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Hoarseness in tuberculosis

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Summary

A study has been made of hoarseness in 26 tuberculous patients in Kandy (Sri Lanka). Tuberculous inflammation of the vocal cord or ventricular fold was its cause in 18 of them, in 17 of whom the laryngeal infection was due to direct implantation of tubercle bacilli from lung cavities through cough; in one it was probably haematogenous. Hoarseness in the other eight patients was due to recurrent laryngeal nerve palsy, the nerve being involved in tuberculous lesions, in six instances in the mediastinum and in three instances in the right apical pleura. However caused, hoarseness associated with active inflammatory disease cleared with anti-tuberculous chemotherapy, whereas that associated with fibrosis persisted.

Introduction

Hoarseness of voice is the commonest symptom of tuberculous involvement of the larynx, the causes of which include inflammation of the vocal cord or ventricular fold or cricoarytenoid joint, and lesions in the recurrent laryngeal nerve from fibrosis at the apex of the lung or pressure from mediastinal lymph nodes (Ormerod, 1939). The existence of tuberculous lesions in the larynx was thought, at one time, to reduce the patient's chance of survival by a half, but the use of anti-tuberculous drugs after 1944 produced dramatic improvement in these lesions (Ormerod, 1954). With the advent of effective anti-tuberculous chemotherapy, more and more tuberculous patients continue to live with extensive fibrosis of the lung or mediastinum. Hoarseness of voice which results from this fibrosis is not amenable to anti-tuberculous chemotherapy, as shown in this study of the pathogenesis and progress with anti-tuberculous chemotherapy of tuberculous lesions of the larynx, in 26 patients presenting with hoarseness in Kandy, Sri Lanka.

Material and Method

From January 1976 to December 1980, 26 patients with hoarseness of voice associated with tuberculosis were referred to the Chest Clinic, Kandy, from the Ear, Nose and Throat Department of the General Hospital, Kandy. Twenty-two of them were males and four females. Their age range was 17 to 71 years. The duration of hoarseness was from two weeks to six months. All of them had the following examinations done on them: laryngoscopy, chest X-ray, sputum for acid-fast bacilli (AFB) and Mantoux test. Eighteen of them had biopsies of the laryngeal lesions. Bronchial carcinoma was excluded by bronchoscopy. Syphilis was excluded by serological examination. All 26 patients were treated with streptomycin 750 mg. daily, isoniazid 300 mg. daily and para-aminosalicylic acid 5 g. bd. for three months followed by prolonged therapy with

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streptomycin 1 g. and isoniazid 700 mg. twice a week. The follow-up period was from seven months to two years. For the present study the laryngoscopic findings were correlated with the clinical types of tuberculosis in these patients and with the response of their hoarseness to anti-tuberculous chemotherapy.

Results

Eighteen patients had active forms of chronic pulmonary tuberculosis (CPT), as shown by the presence of cough, X-ray changes (confluent, ill-defined and ring shadows) and AFB in the sputum. Their Mantoux test was positive and they had no previous history of anti-tuberculous chemotherapy. Six patients, who had been treated earlier for sputum-positive CPT with adequate anti-tuberculous chemotherapy, presented with hoarseness of voice for the first time long after anti-tuberculous drugs had been stopped. They had arrested CPT, as shown by the absence of cough, absence of AFB in the sputum and presence of X-ray evidence of fibrosis (shifting of the mediastinum, raised hila and dense streaky shadows in the upper zones). Their Mantoux test was positive. One patient had primary tuberculosis with X-ray evidence of enlarged mediastinal lymph nodes, strongly positive Mantoux and sputum negative for AFB. She had no previous history of anti-tuberculous chemotherapy. One patient had no radiological evidence of pulmonary or mediastinal disease. She had a strongly positive Mantoux, her sputum was negative for AFB, and she too had had no previous anti-tuberculous chemotherapy.

Of the 18 patients with active CPT, 17 had swelling and/or ulceration of the vocal cord or ventricular fold confirmed as tuberculous by histological examination, and one had immobility of the right vocal cord. Of the six patients with arrested CPT, four had immobility of the left vocal cord, one had immobility of the right vocal cord, and one had immobility of both vocal cords. The patient with primary tuberculosis and enlarged mediastinal lymph nodes had immobility of the left vocal cord. The immobile vocal cord in all eight patients was in abduction. The patient with no radiological evidence of pulmonary or mediastinal disease had a swelling of the vocal cord confirmed as tuberculous by histology.

Hoarseness associated with swelling and/or ulceration of the vocal cord or ventricular fold in all 18 patients cleared completely in two weeks to one month of therapy. The hoarseness associated with immobility of the right vocal cord in the man with active CPT, and that associated with immobility of the left vocal cord in the woman with enlarged mediastinal lymph nodes, cleared after two months and five months respectively of anti-tuberculous chemotherapy. At repeat laryngoscopy at that stage movements of the vocal cords in these two cases were normal. Hoarseness in the six patients with immobile vocal cords and arrested CPT persisted despite continued administration of anti-tuberculous chemotherapy. The immobility of the vocal cord was unaffected by therapy in these six cases.

Discussion

Tuberculous inflammation of the vocal cord or ventricular fold accounted for hoarseness in 18 patients (69 per cent) in this series. In 17 of these with active CPT, the infection of the larynx had taken place by the well-recognized method of direct implantation of tubercle bacilli coughed up from cavities in the lung (Hudson, 1957). But in one patient with no radiological evidence of pulmonary or mediastinal disease, the mode of infection was probably by the rare haematogenous route (Hudson, 1957).

The immobility of the vocal cord recorded in eight patients could have been due to recurrent laryngeal nerve (RLN) palsy (Ormerod, 1939; Gupta, 1959; DeWeese and Saunders, 1968; Farmer, Fulkerson and Stein, 1975), or due to fixation of the cricoarytenoid joint by tuberculous inflammation (Ormerod, 1939). The abducted position of the immobile vocal cord and the

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absence of tuberculous involvement of the arytenoid process in all eight cases suggested RLN palsy as the cause of the immobility (Ormerod, 1939). As mentioned by Gupta (1959), RLN palsy is not a rarity in tuberculosis, and in this series it occurred in 31 per cent of cases with hoarseness. In the five cases reported by Gupta (1959) and the two cases reported by Farmer *et al.* (1975), only the left RLN was involved. In this series the left RLN alone was affected in five cases, the right RLN alone was affected in two cases, and both RLN were affected in one case. The cause of the left RLN lesion was fibrosis of the mediastinum associated with CPT (Perry, 1952) in five cases with arrested CPT. In the patient with primary tuberculous mediastinal lymphadenitis the cause of the left RLN lesion was periadenitis associated with lymphadenitis (Gupta, 1959). Inflammatory or fibrosing lesions in the right apical pleura could have damaged the right RLN (DeWeese and Saunders, 1968). In the man with active CPT who had an immobile right vocal cord which regained its mobility with therapy, the inflammatory lesion at the right apical pleura had resolved with treatment. In the other two cases with permanent immobility of the right vocal cord, the right apical lesion had gone on to fibrosis.

Hoarseness of voice due to inflammation of the vocal cord or ventricular fold, following active CPT or haematogenous spread, cleared completely with anti-tuberculous chemotherapy. Likewise hoarseness due to immobility of the right vocal cord associated with the active form of CPT, and that due to immobility of the left vocal cord associated with enlarged mediastinal lymph nodes, also cleared completely with anti-tuberculous chemotherapy. In these 20 patients, hoarseness was the result of active inflammatory disease, but hoarseness due to immobility of the vocal cord, in all six patients with arrested CPT, persisted as it resulted from fibrosis either in the mediastinum or in the right apical pleura.

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