



PRIMARY TUBERCULOSIS PRESENTING AS GINGIVAL ENLARGEMENT. A DIAGNOSTIC IMPEDIMENT -A CASE REPORT

Dental Science

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ABSTRACT

Tuberculosis is one of the major causes of ill health and death worldwide. It is a chronic granulomatous disease caused by various strains of *Mycobacterium tuberculosis*. Tuberculosis lesions of the oral cavity are rare and can be a diagnostic challenge. Most of the cases are secondary to pulmonary disease and the primary form is uncommon. Oral lesions of TB may be in the form of nodules, ulcers or elevated fissures. In this paper we present a case of primary oral tuberculosis affecting the gingiva manifesting as gingival enlargement in a 43 yr male patient. The diagnosis was based on histopathologic examination, CBP, chest X-ray and immunologic investigation with detection of antibodies against *M. tuberculosis*. This case report emphasizes the need for dentists to consider TB in the differential diagnosis of gingival enlargement keeping in view the increase in the incidence of tuberculosis.

KEYWORDS

INTRODUCTION:

Tuberculosis is a chronic granulomatous infectious disease caused by *Mycobacterium tuberculosis*, and can affect any part of body including oral cavity.¹ According to WHO, tuberculosis is responsible for death of approximately 2 billion people each year and it is estimated that between 2002 and 2020, approximately 1 billion people will be newly infected, over 150 million will get sick, and 36 million will die because of tuberculosis.

Tuberculosis has a definite affinity for lungs. However, any part of the body can be affected, including the mouth.² Tuberculosis affecting other than lungs is extra pulmonary tuberculosis.¹ Extra pulmonary tuberculosis accounts to 10-15% of all cases and is rare.³ Lesions of the oral cavity occur in 0.05%-5% of the patients with TB and frequently are secondary affecting more usually in elderly patients. On the other hand primary form is uncommon and more usually affects young patients.⁴ In oral tuberculosis the most commonly affected site is the tongue, other sites include the lip, cheek, soft palate, uvula, gingiva and alveolar mucosa.⁵ The lesions are seen as superficial ulcers^{6,7}, patches,⁸ indurated soft tissue lesions⁹ or even lesions within the jaw in the form of osteomyelitis.

A majority of oral tuberculosis cases manifest as chronic non healing ulcers and so far very few cases of primary tuberculosis manifesting as gingival enlargement have been reported.

CASE REPORT:

A 43 yr old male patient reported to the Department of Periodontics, Government dental college and Hospital, Afzalgunj, Hyderabad, Telangana, India with the chief complaint of non-painful swelling of the gingiva on the front side of both upper and lower arches. The patient gave a history of gingival enlargement over the past 1yr which gradually increased overtime.

On intra oral examination the gingiva was fiery red in colour, pebbled, granular and unusual in appearance. It was non tender and fibroedematous in consistency. Periodontal examination revealed absence of pockets. The rest of the oral cavity was normal.

The patient was of good built, non smoker, non-alcoholic with normal temperature and respiratory rates. Extra oral examination revealed slight swelling of lips and no cervical lymphadenopathy. Based upon the initial clinical examination, a provisional diagnosis of inflammatory gingival enlargement was made.

Since the patient was not on any systemic medication enlargement of gingiva due to drugs was ruled out. Complete blood picture, urine analysis and biochemical tests were within normal limits ruling out the possibility of leukaemic gingival enlargement.

HIV, HbsAg, VDRL tests were negative ruling out infectious etiology of enlargement. But ESR was elevated to 59mm/hr.

When a supragingival debridement under universal precautions did not show any improvement in the overall condition of the gingiva, an incisional biopsy was performed on the upper labial gingiva in relation to maxillary right central incisor.

HISTOPATHOLOGICAL EXAMINATION:

Histopathological examination revealed stratified squamous epithelium with mild hyperkeratosis, parakeratosis, acanthosis and focal areas of ulceration [fig 1a]. Subepithelial stroma showed epithelioid cell granulomas containing Langhans type of giant cell [fig 2c], focal areas of caseous necrosis [fig 2b] and dense lymphoplasmacytic infiltration. These features were suggestive of tuberculous granulomatous lesion.

Sections stained with PAS and Grocotti-gomori stains for bacteria and fungi were -ve. The patient was referred to the medical department for further investigations. To rule out sarcoidosis serum Ca and angiotensin converting enzyme (ACE) levels were checked and found to be in normal range. Chest X-ray (PA view), culture of sputum were negative. Tissue specimens when checked for *Mycobacterium* with special stains i.e, Ziel Neelson and Auramine Rhodamine were also negative. An immunological test to detect antibodies against *Mycobacterium* in patient's serum (ELISA) was positive. In view of these findings a working diagnosis of primary tuberculous gingival enlargement was made.

In consultation with a physician antitubercular therapy was initiated with Isoniazid (10mg/kg of body wt), Rifampicin (10-20mg/kg of body wt) and Pyrazinamide (10-20mg/kg of body wt) for 2 months followed by Isoniazid and Rifampicin for the next 4 months.

DISCUSSION:

Tuberculosis is major cause of ill health and death worldwide. The risk of infection however is much greater among people in lower socioeconomic groups.⁸ Every year approximately 2.2 million individuals develop tuberculosis in India of which 0.87 million are infectious cases and it is estimated that annually there are around 3,30,000 deaths due to TB.⁹ The average prevalence of all forms of TB has been reported to be 5.05/1000.¹⁰ Moreover TB has become the most common opportunistic infection in areas where HIV infection is prevalent.¹¹

Although oral TB has been well documented, tuberculous lesions of the upper aerodigestive tract are rare. Therefore, intraoral TB is generally missed in differential diagnosis, and there are many patients in whom the correct diagnosis and therapy are delayed or missed entirely.

The mechanism of primary inoculation of tuberculous bacilli into the oral mucous membrane is not clearly understood, although ingestion of milk contaminated with *M. Bovis* by children and adolescents has been presumed to be one probable factor.¹² One reason for the rare occurrence of TB of the gingiva may be that intact squamous epithelium of the oral cavity resists direct penetration of bacilli.¹³ This resistance has been attributed to the thickness of the oral epithelium, cleansing action of saliva, local PH and antibodies in saliva.¹⁴ Even if the onset of the infection is by hematogenous spread, injured or inflamed tissue tends to localise blood borne bacteria. However, the mode of entry of the microorganism may be through a break in the mucous membrane caused by trauma.¹⁵ Where the infection involves the bone, the mode of entry is thought to be through an extraction socket. However, there is general consensus that secondary TB spreads by hematogenous route.¹⁶

The case report presented here is one of the first few cases of primary tuberculous gingival enlargement in the literature. The oral lesion in this case is diffuse gingival enlargement instead of the usual manifestation as an ulcer¹⁷ or localised granular mass.¹⁸

Primary tuberculosis typically involves the gingiva and presents as diffuse, hyperaemic, nodular or papillary proliferation of gingival tissues.¹⁹ These findings were corroborated in the present case as it appeared as diffuse gingival enlargement where the gingiva had a reddish, pebbled, erythematous appearance which failed to respond to initial supragingival debridement. When other investigations turned out to be normal, it raised the suspicion and thus an incisional biopsy was performed. The tuberculin test (Mantoux test), immunological test (ELISA) and histopathological report confirmed the diagnosis of gingival tuberculosis.

TB of oral cavity is difficult to differentiate from other conditions on the basis of clinical signs and symptoms alone. If there is no systemic involvement, definitive diagnosis can be made with incisional biopsy for tissue diagnosis and bacteriological examination with tissue culture. A biopsy of oral lesion is confirmatory with identification of caseating granuloma with associated epithelioid cells and giant cells of the Langerhans type during histological evaluation. But the efficiency of demonstration of acid fast bacilli in histological specimen is low, as there is relative scarcity of tubercle in oral biopsies^{20,21}. And also there is a need for rapid and sensitive of *M.tuberculosis* in tissue specimens, as culture techniques lack sensitivity, present technical difficulties and require a wait of 4-6 weeks for results²². A chest X-ray and Mantoux test are mandatory to rule out systemic tuberculosis^{20,23,24}. According to various studies only a small percentage (7.8%) of histopathology stain positive for acid fast bacilli^{15, 25}. Therefore negative result does not completely rule out the possibility of TB. Another concern is the occurrence of mycobacterial infection as a part of AIDS. Histologically an immunocompromised patient may not show granuloma or caseation. This poses a potential problem in diagnosing TB. The prevalence and incidence of TB is similar in HIV +ve and HIV -ve individuals, but the risk of active TB was increased for seropositive patients^{25,26,27}. In Indian population when considering the overall prevalence of tuberculosis, the presence of epithelioid cell granuloma is indicative of disease²⁸. Two cases of primary tuberculosis in which smear and culture for AFB, from oral lesion and sputum was negative, the diagnosis was confirmed solely on the basis of history and histopathologic examination²⁹.

Absence of bacilli in oral cavity can be explained by:

1. High immunity of the patient resulting in destruction of bacilli.
2. Their enclosure by local tissue reaction and the small numbers of the tubercle bacilli in oral lesions which is why direct examination of scrapings with Ziehl Neelson stain are usually negative.
3. Previous long term treatment with antibiotics^{29,30}. Tuberculosis of gingiva is a relatively infrequent finding and should be considered in diagnosis particularly as a non healing lesion that does not respond to usual therapy. In the differential diagnosis various pathological entities like enlargement due to drugs, malignancies, traumatic ulcer, or infection (bacterial, fungal or viral) should be considered. For a dentist oral tuberculosis can be a diagnostic challenge especially when manifestation is in the form of gingival enlargement. Hence, complete physical examination, anti tuberculin test (Mantoux), immunological test (ELISA), biopsy play a crucial role in the successful diagnosis and treatment of oral tuberculosis.

CONCLUSION:

Tuberculosis infection of the gingiva is relatively rare, oral lesions usually are secondary to pulmonary tuberculosis. To characterise oral lesion as primary tuberculosis, thorough examination with appropriate differential diagnosis should be attempted.

With the recent increase in incidence of tuberculosis, clinicians need to be aware of this possibility, consider TB in the differential diagnosis of gingival enlargement and thus play a role in the prompt detection and treatment of the disease.

Figure 1: Frontal View Of Gingival Enlargement



Figure 2: 10 X H&E (Low Power) Slide Showing Multiple Caseating Granulomas With Overlying Epithelium

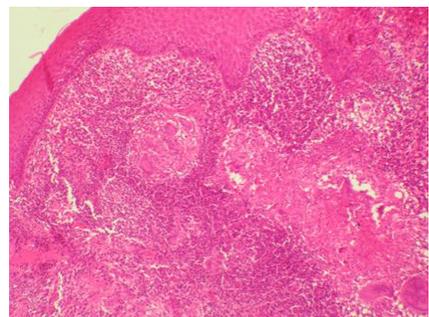


Figure 3: 10 x H& E (Low Power) Cheesy Necrosis And Mixed Inflammatory Infiltrate

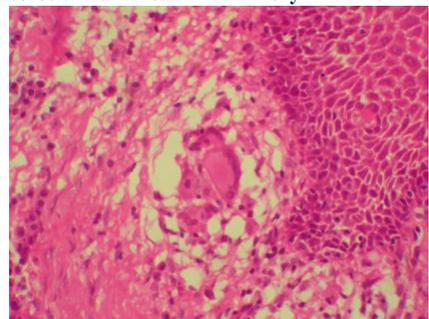


Figure 4 (A) : 40x H& E (High Power) Showing Multinucleated Langhans Cell

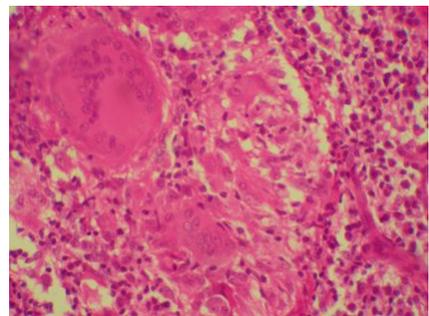


Figure 4 (b): 40x H& E (High Power) Showing Multinucleated Langhans Cell

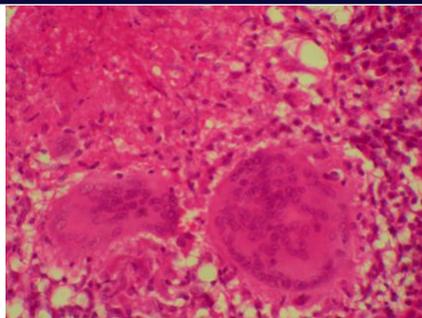


Figure 5: 1yr After Treatment



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