspiration were found in 32% of the patients and may have contributed to the development of bronchiectasis or complicated its progression.

Pulmonary Aspergillosis presents in the form of allergic bronchopulmonary aspergillosis, recurrent wheezing and persistent pneumonia especially in association with prolonged antibiotic therapy [10]. In our case the child presented with recurrent respiratory tract infections with prolonged antibiotic therapy. Gastroesophageal reflux with bronchiectasis with pulmonary aspergillosis was made. However the child's condition worsened and expired after 4 weeks of hospital stay. In the era of antibiotics and vaccinations the role of gastro oesophageal reflex disease in causing recurrent pneumonias and bronchiectasis should not be underestimated. Any child with non resolving or recurrent pneumonias not responding to prolonged antibiotic therapy should be investigated for the gastro oeso-

phageal reflux disease as early diagnosis and timely management can be life saving in these children.

References

1. Orenstein SR, Orenstein DM. Gastro oesophageal reflux and respiratory disease in children. *J Pediatr* 1988; 112: 847-857.
2. Berhman RE, Kleigman R, Jenso HB, eds. *Nelson Textbook of paediatrics*. 18th ed. Philadelphia: W.B. Saunders, 2008: 1547-52.
3. Lewiston NJ. Bronchiectasis in childhood. *Pediatr Clin North Am* 1984; 31: 865-877.
4. Berquest WF, Rachelefsky GS, kadden M, Seigel SC, Katz RM, Fonkalsrud EW, et al. Gastroesophageal reflux associated recurrent pneumonia and chronic asthma in children. *Paediatrics* 1981; 68: 29-35.
5. Banjar H H. Association of bronchiectasis and gastroesophageal disease in children. *Curr Pediatr Res* 2006; 10(1&2):19-23.
6. Jain A, Patwari AK, Bajaj P, Kashyap R, Anand VK. Association of gastro oesophageal reflux disease in young children with persistent respiratory symptoms. *J Trop pediatr* 2002; 48: 39-42.
7. Sutphen JL. Paediatric gastro oesophageal reflux disease. *Gastroenterol Clin North* 1990; 19: 617-629.
8. Karakoc GB, Yilmaz M, Altintas DU, and Kendiri SG. Bronchiectasis: still a problem. *Pediatr pulmonol* 2001; 32: 175-178.
9. Dawson KP, Bakalnova D. Childhood bronchiectasis in a desert location. *Middle East paediatrics* 1996; 1: 6-8.
10. Stevens DA, Kan VL, Judson MA. Practice guidelines for diseases caused by *Aspergillus*. *Clin Infect Dis* 2000; 30: 697-709.

Correspondence:

P. Sriram
Department of Pediatrics,
JIPMER, Puducherry - 605006
India

