# **Endobronchial Tuberculosis Simulating Lung Cancer and Healing without Bronchial Stenosis**

Al-Maslamani M<sup>1</sup>, Ibrahim WH<sup>2</sup>, Chacko K<sup>1</sup> and Al-Khal A<sup>1</sup>

1 Infectious disease division and 2 Department of Medicine, Hamad General Hospital, Doha, Qatar

**Abstract**: Endobroncheal tuberculosis is defined as tuberculous infection of the tracheobronchial tree with microbial and histopathological evidence. The disease is usually mistaken for other lung diseases including lung cancer. Bronchial stenosis is a common complication of this type of tuberculosis despite the use of effective anti-tuberculous chemotherapy. We are presenting a case of endobronchial tuberculosis that simulated lung cancer and healed without residual bronchial stenosis.

Key words: Endobronchial Tuberculosis, Bronchial Stenosis, Lung Cancer.

#### Introduction

Endobronchial tuberculosis (EBTB) is a form of tuberculous infection that involves mainly the trachea and/or bronchi. The disease is often mistaken for more common lung diseases like pneumonia, asthma, lung cancer and other lung diseases. Endobronchial stenosis is a common complication of EBTB despite the use of antituberculous chemotherapy. We are presenting a case of EBTB that manifested with two endobronchial masses, which almost totally occluded the bronchial lumen and resulted in repeated episodes of post-obstructive pneumonia for more than a year prior to diagnosis. Our patient was successfully treated with anti-tuberculous chemotherapy without the use of corticosteroids. Repeated bronchoscopic examination after six months initiating antituberculous treatment revealed complete resolution of the two lesions without residual bronchial stenosis.

### **Case presentation**

A 62 year old Pakistani woman was admitted to Hamad General Hospital with history of fever for 10 days prior to admission. She denied any history of cough, hemoptysis, breathlessness, chest pain, nocturnal sweating or weight loss. Prior to admission she was prescribed a seven-day course of amoxicillin that was changed to cephalexin by her family physician without improvement. For more than a year prior to this admission she had frequent visits to emergency room and a hospital admission for recurrent episodes of fever and cough that were diagnosed as community acquired pneumonia based on the radiographic finding of right lung opacity and the response to antibiotic therapy. Three years earlier she was admitted for abdominal surgery due to a perforated duodenal ulcer and incidentally discovered to have type 2 diabetes for which she was commenced on glibenclamide. She had frequent visits to Pakistan, the last of which was 2 years prior to her admission. Eight months prior to her admission she was refused entry to Canada because of abnormal Chest radiograph. She was a housewife, life-long non-smoker, and never consumed alcohol. She never kept pets or birds.

Physical examination was remarkable for fever of 39.5 Co and dull percussion note over the right lower and mid chest, without added sounds.

Laboratory investigations at admission revealed leucopenia of 1.8 X 103/ul with neutropenia of 0.5 X

103/ul and normal hemoglobin and platelet count. Erythrocyte sedimentation rate (ESR) was 105 /hr. Peripheral blood smear revealed marked leucopenia with severe neutropenia, few toxic, left-shifted neutrophils and reactive lymphocytes without malarial parasites seen (picture of severe infection or drug induced cytopenia). Two samples for blood culture and urine culture did not grow bacteria or fungi. Tuberculin skin test was 14 mm at 72 hours. Bacterial culture of the sputum, 3 sputum samples for acid-fast bacilli smear and culture and Human Immunodeficiency Virus (HIV) testing were all negative.

Chest radiograph revealed right lower and mid zone non-homogenous opacity (Figure 1). Computerized tomography (CT scan) of the chest and abdomen revealed a soft tissue enhancing mass in the lumen of the right main bronchus that was associated with distal parnechymal consolidation and multiple lymph node enlargement (Figure 2).

Bone marrow examination revealed a cellular marrow without evidence of involvement by neoplastic or granulomatous process.

Bronchoscopy was done and showed two large endobronchial masses, one in the right main bronchus and the other in the right upper lobe bronchus with glistening, whitish surface (Figure 3). Endobronchial biopsy was taken from both masses and reveled caseating granuloma with presence of acid-fast bacilli on Zeihl-Nelsen staining (Figure 4).

Bronchoalveolar lavage culture for acid-fast bacilli was negative. The patient was diagnosed as tumorous type EBTB causing post-obstructive pneumonia. Her leucopenia was attributed to antibiotics prescribed by her family physician. She was started on intravenous meropenem for 14 days and antituberculous chemotherapy in the form of Rifampicin, Isoniazide, Ethambutol and Pyrazinamide for 2 months, and then continued on Rifampicin and Isoniazide for further 4 months. No corticosteroid therapy was used at any stage of the treatment.

The patient made a good recovery and was discharged home in a good condition. A repeated bronchoscopy 6 months later revealed complete resolution of the endobronchial masses without evidence of bronchial stenosis (Figure 5).



Figure 1: Chest radiograph showing opacification of the right lower zone

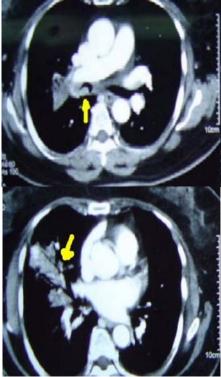


Figure 2: CT scan of the chest showing right bronchial mass with post-obstructive consolidation (arrows)

### **Discussion**

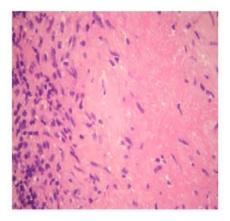
EBTB was first described by the English physician Richard Morten in 1698 [1]. The disease remained infrequently reported and was mainly a postmortem pathological diagnosis until the advent of bronchoscopy in the late 1920s [2]. EBTB is defined as tuberculous infection of the tracheobronchial tree with microbial and histopathological evidence [3,4].

This form of tuberculous infection continues to be an important health problem for three reasons; firstly, its diagnosis is frequently delayed, particularly in developed countries, as the decreased incidence itself diminishes the suspicion of tuberculosis. Secondly, bronchial stenosis is a serious complication of the disease that may develop despite efficacious antituberculous chemotherapy and thirdly, it is often misdiagnosed as bronchial asthma or lung cancer [5,6,7].





Figure  $\overline{\bf 3}$ : Bronchoscopic picture showing a mass in the right main bronchus and another one in the right upper lobe bronchus



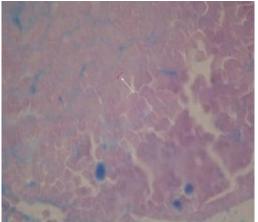


Figure 4: Histopathologic slides showing caseating granuloma with acid-fast bacilli (arrow)





Figure 5: Bronchoscopic picture of the right main bronchus and the right upper lobe bronchus after six months of treatment showing disappearance of the masses with no residual stenosis

The exact pathogenesis of EBTB is unknown, however, direct extension from adjacent pulmonary parenchymal lesion, implantation of the organisms from infected sputum, dissemination from the blood or erosion of a lymph node into the bronchus have all been suggested as possible mechanisms of the disease [3,8]. Chung and Lee identified seven subtypes of EBTB on bronchoscopic appearance [5]; the actively caseating type (43.0%), the edematous-hyperemic type (14.0%), the fibrostenotic type (10.5%), the tumorous type (10.5%), the granular type (11.4%), the ulcerative type (2.7%), and the non-specific bronchitic type (7.9%). Among these subtypes, the prognosis of tumorous EBTB was found to be most grave and unpredictable. The majority of the tumorous EBTB cases studied by Chung and Lee changed to fibrostenotic type within 3 months of treatment. Tumorous EBTB is characterized by an endobronchial mass whose surface is covered by caseous material and totally or near totally occludes the bronchial lumen. This form of EBTB is frequently mistaken for lung cancer, adenoma or carcinoid tumors.

The two most important goals when treating EBTB are to eradicate tubercle bacilli and to prevent bronchial stenosis. Although anti-tuberculous chemotherapy is effective in controlling infection, it does not prevent residual bronchostenosis [5,4]. Oral corticosteroids have been used empirically to prevent bronchial stenosis, nevertheless, their role remains uncertain and

controversial [9,10]. Many patients with EBTB will require aggressive treatment like repeated dilatation, the use of stents or even resection to treat bronchial stenosis [11]. Some previous studies have suggested that delay in diagnosis of EBTB is an independent predictor of the development of persistent airway stenosis [12].

Our patient presented with two tumorous growths that almost completely obstructed the lumens of the right main bronchus and right upper lobe bronchus and resulted in recurrent episodes of post-obstructive pneumonia for more than a year prior to diagnosis. Considering the prognosis of tumorous type EBTB and the delay in diagnosis as a predictor for the development of bronchial stenosis, healing without stenosis in our case seems interesting. It may suggest that the exact mechanism behind the tendency of some patients and certain types of EBTB to develop bronchial stenosis is not yet completely understood. Future research should focus on the pathogenesis of bronchial inflammatory reaction induced by EBTB in order to understand this mechanism.

## References

- 1. Hudson EH. Respiratory tuberculosis: clinical diagnosis. In: Heaf ERG,ed. Symposium on Tuberculosis. London: Cassell and Co.1957; P:321-464.
- 2. Seiden H S and Thomas P. Endobronchial tuberculosis and its sequelae. CMAJ 1981; 124:165-169.
- 3. Kashyap S, Mohapatra PR, and Saini V. Endobronchial tuberculosis. Indian J Chest Dis Allied Sci. 2003; 45:247-256.
- 4. Hoheisel G, Chan BK, Chan CH, Chan KS, Teschler H and Costable U. Endobronchial tuberculosis: Diagnostic features and therapeutic outcome. Respir Med. 1994; 8:593-597.
- 5. Chung HS and Lee JH: Bronchoscopic assessment of the evolution of endobronchial tuberculosis. Chest 2000; 117:385-392.
- 6. Chung HS, Lee JH, Han SK, et al. Classification of endobronchial tuberculosis by the bronchoscopic features. Tuberc Respir Dis. 1991; 38:108-115 (in Korean).
- 7. Albert RK and Petty TL. Endobronchial tuberculosis progressing to bronchial stenosis. Chest. 1976; 70:537-539.
- 8. Smart J. Endobronchial tuberculosis. Br J Dis Chest. 1951; 45:61-68.
- 9. Park IW, Choi BW and Hue SH. Prospective study of corticosteroid as an adjunct in the treatment of endobronchial tuberculosis in adults. Respirology. 1997; 2:275-281.
- 10. Kim YH, Kim HT, Lee KS, Uh ST, Cung YT and Park CS. Serial fiberoptic bronchoscopic observations of endobronchial tuberculosis before and early after antituberculous chemotherapy. Chest. 1993; 103:673-677.
- 11. Han JK, Im JG, Park JH, Han MC, Kim YW and Shim YS. Bronchial stenosis due to endobronchial tuberculosis: successful treatment with self-expanding metallic stent. AJR Am J Roentgenol. 1992; (159)5:971-972.
- 12. Um S-W, Yoon YS, Lee S-M, et al. Predictors of persistent airway stenosis in patients with endobronchial tuberculosis. Int J Tubercc Lung Dis. 2007; 11(12):57-62.