

# PRIMARY GINGIVAL TUBERCULOSIS MANIFESTING AS GINGIVAL ENLARGEMENT: A RARE CLINICAL ENTITY

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## ABSTRACT

Tuberculosis is a specific granulomatous infectious disease and a major cause of death in developing countries. Since the introduction of effective drug therapy, tuberculous lesions of the oral cavity have become so rare that this manifestation of the disease is often forgotten and pose a diagnostic problem. Oral lesions usually appear as secondary to primary tuberculosis infection elsewhere. The lesion may take the form of nodules, ulcers, or elevated fissures. We report a case of primary tuberculosis of gingiva, manifesting as gingival enlargement, which is a very rare finding in tuberculosis. Diagnosis was based on histopathologic examination, complete blood count, chest X-ray, and immunologic investigations with detection of antibodies against *Mycobacterium tuberculosis*. The possibility of gingival enlargement due to drugs, leukemia, fungus and sarcoidosis was ruled out. Antituberculous therapy over 6 months was followed by surgical excision of the enlargement under local anesthesia. This case report also emphasizes the need for clinicians to be aware of this possibility, consider tuberculosis in the differential diagnosis of gingival enlargement, and thus, play a role in the early detection of this disease.

**Keywords:** Caseating necrosis, chronic granulomatous infection, epithelioid cells, Langhans-type giant cell.

## INTRODUCTION

Tuberculosis is a chronic granulomatous infectious disease caused by *Mycobacterium tuberculosis* and is a major health problem in most developing countries. It can affect any part of the body including the oral cavity, though extra pulmonary tuberculosis is rare, occurring in 10% to 15% of all cases<sup>1</sup>. Oral tuberculosis can be primary or secondary. Primary oral tuberculous lesions are extremely rare and generally occur in young adults. It usually involves gingiva and is associated with caseation of the dependent lymph nodes; the lesion itself

remains painless in most cases<sup>2</sup>. In contrast, secondary oral tuberculosis is common and is usually seen in older adults<sup>3</sup>. The most commonly affected site is the tongue, followed by palate, lips, buccal mucosa, gingiva, and frenulum<sup>4</sup>. Tuberculous lesions may present as superficial ulcers, patches, indurated soft tissue lesions, or even lesions within the jaw in form of osteomyelitis<sup>5</sup>. There have been very few published reports of primary tuberculosis of the oral cavity manifesting as gingival enlargement. We report a case of primary tuberculous gingival enlargement as an early presenting sign of tuberculosis, with no

regional lymph node involvement and no evidence of systemic tuberculosis.

### CASE REPORT

A 28 years old male patient reported to the Department of Periodontics in Dental College Azamgarh (UP), with progressive, non-painful swelling of lower gingiva for the past 6 months. The patient had a history of rising temperature in the evening and weakness over the past 3 to 4 months, loss of appetite, and a weight loss of about 5 kg during the past 6 months. On examination, he was of good build, pulse temperature, and respiration rates were normal. Extraoral examination revealed no significant cervical lymphadenopathy.

The initial intraoral clinical examination showed diffuse enlargement of mandibular gingiva extending from right 1<sup>st</sup> premolar to left 1<sup>st</sup> premolar region (Fig 1). Oral hygiene of the patient was very poor. It appeared to be inflammatory gingival enlargement. The colour of the gingiva was brownish pink. On palpation, the swelling was slightly tender but firm and had a tendency for bleeding on provocation. The differential diagnosis included enlargement due to drugs (e.g., phenytoin, nifedipine, cyclosporine, etc.), infection (bacterial, fungal and viral) and hematologic malignancy, such as leukemia. The possibility of drug-induced enlargement was ruled out based on the medical history. Results of a complete blood count were within normal limits except for a marginal rise in leukocyte count ( $14 \times 10^9/L$ ) and an elevated erythrocyte sedimentation rate (ESR) of 61 mm/h (Westergren method), which ruled out leukemia-associated enlargement and raised the possibility of one of the commoner causes of high ESR, tuberculosis. IOPA X-rays revealed slight crestal bone loss without any periodontal or periapical pathology. The patient was then advised tuberculin test, chest X-ray, sputum culture, and immunoglobulins test for tuberculosis.

A tuberculin (Montoux) test was positive, suggesting tubercular infection. Chest radiography (posteroanterior view) revealed no abnormalities. Culture of sputum was negative for *M. tuberculosis*. An immunologic test to detect antibodies against *Mycobacterium* in the patient's serum (ELISA) was positive.

An incisional biopsy from the mandibular labial gingiva adjacent to the central incisors was performed. Histopathologic examination revealed clusters of epithelioid cells, caseating necrosis, and numerous Langhans-type giant cells surrounded by a chronic inflammatory type of infiltrate (Fig 2).

Thus the diagnosis of primary gingival tuberculosis was confirmed and, in consultation with a physician, antitubercular therapy was initiated with isoniazid (10 mg/kg body weight), rifampicin (10–20 mg/kg), pyrazinamide (20–35 mg/kg) and ethambutol (25 mg/kg) for 2 months followed by isoniazid (10 mg/kg) and rifampicin (10–20 mg/kg) for the following 4 months. The possibility that dentists may contact an infection by contact with living tubercle bacilli in the mouths of patients who have oral tuberculosis or pulmonary tuberculosis is a problem of great clinical significance. During this period, the patient was instructed not to undergo any surgical procedure within the oral cavity and was warned about the chance of transmitting the disease to others via salivary contamination. Further, basic periodontal therapy, which included scaling and root planing, and oral hygiene were instituted (Fig 3). This resulted in significant regression of the enlarged gingivae in both arches. After completion of the 6-month regimen, internal bevel gingivectomy was performed to shape and contour the residual enlargement under universal aseptic conditions. No recurrence of the

lesion occurred during 6 months follow-up (Fig 4).

## DISCUSSION

Tuberculosis remains the leading cause of death worldwide. The vulnerability to tuberculosis in developing countries results from poverty, economic recession and malnutrition. Extrapulmonary tuberculosis like tuberculosis of gingiva is an uncommon condition. The reason for its rare occurrence may be that the intact squamous epithelium of the oral cavity resists direct penetration by bacilli<sup>6</sup>. This resistance may also be attributed to the thickness of the oral epithelium, the cleansing action of saliva, local pH, and antibodies in saliva<sup>1</sup>. The mode of entry of the organism may be through a break in the mucous membrane caused by local trauma<sup>6</sup>. Histopathologic examination revealing a granulomatous lesion and identification of the bacilli using special stains lead to presumption of a diagnosis of tuberculosis. But there is a need for rapid and sensitive detection of *M. Tuberculosis* in tissue specimens, as culture techniques lack sensitivity, present technical difficulties, and require a wait of 4–6 weeks for results<sup>5</sup>. The differential diagnosis of our case included enlargement and ulceration due to infection (bacterial, fungal, and viral) and hematologic malignancy, such as leukemia. Results of a complete blood count were within normal limits, except for a marginal rise in leukocyte count and an elevated ESR, which ruled out leukemia-associated enlargement and raised the possibility of one of the more common causes of high ESR---tuberculosis. Microscopic examination raised the possibility of chronic granulomatous infection, including *M. tuberculosis*, fungal etiology, or sarcoid granuloma. Sections stained with periodic acid-Schiff and Grocott-Gomori stains for bacteria and fungi were negative. Levels of serum calcium and angiotensin-converting enzyme were not elevated, which ruled out sarcoidosis. For many years, tuberculosis

has been recognized as an occupational risk for health care workers, especially the dentists.

The tongue and gingiva are the most common sites of infection in patients with oral TB, followed by that of tooth sockets, soft palate, floor of mouth, lips and buccal mucosa. Primary gingival involvement is more common in children and adolescents than adults. It usually presents as a single painless indolent ulcer, which progressively extends from the gingival margin to the depths of the adjacent vestibule and is often associated with enlarged cervical lymph nodes<sup>7</sup>. Oral lesions can also present as nodules, fissures, plaques, vesicles, tuberculomas or granulomas and the last two are usually caused by underlying pulmonary disease. They may be single or multiple, painful or painless and usually appear as irregular, well circumscribed ulcer with surrounding erythema without induration and satellite lesions are commonly found<sup>8</sup>. Primary tuberculosis of gingiva presenting as gingival enlargement is very rare, we could find only one case documented in the literature of primary tuberculous gingival enlargement which was reported by Karthikeyan BV, Pradeep AR, Sharma CG in 2006<sup>9</sup>. In this case also tuberculous lesion appeared as diffuse gingival enlargement instead of the usual manifestation as an ulcer or localized granular mass.

## CONCLUSION

With the decreased incidence of TB, unusual manifestations of oral TB are likely to be missed. The clinician needs to be aware of these various lesions and should consider TB in the differential diagnosis of any gingival enlargement lesions of the oral cavity and thus can contribute in control of tuberculosis through early detection and referral of patients to physicians for proper treatment.

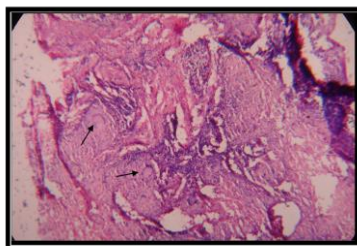
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## FIGURES:



Figure 1



Photomicrograph showing Langhan type of giant cells (10x)

Figure 2



Figure 3



Figure 4