Endobronchial Tuberculosis-is Antitubercular drug only sufficient?

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ABSTRACT
Endobronchial tuberculosis (EBTB) is a highly infectious disease that remains a diagnostic challenge in clinical practice. It also presents as a troublesome therapeutic problem due to its sequelae of cicatricial stenosis. Diagnosis of EBTB is frequently delayed until the onset of serious bronchial stenosis with resultant atelectasis and bronchiectasis. Antituberculous chemotherapy is effective in controlling the infection, but does not prevent residual bronchostenosis. Early treatment with steroid therapy is effective in certain groups of EBTB.

Keywords: Endobronchial tuberculosis, Bronchoscopy, Antitubercular treatment, Steroids, Stenosis.

INTRODUCTION
Endobronchial tuberculosis (EBTB) is a highly infectious disease that remains a diagnostic challenge in clinical practice. Early treatment with steroid therapy is effective in certain groups of EBTB1. Endobronchial tuberculosis (EBTB) is defined2 as a specific inflammation of the tracheobronchial tree caused by the mycobacterium tuberculosis. The definitive diagnosis depends on culture of mycobacterium from bronchoscopic material. EBTB is present in 10-40% of patients with active tuberculosis and causes some degree of bronchial stenosis in more than 90% of the patients3. It is a highly infectious disease that poses a diagnostic challenge because the disease presents with non specific clinical findings and the lesion may not be evident on the chest X-ray3. Frequently, the diagnosis is delayed because of its decreased incidence now a days. Management poses a great challenge due to its sequelae of bronchial stenosis5. Early diagnosis and prompt treatment may prevent the development of complications. Steroid treatment for bronchostenosis remains controversial. Interventional bronchoscopy or surgery should be considered for the management of stenosis that occurs despite medical treatment5.

CASE REPORT
A 34 year, female patient presented with history of non productive cough since 4 months, sore throat, and progressive shortness of breath on exertion since 2 months. She reported no symptoms of night sweats, haemoptysis or weight loss and she had no history of contact with tuberculosis. She had evening rise fever, blood pressure was 126/84 mm Hg, pulse rate was 78 per minute, and respiratory rate was 13 breaths/min. Results of the physical examination were unremarkable. She had been referred to us from private sector and was put on inhaled bronchodilators but not relieved. The patient’s purified protein derivative skin test was positive, with an induration of 24 mm. Her erythrocyte sedimentation rate was 82 mm/h. Sputum for AFB was negative. Her Spirometry and chest x-Ray was normal. Gradually over the duration of 4 months patient developed hoarseness of voice and chest pain along with stridor. Repeat chest x-ray was done which was suggestive of left hilar widening. RE sputum for AFB was negative. We went for CECT chest which was suggestive of multiple branching nodular opacities in Rt middle, Rt lower and anterior & apicoposterior segment of left upper lobe. Non enhancing hypodense area in the right bronchus intermedius and lower lobe bronchus suggestive of narrowing 70 to 90%. So endobronchial spread was a clue on CT report. Few heterogeneously enhancing lymphnodes were in pretracheal, Rt paratracheal and subcarinal areas showing possibility of Koch’s etiology. Her complete blood count and serum electrolyte levels were within normal limits. HIV & HbsAg was negative. Then to confirm the diagnosis we planned Bronchoscopy, findings of which were white patches on vocal cords, trachea and bronchial wall was completely covered with necrotic mucosa. Bronchial lavage for AFB stain by ZN and culture was positive for mycobacterium tuberculosis complex which was sensitive to all first line ATT. Bronchial biopsy of endobronchial lesion revealed multiple necrotizing granulomas without evidence of malignancy. She was put on 4 drugs ATT CAT 1 with steroids 1mg/kg in tapering doses for 3 months. She did not improved at end of 6 mths of CAT 1 in terms of symptoms. So we decided to repeat bronchoscopy and BAL was again positive for AFB by ZN stain. It was suggestive of tracheal stenosis approx 60% of the lumen). Patient was shifted to CAT II under RNTCP considering it as treatment failure. Now patient has completed 7 mths.
of therapy and quite improved in terms of symptoms, has gained weight. Patient does not have stridor at present.

Figure 1: Normal chest X-ray

Figure 2: “Tree-in-bud” appearance in CT scan

Figure 3: Marked narrowing of bronchus

Figure 4: Erythema, mucosal granulation

DISCUSSION

Endobronchial tuberculosis (EBTB) is the tuberculous infection of the tracheobronchial tree, with microbial and histopathological evidence. The incidence of EBTB among postmortem specimens with modern treatment has declined to 10% of cases of pulmonary tuberculosis. The clinical presentation of EBTB is variable. The clinical features depend on the site and the extent of involvement and may occur in the absence of recognized symptoms. It may have an insidious onset, simulating lung carcinoma, or an acute onset mimicking asthma, foreign body aspiration or pneumonia. Physical examination may reveal no abnormalities in one third of the patients. Examination of the respiratory system may detect rales, decreased breath sounds, a localized monophasic wheeze, bronchi and bronchial breathing. Persistent unilateral wheeze is indicative of EBTB. Stridor may occur with ulceration and cicatrization of the larynx or trachea. Chung and Lee have classified EBTB into seven subtypes according to the bronchoscopic findings: actively caseating, oedematous-hyperaemic, fibrostenotic, tumorous, granular, ulcerative and the nonspecific bronchitic type. A clear chest X-ray does not exclude the diagnosis because the X-ray appearances may be normal in 20% of patients. Bronchial stenosis, bronchiectasis and broncholiths may occur as late complications of endobronchial disease. A “tree-in-bud” appearance is a characteristic in high resolution computed tomography Thorax (HRCT) which represents caseation necrosis within and around the bronchioles. Miliary tuberculosis and endobronchial involvement may coexist. Other abnormalities on HRCT in decreasing order of frequency are 4-8 mm diameter nodules with poorly defined margins, lobular areas of consolidation and thickening of the interlobular septa. A low yield is reported of positive sputum smear examination for AFB in the diagnosis of endobronchial tuberculosis. Bronchoscopic examination is the key to diagnosis. A bronchoscopic biopsy and BAL for AFB culture is the most reliable method for the diagnosis of EBTB. Needle aspiration may provide only a cytological diagnosis. Bronchial biopsy is positive 30-84% of patients. The treatment of EBTB is the same as that of pulmonary tuberculosis. Antituberculous chemotherapy is effective in controlling the infection but may not preclude residual bronchostenosis. Corticosteroid therapy for the prevention of bronchial stenosis remains controversial. Corticosteroids more likely to be useful in the earlier stages of EBTB when hypersensitivity is the predominant mechanism. They are not likely to be helpful in advanced cases when extensive fibrosis is present. The usual dose of corticosteroids is 40-60 mg (1 mg/kg body weight) daily for 4-6 weeks, tapered gradually over the next few weeks. The effectiveness of treatment in preventing endobronchial stenosis is debatable as 60-95% of adequately treated patients have developed bronchial stenosis. Patients with tracheal involvement may have serious sequelae such as respiratory failure, collapse of the lung, bronchiectasis and post obstructive pneumonitis. Early diagnosis with prompt treatment is important to prevent the serious complications of EBTB. Laser resection or electrosurgery may be performed to prevent further stenosis. If the fibrostenosis is long an endobronchial stent can be placed after balloon dilatation, which is only a temporary measure. Dumen stents are appropriate for caseating types since removal or placement is always possible. Ultraflex stents should not be used because their removal is difficult. Restenosis due to granulation tissue is treated by laser or electrocoagulation. Surgical resection may be indicated in subjects unresponsive to interventional bronchoscopic treatment. Bronchoplastic surgery is performed for tracheal or major bronchial strictures in order to preserve lung function.

REFERENCES

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