

A rare, unusual presentation of tuberculosis in oral cavity-A case report

Anuradha Ganesan^{1,*}, Gautham Kumar N², Krithika CL³, Yesoda Aniyam K⁴

^{1,3,4}Department of Oral Medicine & Radiology, SRM Dental College, Ramapuram, Chennai, India.

²Department of Periodontics, Madha Dental College & Hospital, Kundrathur, Chennai-600069, India.

Abstract

Tuberculosis (TB) is a chronic granulomatous communicable infection of global health concern caused by various strains of mycobacteria, usually mycobacterium tuberculosis in humans. The most affected organ in TB is the lungs. TB also may involve the central nervous system, lymph nodes, hepatosplenic, genitourinary, musculoskeletal regions and gastrointestinal system. Oral TB has a rare occurrence and is considered to account for 0.1 -5% of all TB infections. Oral tubercular lesions are rare, difficult to diagnose, and pose a potential infectious hazard to dental personnel engaged in the treatment. This case report is of a 54-year-old male patient who presented with an ulceration in the lower labial mucosa and alveolar mucosa in relation to 31-37. This case report also highlights the importance and awareness of oral TB lesions among health care professionals.

Keywords: Tuberculosis, TB, Oral cavity, Mycobacteria, Lesions

Case study

*Corresponding Author, e-mail: anug77@yahoo.com

1. Introduction

Tuberculosis (TB) is a chronic granulomatous communicable infection of global health concern caused by various strains of mycobacteria, usually mycobacterium tuberculosis in humans. There is an extremely high prevalence of the disease in Asia and India. It accounts for nearly one third of global burden of tuberculosis [1]. The upward trend in the number of cases is due to the development of drug resistant strains and due to the emergence of HIV seropositive patients. The most affected organ in TB are the lungs. TB also may involve the central nervous system, lymph nodes, hepatosplenic, genitourinary, musculoskeletal regions and gastrointestinal system [2]. Oral TB has a rare occurrence and is considered to account for 0.1 -5% of all TB infections. The lesions are usually secondarily inoculated with infected sputum or due to haematogenous spread [3]. Oral TB can occur at any site in the oral cavity, but tongue is the most common site. Other sites include the palate, lips, buccal mucosa, gingiva and floor of the mouth [4,5]. Oral lesions can have a variety of clinical manifestations and can be a challenge for diagnosis. This case report is of a 54-year-old male patient who presented with an ulceration in the lower gingiva and alveolar mucosa in relation to 31-37. This case report also highlights the importance and awareness of oral TB lesions among health care professionals.

2. Case Report

A 54-year-old male patient reported to Oral Medicine and Radiology clinic with the chief complaint of pain and an ulcer in the gingiva in relation to lower left back tooth region for the past 6 months. History of this illness revealed that the ulcer started as a small one 6 months back and progressively increased in size to reach the present size and was associated with continuous pain since 1 month. There was no evening rise of temperature and the patient noticed weight loss since 7-8 months. Patient had visited a dental physician and was advised topical and systemic medications and scaling of teeth and periodontal treatment. Since the lesion did not show any signs of healing even after medications and dental treatment, the patient visited our dental clinic.

The medical history revealed that the patient was diabetic and hypertensive and was on regular medication. Family history was non-contributory, and the patient did not have any habit of smoking or chewing tobacco. On examination of the lymph nodes, the right and left submandibular lymph nodes were enlarged, mobile and firm in consistency.

On intraoral examination, there was an ulcerative lesion in the lower left labial mucosa, gingiva and alveolar mucosa in relation to 31-37 region [Fig 1].

The mucosa covering the lesion was rough and irregular and was erythematous. It measured about 3x6 cm with irregular margins and the edges were raised. The floor of the ulcer was covered with slough. On palpation, all the inspectory findings were confirmed and the ulcer was tender and firm in consistency. There was also mild bleeding present on periodontal probing with Grade II mobility of 33-36. Based on these clinical findings, a provisional diagnosis of non-healing ulcerative lesion in relation to 31-37 was given. Differential diagnosis included malignant ulcer, ulcers due to fungal and bacterial infection. An intra oral periapical radiograph and an orthopantomograph was taken and it showed generalised bone loss suggestive of chronic generalised periodontitis [Fig 2]. An incisional biopsy of the lesion was advised and under high power, the section showed stratified squamous epithelium and underlying connective tissue stroma. Few areas in the underlying connective tissue stroma had formation of granuloma consisting of central necrosis surrounded by epithelioid cells, lymphocytes and multi nucleated giant cells resembling Langhans giant cells [Fig 3]. Connective tissue stroma exhibited dense infiltration of mixed inflammatory cell infiltrate with neutrophils, plasma cells and lymphocytes. The histopathological features were suggestive of granulomatous tuberculosis ulcer of gingiva. The patient was advised for routine haematological investigation which showed elevated erythrocyte sedimentation rate (54mm/1st hour-Wintrobe) and positive Mantoux test. The chest radiograph showed areas of patchy consolidation and foci [Fig 4].

A diagnosis of extra pulmonary TB involving oral cavity was considered and the patient was treated with anti-tubercular therapy. Anti-tubercular regimen which included Isoniazid (H), Rifampicin (R), Pyrazinamide (Z) and Ethambutol (E) for a duration of two months followed by a regimen of Isoniazid (H), Rifampicin (R) and Pyrazinamide (Z) for four months, dosage based on the body weight of the patient. By the end of 2 months of therapy, the ulcerative lesion showed satisfactory healing [Fig 5] and by the end of 4 months, the ulcer had completely healed [Fig6].

3. Discussion

Tuberculosis is a disease characterized by granulomatous lesions caused by *Mycobacterium Tuberculosis* (Mtb) an aerobic, non-motile, non-capsulated, non-spore forming acid fast bacilli. History of Tuberculosis goes back 15,000 to 20,000 years ago in the relics from ancient Egypt, China and India. Archaeologists have detected spinal tuberculosis as Pott's disease in Egyptian mummies and was known as King's evil. In the 18th century, it was termed white plague when it reached its peak prevalence. It was considered as a stigma in the society and even compared to a 'devouring dragon' in some parts of Europe.[6] After Robert Koch demonstrated the causative organism in 1882, Edward Livingston Trudeau in 1884 started the concept of isolating these patients from the society, treating them with rest and nutrition. Later Albert Calmette and Camille Guerin in Lille, France in 1908 invented, *Bacillus Calmette Guerin* and this vaccine was first used in humans in 1921. The discovery of this vaccine made a revolution and now is counted in WHO's list of most essential medication for basic health system. The WHO Global Tuberculosis Report 2022 showed an global estimate of 10.6 million people affected

with TB in 2021, equivalent to 134 cases per 100 000 population. India with 28% cases was among the eight countries accounting for more than two-third (68.3%) of the total TB patients' count. [7].

Pulmonary TB (PTB) is a multibacillary disease and sputum of these patients contains a larger number of bacilli. On the other hand, oral TB is a paucibacillary disease and concentration of acid-fast bacilli (AFB) is significantly less in saliva. The resistance of striated muscles to bacterial invasion, saprophytes, and thickness of protective epithelial covering are the factors contributing to decreased susceptibility for the development of oral TB. Another very important factor for oral TB susceptibility is a breach of the oral mucosa, which may lead to the colonization of bacteria [8]. Immunocompromised states, such as HIV, diabetes mellitus, malnutrition, malignancies prolonged corticosteroids therapy, and chronic renal failure, may also pose a threat to the development of TB [9]. Oral TB lesions are rare and can be either primary or secondary in occurrence. Primary lesions occur in younger patients often associated with enlarged cervical lymph nodes. Secondary oral TB usually coexists with pulmonary disease, may occur in all age groups and the most likely route of inoculation is the entry of organisms in the sputum and, from there, entry into the mucosal tissue through a small break in the surface. It is possible that the organisms may be carried to the oral tissues by a haematogenous route, to be deposited in the submucosa, and subsequently to proliferate and ulcerate the overlying mucosa. TB lesions in the oral cavity present as slow growing, single, indurated, irregular, radiating, superficial, or deep painful ulcer with undermined edges covered by inflammatory exudates. Other clinical presentations include nodules, fissures, tuberculomas, and granulomas. Lesions may be single or multiple, painful or painless. Skin, cervical lymph nodes, and salivary glands are also frequently involved. TB may involve the jaws causing tuberculous osteomyelitis or rarefying osteitis [10]. In this case, patient presented with a ulcerative lesion in the gingiva and mobility of the teeth and extensive bone loss similar to a case reported by Jingta P K et al [11]. Even with an increase number of oral TB cases reported, the lack of pathognomic signs makes it a diagnostic challenge. Since most early lesions resemble a neoplasm or inflammatory lesion, due to the lack of systemic manifestations, anti-inflammatory treatment and surgical resection are advocated. Other lesions that can be considered as a differential diagnosis for a chronic non-healing ulcer are malignancy, ulcers in HIV infection, cicatricial pemphigoid, syphilis, and deep mycotic infection such as Histoplasmosis, Wegener's granulomatosis, and Sarcoidosis [12,13].

Various investigations can be used to diagnose TB, which include medical imaging, Ziehl Neelsen staining, testing the patient's immune response [tuberculin skin testing – Mantoux Test and interferon gamma release assays], histopathology. Biopsy from oral lesions remains the gold standard for confirmation of oral TB lesions and the histopathological presentation in TB is mainly due to cell-mediated hypersensitivity reaction. Granuloma formation is seen in the area of infection along with epithelioid cells, lymphocytes, and multinucleated giant cells. A single granuloma is called as "tubercle". Langhans giant cells show a horseshoe-shaped arrangement of the nuclei. Demonstration of acid fast bacilli in histological specimens are low due to the scarcity of tubercle bacilli in oral biopsies,

however negative results do not rule out the possibility of TB. PCR is the most advanced diagnostic procedure to identify *Mycobacterium tuberculosis* DNA [14]. The treatment of oral TB lesions is similar as systemic TB. Currently, the most effective regimens require a combination of 4 drugs [isoniazid (INH), rifampicin (RIF), pyrazinamide (PZA), and ethambutol (ETO)] administered daily for the first 2 months, followed by an additional 4 months with 3 drugs (INH, RIF and ethambutol). The difficulty of this regimen prompted the WHO to launch a new global strategy for TB control known as “Directly Observed Therapy, Short course” (DOTS) in 1997. The central component of this strategy is a direct observation by trained personnel, which secures both patient compliance with the drug regimen and decreases the likelihood of drug resistance. The management of TB is strenuous because of the two primary factors: persistence and resistance. In spite of the fact that antibiotics are accessible, *Mtb* is extremely persistent, possibly because the bacterium encourages chronic inflammation that sequesters it inside the tissues, defending it against drug exposure. Thus, drug treatment must be extended to completely damage the bacterium and prevent relapse. Drug resistance is the result of genetic mutations that cause a heritable loss of drug susceptibility. Even though resistance to a single drug does not render therapy unsuccessful, multidrug-resistant strains make TB much more expensive and difficult to treat. For this reason, they require newer and more effective drugs that achieve multiple goals in improving TB controls that are imperative. There are two types of resistance commonly observed in the context of TB: multidrug resistant TB (MDR) and extensively drug resistant (XDR). MDR-TB is described as *Mtb* resistant to the most effective first-line anti-TB drugs, isoniazid, and rifampicin, whereas XDR-TB has additional multidrug resistance to the most potent second-line agents,

injectable drugs (aminoglycosides and/or cyclic polypeptides-capreomycin, amikacin, and kanamycin), and fluoroquinolones [15].

Dental healthcare professionals are at risk of getting exposed to TB due to splatter, aerosols, or infected blood. As various severe diseases are air-borne, blood-borne, or can extend through the contact of other body fluids, and it is not possible to know which certain patients are infected, it is pertinent to avoid direct contact with body fluids, blood, and mucous membranes. Only urgent and necessary dental procedures should be carried out in patient with active TB. High levels of operator disinfection and instrument sterilization should be provided. For recognized active TB patients, TB isolation rooms that are properly equipped rooms with functional air evacuation, negatively pressured correlative to the corridors with air either debilitated to the outside or HEPA-filtered if recirculation is mandatory, with high volume suction to reduce the aerosol production [16].

Use of rubber dams limits aerosol contact, but should not be used in a patient with persistent cough. Personal protective gears (head caps, eye shields, face masks, gloves, and surgical gowns), and maintenance of proper hand hygiene should be followed, including use of particulate face masks as against standard masks which do not protect against TB. A wet mask should be changed immediately, either inter or intra appointment [17]. Other oral instruments and handpieces should be cleaned and autoclaved routinely. The aim of the dental infection-control program is to bestow a safe working environment that minimizes the risk of both healthcare-related infections between patients and occupational exposures among dental team members [18].



Figure 1. Ulcerative lesion in the labial mucosa and gingiva 31-37 region



Figure 2. Orthopantomograph showing bone loss

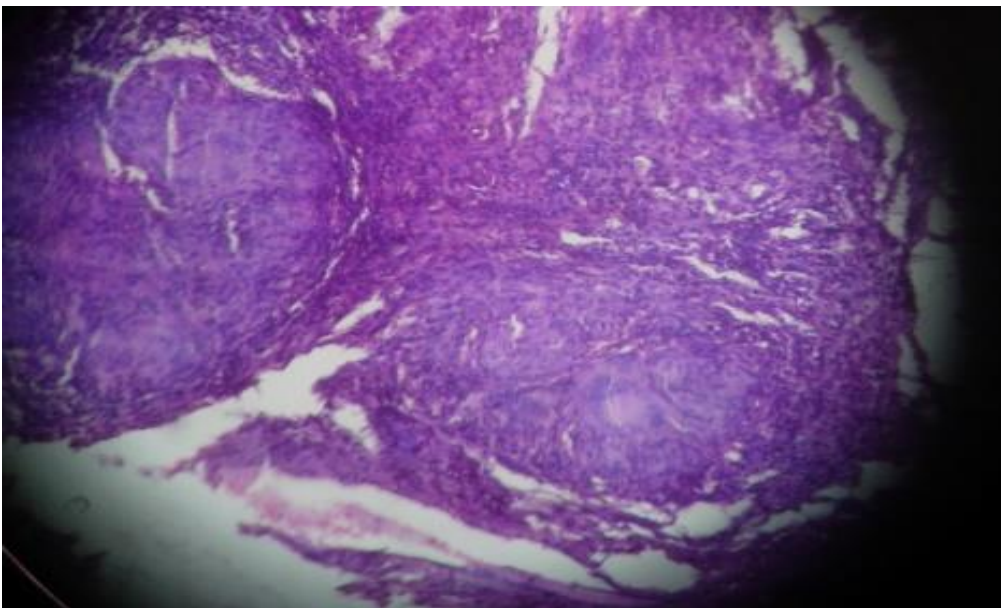


Figure 3. Photomicrograph showing langhans cells, epitheloid cells, and lymphocytes

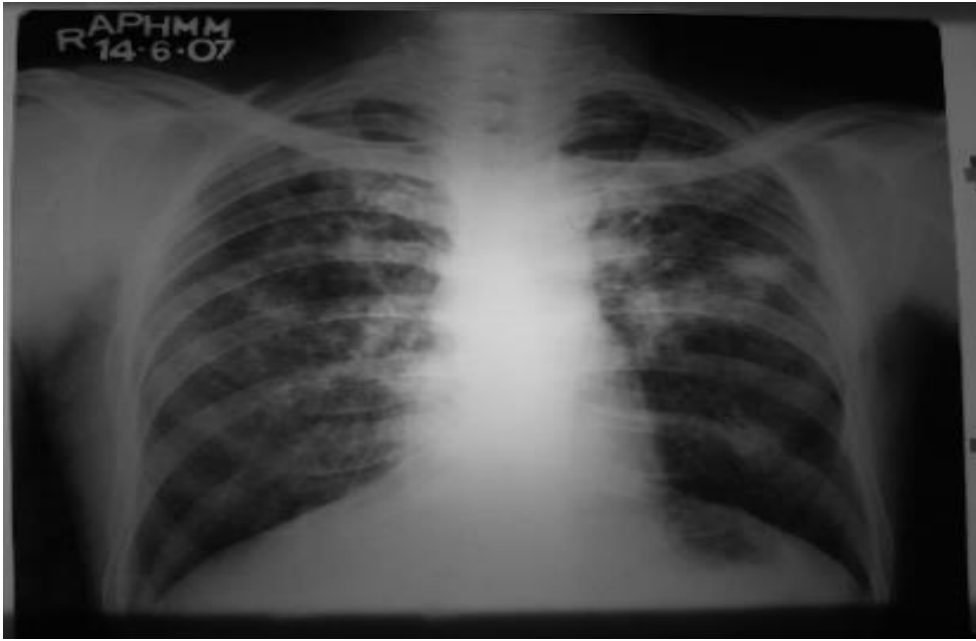


Figure 4. Chest radiograph shows signs of consolidation.



Figure 5. Shows healing tuberculous ulcer (two months).



Figure 6. Shows healed tuberculous ulcer (four months)

4. Conclusions

Oral tubercular lesions are rare, difficult to diagnose, and pose a potential infectious hazard to dental personnel engaged in the treatment. So, each and every persistent and atypical oral lesion must be examined carefully to intercept and prevent the disease early. Intercepting the disease early will advance the morbidity and mortality of the patients. So, it becomes the responsibility of the dentist to include TB in the differential diagnosis of suspicious oral lesions to avoid delay in the treatment of this disease.

References

- [1] V.G. Rao, M.Muniyandi, J.Bhat, R. Yadav, R. Sharma. (2018). Research on tuberculosis in tribal areas in India: A systematic review. *Indian Journal of Tuberculosis*.65:8-14
- [2] P. Pang, W. Duan, S. Liu, S. Bai, Y. Ma, R. Li, et al. (2018). Clinical study of tuberculosis in the head and neck region-11 years' experience and a review of the literature. *Emerging Microbes and Infections*. 7:1-10.
- [3] P. Jain, I. Jain. (2014). Oral manifestations of tuberculosis: Step towards early diagnosis. *Journal of Clinical and Diagnostic Research*. 8:18-21.
- [4] A. Ganesan, G. Kumar. (2017). Scrofuloderma: A rare cutaneous manifestation of tuberculosis. *Journal of Indian Academy of Oral Medicine and Radiology*.29:223-6
- [5] F.A. Ito, CR.de Andrade, P.A. Vargas, J. Jorge, M.A. Lopes. (2005). Primary tuberculosis of the oral cavity. *Oral Diseases*.11:50-3.
- [6] R. Jaiswal, M. Badni, A. Singh, P. Singh (2011). Oral tuberculosis involving maxillary gingiva. *National Journal of Maxillofacial Surgery*. 2:175-6.
- [7] WHO: 2022. *Global Tuberculosis Report (2022)* Geneva, Switzerland: World Health Organization.
- [8] F.N. Pekiner, G. Erseven, MO Borahan, B. Gumru (2006). Natural barrier in primary tuberculosis inoculation: Oral mucous membrane. *The International Journal of Tuberculosis and Lung Disease*. 10:1418.
- [9] A. Ganesan, A. Muthukrishnan, V.P. Veeraghavan, N.G. Kumar. (2022). Effectiveness of Salivary Glucose as a Reliable Alternative in Diagnosis of Type 1 Diabetes Mellitus: A Cross-Sectional Study. *J Pharm Bioallied Sci*. Jul;14(Suppl 1): S557-S562.
- [10] S. Sharma, J. Bajpai, P.K. Pathak, A. Pradhan, P. Singh, S. Kant. (2019). Oral tuberculosis - Current concepts. *Journal of Family Medicine and Primary Care*. 8:1308-12.
- [11] P. Jhingta, P. Machhan, D. Sharma, S. Vaid, V. Bhardwaj, N. Gupta. (2015). Primary isolated gingival tuberculosis: A rare case report. *International Journal of Health and Allied Sciences*. 4:45
- [12] P.M. Kamath, V.S. Shenoy, M. Nirupama, V. Prasad, N.A. Majeed. (2015). Tuberculosis of Waldeyer's ring with an atypical presentation as chronic adeno-tonsillitis. *Journal of Clinical and Diagnostic Research*.9:MD01-2
- [13] A. Von, A. Husain. (2001). Oral tuberculosis. *British Dental Journal*. 190:420-2.

- [14] A. Konstantinos. (2010). Testing for tuberculosis. *Australian Prescriber* .33:8-12.
- [15] P. Nahid, S. Dorman, N. Alipanah, P. Barry, J. Brozek, A. CattamanchiA et al. (2016). Executive summary: Official American thoracic society/Centers for disease control and prevention/infectious diseases society of America clinical practice guidelines: Treatment of drug susceptible tuberculosis. *Clinical Infectious Disease* .63:853-67.
- [16] M.R. Popescu, I.E. Plesea, M. Olaru, I.R. Strâmbu, A.I. Fronie, I.O. Petrescu et al. (2015). Morphological aspects in tuberculosis of oral cavity—Our experience and a review of the literature attempt. *Romanian Journal of Morphology and Embryology* .56:967-87.
- [17] H. M. Blumberg, W.J. Burman, R.E. Chaisson, C.L. Daley, S.C. Etkind, L. N. Friedman et al. (2003). American thoracic society, centers for diseases control and prevention, infectious disease society of America. Treatment of tuberculosis. *American Journal of Respiratory and Critical Care Medicine* .167:603-62.
- [18] A. Srividya, A. Kannan, C.L. Krithika. (2021). Knowledge, Attitude and Practice Survey on Special Care Dentistry: A Cross-sectional Study. *Journal of Clinical and Diagnostic Research*. Jul, Vol-15(7): ZC14-ZC18