The Journal of Laryngology and Otology
June 1998, Vol. **112**, pp. 579–580

Primary tuberculous tracheitis

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Abstract
We report a case of primary tuberculous tracheitis in an otherwise healthy woman who presented with cough and stridor due to diffuse tracheal narrowing by tuberculous pseudomembranous lesion, which resolved completely with antituberculosis chemotherapy.

Key words: Tuberculosis; Trachea

Case report

A 50-year-old woman was referred to our department by her physician for acute onset of hoarseness, stridor and fever following three months' duration of productive cough. She was a nonsmoker and she had no past history of pulmonary tuberculosis. Indirect laryngoscopy showed mildly oedematous but mobile vocal folds with yellowish membranous tissue extending from the subglottis downwards. There was stridor at rest, and rhonchi in both lung fields. The lateral view of the neck X-ray showed a markedly narrowed trachea, (Figure 1), while the chest X-ray was normal. Sputum smear for acid-fast bacilli had been taken for three consecutive days and all were reported negative.

In view of her respiratory distress and X-ray findings, an emergency tracheostomy was carried out; a tracheostomy tube with a 6 mm inner diameter was inserted, followed by microlaryngoscopy. On microlaryngoscopy, yellowish pseudomembranous necrotic tissue was detected from the subglottis to the anterior and lateral walls of the upper trachea; the lumen above the tracheostomy site was markedly narrowed. Necrotic tissue in the trachea was removed as much as possible to widen the tracheal lumen, revealing inflamed tracheal mucosa.

The tissue section showed necrotic tissue with fibrin and polymorphs only. Tissue smears for bacteria, fungi, and acid-fast bacilli were all negative. the white blood cell count was 21.3×10^9/l, predominantly due to raised neutrophils, and the erythrocyte sedimentation rate was 56 mm per hour. The patient had been started on amoxycillin plus potassium clavulanate empirically by her physician and this was therefore continued.

The patient's general condition apparently improved. Her temperature and white cell count returned to normal in two weeks. Bronchoscopy and rebiopsy were carried out two weeks later. There was some granulation on the tracheal wall; the carina, main bronchi and lobar bronchi were clear. Tissue was again taken for histology and this time it showed vascular granulation tissue with ulcerated inflamed mucosa. There were some epithelial atypia and a few poorly formed granuloma with occasional giant cells. However, stains for acid-fast bacilli and fungus were negative.

Three weeks after admission, her erythrocyte sedimentation rate returned to normal, her airway improved and the tracheostomy tube was removed. She was then discharged home. The provisional diagnosis at this stage was acute bacterial tracheitis.

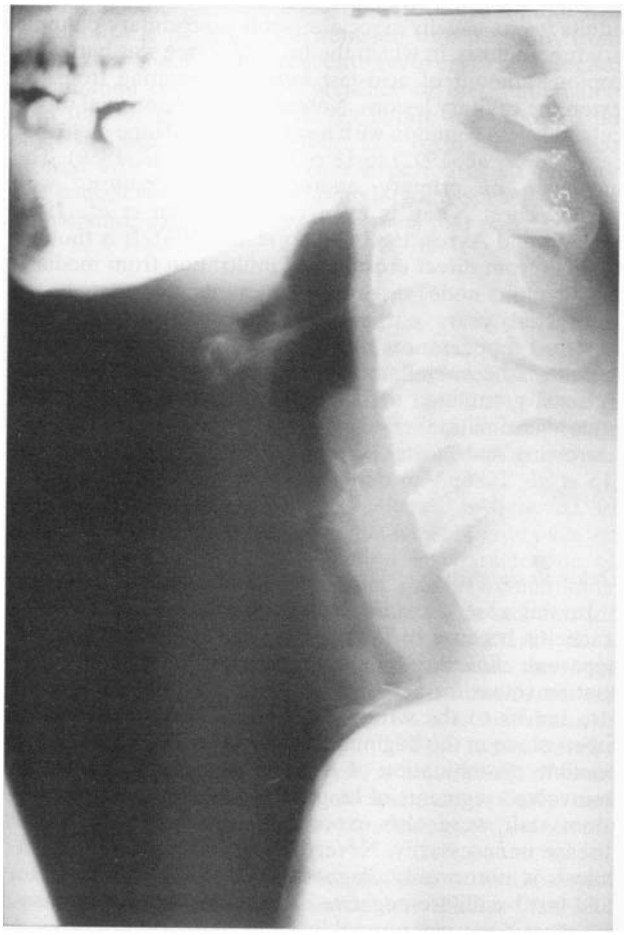

FIG. 1
X-ray neck. The trachea was diffusely narrowed due to a tuberculous pseudomembrane.

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Accepted for publication: 15 April 1998.

The patient was followed up one month later with flexible bronchoscopy that showed some granulation at the anterior tracheal wall but the tracheocutaneous fistula kept on discharging. Six weeks later tissue culture confirmed tuberculosis and the discharge smear from the tracheocutaneous fistula was also positive for acid-fast bacilli. The final diagnosis was tuberculous tracheitis.

She was started on isoniazid 300 mg daily, rifampicin 450 mg daily, pyrazinamide 1.5 gm daily, ethambutal 1.2 gm daily for two months followed by rifampicin and isoniazid for three more months. The tracheocutaneous fistula healed five months after the start of antituberculous chemotherapy. Sputum smears and culture for acid-fast bacilli after completion of chemotherapy were negative. Bronchoscopy two months after completion of chemotherapy showed a normal trachea with no residual stenosis. She remained asymptomatic two years after completion of chemotherapy. The last bronchoscopy which was performed two years after recovery showed only small crescent-shaped stenosis at the right anterolateral tracheal wall.

Discussion

Uncommon presentation and pathogenesis

Endobronchial tuberculosis i.e. involvement of the trachea and/or major bronchi was not uncommon in the pre-antibiotic era and was found to occur in 60 per cent of patients dying from cavitary pulmonary tuberculosis. In adults it was usually associated with postprimary pulmonary tuberculosis, in which the bronchial tree was bathed in copious amount of acid-fast bacilli, originating from the extensive cavitary lesions. Nowadays endobronchial tuberculosis is less common with a reported incidence of 4.1 per cent (Lee *et al.*, 1992) to 18 per cent (Ip *et al.*, 1986). The incidence of primary endotracheal tuberculosis with normal chest X-ray is even rarer. Wathen *et al.*, 1987; Watson and Ayres, 1988; Dilkes *et al.*, 1993). It is thought to arise from direct erosion and infiltration from mediastinal lymph node disease where the dormant bacilli contracted years earlier have regained activity. The reported appearance of tracheobronchial tuberculosis includes mucosal inflammation, cobblestone mucosa, submucosal granuloma and polyps, ulceration, white gelatinous granulation tissue, hypertrophy with luminal narrowing and cicatricial stenosis with pseudomembrane (Ip *et al.*, 1986; Van den Brande *et al.*, 1990; Lee *et al.*, 1992).

Delay in diagnosis

In this case the initial diagnosis was acute bacterial tracheitis because of the raised neutrophil count and the apparent clinical response to amoxycillin. The negative sputum smear for acid-fast bacilli and normal chest X-ray also led us to the wrong diagnosis. Failure to recognize tuberculosis in the beginning led to delay in treatment and possible dissemination of disease by ventilation into the uninvolved segments of lungs. The surgeon and operating room staff were also exposed to this highly infectious disease unnecessarily. Nevertheless, endobronchial tuberculosis is notoriously diagnosed late. Sputum smears for acid-fast bacilli are negative in 45 to 85 per cent of cases; the chest X-ray was normal in 8.3 to 20 per cent of cases. A wrong diagnosis of bronchogenic carcinoma has been made in 30 per cent of patients (Ip *et al.*, 1986; Van den Brande *et al.*, 1990; Lee *et al.*, 1992). Hence the lesson to learn is that a negative sputum smear and clear chest X-ray cannot exclude endobronchial tuberculosis.

Complication of tracheal stenosis

Endobronchial tuberculosis frequently heals with concentric scarring and permanent fibrous tracheal stenosis. Extension of the stenosis into the main, lobar and segmental bronchi may result in atelectasis and secondary pneumonia. Ip *et al.* found variable degrees of bronchial stenosis in 11 out of 12 patients, with a mean follow-up 27 months after completion of antituberculosis chemotherapy (Ip *et al.*, 1986).

The effect of steroids in preventing late stenosis was disappointing (Chan *et al.*, 1990; Van den Brande *et al.*, 1990). Residual bronchostenosis was found in 23 patients out of 44 treated with steroid (Lee *et al.*, 1992). It is likely to be beneficial in the early stages but unlikely to be helpful in late advanced cases when extensive fibrosis is present. The extent of endobronchial involvement may be related to late stenosis. Kim *et al.* classified four types of endobronchial tuberculosis: exudative, ulcerative, cicatricial and bronchoglandular. In the first three types, administration of antituberculosis chemotherapy alone resulted in a favourable response in the early post-treatment period, without residual stenosis (Kim *et al.*, 1993).

In this patient with an exudative lesion, the prognosis is favourable. Only a mild crescent-shaped stenotic web has been observed so far, however, regular follow-up with bronchoscopy is advised as stenosis may progress requiring endoscopic dilatation, laser excision or tracheobronchoplastic procedures.

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