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Persistent pneumonia in an infant with achalasia cardia due to non-tuberculous mycobacterium

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(Key words: Non-tuberculous mycobacteria, Achalasia cardia, Pneumonia, Barium swallow, gastroesophageal reflux disease, Aerosolization, Cystic fibrosis)

Introduction

Non-tuberculous mycobacteria (NTM) are a large family of acid-fast bacteria, widespread in the environment. In children, NTM cause lymphadenitis, skin and soft tissue infections, and occasionally lung disease and disseminated infections[1]. These manifestations can be indistinguishable from tuberculosis (TB) on the basis of clinical and radiological findings and tuberculin skin testing. The immune response to both NTM and *Mycobacterium tuberculosis* is based on cellular immunity and relies on the type-1 cytokine pathway[2]. Treatment of NTM infections is different from the treatment of TB and depends on the strain and anatomical site of infection and often involves antibiotic combinations, surgery, or both. We present a 2-month-old infant with persistent fever and breathing difficulty from 1.5 months of age. Child was diagnosed through broncho-alveolar lavage (BAL) mycobacteria growth indicator tube (MGIT) culture to have NTM and treated with sensitive drugs. There are no reported cases of NTM in infants with oesophageal motility disorders, though common in adults.

Case report

A 2-month-old infant girl had fever and progressive breathing difficulty at 1.5 months of age. She was formula-fed and had failure to thrive (birth weight 3kg, admission weight 3.3kg). X-rays revealed persistent bilateral consolidation (Figure 1). High resolution computed tomography (HRCT) of the chest revealed air space opacification in the right upper and left lower lobes with a dilated thoracic oesophagus (Figure 2). Barium swallow confirmed delayed emptying and achalasia cardia (Figure 3). Botulinum was injected endoscopically for treatment of achalasia along with antibiotics for 21 days. Symptoms recurred within 20 days of discharge with fever and breathlessness needing re-admission. A bronchoscopy done revealed gastro-oesophageal reflux disease and BAL culture revealed multidrug resistant (MDR) *Klebsiella pneumoniae* which was treated with Inj. meropenem and Inj. cotrimoxazole as per sensitivity for 14 days. Though the child was weaned to oxygen support, she had re-appearance of fever and distress. TB culture (MGIT culture) revealed NTM; sensitive drugs rifampin, ethambutol and ofloxacin were given for 1 year, with close follow up and she is well on follow-up (current weight 8.3kg and length 71cm).

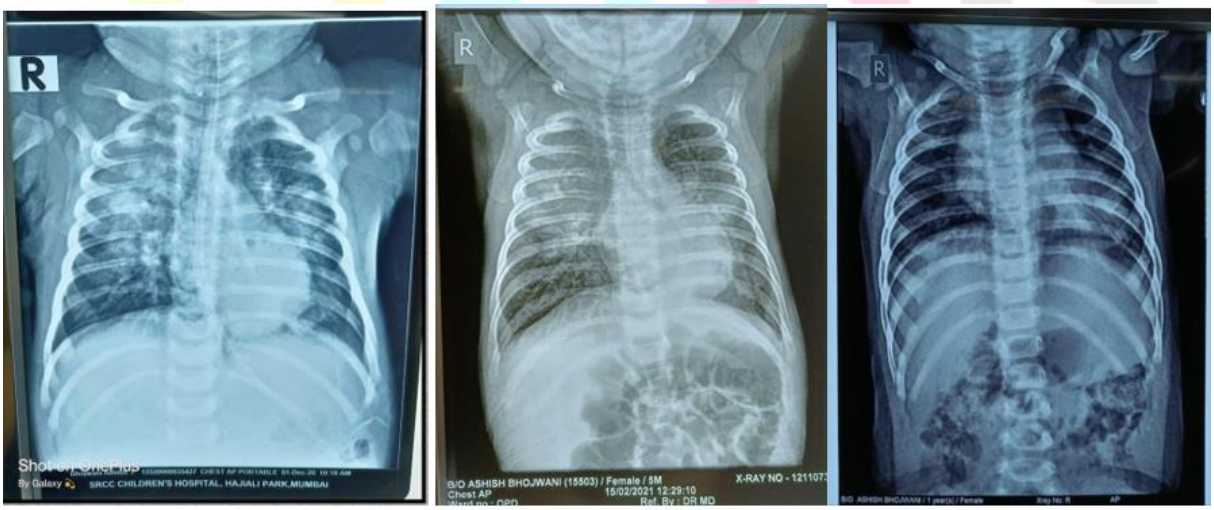

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Figure 1: ***Chest x-rays of patient***

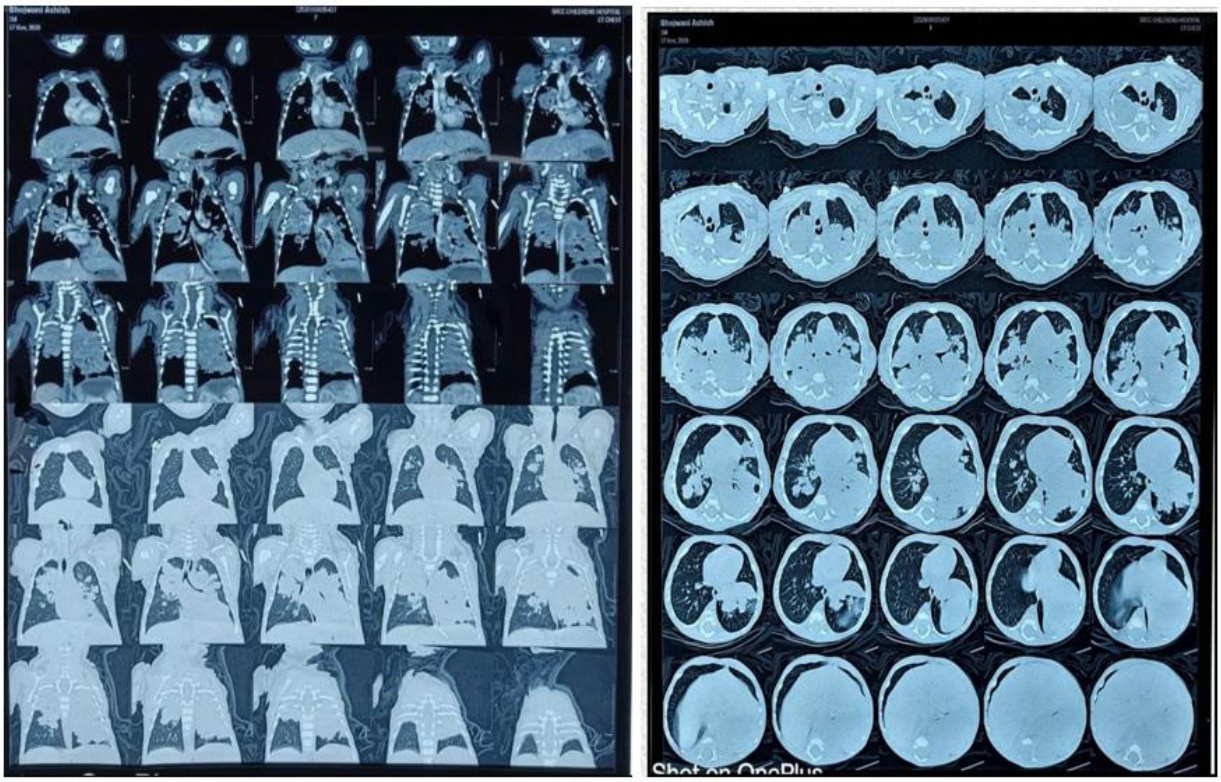

Figure 2: ***High resolution computed tomography of chest***

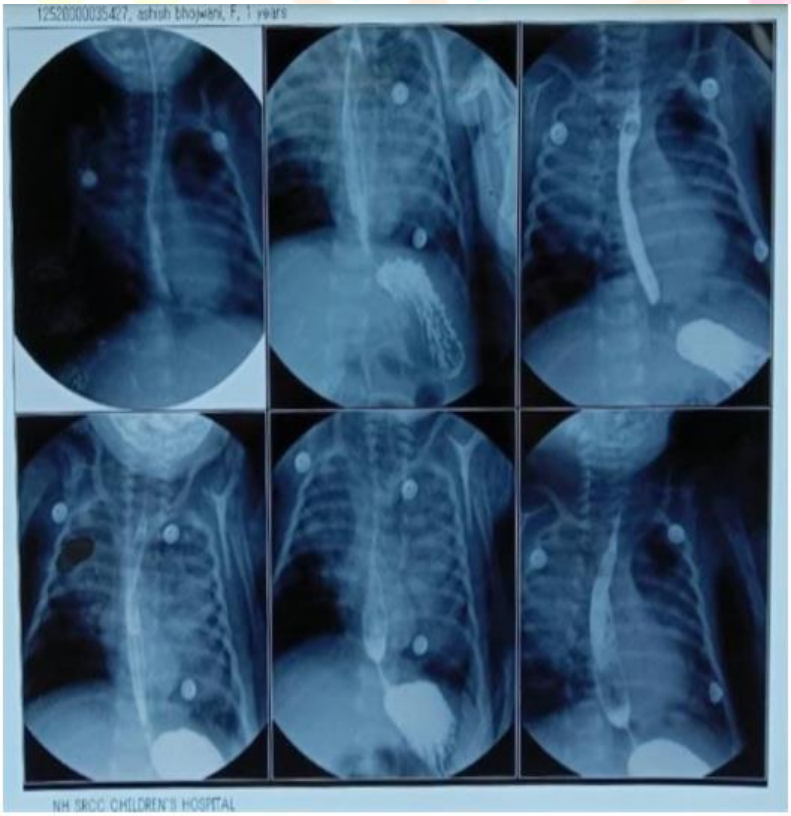

Figure 3: ***Barium swallow showing dilated oesophagus***

Discussion

Recurrent or persistent pneumonia pose a significant challenge to paediatricians. Persistent pneumonia implies a chronic, non-resolving pneumonia. It is defined as persistence of symptoms and radiographic abnormalities for more than 1 month[3]. Recurrent or chronic aspiration may be associated with anomalies of the upper airway, abnormal swallowing, gastro-oesophageal reflux or neuromuscular disorders[4]. Aspirations alter the pulmonary host defenses. Defects in clearance of airway secretions may be because of abnormalities of respiratory mucus as in cystic fibrosis or defects in mucociliary function - which may be due to structural defects of cilia or secondary to various infections. Compression of the airway (intrinsic/extrinsic) interferes with the clearance of airway secretions.

Pulmonary infections due to NTM are increasingly recognized worldwide. Although over 150 different species of NTM have been described, pulmonary infections are most commonly due to *Mycobacterium avium complex* (MAC), *Mycobacterium kansasii*, and *Mycobacterium abscessus[5]*. The identification of these organisms in pulmonary specimens does not always equate with active infection; supportive radiographic and clinical findings are needed to establish the diagnosis. It is difficult to eradicate NTM infections.

Nearly everyone is presumed exposed to NTM, yet most do not develop clinical signs of infection[5]. The factors predisposing to infection are not well understood, but likely are due to an interaction between host defense mechanisms and the load of clinical exposure. Although the exact route of NTM infection is not established with certainty, based on NTM environmental distribution, it is very likely that the organism is ingested, inhaled, or implanted. Cervical lymphadenitis due to NTM occurs more frequently in children, coincident at the time that they are exploring outdoors and there is trauma to gums due to erupting teeth[6]. Thus, it is assumed that NTM enter the tissues via the mouth. Aerosolization of droplets small enough to enter the alveoli is the likely route of acquisition of pulmonary disease. Bathroom showers have been implicated as a primary source of exposure to aerosolized NTM[7]. Unlike TB, the isolation of NTM in pulmonary specimens does not equate with disease.

In an effort to standardize the definition of NTM infection, the American Thoracic Society (ATS) and the Infectious Disease Society of America (IDSA) jointly published guidelines in 2007[8]. The diagnosis of NTM pulmonary infection requires the presence of symptoms, radiologic abnormalities, and microbiologic cultures in conjunction with the exclusion of other potential etiologies. Clinical symptoms vary in scope and intensity but chronic cough, often with purulent sputum, is common. Hemoptysis may also be present. Systemic symptoms including malaise, fatigue, and weight loss may occur often in association with advancing disease. Various radiographic patterns may be seen in patients with NTM pulmonary infections. Fibro-cavitary disease is commonly identified on chest roentgenograms[8]. Characteristic findings include thin-walled cavities with an upper lobe distribution and surrounding pleural abnormalities. There is no radiographic finding to reliably differentiate fibro-cavitary NTM from TB. NTM in conjunction with nodular bronchiectasis may be visible on chest radiograph, but is best appreciated on high resolution chest computed tomography (HRCT). Characteristic findings include clusters of small nodules usually less than 0.5 mm—the so-called tree-in-bud sign. Larger nodules, with or without cavitation, may occur, which are suspicious for malignancy. Uptake of 18F fluorodeoxyglucose (FDG) on PET scan has been described in nodules due to NTM[8]. Infected areas of lung parenchyma may demonstrate atelectasis or cystic or saccular bronchiectasis..

Treatment is with antibiotics based on the organism and sensitivity pattern. In advanced cases, surgery such as video-assisted thoracoscopic surgery (VATS) may be needed for pulmonary resection[9]. Monitoring patients for signs of drug toxicity is required during therapy. Attention to adequate nutritional intake and weight stability is important as gastrointestinal side effects, such as nausea and vomiting, are commonly seen in conjunction with the macrolide agents, rifampin, and rifabutin.

Conclusion

The incidence of NTM infections surpasses that of TB infections in developed countries. Although infection may occur in virtually any organ, pulmonary infections are most common. *M. avium*, *M. kansasii*, and *M. abscessus* are the most frequently identified organisms causing lung disease. The isolation of an NTM organism does not necessarily equate with active infection; clinical, radiologic, and microbiologic parameters are all needed to establish the diagnosis of infection. Eradication of disease with drug therapy requires prolonged combination therapy. Surgical resection is often indicated in localized disease, in the presence of drug resistant organisms, or in some cases, of failure of medical therapy.

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