Tuberculous Parotid Lymphadenitis: A Rare Case Report

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ABSTRACT
Tuberculosis of parotid lymph nodes is a rare condition. Although uncommon, it must be included in differential diagnosis of a discrete parotid gland swelling or mass. Early recognition of the disease entity and awareness of its potential existence can spare the patient unnecessary surgical intervention. A 13-year-old female patient presented with a painless swelling in left preauricular region. Ultrasonographic examination showed presence of calcified lymph nodes with central necrosis. Fine needle aspiration cytology showed epitheloid cells, foci of calcification and areas of caseation necrosis suggestive of tuberculosis. Standard antituberculous regimen was planned for 6 months with rifampicin, ethambutol, isoniazid and pyrazinamide. Patient discontinued the antituberculous regimen after free of clinical symptoms at the end of the 5th month. Recurrence of the tuberculosis was seen 2 weeks after the discontinuation of the treatment. Completed retreatment for 8 months and follow-up shown patient free of tuberculosis.

Keywords: Tuberculosis, Parotid lymphadenitis.

INTRODUCTION
Tubercular involvement of the parotid gland is extremely unusual even in countries with high incidence of this infection such as India (Handa et al 2001). It most commonly presents as a localized mass, resulting from infection of intracapsular or pericapsular lymph nodes (Rowe-Jones et al 1992). It may also present as parenchymatous tuberculosis (Bhat and Stansbie, 1996), periauricular fistula/sinus (Zheng and Zhang, 1995) or as an abscess (Chatterjee et al 2001). The diagnosis of this condition has been established on the basis of histopathological evaluation of the excision biopsy specimen in the past, but, a diagnosis based on fine needle aspiration cytology (FNAC) and medical management have been shown to be curative without the need of surgical intervention (Handa et al 2001).1

Tuberculous intraparotid lymphadenopathy has been described by Kumvilla et al and Ubbi et al among others. Ubbi et al reported four cases of tuberculosis in the parotid gland in an immigrant community. El-Hakim and Langdon presented a case of tuberculosis of a submandibular lymph node masquerading as a salivary gland tumor. The salivary gland register (1965-1981) contains only two cases of parenchymatous tuberculosis of the parotid gland, but contains 46 cases of intraglandular or periglandular lymph node involvement. Van der Walt, in his study on granulomatous sialadenitis, mentions tuberculous involvement of major salivary glands but recognizes that the condition is rare.2

CASE REPORT
A 13-year-old female patient presented to Department of Oral Medicine and Radiology of Narayana Dental College and Hospital, Nellore, with a swelling in left preauricular region since 1 month (Fig. 1). The swelling was slowly progressive in size. There was history of fever since 1 week. No history of cough or weight loss. No significant past medical and family

Fig. 1: Swelling in the preauricular region
history. Local examination revealed a firm, non-mobile, tender swelling which was 3 × 3 cm in diameter extending anteroposteriorly 2 cm from left angle of mouth to 1 cm from left earlobe. Superior-inferiorly, the lesion extended from ala-tragus line to lower border of mandible. No sinus opening or discharge from swelling was present. Left submandibular lymph nodes were palpable, tender and mobile. Facial nerve function was normal. Provisional diagnosis was given as sialadenitis of left parotid.

The routine blood investigations were in the normal limits apart from raised ESR (30 mm/1hr). Screening tests for HIV, HBsAg, HCV was negative. Mantoux test was also negative. Posteroanterior view of chest radiograph shows normal radiological findings. Sialogram of left parotid showed no calcification was seen in the region of duct or gland. Fine needle aspiration cytology (FNAC) from the lesion showed epitheloid cell clusters, foci of calcification and areas of caseation necrosis suggestive of tuberculous parotid lymphadenitis. Secondary infection due to FNAC was developed and was controlled with antibiotic therapy (Fig. 2). Ultrasound examination showed two calcified parotid lymph nodes with central necrosis (Fig. 3).

Patient was referred to regional tuberculosis center and standard antituberculous regimen was planned for 6 months with rifampicin, ethambutol, isoniazid and pyrazinamide. Patient discontinued the antituberculous regimen after free of clinical symptoms at the end of the 5th month. Recurrence of the tuberculosis was seen 2 weeks after the discontinuation of the treatment. Completed retreatment for 8 months with streptomycin, rifampicin, ethambutol, isoniazid and pyrazinamide and follow-up shown patient was free of clinical signs and symptoms and subsequently regional tuberculosis center reported patient is free of tuberculosis (Fig. 4).

DISCUSSION

Evidence of TB is present in ancient mummies with spinal deformities and calcified lung lesions characteristic of the disease. Hippocrates identified TB as phthisis, and he advised followers to avoid treating late-stage disease to avoid damage to their reputations, because it was almost always fatal. The earliest recorded symptoms of pulmonary TB (cough), expectoration, hemoptysis and wasting were found in the library of Ashurbanipal (668 to 626 BC), king of Assyria. Later descriptions of TB labeled the disease as lupus vulgaris (TB of the skin), Pott disease (TB of the bones) or, simply consumption. TB was romanticized as the ‘Gentle Death’ in the 19th century, because the victim often appeared to slowly and gracefully fade away. Tuberculosis is a necrotizing granulomatous disease with protean manifestations and a wide distribution. The lungs are most commonly affected. Extrapulmonary sites may be involved during the early course of the disease, with seeding of the mycobacteria manifesting itself as multiple lesions. Among the sites commonly affected are the kidneys, bones, meninges and cervical lymph nodes.
TB of the head and neck has been documented extensively. A 10-year retrospective study in India documented 165 cases of TB of the head, neck and oral cavity. Of these patients, 121 (73.3%) had isolated tubercular lymphadenitis; (14.5%) had laryngeal TB, and four (2.4%) had tubercular otitis media. Three (1.8%) had cervical spine involvement; three (1.8%) had parotid gland involvement, and eight (5%) had oral cavity involvement. One patient had temporomandibular joint involvement and one had TB of the nose. Only about 0.05 to 5% of patients who have active TB will present with oral lesions. Localized pain is the most common symptom, with odynophagia present in 61.1% of patients who have oral TB. Squamous cell carcinoma, traumatic ulceration, primary syphilis and pulmonary fungal diseases may show a similar clinical presentation and should be included in the differential diagnosis.  

Extrapulmonary tuberculosis may present in concurrence with a focus in the lungs or may present primarily without pulmonary involvement. The latter situation may provide a difficulty in diagnosis due to the absence of systemic signs and symptoms of the disease (O’Conell et al 1993). Tuberculosis of the parotid gland is rare and may pose a significant difficulty in diagnosis, as it is difficult to distinguish from the tumors of parotid gland that are comparatively much commoner (Handa et al 2001). It may also occur either secondary to a primary focus in the lung, as a result of hematogenous spread or as an autoinfection from the oral cavity. It may occur as one out of the two pathological forms the localized form, because of the involvement of intra- or periglandular lymph nodes is the more common form with pathogenesis similar to scrofuloderma, whereas, the diffuse form involving the parenchyma may be secondary to the nodal infection, and is rare (Bhat and Stansbie, 1996). Clinically, the most common mode of presentation is as a localized swelling with gradual enlargement (Zheng and Zhang, 1995). The patient may also present with preauricular discharging fistulae as a result of spontaneous or surgical drainage of a fluctuant swelling (Zheng and Zhang 1995). The source of infection in parotid tuberculosis is controversial. There are different postulations on the source of infection. Extension of infection along Stenson’s duct from oropharynx and vascular mode of spread from primary focus in the body or through wounded oral mucosa are some of postulations. According to Berman and Fein, spread by lymphatic vessels, particularly from infected tonsils and external auditory canal, plays an important role. 

Tuberculous sialadenitis may develop secondary to infection in the oral cavity. Direct extension to salivary gland parenchyma by the bacillus may occur by way of the glandular ductal system. The parotid is the major salivary gland most commonly infected in this manner. Tuberculous disease is impossible to distinguish clinically from other diffuse inflammatory disease of the salivary glands if a culture of the glandular secretions from the Stenson’s duct or saliva are negative for AFB. Less than 200 cases have been reported since the first description of this condition. The differential diagnosis includes actinomycosis, parotitis, mumps, sarcoidosis, Sjogren syndrome and sialosis. Investigations should include chest X-ray and skin testing.  

The diagnosis of parotid gland tuberculosis is based on the clinical presentation of the patient combined with investigations like a contrast-enhanced CT scan, FNAC and histopathological evaluation. In the past, the diagnosis was invariably established on the basis of an excision biopsy (Maynard 1967). In the recent years, FNAC has become more acceptable as a means of diagnosing the pathology and nature of salivary gland masses without the complication of implantation (Weiner and Pahor, 1996; Handa et al 2001). The valuable investigation in this case appeared to be the FNAC, which yielded the diagnosis. CT has been found to be the imaging modality of choice for most pediatric parotid disease. Associated with facial nerve involvement could be evaluated by magnetic resonance imaging. There is a rising trend of HIV disease among tuberculosis patients in urban and rural areas in India. Clinical and radiological manifestations of HIV and tuberculosis are in consonance with the time of occurrence of either disease, time of diagnosis, CD4 lymphocyte count, tuberculin anergy and the presence of coexisting or opportunistic infections. Sonographically guided FNAC may provide diagnostic information by means of smears and acid-fast stains, which may show AFB, lymphocytes and epithelial granulomas, or Langerhans giant cells with caseous debris. Detection of M. tuberculosis DNA in circulating mononuclear blood cells holds promise as a rapid screening assay and an adjunct to routine techniques. The PCR has been evaluated for more than a decade for the diagnosis of pulmonary and extrapulmonary TB, including TB-L.  

Rapid identification of mycobacterium to the species level was developed on the basis of evaluation by the polymerase chain reaction (PCR) of the gene encoding for the 65-kDa protein. The method involves restriction enzyme analysis of PCR products obtained with primers common to all mycobacteria. Using two restriction enzymes, Bst EII and HaeIII, medically relevant and other frequent laboratory isolates. PCR is the most sensitive single technique available to date for the demonstration of M. tuberculosis in specimens derived from patients with a clinical suspicion of tuberculous lymphadenitis. Diagnostic tests and thought processes in granulomatous diseases were demonstrated in Flow Chart 1. Standard antitubercular drug treatment was given to this patient through DOTS (directly observed therapy short-course) in alternate days which include rifampicin (450 mg), isoniazid (600 mg), ethambutol (1200 mg), pyrazinamide (1500 mg), every alternate day up to 2 months as intensive phase. In continuous phase, rifampicin (450 mg) and isoniazid (600 mg) were given up to 6 months (Table 1).
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Table 1: Recommended dose of each drug in a range-per mg/kg body weight

<table>
<thead>
<tr>
<th>Body weight</th>
<th>Drugs</th>
<th>30 kg</th>
<th>40 kg</th>
<th>50 kg</th>
<th>60 kg</th>
<th>70 kg</th>
<th>80 kg</th>
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<tr>
<td></td>
<td>Isoniazid</td>
<td>120 mg</td>
<td>200 mg</td>
<td>250 mg</td>
<td>300 mg</td>
<td>350 mg</td>
<td>400 mg</td>
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<td></td>
<td>Rifampicin</td>
<td>300 mg</td>
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<td>500 mg</td>
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<td>500 mg</td>
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<td></td>
<td>Pyrazinamide</td>
<td>750 mg</td>
<td>1000 mg</td>
<td>1250 mg</td>
<td>1500 mg</td>
<td>1750 mg</td>
<td>2000 mg</td>
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<td></td>
<td>Streptomycin</td>
<td>450 mg</td>
<td>600 mg</td>
<td>750 mg</td>
<td>900 mg</td>
<td>1050 mg</td>
<td>1200 mg</td>
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<td></td>
<td>Ethambutol</td>
<td>400 mg</td>
<td>600 mg</td>
<td>750 mg</td>
<td>900 mg</td>
<td>1050 mg</td>
<td>1200 mg</td>
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Flow Chart 1: Diagnostic tests and thought processes in granulomatous diseases

REFERENCES