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Endobronchial tuberculosis – is corticosteroid treatment useful? – a report of 8 cases and review of the literature

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Summary: Eight patients with endobronchial tuberculosis diagnosed on bronchoscopy were treated with antituberculosis drugs and a course of prednisone. The progress of the endobronchial lesions was assessed on repeated examinations. The course of the disease was variable and the endobronchial narrowing improved in two patients only. Hypersensitivity reactions associated with initiation of antituberculosis treatment may constitute a special group where corticosteroid is indicated. In other situations, the usefulness of corticosteroid for treatment of endobronchial tuberculosis is not well documented.

Introduction

Endobronchial tuberculosis, while relatively common before the advent of effective antituberculosis chemotherapy,[1] is infrequently encountered nowadays. The pathogenesis of the disease is unclear. It may result from rupture of caseous material from lymph nodes, lymphatic spread along the bronchial tree, haematogenous spread or direct implantation of tubercle bacilli from parenchymal focus. The presentation of the disease is variable. It may be acute, simulating asthma[2] or foreign body aspiration,[3] insidious, simulating lung cancer,[4] or delayed, appearing for the first time long after completion of antituberculosis treatment.[5] The course and prognosis of the disease is as variable, ranging from complete clearance to severe bronchostenosis.[6] Although corticosteroid has been given empirically in an attempt to prevent fibrosis, the value of corticosteroid in the treatment of endobronchial tuberculosis is uncertain.

Because short courses of corticosteroid are 'safe' under the cover of antituberculosis drugs and the consequences of bronchostenosis may be serious, we are inclined to give corticosteroid to all cases of endobronchial tuberculosis with significant involvement of the major bronchi although we are not certain of its value. Furthermore, the number of cases of endobronchial tuberculosis is too small and the clinical features too variable to allow proper randomization of corticosteroid treatment. It is the purpose of the present study to follow the progress of eight cases of endobronchial tuberculosis treated with corticosteroid and to evaluate its usefulness in the prevention of bronchostenosis.

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Patients and methods

Eight cases of endobronchial tuberculosis of the major bronchi were diagnosed between January 1988 and December 1989 in Prince of Wales Hospital and Haven of Hope Hospital in Hong Kong. Endobronchial tuberculosis involving the subsegmental bronchi were excluded because of the inaccuracy in assessing the progress of these small lesions. Half of the patients were male. Their mean age was 29 years with a range from 18 to 50. The clinical, radiological and bronchoscopy features of the 8 cases are summarized in Table I.

The diagnosis of endobronchial tuberculosis was established with the combination of: (1) significant airway inflammation and/or narrowing seen on bronchoscopy, preferably with histological confirmation of tuberculous involvement, and (2) positive culture of tubercle bacilli in sputum or bronchial aspirate.

After treatment with antituberculosis drugs for the initial 2 to 6 weeks, all cases were given prednisone 0.75 mg/kg tapering over 4 to 6 weeks in an attempt to prevent the development of bronchostenosis. The progress of the endobronchial lesions was assessed on repeated radiological and bronchoscopic examinations and lung function testing. Bronchoscopy was performed for 2 to 4 times at 3 to 6 month intervals with the Olympus BF-P10 fibreoptic bronchoscope. When they had been put on antituberculosis treatment and sputum was smear negative for tubercle bacilli, resting lung function tests were measured with the dry bellow spirometer (Vitalograph). The duration of follow up was between 8 weeks and 2 years. Patient 6 was assessed for 8 weeks only because she underwent a

Table I Clinical, radiological and bronchoscopic features of the 8 cases of endobronchial tuberculosis

Patient number	*Sex/Age*	*Clinical features*	*Duration of symptoms*	*Bacteriological confirmation*	*Radiological description*	*Indication for bronchoscopy*	*Bronchoscopic description*	*Bronchial biopsy*
1	F/18	Cough	6 months	Sputum AFB S,C +ve	Left lung shadowing	Localized wheeze	Pinhole narrowing of LMB	Not done
2	M/26	Cough	10 days	Bronchial aspirate C +ve	RLL consolidation	Persistent pneumonia	Nodular lesion on RMB & RLL bronchus	Granuloma ZN stain −ve
3	F/23	Cough, weight loss	8 weeks	Sputum S,C +ve	RML consolidation	Atypical site of tuberculosis	Narrowing of RML bronchus	Granuloma ZN stain +ve
4	M/50	Cough, weight loss	6 weeks	Sputum S,C +ve	LUL cavitation	No improvement after treatment	Narrowing of LUL bronchus	Granuloma ZN stain −ve
5	M/26	Cough, chest pain	4 weeks	Sputum S,C +ve	LUL consolidation	Suspicion of left hilar shadow	Narrowing of LMB	Chronic inflammation ZN stain+ve
6	F/28	Cough, dyspnoea	6 months	Sputum S,C +ve	Collapsed left lung	LMB obstruction	Narrowed LMB with caseous material	Caseating granuloma ZN stain +ve
7	F/20	Cough	4 months	Sputum S,C +ve	Left lung shadowing	LMB obstruction	Narrowed LMB	Acute inflammation ZN stain +ve
8	M/41	Cough	2 weeks	Sputum S,C +ve	Bilaterial upper zone infiltrate	Collapse of RUL after treatment	Inflamed RUL	Granuloma ZN stain −ve

AFB - acid fast bacilli, S - smear, C - culture, ZN - Ziehl Neelsen, RMB - right main bronchus, RUL - right upper lobe, RML - right middle lobe, RLL - right lower lobe, LMB - left main bronchus, LUL - left upper lobe.

left pneumonectomy subsequently for uncontrolled infection.

Results

The final outcome of the endobronchial lesions is summarized in Table II. Pinhole size narrowing of the left main bronchus was seen in patient 1 at presentation. Bronchial biopsy was not done at that time for fear of worsening the bronchial narrowing. She was treated with prednisone for 6 weeks and antituberculosis drugs for 7 months, but repeated bronchoscopy over 2 years showed no change in the bronchostenosis (Figure 1). Patient 6 had severe narrowing of the left main bronchus, just admitting the 4.8 mm diameter fibreoptic bronchoscope. She underwent left pneumonectomy because of difficulty in clearing the infection in the left lung (Figure 2) despite adequate antituberculosis treatment and sensitivity of the tubercle bacilli to the first line antituberculosis drugs. Patient 7 had inflammation but little narrowing of the left main bronchus seen on bronchoscopy at presentation. A course of prednisone and antituberculosis drugs for 6 months was given. There was gradual resolution of the radiological abnormality. However, she presented 18 months later with recurrent pneumonia. Chest radiograph then showed collapsed left lung and bronchoscopy revealed pinhole size narrowing of left main bronchus. Bronchial biopsy and aspiration showed no evidence of reactivation of tuberculosis. After clearing of mucosal oedema, patients 3, 4 and 5 were left with a moderate degree of bronchial narrowing. Patients 2 and 8 had the shortest duration of symptoms at presentation, 10 and 14 days respectively. Both of them had marked resolution of the endobronchial lesions. Patient 2 probably had a hypersensitivity reaction to the antituberculosis drugs and the usefulness of corticosteroids in such circumstances has been described previously.[7] The role of corticosteroid in patient 8 was not clear.

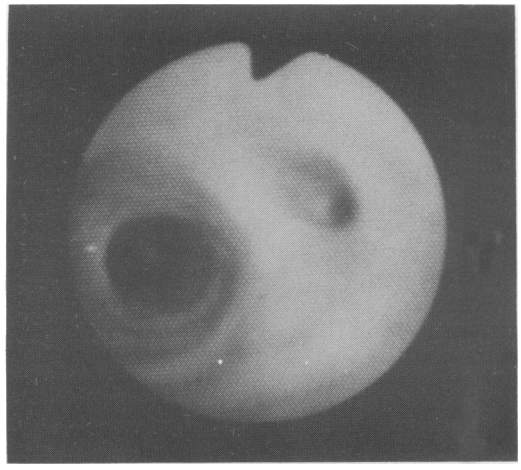

Figure 1 Bronchoscopy of patient 1 showing persistent bronchostenosis of the left main bronchus just distal to the carina.

The lung function tests of all the patients exhibited a restrictive lung defect with a high forced expiratory volume in one second (FEV_1)/forced vital capacity (FVC) ratio (range 81.9 to 99.3% of predicted). Measurement of FVC appeared to be a good indicator of their ventilation defect. The initial and latest FVC measurements of the patients are depicted in Table II.

At completion of treatment, patient 1 complained of exertional dyspnoea and diminished exercise tolerance. Patient 4 had a mild degree of dyspnoea probably as a result of the concomitant parenchymal fibrosis from tuberculosis involvement. Patient 7 had been admitted twice to hospital because of recurrent chest infections. The other five patients were asymptomatic.

Table II Final outcome of the 8 cases of endobronchial tuberculosis

Patient number	*Duration of follow up*	*FVC (% pred) Initial*	*FVC (% pred) Final*	*Outcome*
1	2 years	56	42	Pinhole narrowing of LMB
2	1 year	70	71	Little residual stenosis
3	8 months	77	84	Moderate narrowing RML bronchus
4	7 months	48	49	Moderate narrowing LUL bronchus
5	7 months	29	31	Moderate narrowing LUL bronchus
6	8 weeks	35	Not done	Left pneumonectomy done
7	18 months	Not done	57	Pinhole narrowing of LMB
8	5 months	56	67	Much improved in RUL bronchus inflammation and narrowing

LMB - left main bronchus, LUL - left upper lobe, RUL- right upper lobe, RML - right middle lobe.

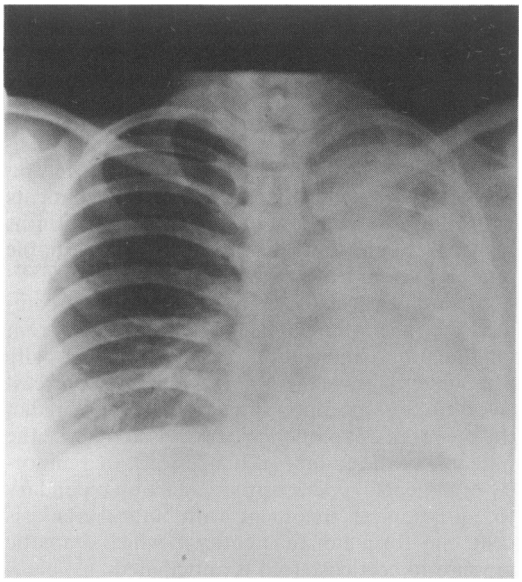

Figure 2 Chest radiograph of patient 7 shows failure of resolution of consolidation of left lung despite adequate antituberculosis treatment.

Discussion

In the early era of antituberculosis chemotherapy, tuberculosis of the trachea and major bronchi was common and a vast number of cases were described.[8-10] However, with the improvement in living standards and medical care, and the advent of BCG vaccination and effective antituberculosis drugs, the incidence of endobronchial tuberculosis has become much less common. On reviewing the literature, just over 50 cases of tuberculosis involving the trachea and major bronchi have been reported in the last decade (Table III). The largest series was that of Ip *et al.*[6] with 20 cases collected from 1978 to 1985, but only 11 of their patients could be recalled for assessment of residual bronchostenosis. Out of 52 reported cases of endobronchial tuberculosis, only 11 had been given a corticosteroid. Improvement of the endobronchial lesion was noted in 4 patients in the group treated with corticosteroid. It was not known if the improvement in those 4 cases was due to corticosteroid or natural course of the disease process. The information in the group not given corticosteroid was inadequate to assess the final outcome. Therefore, the usefulness of corticosteroid remains unsettled.

Corticosteroid as prednisone was given in all 8 subjects reported here. The present study agrees with previous reports that the course of endobronchial tuberculosis is highly variable and the effect of corticosteroid is difficult to predict. Hypersensitivity reaction associated with antituberculosis treatment is a distinct situation where corticosteroid is of potential benefit. Four cases of endobronchial tuberculosis belonging to this category have been reported.[2,7] The condition is characterized by acute deterioration after initiation of antituberculosis treatment, probably related to the killing of mycobacteria and release of cell wall material causing an acute hypersensitivity reaction. Similar examples are the enlargement of lymph nodes in tuberculous adenitis and the development of intracranial tuberculoma on antituberculosis treatment.[18] In other situations, the effect of corticosteroid is less well documented.

The heterogenicity in presentation and response to treatment in endobronchial tuberculosis is not

Table III Review of 52 cases of endobronchial tuberculosis of trachea or major bronchi reported in the last decade and their outcome with reference to the use of corticosteroid

Author/year	*Number of patients*	*Use of corticosteroid*	*Outcome*
Lynch *et al.*[11] 1980	1	No	Improved
Seiden *et al.*[12] 1981	1	No	Residual stenosis
So *et al.*[13] 1983	1	Yes	Unknown
Matthews *et al.*[4] 1984	4	No	Unknown
Ip *et al.*[6] 1986	20	3 patients	Residual stenosis in all except one case not given corticosteroid
Wathen *et al.*[14] 1987	1	Yes	No residual stenosis
Volchaert *et al.*[15] 1987	3	No	No residual stenosis in one, pneumonectomy in one, unknown in one
Watson *et al.*[16] 1988	1	Yes	No residual stenosis
Williams *et al.*[2] 1988	2	Yes	No residual stenosis
	1	No	Lost to follow up
Caglayan *et al.*[3] 1989	1	No	Improved
Kalyoncu *et al.*[17] 1989	15	3 patients	Unknown
Chan *et al.*[7] 1989	1	Yes	Improved

surprising if we consider the complexity of its pathogenesis.[1] The tuberculous process starts as small foci in the subepithelial portion of the wall, surrounded by a zone of vascular granulation tissue. Bronchial stenosis is the result of a chronic tuberculous process with a combination of destruction and repair. In those cases of chronic endobronchial tuberculosis with abundant fibrous tissue formation, corticosteroid is unlikely to be helpful. In cases where airway narrowing is predominantly due to inflammatory cells and oedematous fluid accumulation, corticosteroid may facilitate the clearing of inflammation and improve the airway narrowing. A corollary of the effect of corticosteroid on endobronchial tuberculosis may be observed in the treatment of tuberculous pleural effusion. Corticosteroid hastens the initial clearing of pleural fluid but has no effect on residual pleural thickening.[19]

It is difficult to predict from clinical, radiological or bronchoscopic findings whether the airway narrowing is predominantly due to fibrosis or inflammation. We may postulate that patients with the longer duration of symptoms (e.g. patients 1 and 6) have greater degree of tissue destruction and subsequently worse prognosis. In patients with the shorter duration of symptoms (patients 2 and 8) most of the inflammatory changes may be reversible and hence have better prognosis. However, on reviewing the literature, there were two patients with duration of symptoms for 5 and 8 months respectively and both of them had a favourable outcome on antituberculosis treatment alone.[11,15]

In summary, endobronchial tuberculosis comprises a spectrum of disorders ranging from inflammatory infiltration, which is potentially reversible, to permanent narrowing due to excessive fibrous tissue formation. It is difficult to predict the predominant pathogenic process and hence the outcome of the endobronchial lesions. In a minority of patients, hypersensitivity reaction secondary to initiation of treatment with antituberculosis drug may form a distinct entity in which dramatic reponse to corticosteroid is anticipated.

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