



RESEARCH ARTICLE

A CASE OF CENTRAL TRACHEAL MASS DIAGNOSED AS ENDOBRONCHIAL TUBERCULOSIS

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ABSTRACT

Endobronchial tuberculosis is rare nowadays. Endobronchial tuberculosis (EBTB) is known to complicate pulmonary tuberculosis and can occur in the absence of parenchymal lesion, and is less recognised which can lead to difficulties in diagnosis. We report a 60-year-old woman who presented with symptoms of cough, shortness of breath and wheezing simulating bronchial asthma. A rare case of endobronchial tuberculosis presented as central tracheal mass in CT scan suspecting malignancy. Patient was sputum smear negative for acid fast bacilli. Diagnosis was possible only with fiberoptic bronchoscopy along with cartridge-based nucleic acid amplification test.

INTRODUCTION

Endobronchial tuberculosis (EBTB) was first of all described by Richard Morton in 1689. Endobronchial tuberculosis is a health problem which may be misdiagnosed as lung cancer. It is an infectious disease and definitive diagnosis depends on detection of mycobacterium from bronchoscopic material. Based on bronchoscopic assessment of endobronchial lesions, Chung and Lee (2000) classified EBTB into seven subtypes: actively caseating, edematous-hyperemic, fibrostenotic, tumorous, granular, ulcerative, and nonspecific bronchitic; tumorous being the less common type. We report a case of tumorous type of EBTB in an old female of 60 years who presented with an endobronchial growth at carinal region which initially simulating as a malignancy.

Case Report

A 60 yr. old married female (non-smoker, non-alcoholic) was admitted to our inpatient department with complaints of dry cough since 6 months, shortness of breath since 2 months and generalized weakness since 15 days. Cough was insidious in onset, gradually progressive and non-productive in nature with increased severity at night and early morning, no seasonal variation and not associated with haemoptysis.

Shortness of breath was insidious in onset, gradually progressive, initially while exertion then at rest since 5 days and subsided on taking rest. There was no seasonal and diurnal variation present. There was no history of fever, chest pain, loss of appetite and loss of weight. In 1994, patient was treated for obstructed jaundice and gall bladder was removed. In 2006, patient had history of facial nerve palsy which was recovered. In 2009, diabetes and hypertension was diagnosed for which she was on metformin 500mg BD and glibenclamide 5mg, and telmisartan and amlodipine, respectively. There was no history of contact of tuberculosis, no history of chulha or biomass fuel exposure and no history of any allergies. Family history was insignificant.

Examination

Patient was well built and nourished. There was a sign of pallor but no cyanosis, jaundice, clubbing or peripheral lymphadenopathy. Patient was afebrile and her vitals were: pulse-112/min, BP-140/90mmhg, RR-25/min, SPO₂-92% on O₂@4l/min. The examination of respiratory system reveals bilateral rhonchi in mammary, infrascapular, infraaxillary region with resonant percussion note in the same region and trachea was centrally placed. Other systemic examination findings were unremarkable.

Investigation

Complete hemogram revealed anaemia (Hb-10.1 gm%, RBC-3.86 million/mm³) and neutrophilic leucocytosis (TLC:

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12,700/mm³, DLC: N₇₀). Blood urea: 17 mg/dl, serum creatinine 0.69 mg/dl. Other blood biochemistry was normal. HIV serology was non-reactive. Her two sputum sample for acid fast bacilli was negative. Chest X-ray revealed bilateral lower zone haziness.

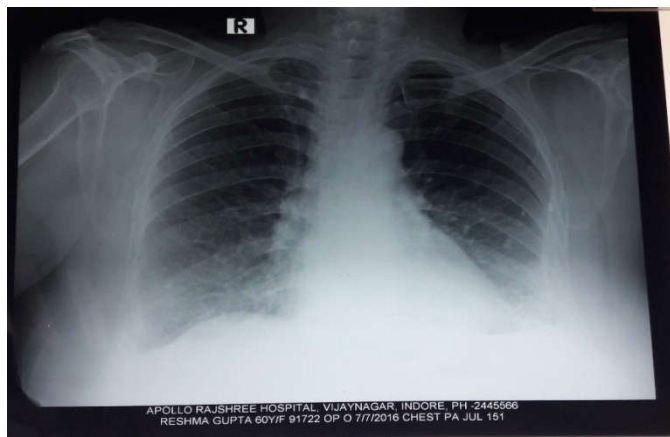


Figure 1. Chest X ray

CECT thorax showed Irregular circumferential wall thickening of tracheal carina with irregular mucosal ulceration and few nodular calcification, contiguous mediastinal soft tissue infiltration with loss of fat planes with small adjacent portion of azygous vein, SVC and right pulmonary artery, enlarged lymph node in the aortopulmonary window measuring 19 x 16 mm, bilateral patchy basal pleural thickenings with sub pleural atelectasis and left basal pleural calcification. Fiberoptic bronchoscopy revealed highly irregular mass infiltrating the carina, firm and compressing both main stem bronchus and polypoid tumour like growth infiltrating carina i.e., central tracheal mass.



Figure 2. Bronchoscopic view of growth at carinal region

BAL sample for AFB, gram staining, cytology, CBNAAT and C/S was sent. Bronchial brushing and biopsy was also sent for histopathology. BAL fluid gram staining and C/S showed growth of *Pseudomonas aeruginosa* (ESBL producing) sensitive to cefepime, colistin, doripenem, gentamicin and levofloxacin. *Mycobacterium tuberculosis* was detected on CBNAAT. BAL cytology showed few small clusters and scattered respiratory and squamous epithelial cells along with neutrophils and alveolar macrophages on background of mucin and degenerated cells. Atypical cell not seen. Brushing shows few small clusters and scattered respiratory epithelial cells along with neutrophils and alveolar macrophages on background of mucin and degenerated cells.

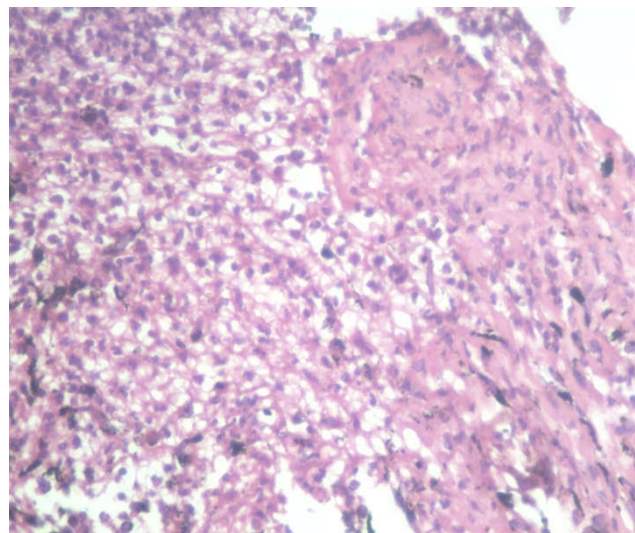


Figure 3. Histopathological slide of Bronchial Brushing

Treatment Patient was put on intravenous antibiotics, but there was no signs of improvement. After review of BAL CBNAAT reports, Category I ATT (2H3R3Z3E3/ 4H3R3) started (DOTS) and patient showed improvement following treatment with antitubercular drugs.

DISCUSSION

The Endobronchial tuberculosis is defined as tuberculous infection of the tracheobronchial tree with microbial and histopathological evidence (Hoheisel, 1994). It can affect any part and layer of the tracheobronchial wall. According to various studies, 10–38.8% EBTB patients was present with active pulmonary tuberculosis (Lee, 1992). However, few authors have reported low incidence (5.88%), because routinely bronchoscopy was not performed in all cases of pulmonary tuberculosis (Lee, 2000). EBTB has females predilection (Lee, 2000) which is usually explained by implantation of organisms from infected sputum, since women do not generally expectorate sputum because of their sociocultural factors. Majority of patients are affected in second or third decade (Lee, 2000). In addition, there is a second peak in old age also as reported by van den Brande *et al.* [1990]. This fact support our study, as our patient was female and she belonged to geriatric age group.

The pathogenesis of EBTB is not fully understood, may due to direct implantation of tubercle bacilli from parenchymal focus, hematogenous spread, rupture of tuberculous intrathoracic lymph node into the bronchus, direct infiltration from adjacent tuberculous mediastinal lymph node, and lymphatic spread along the bronchial tree by lymphatic drainage (Smart, 1951). Most EBTB cases have pulmonary lesions, and approx. 10% to 20% cases may have normal chest radiographs (Lee, 2004). Symptoms and signs of EBTB are usually non-specific and mimic various respiratory diseases like bronchial asthma (Lee, 2004), pneumonia (Kim, 2008), foreign body aspiration (Park, 2000), and malignancy (Singla, 2007). It is difficult to diagnose EBTB on clinical presentation alone and hence, missed frequently. Consideration and awareness of EBTB as differential diagnosis in suspected cases might be helpful in further planning the strategy for definitive diagnosis. To solve dilemma of EBTB, demonstration of acid fast bacilli in sputum, bronchoscopy and computed tomography are essential

for accurate diagnosis. The diagnosis of endobronchial tuberculosis is delayed because of their non-specific clinical manifestation. High index of suspicion is necessary when making the diagnosis in sputum negative patients and patients who are not responding to antibiotics. Bronchoscopy should be performed as soon as possible in suspected patients. In our case report, patient was negative for sputum for AFB initially, and not responsive to antibiotics. Fiberoptic bronchoscopy showed polypoidal growth at carinal region and BAL sample CBNAAT confirmed the diagnosis. Cartridge based nucleic acid amplification test (CBNAAT) does not need any specific pre-requisites for its set-up, requires little technical training, no biosafety risks, use as a rapid diagnostic test and results for both presence of MTB and rifampicin resistance in less than 2 hours, in comparison to conventional drug-sensitivity testing which give results in 8–10 weeks (Boehme *et al.*, 2010). This case report is a reminder that EBTB diagnosis must be considered even though the chest radiograph is normal and patient not responding to antibiotics. The EBTB may also mimic other conditions, such as bronchial asthma or malignancy. Bronchoscopic examination, proper and adequate sampling for acid-fast bacilli smear and culture for Mycobacterium tuberculosis, CBNAAT as well as histology samples, would help in definite diagnosis. Early establishment of diagnosis of endobronchial tuberculosis and their aggressive treatment must be favour change in the course of the disease, increase the chances of improvement and decrease the complication of EBTB.

Declarations

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