Primary Tuberculosis of Parotid Gland Mimicking Parotid Tumour


Abstract
Primary tuberculosis of parotid gland is rare. Its occurrence may pose a diagnostic problem in differentiating it from parotid tumours. It occurs without any primary focus in lungs or any pulmonary symptom. We report a case of 17 year old female who presented with clinical picture of parotid tumour. Ultrasound showed picture of nodule in parotid. Fine needle aspiration cytology (FNAC) was inconclusive. Superficial parotidectomy was done. Histopathology revealed tuberculosis of parotid gland. Four drugs Antikoch’s treatment (AKT) was given for two months and followed by two drugs for four months.

Follow up of 6 months has shown patient to be symptom and disease free.

Introduction
Tuberculosis in the parotid gland is an infectious disease. It manifests itself by an increase in gland volume; mainly involving glandular lymph nodes, making it lobulated and causing lymphadenitis. Primary tuberculosis of parotid gland is rare even in countries like India where tuberculosis is endemic. It is difficult to differentiate parotid tumours from tuberculous parotitis due to similar presentation. Although rare, tuberculous parotitis should always be kept as a differential diagnosis of tumours that increase parotid volume. We present a similar case of 17 years female with tuberculous parotitis mimicking parotid tumour.

Case Report
A 17 year old female presented with right preauricular swelling since 2 years. She had no history of pain, fever or weight loss. No history of Koch’s or Koch’s contact. On examination there was a swelling in the right preauricular region, measuring 2 x 2 cms, with well defined margins, smooth surface and firm in consistency. It appeared free from skin and underlying structures. No neck nodes were palpable. There were no signs of facial nerve palsy. Ultrasonography (USG) of the local part was suggestive of 2 x 2 cm heterogeneous lesion in right parotid. All routine investigations were within normal limits apart from ESR-45 mm at one hour. X-ray chest was within normal limit. A provisional diagnosis of pleomorphic adenoma was considered. Fine needle aspiration cytology (FNAC) was inconclusive. Patient was posted for superficial parotidectomy.

Intraoperatively there was a 2 x 2 cm nodule in parotid, on dissection cheesy material came out of the swelling. A superficial parotidectomy was done and specimen sent for histopathological and microbiological examination. Histopathological examination of the swelling was suggestive of a tubercular infection of parotid with caseation seen with lymphocytosis and Langhan’s giant cell and epithelioid cells (Figs. 1 and 2). On culture sensitivity of the aspirate no organism was isolated. Patient was started on four drug Antikoch’s treatment (AKT) (i.e. isoniazid, rifampicin, pyrazinamide, ethambutol) for two months followed by two drugs (i.e. isoniazid, rifampicin) for four months. Patient was on follow up for six months. Patient recovered well and had no complains or recurrence.
Discussion

Tuberculosis of parotid gland is rare. Granulomatous lesion of parotid may present as nodule and make the diagnosis difficult. Primary parotid tuberculosis is defined as tuberculosis of parotid gland without any other identifiable primary focus i.e. pulmonary or lymph node.

The source of infection to parotid is controversial. Saliva is said to have inhibitory effect on mycobacterium. Van Stubenrauch postulated extension of infection along Stenson's duct from the oropharynx and Bockhorn postulated a vascular mode of spread from any primary focus in the body or through wounded oral mucosa. According to Berman and Fein it is spread by lymphatic vessels, particularly from infected tonsils and the external auditory canal, which plays an important role. Carmody formulated a canalicular mode of spread from infected molar teeth. The most commonly implicated agent is mycobacterium bovis. Atypical mycobacterium rarely infects the parotid.

Primary tuberculosis of parotid presents in two forms, first acute inflammatory lesion mimicking sialadenitis which is more common, consisting of small and large abscesses, the parotid tissue is oedematous, friable and indurated at places. Second presentation is chronic tuberculous lesion which is circumscribed, these present as slow growing mass over months to years, gradually increasing in size with no symptoms apart from swelling, on clinical examination it is impossible to distinguish them from parotid neoplasms. Our case was most probably a chronic tuberculous lesion presenting as asymptomatic swelling.

In absence of active tuberculosis or history of tuberculosis or any clinical evidence, the tuberculous parotid swellings are mistaken for parotid tumour. Fine needle aspiration is advocated as reliable and useful technique for diagnosis of tuberculosis of parotid. In parotid lesions it has a sensitivity of 81% to 100% and specificity of 94% to 100%, but they are not always contributory as areas of necrosis may be seen in tumours as well as tubercular infection. In our patient fine needle aspiration cytology was inconclusive, may be because it was not done from the representative area.

![Fig. 1: Showing caseous necrosis in substance of parotid gland with Langhan's giant cell and epitheloid cells (Low Power Field).](image1)

![Fig. 2: Showing caseous necrosis in substance of parotid gland with Langhan's giant cell and epitheloid cells (High Power Field).](image2)
culture the aspirate but this requires initial suspicion and may require long time to obtain result.

As a negative FNAC does not rule out tuberculosis or tumour of parotid, it becomes necessary to utilize other diagnostic aids such as imaging and exploration. Imaging studies include USG, computed tomography (CT Scan) and magnetic resonance imaging (MRI). But there are no specific signs of tuberculosis in parotid with any of these imaging techniques. Since tuberculosis may involve multiple sites in parotid and periparotid region MRI is better than CT or USG. Incisional biopsy should not be done as it results in cutaneous fistula formation. Excisional biopsy becomes mandatory when other modalities are not contributory. In a meta-analysis by Lee and Liu of 49 patients of tuberculous parotitis, FNAC was helpful in 10 cases and excision was done in 34 patients. In our patient FNAC and USG were unrewarding and a superficial parotidectomy was done in order to make the diagnosis.

When tuberculosis of parotid is diagnosed on fine needle aspiration, patient can be given anti-tubercular treatment and followed up without surgery. On most instances preoperative diagnosis of parotid tuberculosis is not possible; it is diagnosed on histopathological examination done postoperatively. After a histopathological diagnosis is made a full course of AKT has to be given, as was done for our patient.

With increase in incidence of retroviral infection the incidence of tubercular parotitis is increasing. In conclusion tubercular parotitis should always be considered as differential diagnosis in parotid swellings.

References