TUBERCULOSIS OF PAROTID GLAND IN A HIV POSITIVE PATIENT- A CASE REPORT

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Abstract

A 35 year old HIV positive married male patient was reported with the chief complaint of watery discharge from the swelling in the left parotid region since a week. Detail history revealed that patient was diagnosed as having tuberculosis seven months back, but he discontinued antituberculosis therapy after 2 months as there was slight improvement in his symptoms. Sputum examination, OPG, Chest PA view, CT scan, FNAC of left parotid gland was advised. After these investigations a final diagnosis of tuberculosis of parotid gland in a HIV positive patient was made. Patient was referred to DOT centre at Bableshwar for antituberculosis treatment where he was treated for 6 months. Two months after initiation of therapy sinus had healed, parotid swelling regressed and lymph nodes also reduced in size. Post therapy sputum examination was negative.

Key words: *HIV, Tuberculosis, Parotid gland.*

Introduction

Since the first report of HIV infection in India in a commercial sex worker of Tamil Nadu in1986, the virus has spread all over the country though there is geographic variation. There are estimated 5.1 million people infected with HIV in India. India has the highest total burden of tuberculosis in the world with an estimated 1.85 million incident cases in 2005.[1]

One third of the 42 million people living with HIV/ AIDS worldwide are co-infected with tuberculosis.[1] Approximately about two million HIV infected persons living in India are co-infected with tuberculosis.

Tuberculosis is one of the main causes of death in HIV positive patients involving multiple organs, particularly lungs. Tubercular involvement of parotid gland is extremely unusual even in countries with high incidence of this infection such as India. Less than 200 cases have been reported since the first description of this condition by Von Stubenrauch in 1894.

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Case report

A 35 yr old married male patient reported with the chief complaint of watery discharge from the swelling in the left parotid region since a week. Medical history revealed that patient was diagnosed as HIV positive 3 years ago and was on antiretroviral therapy. Detail history revealed that his coworker was diagnosed as having tuberculosis 7 months back. Few days later patient experienced cough for which he visited a general physician who after investigations (viz. sputum examination and chest radiograph) diagnosed him as suffering from pulmonary tuberculosis. Patient was then put on antituberculosis therapy but he discontinued the same after 2 months as there was very slight improvement in his symptoms.

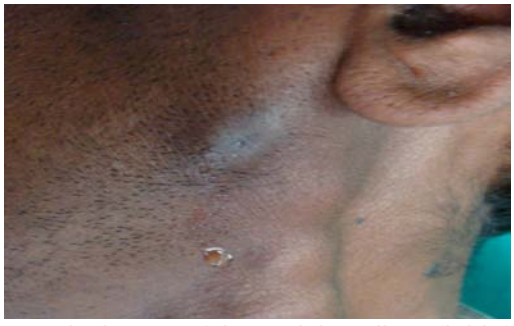

Fig 1: Discharge of brownish yellow fluid from left parotid gland.

On extra oral examination bilateral diffuse parotid enlargement was noted which was soft to firm in consistency and there was a discharge of brownish yellow fluid from left parotid (Fig.1). A sinus opening was seen near left angle of mandible. Skin overlying the swelling was normal. Multiple large lymph nodes were palpable in right submandibular region which were tender on palpation (Fig.2). Left jugulodigastric as well as inguinal lymph nodes were also palpable. Intraoral examination did not reveal any significant findings.

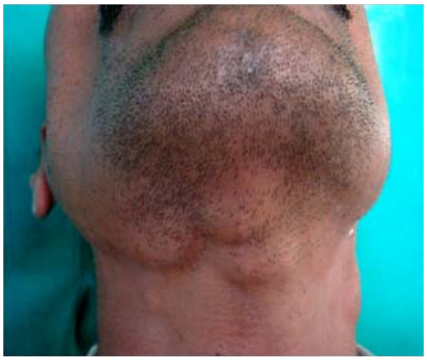

Fig 2: Enlarged right submandibular lymph nodes

Investigations

Sputum examination revealed presence of acid - fast bacilli on Ziehl- Neelsen staining. From this he was classified as category I patient. Orthopantomograph did not reveal any odontogenic cause for the swelling. PA view of chest revealed multiple nodular ill defined infiltrates in both lung fields. Right hilum was bulky suggestive of lymphadenopathy. Overall picture was suggestive of bilateral pulmonary tuberculosis (Fig.3).

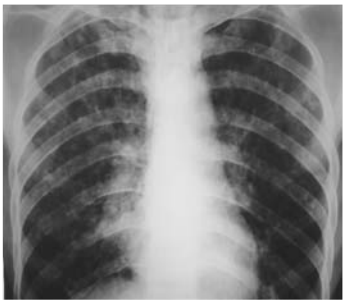

Fig 3: PA view of chest

CT scan revealed multiple heterogenous moderately enhancing soft tissue density lesions in superficial as well as deep lobe of parotid glands suggestive of bilateral intraparotid lymph nodes. Post contrast study showed enlarged lymph nodes showing heterogenous ring enhancement with central non enhancing areas (Fig.4). The picture was suggestive of infective tubercular etiology.

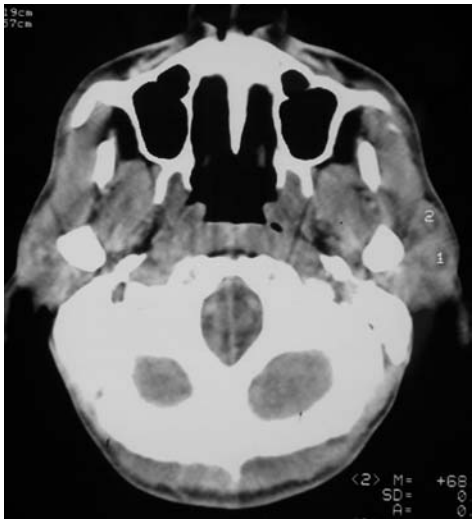

Fig 4- Post contrast Axial CT scan

FNAC of left parotid showed moderately cellular smears comprised of few granulomas consisting of epitheloid cells, macrophages and neutrophils, lymphocytes and RBC's.

A final diagnosis of tuberculosis of parotid glands in a HIV positive patient was made. Patient was referred to DOT centre at Bableshwar where he was treated for 6 months. For initial 2 months he was put on rifampicine, ethambutol, isoniazid and pyrazinamide on alternate days. For next four months he was given rifampicine and isoniazid on alternate days. Two months after initiation of therapy sinus healed, parotid swelling reduced and lymph nodes also regressed in size (Fig.5). Post therapy sputum examination was negative.

Discussion

With the introduction of P-aminosalicyclic acid (1929), streptomycin (1944), isoniazid ((1952), pyrazinamide (1954), cycloserine (1955), ethambutol (1962) and rifampicine (1963) as

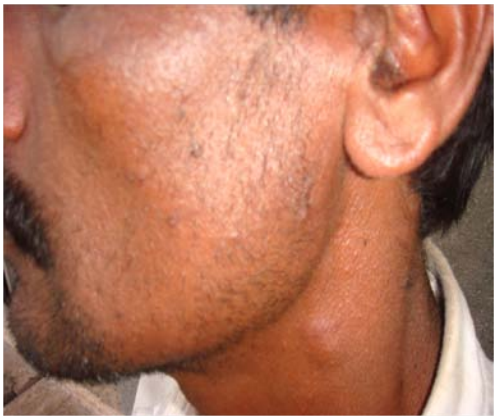

Fig 5- Healed sinus over left parotid region

antituberculosis agents hope dawned on the gloomy world of patients of tuberculosis. The situation further brightened with introduction of the directly observed therapy short course (DOTS) strategy. The co-infection of tuberculosis - HIV and emergence of MDR strains have added complexities to treatment.

Extrapulmonary tuberculosis may present in concurrence with a focus in the lungs or may present primarily without pulmonary involvement. Mycobacterial disease commonly presents in the head and neck as a unilateral mass lesion consisting of multiple matted enlarged lymph nodes. Extrapulmonary form of the disease accounts for approximately 20% of overall active tuberculosis.

More than 50% of AIDS patients will have extrapulmonary lesions and can be seen in the lymph nodes (55-60%), bones (8-9%), gastrointestinal tract (7-8%), meninges (1-2%), skin (1-2%), and kidneys. Pain, circumferential reduction of movements at the joints, fever, headache, irritability, behavioural changes, focal or generalized seizures, abdominal pain, diarrhoea, weight loss, nausea, vomiting, constipation, haematuria, asymptomatic enlargement of lymph nodes, hoarseness of voice are the various clinical manifestations of tuberculosis depending on the organ involved. In our case only enlarged lymph nodes were present. Hence involvement of other organs was ruled out clinically.

Head and neck involvement is not rare. The most common extrapulmonary sites in head and neck are the cervical lymph nodes followed by larynx and middle ear. Less common sites include the tonsil, nasal cavity, nasopharynx, oral cavity, parotid gland and esophagus. Most common intraoral sites are tongue, gingiva, palate and lips.[2]

Salivary gland appears to be rarely infected with tuberculosis. This may be because saliva has an inhibitory effect on mycobacteria. Tuberculosis is normally confined to the intraglandular and periglandular lymph nodes in the major glands and invasion of the parenchyma is usually a secondary spread from the nodes themselves.

Tuberculous intraparotid lymphadenopathy has been described by Ubbi et al among others.[3] Similar finding was observed in our case.

Involvement of parotid gland and lymph nodes may develop in two ways. First, a focus of mycobacterial infection in the oral cavity liberates the mycobacterium that ascends into the salivary gland via its duct or passes to its associated lymph nodes via lymphatic drainage. The second pathway involves hematogenous or lymphatic spread from a distant primary lung focus[4]

HIV-infection is among the strongest risk factors for progression of latent tuberculosis infection to active disease. The hallmark of HIV infection is a progressive depletion and dysfunction of CD4 T lymphocytes coupled with defects in macrophages and monocyte function. Because CD4 T lymphocytes and macrophages have a central role in antimycobacterial defenses, dysfunction of these cells places patients with HIV infection at high risk of primary or reactivation tuberculosis disease.

In the past the diagnosis was invariably established on the basis of histopathological examination. In the recent years FNAC, contrast enhanced CT scans has become more acceptable as a means of diagnosing the pathology. On contrast enhanced CT scan, the presence of thick walled rim enhancing lesion with a central lucency is suggestive of tubercular pathology.[5] Similar finding was observed in our case which is consistent with the findings reported by A. Sethi.[6] In parotid lesions FNAC has a sensitivity of 81-100% and specificity of 94-100%.

In 2000, there were globally an estimated 3,50,000 deaths from HIV related tuberculosis. Delays in the diagnosis of tuberculosis have been associated with worst outcomes, so initiation of treatment as soon as tuberculosis is suspected is very important.

Anti-TB drug treatment is the same for HIV-positive and HIV-negative TB patients, with one exception: do not give thioacetazone to HIV-positive TB patients (increased risk of severe and sometimes fatal skin reactions)**.** Medical management of the tuberculosis involves the use of isoniazid, rifampicine, pyrazinamide, streptomycin, ethambutol, thioacetazone. DOTS is the internationally recommended strategy for TB control adopted as RNTCP (Revised National TB Control Programme) in India since 1997. The DOTS strategy can prevent emergence of multidrug-resistance tuberculosis.

Refernces

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