ENDOBRONCHIAL TUBERCULOSIS -A REPORT OF 5 CASES

S K Teo

ABSTRACT

Endobronchial tuberculosis is not as well-known to internists as tuberculosis involving the lung parencyma. Five cases with this conditon are reported to illustrate the varied clinical manifestations. The presenting features of the 5 cases were lobar or lung collapse, unresolved pneumonia, dyspnoea and stridor. Bronchostenosis developed in 2 patients many years following chemotherapy, while stenosis of the trachea developed in one patient during chemotherapy. In another patient, the tuberculous granulation tissue simulated a lung cancer at bronchoscopy. Diagnosis can be difficult as endobronchial tuberculosis can occur in the absence of chest X-ray abnormality and sputum smear may also be negative for acid fast bacilli (AFB). Therefore, bronchoscopy should be done when the condition is suspected in a patient who has unexplained cough, wheezing, dyspnoea or haemoptysis. The modalities of treatment for fibrostenosis of a large airway include surgical resection followed by anastomosis, balloon dilatation, laser photoresection or a combination of both procedures.

Keywords: Endobronchial tuberculosis, manifestations, diagnosis, management

SINGAPORE MED J 1990; Vol 31: 447 - 450

Endobronchial tuberculosis (ESTB) is a disease better known to the physicians of the eighteenth and nineteenth century. It was first described by the English physician, Richard Morton, in 1689. With the advent of effective chemotherapy, much interest has centred on tuberculosis involving the parenchyma but little attention has been given to tuberculosis involving the trachea and bronchi.

The purpose of this paper is to familiarise doctors with the diverse manifestations, diagnosis and management of the condition.

CASE REPORTS

Case 1

A 58-year-old Chinese man was referred for investigation of collapse of the left lung. He had been treated 20 years previously for pulmonary tuberculosis (PTB). Sputum smear and culture examination were negative for acid fact bacilli (AFB). Bronchoscopy showed widening of the carina. The lower tracheal wall on the left side and the wall of the left main bronchus just distal to the carina was noted to collapse inwards preventing further passage of the fibreoptic bronchoscope. Foul smelling pus was present. Biopsy of the bronchial mucosa showed acute inflammation with hyperplasia and metaplasia. No malignant cells were seen. AFB were detected by Ziehl-Neelsen stain.

He was treated with a 9-month course of isoniazid and rifampicin with ethambutol given for the first 2 months.

Department of Tuberculosis Control Tan Tock Seng Hospital Moulmein Road Singapore 1130

S K Teo, MBBS, MRCP (UK), FCCP, AM (S'pore) Physician and Head

He was well two and a half years after stopping treatment. The collapse of the bronchial wall was most likely due to destruction of the cartilage.

Case 2

A 48-year-old woman was referred in 1986 because of right upper lobe collapse. She had been coughing for five to six years. She had a history of PTB 20 years ago(1966) and was given antituberculosis treatment for one and a half years. Review of her chest X-rays taken in 1974, 1976 and 1986 (Fig 1a, b and c) showed gradual but progressive collapse of the right upper lobe.

Bronchoscopy showed a pin hole stenosis of the right upper lobe bronchial orifice. Bronchial aspirate was negative for AFB. She was well for 3 years while on follow.up.

Case 3

The patient was a 50-year-old secretary. She presented in 1985 with fever and cough for 3 weeks which started as an upper respiratory tract infection. She was treated by several private practitioners without much improvement. She was admitted and investigated as a case of unresolved pneumonia (Fig 2a). Her sputum smears and cultures were negative for AFB. Bronchoscopy showed severe narrowing of the right main bronchus. Bronchogram was done using a fine catheter. It showed a 1 cm stricture of the right main bronchus just distal to the carina with bronchiectasis and marked collapse of the middle and lower lobes and mild bronchiectasis of the upper lobe (Fig 2b). She gave a history of being treated for tuberculous pleurisy 20 years ago and a chest X-ray taken 1 year ago for hypertension showed no abnormalities. She was treated with a prolonged course of antibiotics. In view of the absence of any respiratory symptoms prior to her illness and the good response to treatment, it was felt that she should





1 (b)



1 (c)



be observed closely as surgical treatment may necessitate doing a pneumonectomy. An attempt was made to dilate the stricture by balloon catheter. Although there was little increase in the size of the bronchial lumen immediately after the procedure, the patient has remained well over the last 4 years. The severe stenosis has probably developed very gradually over many years.

Case 4

A 59-year old male presented in 1987 with cough for 2 months. Chest X-ray showed far advanced pulmonary tuberculosis (Fig 3a). Sputum smear was positive for AFB. Antituberculosis chemotherapy was started with streptomycin, isoniazid, rifampicin and pyrazinamide. He was admitted 3 weeks later for severe dyspnoea and stridor of 3 days' duration. Overpenetrated view of the trachea and main bronchi showed narrowing of the lower end of the trachea. Tomogram of the trachea showed significant narrowing at the level of T2-3 (Fig 3b).

He was given hydrocortisone 200 mg 6 hourly followed by prednisolone 10 mg tds, CT scan of thorax was done

Fig 2a Chest X-ray showing consolidation of right lower zone.



Bronchogram showing stricture of the right main bronchus near its origin with bronchiectasis of the lower and middle lobes and mild bronchiectasis of the upper lobe.



when he was less dyspnoeic. It showed severe narrowing of 1 cm length in the lower end of the trachea. Bronchosocpy showed severe concentric narrowing of the tracheal wall.

He was given isoniazid and rifampicin for a total of 7 months supplemented with pyrazinamide for 8 weeks. Streptomycin was stopped after 4 weeks because of pain. Prednisolone was given for a total of 8 weeks. His sputum converted after 3 months and chest X-ray showed good radiological resolution. He was able to lead an independent life. However, he continued to be breathless when he had to walk rapidly but did not want to undergo resectional surgery for his tracheal stenosis.

Fig 3a Chest X-ray showing far advanced PTB involving both lungs.



Fig 3b Tomogram of the trachea showing severe narrowing of the lumen at level of T2-T3 vertebra.



Case 5

A 56-year-old Sikh female presented in 1988 with cough and dyspnoea on exertion for 2 months.She had no fever,

hemoptysis or weight loss. Air entry was diminished at the right lung base. Sputum smear examination was negative for AFB. Chest X-ray showed collapse and consolidation of the middle and lower lobes of the right lung (Fig 4). Bronchoscopy showed obstruction of the right intermediate bronchus by granulation tissue which looked like tumour tissue. Biopsy of the bronchial tissue showed acid fast bacilli. She was treated with ethambutol, isoniazid and rifampicin for 2 months followed by isoniazid and rifampicin. Sputum culture was subsequently reported to be positive for AFB. She responded well to treatment. Chest X-ray showed re-expansion of the collapsed lobes after 2 months of therapy.

Fig 4 Chest X-ray showing collapse and consolidation of right middle and lower lobes.



DISCUSSION

Tuberculosis involving the tracheobronchial tree is not uncommon. In the 1940s, Auerbach reported an incidence of 42% in 1000 autopsies⁽¹⁾ while Salkin found an incidence of 15.5% in 622 patients examined by rigid bronchoscopy⁽²⁾. A recent study of smear negative PTB by fibreoptic bronchoscopy reported an incidence of 18%⁽³⁾.

The cases reported herein illustrate the diverse manifestations of EBTB. A persistent cough is the main symptom, while the other presenting features include lobar or whole lung collapse, unresolved pneumonia and stridor. The stridor in Case 4 was due to stricture of the trachea. Although bronchial stenosis may give rise to localized wheezing, it was not a feature in the present series. The other symptoms which have deen reported include fever, hemoptysis and excessive production of foul smelling sputum when there is associated bronchiectasis.

Diagnosis can be difficult. The sputum smear may be negative for AFB as shown in Cases 1 and 5 and the chest X-ray may not show any abnormality even though bronchial wall disease is present⁽⁴⁾. Occasionally, the sputum smear may be positive for AFB when the chest X-ray is normal. The diagnosis of bronchial stenosis may not be suspected as it is not often appreciated that this complication may develop many years after successful chemotherapy⁽⁵⁾ as illustrated by Case 2 and Case 3.

The most important procedure in the diagnosis of EBTB is bronchoscopy. The bronchial wall abnormalities seen at bronchoscopy range from mucosal redness and swelling to shallow or deep ulcerations and granulation tissue, which can progress to fibrosis and stenosis. During bronchoscopy, bronchial aspirate can also be collected and examined for AFB by smear and culture. Smear examination of the bronchial aspirate for AFB gave a higher diagnostic yield compared with that of sputum smear examination⁽⁶⁾.

In patients with bilateral lung lesions, endobronchial disease was found on the side with cavitation⁽¹⁾. This was attributed to the discharge of AFB from the carvity and its subsequent implantation in the brochial mucosa. The frequent involvement of the lower lobes where secretions tend to be retained, also supports the implantation theory. Other modes of spread, especially in patients with no lung cavitation, include direct extension from a parenchymal focus or spread to the bronchi from a parenchymal focus by lymphatics, erosion of a lymph node into the bronchial lumen and rarely, haematogenous spread.

Endobronchial tuberculosis can masquerade as a lung cancer (7) especially when a patient, who is also a heavy smoker, presents with a segmental or lobar collapse. The tuberculous granulation tissue may resemble a malignant tumour even under bronchoscopic examination. Therefore it is important to look for AFB histologically and bacteriologically in the bronchial biopsy specimen.

Tomography and CT scan can also help to visualise and determine the extent of stenosis in the trachea and main stem bronchi. Underlying bronchiectasis can be diagnosed by doing a bronchogram as shown by Case 3.

Stenosis can be treated by resection followed by anastomosis or sleeve resection. The role of steroid therapy in preventing stenosis is uncertain with no beneficial effect reported in one study⁽⁶⁾. The effect of steroids may depend on the stage and pathogenesis of the endobronchial lesion. Steroids have been reported to be effective when given to patients who developed endobronchial disease during chemotherapy^(8,9). The endobronchitis is believed to be due to a hypersensitivity reaction to tuberculoprotein released by the killing of mycobacteria. The reaction is suppressed by steroids. In recent years, various methods of treating bronchial or tracheal stenosis (of different aetiology) have been reported such as balloon catheter dilatation, taser therapy or both with encouraging results⁽¹⁰⁻¹²⁾.

CONCLUSION

Endobronchial tuberculosis is important to recognise as it may lead to severe stenosis of the main stem bronchi or trachea. The diagnosis should be suspected and bronchoscopy performed when a patient has unexplained cough, wheezing, hemoptysis, atelectasis or radiological evidence of airway obstruction such as hyperinflation, localised obstructive emphysema, presence of a tensioncyst or fluctuation in the size of a cavity (13). A negative sputum smear and a normal chest X-ray do not exclude the presence of endobronchial tuberculosis. Surgery can be offered to patients with stenosis of the large airway.

REFERENCES

- 1. Auerbach O. Tuberculosis of the trachea and major bronchi. Am Rev Tuberc 1949; 60: 604-20.
- 2. Salkin D, Cadden AV, Edson RC. The natural history of tuberculosis tracheobronchitis. Am Rev Tuberc 1943; 47: 351-69.
- 3. So SY, Lam WK, Yu DYC. Rapid diagnosis of suspected pulmonary tuberculosis by fibreoptic bronchoscopy. Tubercle 1982; 63: 195-200
- 4. Volckaert A. Endobronchial tuberculosis: report of three cases. Eur J Respir Dis 1987; 99-101.
- 5. Albert RK, Petty TL. Endobronchial tuberculosis progressing to bronchial stenosis. Fibreoptic bronchoscopic manifestations. Chest 1976; 70 : 537-9.
- 6. Ip MSM, So SY, Lam WK, Mok CK. Endobronchial tuberculosis revisited. Chest 1986; 89 (5): 727-30.
- Matthews JI, Matarese SL, Carpenter JL. Endobronchial tuberculosis simulating lung cancer. Chest 1984; 86: 642-4.
- Williams DJ, York EL, Nobert EJ, Sproule BJ. Endobronchial tuberculosis presenting as asthma. Chest 1988; 93: 836-8.
- 9. Chan HS, Pang JA. Effect of corticosteroids on deterioration of endobronchial tuberculosis during chemotherapy. Chest 1989; 96: 1195-6.
- 10. Cohen MD, Weber TR, Rao CC. Balloon dilatation of tracheal bronchial stenosis. ARJ 1984; 142: 477-8.
- 11. Carlin BW, Harrell JH, Moser KM. The treatment of endobronchial stenosis using balloon catheter dilatation. Chest 1988; 93: 1148-51.
- 12. Liu AC, Mehta AC, Golish JA. Upper airway obstruction due to tuberculosis. Treatment by photocoagulation. Postgrad Med 1985; 78, 2: 275-8.
- 13. Barsby BE. Treatment of lung cavitives and endobronchial tuberculosis. Edinburgh and London: E & S Livingstone, 1959.