

Concurrent Tuberculosis of the Larynx and the Tonsil

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Tuberculosis of the larynx and the tonsil were not relatively uncommon diseases in the past, but they have become rare diseases with the advent of effective antituberculous chemotherapy. Especially cases secondary to tuberculosis elsewhere in the body are indeed uncommon. We have recently seen a case of tuberculosis of the larynx associated with that of the tonsil which is thought to have developed secondary to miliary tuberculosis.

Key Words: Laryngeal tuberculosis, tonsillar tuberculosis, miliary dissemination.

The incidence of tuberculosis of the larynx and the tonsil has been greatly reduced with chemotherapy, although it has not been completely eradicated. Laryngeal and tonsillar tuberculosis may be secondary to tuberculous lesions elsewhere in the body, especially the lung, or a primary affection from inhaled tubercle bacilli. Most authorities agree that the former is the usual route and the latter is very rare.

Rohwedder (1974) has described that recent chemotherapeutic advances have reduced the incidence of upper aero-digestive tract tuberculosis so dramatically that tuberculosis is usually forgotten when patients present with otolaryngological symptoms, for example, hoarseness, sore throat, odynophagia, otalgia and otorrhea etc.

This paper presents a case of concurrent tuberculosis of the larynx and the tonsil which is suspected to be secondary to miliary tuberculosis and a brief review of the literature.

CASE REPORT

A 20 year old female was admitted to the hospital in March 1986 with the chief complaint of sore throat and hoarseness. She sought medical attention for

hoarseness that had developed during the preceding 3 months and was first seen in the ENT department. Six months earlier, she had experienced sore throat and frequent coughing with copious sputum. Her voice became progressively hoarse during the last 3 months. There was no relevant past or family history, and no history of vocal abuse or contact with tuberculosis.

On physical examination, her general appearance was slightly cachectic. Throat examination revealed moderately thickened beef-red anterior tonsillar pillars and posterior pharyngeal wall. Exophytic yellow-gray necrotic vegetations were present, extending from the left anterior tonsillar pillar to the base of the left palatine tonsil which bled easily on touch.

Indirect laryngoscopy showed a diffuse ulcerative infiltration of both vocal cords which was more severe at the posterior commissure. The arytenoids and false cords were moderately swollen and slightly red. The mobility of the true cords was normal.

The hemoglobin was 8.4gm/dl; hematocrit, 25.8%, white cell count, 5400; and ESR, 70mm/hr (corrected ESR, 38mm/hr). Many WBC and 20/HPF RBC were detected on urinalysis. There was no abnormal liver function, but alkaline phosphatase was 482 U/L. BUN and creatinine were not remarkable. Direct smear for AFB of sputum was negative.

An X-ray film of the chest (Fig. 1) disclosed discrete fine nodular and/or mottled densities in both lungs, which were most likely miliary tuberculosis. Because of abnormal urine findings, intravenous pyelography (Fig. 2) was taken, which revealed a non-functioning and enlarged left kidney. Hydronephrosis of the left

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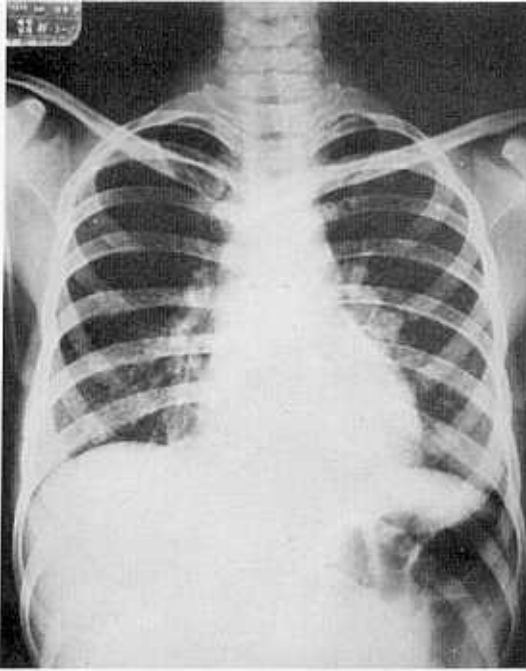


Fig. 1. Chest X-ray shows discrete fine nodular and/or mottled densities in both lungs, which is most likely suggestive of miliary tuberculosis.

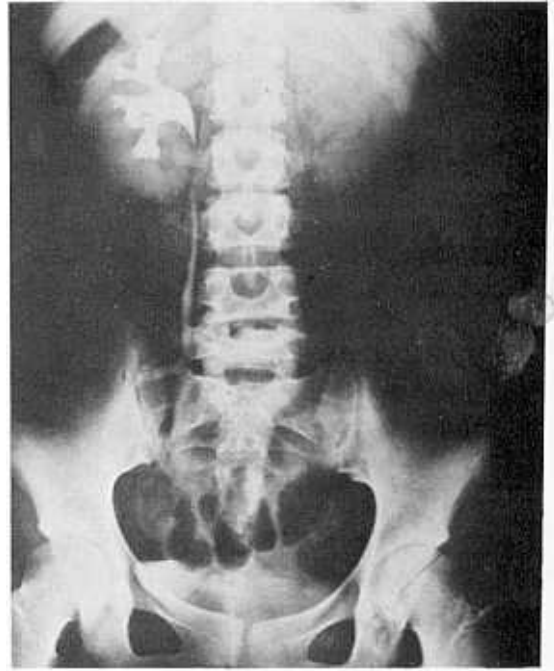


Fig. 2. IVP shows an enlarged left renal shadow with a non-functioning left kidney and osteolytic bony changes with surrounding partial sclerotic changes in the left inferior ramus of the ischium. A narrowing of the disc space between L4 and L5 vertebral bodies is noted with partial osteolytic changes in L3 and L5 vertebral bodies.

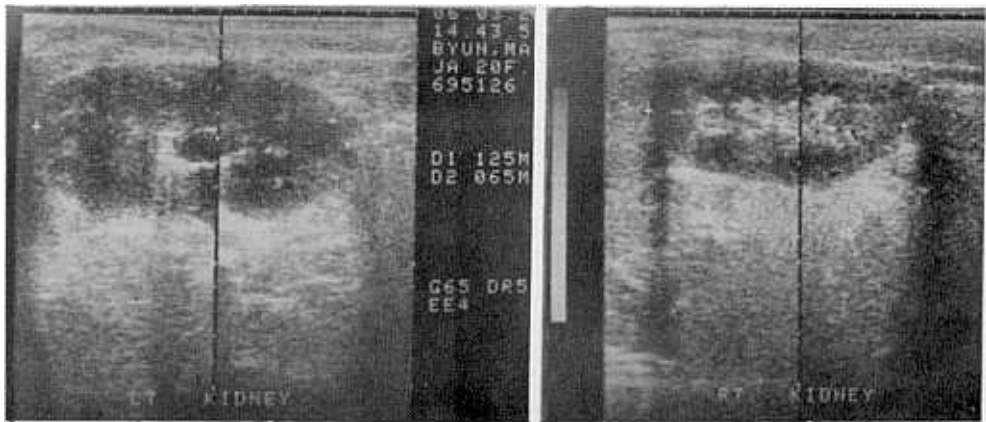


Fig. 3. Ultrasonogram reveals an enlarged left kidney with dilatation of the renal pelvis and calyceal systems which is suggestive of hydronephrosis.

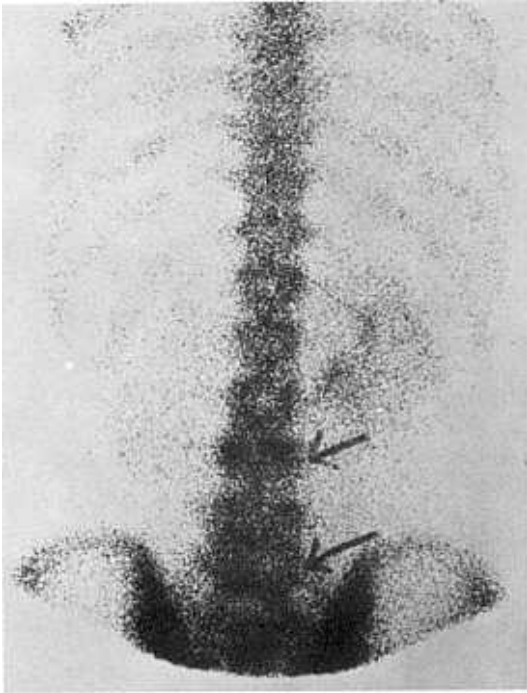


Fig. 4. A bone scan shows abnormal increased radioactivity in L3 and L5 vertebral bodies.

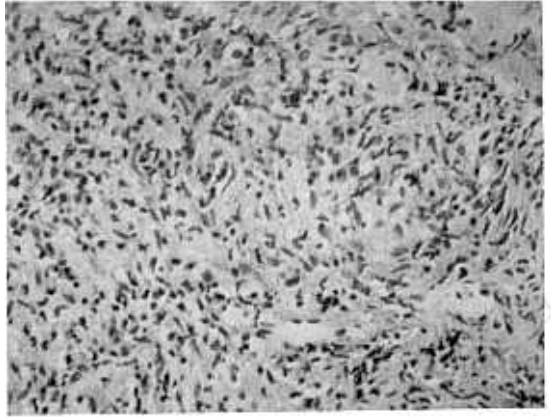


Fig. 6. The vocal cord is ulcerated and replaced by scattered tubercles composed of epithelioid cells and Langhans giant cells with central caseating necrosis (H & E, $\times 100$).

IVP films. These lesions revealed increased radioactivity on bone scan (Fig. 4).

Biopsies of the tonsil and the larynx were obtained. They were rather friable and bled easily. Microscopic sections from the tonsil (Fig. 5) and the larynx (Fig. 6) revealed caseation necrosis with granulomatous lesions.

Treatment was begun with isoniazid, rifampin and streptomycin injection under the diagnosis of laryngeal, tonsillar and miliary tuberculosis, as well as an impression of tuberculosis of the left kidney and two bony sites. The typically rapid improvement due to chemotherapy for tuberculous laryngitis and tonsillitis was evident early in the third week of treatment. After four weeks the patient was no longer hoarse. No laryngeal and tonsillar disease could be identified after two months of treatment, although the chest X-ray film was essentially unchanged.

DISCUSSION

Primary laryngeal tuberculosis is an extremely rare condition and most cases of laryngeal involvement are secondary to pulmonary tuberculosis. The tubercle bacilli reach the laryngeal mucosa through a heavily contaminated sputum, although the hematogenous route or lymphatic spread may also be the source in a small number of cases (Chodosh *et al.* 1970).

Primary tuberculosis of the tonsil is rare, and cases secondary to tuberculosis elsewhere in the body are indeed uncommon. When the latter does occur,

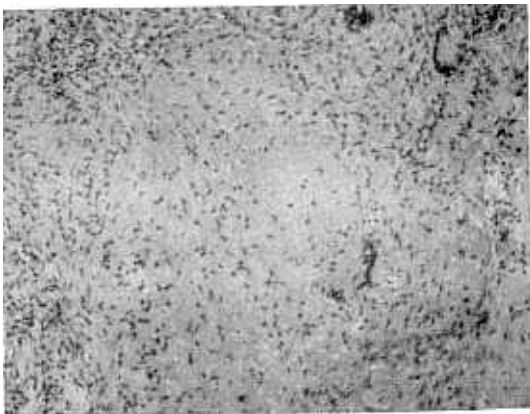


Fig. 5. The section from the tonsil shows an ill-defined epithelioid cell aggregation forming a tubercle (H & E, $\times 200$).

kidney was confirmed by ultrasonography (Fig. 3). There were osteolytic lesions with somewhat sclerotic changes in the left inferior ramus of the ischium and the third and the fifth lumbar vertebral bodies on

however, the patient's pulmonary infection has already been discovered in virtually all cases (Sanford *et al.* 1966). There is, however, a general agreement that tonsillar tuberculosis, whether primary or secondary to an infection established elsewhere in the body, is seldom other than clinically occult (McDowell 1953). In fact, studies in the past on routine tonsillectomy specimens have revealed a significant percentage of tubercle bacilli without active clinical disease. The vast majority of investigators (Aronheim 1938; Weller 1921; Rather 1943) feel that tonsillar tuberculosis is secondary to pulmonary tuberculosis. When the tonsils are involved secondary to pulmonary disease it can be by exogenous (infected sputum) or endogenous (blood stream) extension. Which method predominates in individual cases is not certain. It has been reported that when the crypts are primarily involved, exogenous extension by infected sputum has occurred (usually unilateral), but when the germinal centers are primarily involved, endogenous extension via the blood stream has occurred (usually bilateral).

In Korea a number of papers have separately reviewed tuberculosis of the larynx or the tonsil (Lee *et al.* 1976; Han *et al.* 1978; Jun *et al.* 1980; Lim *et al.* 1983; Lee *et al.* 1984), but concurrent tuberculosis of the larynx and the tonsil which is suspected to be secondary to miliary tuberculosis is rare. In general, secondary tuberculosis is that phase of tuberculous infection which arises in a previously sensitized individual, whether the tubercle bacilli are derived from endogenous or exogenous sources.

Endogenous tuberculosis may occur in any one of the organs or tissues commonly affected by miliary dissemination. Most likely, in the course of lymphatics or hematogenous dissemination of bacilli, organisms are rapidly destroyed in all other sites save for the particular tissue involved in the isolated tuberculosis process. The most common sites of such isolated-organ tuberculosis are the cervical lymph nodes, meninges, kidneys, adrenals, bones, the fallopian tubes and epididymides.

When the pulmonary lesions erode into the bronchi, the material may be coughed up and seed the mucosal lining of the bronchioles, bronchi, and trachea, or organisms may become exogenously implanted in the larynx and the tonsil to produce laryngeal and tonsillar tuberculosis. The larynx and the tonsil are infected by the bacilli in the sputum coughed up from diseased lungs. The more there are, the greater the risk of the disease taking hold. From time to time, reports of cases thought to be examples of primary laryngeal and tonsillar tuberculosis have appeared but have been received with skepticism.

Caseating tuberculous foci including hilar lymphnodes may ulcerate into blood vessels and cause a tuberculous bacillaemia, or the blood stream may be invaded by way of the lymphatics. In these events, miliary tuberculosis will usually occur but it is rare for the larynx and the tonsil to be involved.

According to Dworetzky *et al.* (1971) the incidence of upper airway tuberculosis has decreased from 26 per cent in 1914 to 3.6 per cent in 1941. Auerbach (1946) found laryngeal involvement in 37.5 per cent of 811 tuberculosis patients who were autopsied between 1940 and 1946. These figures suggest that, even when common, the condition frequently was not recognized clinically. Nedwick's observations (1970) from a large Veterans Administration Hospital in suburban Detroit, Michigan, indicates that the incidence of tuberculous laryngitis fell from 1.7 per cent of all tuberculosis admissions between 1949 and 1955 to 0.4 per cent of such admissions from 1956 to 1968.

Concerning the incidence of tonsillar tuberculosis, Newhart *et al.* (1934) reviewed the statistics of 14 different reports comprising a grand total of 30,676 cases. The incidence of tuberculous involvement varied from 0.52 per cent to 8.0 per cent. Of these 30,676 cases, the average incidence was 2.03 per cent. Cap'o (1947) reported that 2.2 per cent of the 2,500 tuberculous patients he examined had active tonsillar tuberculosis, and 92 per cent of these were associated with laryngeal tuberculosis.

There is general agreement that collapse therapy and improvements in general patient care had reduced the incidence of upper airway tuberculosis before the era of effective chemotherapy.

The treatment of laryngeal and tonsillar tuberculosis is now the treatment of pulmonary infection with appropriate combinations of anti-tuberculous drugs and will be in the hands of the chest physician. Hunter *et al.* (1981) pointed out that hoarseness and sore throat were resolved completely in three to four weeks if corticosteroids were given in combination. However rapid resolution of symptoms and signs was observed in three weeks in our patient without corticosteroids although the disease had a disseminated pattern. It is now our policy to not give corticosteroids routinely in the management of laryngeal and tonsillar tuberculosis even though cases are suspected of following miliary tuberculosis.

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