Review Article

Endobronchial Tuberculosis: An Overview

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Abstract

Endobronchial tuberculosis (EBTB) is a special type of pulmonary tuberculosis and its incidence has increased in recent years. The EBTB often injures the tracheobronchial wall and leads to tracheobronchial stenosis resulting in intractable tuberculosis and make patients become chronic infection sources of tuberculosis, or may even cause pulmonary complications and resulting in death. The etiological confirmation of Mycobacterium tuberculosis is most substantial for diagnosis. However, because the positive rate of acid-fast bacillus staining for sputum smears is low and the clinical and radiological findings are usually non-distinctive, the diagnosis of EBTB is often mistaken and delayed. For early diagnosis, a high index of suspicion is required and the bronchoscopy should be performed as soon as possible in suspected patients. The eradication of Mycobacterium tuberculosis and the prevention of tracheobronchial stenosis are two most substantial treatment goals. For this, the diagnosis must be established early and aggressive treatments must be performed before the disease progresses too far.

Keywords: Endobronchial tuberculosis; Mycobacterium tuberculosis.

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Introduction

Pulmonary tuberculosis is one of the major health problems worldwide.[1] In spite of much progress in diagnosis and therapy, this problem still remains.[2] Moreover, there has been a resurgence of pulmonary tuberculosis recently, which is related to the HIV epidemic[3], multidrug-resistant strains, poverty, immigration, and shortness in the prevention and treatment system.[4] About 10% to 40% of patients with active pulmonary tuberculosis had EBTB as reported in previous studies.[5,6] It has been defined as tuberculous infection of the tracheobronchial tree with microbial and histopathological evidence[7] or a complication of progressive primary tuberculosis.[8] Endobronchial tuberculosis (EBTB) is a special form of pulmonary tuberculosis[9] not easily recognizable, often dangerous for its consequences and potentially a source of spread of infection in the community. The diagnosis of typical pulmonary tuberculosis is easily confirmed by bacteriological studies and radiological findings. However, the diagnosis of EBTB is more difficult because of varied clinical manifestations. The long term sequelae of endobronchial tuberculosis is bronchostenosis, which is mainly determined by the extent of disease progression and closely related to the formation of granulation tissue.[10,11] Once fibrostenosis develops or extensive granulation tissue appears, marked bronchostenosis is inevitable despite efficacious anti-tuberculosis chemotherapy.[11,12] The incidence rate of bronchostenosis may reach up to 68% in initial 4 to 6 months of the disease and rises further with the course of disease elongating.[13,14] Severe bronchostenosis may cause pulmonary complications such as pulmonary infection, atelectasis, bronchiectasis, and even death by inducing respiratory failure and asphyxia. Additionally, the bronchostenosis may cause intractable tuberculosis and make patients to become chronic infection sources of tuberculosis. The eradication of tubercle bacilli and the prevention of bronchostenosis are the two most important goals of EBTB treatment. To fulfill these treatment goals, the diagnosis of this disease must be established early and aggressive treatment should be started before the disease progresses further. This review article is aimed at detection of early diagnosis and initiation of effective therapy.

Pathogenesis: Five potential mechanisms have been suggested for the development of endobronchial infection due to M.tuberculosis[10]: (1) direct extension from adjacent parenchymal focus; (2) implantation of organisms from the infected sputum; (3) hematogenous dissemination; (4) lymph node erosion into the bronchus; and (5) through lymphatic drainage from parenchyma to the peribronchial region. Myerson has suggested alternative mechanism of retrograde passage

of tubercle bacilli through lymphatics from bronchioles and subsegmental bronchi. Perforation of seperate lymphnode into the bronchi has been considered in some adults[9]. Initially, the mass protrudes into the bronchial wall and may obstruct the lumen; the node may be seen as a greyish–yellow mucosa, and the lumen wall appearing as hemorrhagic and granulating. A fistula may develop from which cassous material extrudes, forming caseous lumps in the sputum. Computed tomographic (CT) scan, x-ray chest, or bronchograms may demonstrate excavation of the node radiographically. Finally, fibrosis develops with scarring of the bronchial wall resulting in bronchostenosis.

Clinical Features: EBTB may have insidious onset, simulating bronchogenic carcinoma, or may be acute, mimicking asthma, foreign body aspiration and pneumonia. Symptoms of EBTB may develop even after completion of therapy. EBTB is more common in young adults with a female predominance.[15] Fifteen percent of geriatric patients may also have EBTB.[16] Endobronchial tuberculosis can present with a variety of nonspecific signs and symptoms, which include cough (usually nonproductive), dyspnea, anorexia,weight loss, hemoptysis, chest pain, and hoarseness. The barking cough that is not responsive to an antitussive medication, but responds to steroids along with anti-tuberculosis treatment may be a feature of EBTB. Localized wheezing can occur if there is a stenosing effect by the endobronchial lesion.[17] Bronchorrhea can occur in active endobronchial tuberculosis.[18] Sputum production is variable. Hemoptysis may occur but is seldom massive. Lymph node rupture may cause chest pain that may be sharp or dull in sternal or parasternal region. Dyspnea is often associated with atelectasis of the lung. Physical examination may reveal diminished breath sounds and localized low-pitched wheeze or rhonchi. Up to 25 to 35% of EBTB pateints may have features of collapse. Classical monophonic wheeze may be heard in about 15% of the patients[19]. There may be expectoration of tracheal cartilage[20]. Dull and sharp chest pain can occur anteriorly due to enlargement and rupture of the lymph nodes. Constitutional symptoms including fever, weight loss, anorexia and night sweat are not usually prominent in EBTB[21]. Serious sequelae especially with tracheal involvement such as respiratory failure, collapse of dependent portion of lungs, failure of endobronchial intubation, or death by suffocation have been reported.[22,23]

Diagnosis: Although sputum examination is the essential and first step towards the diagnosis of EBTB, bronchoscopy and computed tomography are the methods of choice for accurate diagnosis of bronchial involvement and assessment for surgical intervention. Fibreoptic bronchoscopy is indicated in patients in whom chest radiographs, physical signs or symptoms suggest the possibility of endobronchial tuberculosis. Due to the potential utility of early antituberculosis treatment and use of corticosteroids in reducing the lymph node size and mucosal swelling, the above procedures should be carried out at the earliest. Typical bronchoscopic finding is the presence of white gelatinous granulation tissue. The mucosa is nodular, red, vascular and sometimes ulcerated. It may simulate a bronchogenic carcinoma[24]. Nucleic acid amplification tests, such as PCR and other methods for amplifying DNA and RNA, may facilitate rapid detection of M. tuberculosis in respiratory tract specimens. Recommendations for interpretation and use of nucleic acid amplification have been recently updated by the centers for disease control and prevention.[25]

Sputum Examination: All suspected patients should be subjected to sputum smear and culture examination for M. tuberculosis. Yield of sputum smear for AFB is not as high as in parenchymal involvement even in an optimal laboratory setup with meticulous sputum examination. In recent studies, sputum positivity in EBTB has been demonstrated from 16 to 53.3 percent[26-28].However, EBTB with ulceration and mucosal involvement has higher sputum positivity and yield is even greater with sputum culture when compared to smear (73.6 vs 53.3%).[28]

Chest Radiograph: 10 to 20 percent patients with EBTB may have a normal chest radiograph[13]. Thus, a clear chest radiograph does not exclude the diagnosis of endobronchial TB. Bronchial stenosis occurs in 10-40 percent of patients with active pulmonary tuberculosis[24]. Radiologic manifestations of tuberculous bronchostenosis include persistent segmental or lobar collapse, lobar hyperinflation, obstructive pneumonia and mucoid impaction[29]. Volume loss on chest radiography may indicate development of bronchial stenosis, and fibreoptic bronchoscopy should be considered in such cases.[29] Erosion of calcified hilar nodes into adjacent bronchi, known as broncholithiasis, may also result in segmental collapse or over inflation[30].Bronchiectasis is another complication of EBTB, usually involving upper lobes.

Computed Tomography (CT) Scan: Volumetric computed tomography has the advantage of acquiring both multiplanar and three-dimensional (3D) images, thus enabling precise diagnosis and evaluation of the extent of disease involving the airways. Multiplanar and 3-D images appear to be useful for global understanding of the status of the tracheobronchial tree, particularly for evaluation of focal stenosis of the airways.[31] Characteristic HRCT findings of EBTB are patchy asymmetric centrilobular nodules and branching lines that may have unilateral or bilateral distribution.[32,33] Multiple branching linear structures of similar caliber originate from a single stalk (the ‘tree-in-bud’ appearance). The stalk is thought to represent a lesion that affects the last order bronchus within the

secondary pulmonary lobule and the bud is thought to represent a lesion that is in the bronchioles and alveolar ducts. In CT scan, there are foci of ill-defined nodular densities which are peribronchiolar in location and markedly variable in size, including the lesions as small as2-3 mm. These nodular densities may become confluent.[34] Tuberculous mediastinal lymph nodes are usually of low density (necrotic) on chest CT. CT is also a useful adjunct to direct endoscopic visualization, CT accurately depicts the bronchial abnormality in 93% to 100% cases. CT findings include isolated (41% to 43%) lung segmental bronchial narrowing with concentric wall thickening, complete endobronchial obstruction (32%) and extrinsic obstruction by adjacent adenopathy (23% to 50%).[35] Usually, tuberculosis heals following therapy resulting in scar formation in the parenchyma. Such scars cause distortion of bronchovascular bundles, irregular fibrotic bands and small irregular nodular densities with surrounding cicatricial emphysema. These changes attributable to healed lesions have been noted in the areas remote from the endobronchial lesion.

Fibreoptic Bronchoscopy(FOB):

Bronchoscopic sampling has been the key to the diagnosis of EBTB, producing more than 90 percent yield on smear as well as on culture.[6-13] In diagnosing EBTB, the experienced bronchoscopist is required. Even if biopsy fails to supply tangible results, the bronchoscopic changes, supported by clinical and radiological findings, may be sufficient to establish the diagnosis of EBTB.A bronchoscopic biopsy is the most reliable method for diagnosing EBTB[36], because a needle aspiration can provide only a cytological diagnosis. However, needle aspiration can be used for obtaining materials from segments of a lobe where the forceps cannot reach. Different bronchoscopic specimens including biopsy, brushing and washings should be obtained as practicable. These specimens provide variable yield. Bronchial biopsy may be positive in 30.35% to 84% patients. Similarly, bronchial washings have also yielded variable results ranging from 10% to 37.5 percent[7].A widely accepted classification defining EBTB by FOB has the following seven subtypes :(i) actively caseating, (ii) edematous-hyperemic,(iii) fibrostenotic, (iv) tumorous, (v) granular, (vi) ulcerative, and (vii) nonspecific bronchitis.[28] The prominent lymph nodes are seen as grayish-yellow masses through the bronchial mucosa. Hemorrhage, granulation tissue fistula formation and caseous material draining into bronchus may also be seen. Early bronchoscopic findings consist of erythema, mucosal granularity including discrete submucosal tubercles, and shallow mucosal ulcers. Findings suggestive of more advanced disease are deep ulcers, hyperplastic inflammatory polyps, tumour like collections of granulation tissue and bronchial stenosis.[37] The exudative lesion consists of mucosal erythema, swelling and white caseous exudates with or without granulation tissue. The lesions are described as ulcerative when bronchial mucosa shows predominantly ulcerations. The cicatricial lesions have hypertrophic mucosa and distorted lumen with or without polypoid lesions. The bronchoglandular lesions are manifested as one or more eccentric round indentations into bronchial lumen on bronchoscopic examination. Such lesions have involvement of peribronchial or paratracheal lymph node(s) on CT.[23] Diffuse mucosal congestion with edema (suggestive of inflammation) and mass lesion were most common bronchoscopic findings in three different studies.[38]

Treatment: Early diagnosis and a correct therapy may favourably change the course of EBTB. The treatment must be standardized according to the internationally accepted guidelines. The role of steroids is quite controversial, however, and when orally administered, they could reduce the inflammatory reactions with positive reflexes both on the early and late bronchial stenosis.[39] Alternatively, a positive role of topical administrations of steroids through repeated bronchoscopic sessions, or with the use of aerosolized steroids and isoniazid have been described. When a medical treatment is not sufficient to prevent the development of a stenosis it is necessary to implant a stent or to turn to surgical treatment with bronchoplastic surgery.[40] Endobronchial laser and curettage can yield interesting positive results provided the correct indications are previously set.[41, 42] The treatment of EBTB is the same as that for pulmonary tuberculosis. Five standard first line drugs are used for the treatment of EBTB: isoniazid (INH), rifampin (RIF), Ethembutol (EMB), pyrazinamide (PZA) and streptomycin (STR). It is necessary to know the dosages and adverse reactions caused by these drugs. Full assessment for drug susceptibility or resistance is essential. Patients should be evaluated at least monthly for adverse reactions to these drugs. It is helpful to have a baseline measurement of hepatic enzymes, bilirubin, complete blood count, serum creatinine and uric acid prior to initiating treatment. If EMB is included in the regimen, visual acuity and red-green colour perception should be evaluated. Hearing tests should be performed prior to initiation of STR. A 6 month treatment consisting of INH, RIF and PZA for the first two months, followed by INH and RIF for the next 4 months is the treatment for patients with fully susceptible organisms. In drug resistant tuberculosis, treatment must be based on susceptibility results.[43] Since bronchial stenosis is relatively common and may occur despite adequate antituberculous and steroid therapy, interventional bronchoscopic or surgical management may be indicated. The role of corticosteroids in reversing bronchostenosis is controversial.[44] Corticosteroids are 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. Before corticosteroids are given, adequate antituberculosis chemotherapy should have been started. The usual dose of corticosteroids is 40-60 mg daily for 4-6 weeks, tapered gradually over the next few weeks.[45] The steroids may acutely reduce bronchial narrowing and reduce the extent of poststenotic lung damage. They may also reduce the long term evolution of high-grade bronchial stenosis.[46] Verhaege et all demonstrated resolution of EBTB with submucosal methylprednisolone injection.[47-49] Rikimaru showed that healing time of ulcerous lesions was shorter and bronchial stenosis was less severe in patients treated with aeresol streptomycin and dexamethasone.[41] A major issue in the treatment of EBTB is evaluation of bronchoscopic findings during treatment. The therapeutic outcome of each type of EBTB can be predicted by follow-up bronchoscopy during the initial three months, with the exception of the tumorous type. The tumorous type may show diverse progress and unexpected changes. The evolution of the lesions is complicated and stenosis may develop at a later time. CT may also be very useful in the evaluation of bronchial stenosis or obstruction.In tumorous type bronchial stenosis, the prognosis is grave if the condition is not treated aggressively. Laser resection or electrosurgery may be performed to prevent further stenosis.[50] If the fibrostenosis is long, an endobronchial stent can be placed after balloon dilatation, which is only a temporary mesasure. In the caseating, oedematous and tumorous forms of EBTB, the therapeutic results of stent placement are poor because of severe inflammation. Dumon stents are appropriate since removal or placement is always possible. Ultraflex stents should not be used because their removal is difficult[49].Restenosis due to granulation tissue is treated by laser or electrocoagulation. Stents may be removed one year after placement. In granular, ulcerative or nonspecific EBTB, significant bronchostenosis does not develop. Laser resection and surgery are the gold standard for the treatment of bronchial stenosis and long term appropriate antituberculosis treatment should be administered for nine to twelve months to prevent restenosis.[50] The majority of patients with EBTB have negative results on sputum smears within 2 months of therapy. Despite treatment, bronchial stenosis is a commonly reported complication of EBTB, and prospective studies evaluating the use of corticosteroids to prevent bronchial stenosis have not reported success.

Conclusion

The diagnosis of EBTB depends on the presence of specific endobronchial inflammatory lesions and culture of mycobacterium from bronchoscopic samples. The treatment strategy should be individualized according to the presumptive natural course of the subtype of EBTB detected on the initial bronchoscopic examination. The desired therapeutic outcome of EBTB is healing without significant sequelae. While on the other hand, there may occur stenosis and bronchial obstruction. All subtypes fall between these two end points and may transform or progress into other subtypes during treatment. Early diagnosis and prompt treatment of EBTB is important to minimize or prevent bronchial stenosis which may occur despite effective antituberculous treatment. The role of steroids in the prevention of bronchostenosis is controversial. The therapeutic outcome of each subtype can be predicted by bronchoscopy in the initial three months. The evolution of the tumorous subtype is usually complicated and severe bronchostenosis may develop. In such patients, aggressive treatment should be performed before the stenosis becomes irreversible and long term follow-up is advisable. Bronchoscopy is mandatory not only for the initial diagnosis but also for follow-up and to prevent bronchostenosis. For the treatment of bronchostenosis that has already developed, interventional therapeutic modalities such as electrocautery, laser therapy or stent insertion should be considered. Drug treatment should be given for active inflammation before interventional procedures and should be continued for a minimum of three months to prevent recurrence. Laser photoresection and electrosurgery are other effective treatment modalities for tuberculous bronchial stenosis. 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. Appropriate antituberculous treatment should be given for at least nine months to prevent recurrence or restenosis in such patients.

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