



## ORAL TUBERCULOSIS- A NEED FOR SPY EYE'S

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### ABSTRACT

Tuberculosis is a chronic infectious disease which can affect any part of the body including the mouth. The involvement of oral cavity in tuberculosis is rare and the non specific nature of its clinical presentation makes the diagnosis of tuberculosis delayed. The purpose of this article to emphasize the importance of early diagnosis tuberculosis involving the oral cavity by medical and dental practitioners and considering them in the differential diagnosis of suspicious oral ulcers and thus preventing the morbidity and mortality associated with this life threatening infectious disease.

**KEYWORDS:** Oral tuberculosis, Ulcer, Osteomyelitis, Saliva, Primary lesions, Secondary lesions



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## INTRODUCTION

Tuberculosis is a major cause of ill health and death worldwide, but has been declining in incidence in industrialized countries until recently.<sup>1</sup> It is a chronic infectious granulomatous disease caused mainly by *Mycobacterium tuberculosis*, an acid-fast bacillus that is transmitted primarily through the respiratory route through inhalation of infected airborne droplets containing the bacillus, *M. tuberculosis*. Less commonly, TB is caused by exposure to *Mycobacterium bovis* through ingestion of unpasteurized, infected cow's milk or other atypical mycobacteria.<sup>2</sup> History of Tuberculosis goes back to some 15,000 to 20,000 years ago. It has been found in relics from ancient Egypt, China and India. Archeologists have detected spinal tuberculosis as Pott's disease in Egyptian mummies.<sup>3</sup> It was known as King's evil. In the 18<sup>th</sup> century, it reached its peak prevalence of, as much as, 900 deaths per 100,000 and was termed as white plague. It was considered as a stigma in the society and even compared to a 'devouring dragon' in some parts of Europe. After Robert Koch demonstrated the causative organism on 24<sup>th</sup> March 1882, Edward Livingston Trudeau in 1884 started the concept of isolating these patients from the society, treating them with rest and nutrition. In the year 1904, National Tuberculosis Association (American Lung Association) came into being.<sup>3</sup> Later, Bacillus Calmette Guerin was invented by Albert Calmette and Camille Guerin in Lille, France in 1908. But it was first used in humans in 1921.<sup>3</sup> It has been a worldwide major health problem for centuries. Although the disease's prevalence reduced decades ago, it still has extremely high prevalence in Asian countries. India accounts for nearly one third of global burden of tuberculosis.<sup>4</sup> According the most recent report of WHO (2013), nearly 8.6 million people around the world became infected with TB disease. There were around 1.3 million TB-related deaths worldwide. An estimated 1.1 million (13%) of the 8.6 million people who developed TB in 2012 were HIV-positive. Increasing problems with TB may well continue because of the continuing emergence of MDR

strains of *M. TB*, which is a major threat, particularly with HIV- and AIDS-infected patients, among whom, mortality rates are high. Globally in 2012, an estimated 4.5 lacs people developed MDR-TB (Multi-drug resistant Tuberculosis) and there were estimated 1.7 lacs deaths from MDR-TB. The majority of cases worldwide in 2012 were in the South-East Asia (29%), Africa (27%) and Western Pacific (19%) regions. India and China alone accounted for 26% and 12% of total cases, respectively.<sup>5</sup> Pulmonary TB is the most common form of disease. However, TB can also occur in the lymph nodes, meninges, kidneys, bone, skin, and in the oral cavity.<sup>1,6</sup> Oral lesions of TB are nonspecific in their clinical presentation and are present before systemic symptoms became apparent. Oral lesions are seen in 0.05 to 5% of the patients with TB and may be either primary or secondary. Primary forms generally are uncommon and occur in younger patients with frequently associated caseation of the draining lymph nodes. Secondary lesions are more common and are seen mostly in older persons.<sup>7</sup> In dental clinics, oral health workers are at high risk for *M. tuberculosis* infection because of close contact with patients and aerosol spread during the dental treatment process.<sup>6</sup> This review is an attempt to assess the need of the hour to early diagnose manifestations and symptoms of T.B in the oral cavity as documented in the earlier archives published till date and thus reduce the risk of exposure to the patients contacts.

## DISCUSSION

### ORAL MANIFESTATIONS

Although oral tuberculosis has been well documented, tuberculous lesions of the upper aerodigestive tract have become rare. Tuberculous lesions of the mouth may be either primary, or secondary to pulmonary tuberculosis, with secondary lesions being more common.<sup>1</sup> Saliva is considered to have a significant role which explains the paucity of oral lesions, despite the large numbers of bacilli present in sputum contacting the oral mucosa in a typical case of pulmonary tuberculosis.

Other attributing factors to relative resistance of oral cavity for TB are presence of saprophytes, resistance of striated muscles to bacterial invasion, and thickness of protective epithelial covering. It is believed that the organisms enter the mucosa through small breaches in the surface epithelium which makes it a favourite site for colonization of bacteria. Local factor that may facilitate the invasion of oral mucosa includes poor oral hygiene, leukoplakia, local trauma, and irritation by clove chewing, etc. Self-inoculation by the patient usually results from infected sputum or by hematogenous or lymphatic dissemination.<sup>8,9,10</sup> Conditions that predispose to the disease include crowded urban living, drug abuse, poor health and hygiene, poverty. Viral infections like HIV with or without the development of AIDS, cause immunosuppression which has lately emerged as a very significant risk factor for development of TB.<sup>11</sup> The World Health Organization (WHO) estimates approximately 20 million active cases of tuberculosis, 80% of which occur in the developing countries. The regions with the highest incidence of tuberculosis are the Indian subcontinent, South- East Asia, and Africa. The epidemiology of tuberculosis differs considerably with ethnicity. Age has also been implicated as an important risk factor, as well as differing ethnic and socio-economic grouping.<sup>12</sup> Poorer populations are twice as likely to have tuberculosis and three times less likely to access care for this disease.<sup>13</sup> TB oral lesions are a relatively rare occurrence. The oral lesions may be present in a variety of forms, such as ulcers, nodules, tuberculomas, and periapical granulomas.<sup>5,6,14</sup> Oral TB lesions may be either primary or secondary in occurrence. Primary lesions are uncommon, seen in younger patients, and present as single painless ulcer with regional lymph node enlargement. The secondary lesions are common, often associated with pulmonary disease, usually present as single, indurated, irregular, painful ulcer covered by inflammatory exudates in patients of any age group but relatively more common in middle-aged and elderly patients.<sup>15,16,17</sup> Although the tongue is the commonest site for oral tuberculous lesions,<sup>18</sup> they may also occur on the gingiva,

floor of mouth, palate, lips and buccal mucosa.<sup>19</sup> Tuberculosis of the tongue has also presented as macroglossia.<sup>20</sup> It may also be present around the upper aerodigestive tract as parotitis,<sup>21,22</sup> intra-osseous lesions,<sup>23,24</sup> preauricular swelling and trismus,<sup>25</sup> tracheitis<sup>26</sup> and laryngitis.<sup>1</sup> When oral TB occurs as a primary lesion, an ulcer is the most common manifestation usually developing along the lateral margins of the tongue which rest against rough, sharp, or broken teeth or at the site of other irritants. Patients with oral tubercular lesions often have a history of preexisting trauma. Any area of chronic irritation or inflammation may favor localization of the *Mycobacterium* associated with the disease.<sup>27</sup> Deep tubercular ulcers of the tongue are typical in appearance with a thick mucous material at the base. These tongue lesions are characterized by severe unremitting and progressive pain that profoundly interferes with proper nutrition and rest. Classically, tubercular ulcers of the tongue may involve the tip, lateral margins, dorsum, the midline, and base of the tongue. They are irregular, pale, and indolent with inverted margins and granulations on the floor with sloughing tissue.<sup>28</sup> Tuberculous lymphadenitis constitutes a component of head and neck disease in up to 90% of patients and presents as single or multiple enlarged lymph nodes that may be firm, fluctuant, or matted with fistula formation.<sup>29,30,31</sup> The oral manifestations of TB can also be in the form of superficial ulcers, patches, indurated soft tissue lesions, or even lesions within the jaw that may be in the form of TB osteomyelitis or simple bony radiolucency.<sup>14</sup> 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. 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>32</sup>

### **DIFFERENTIAL DIAGNOSIS**

The differential diagnosis of a tuberculous ulcer of the oral cavity includes aphthous ulcers, traumatic ulcers, syphilitic ulcers and malignancy, including primary squamous cell carcinoma, lymphoma and metastases. As reported here, the most likely clinical diagnosis is that of squamous cell carcinoma, in which case biopsy is mandatory. It is most likely that tuberculosis was only considered when the histological specimen reveals a granulomatous lesion. This would then lead to consideration of other orofacial granulomatous conditions such as sarcoid, Crohn's disease, the deep mycoses, cat-scratch disease, foreign-body reactions, tertiary syphilis and Melkersson-Rosenthal syndrome.<sup>1</sup>

### **INVESTIGATIONS**

Oral cavity TB is difficult to differentiate from other conditions on the basis of clinical signs and symptoms alone. If there is no systemic involvement, one should go for excisional biopsy for tissue diagnosis and bacteriologic examination with culture for a definitive diagnosis. The efficiency of demonstration of acid fast bacilli in histological specimens is low, as there is a relative scarcity of tubercle bacilli in oral biopsies.<sup>16,33</sup> A biopsy of an oral lesion is confirmatory but in majority of the cases, a single biopsy may not suffice because the granulomatous changes may not be evident in early lesions. The lesion is eventually disclosed by repeat biopsies. The differential diagnosis is made with the identification of a caseating granuloma with associated epithelioid cells and giant cells of the Langerhans type during histological evaluation of biopsied tissue. Deeper biopsies are always advocated for ulcers of the tongue; a superficial biopsy may not reveal the etiology due to epithelial hyperplasia. A chest x-ray and a Mantoux skin test are mandatory to rule out systemic TB.<sup>16,34,35</sup> According to various studies only a small percentage (7.8%) of histopathology specimens stain positive for acid fast bacilli.<sup>32,36</sup> Therefore, a negative result does not rule out completely 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. HIV-1-associated TB is reaching epidemic proportions in many African countries. The prevalence and incidence of TB is similar in both HIV-positive and HIV-negative individuals, but the risk of active TB was elevated only for seropositive subjects. Increasing problems with TB may well continue because of the continuing emergence of MDR strains of *M. TB*, which is a major threat, particularly with HIV- and AIDS-infected patients, among whom, mortality rates are high.<sup>14,32,37</sup> Pandit et al., when considering the overall prevalence of tuberculosis in the Indian population, the presence of epithelioid cell granuloma is indicative of the disease. It is also reported that the percentage of cases showing AFB positivity declines when more epithelioid cell granulomas are observed.<sup>38</sup> Dimitrakopoulos et al. reported two cases of primary tuberculosis of the oral cavity where smears and culture for AFB, from the oral lesion and the sputum, were negative.<sup>39</sup> They confirmed the diagnosis solely on the basis of history and histopathologic examination, which only revealed giant cells and epithelioid cells. This may be due to (a) high immunity of the patient resulting in the destruction of the bacilli, (b) their enclosure by local tissue reaction and the very small numbers of tubercle bacilli in oral lesions, which is why direct examination of scrapings stained with the Ziehl-Neelsen stain are usually negative, and (c) previous long-term treatment with antibiotics.<sup>31,39</sup>

### **DENTISTS ARE AT RISK**

As stated, contact tracing follows the protocol of the Joint Tuberculosis Committee of the British Thoracic Society. For patients with pulmonary tuberculosis, close contacts are those sharing a house, and are most at risk. Occasionally a contact at work is close enough to be equivalent to a household contact. Casual contacts are most occupational contacts and need only be examined if they are unusually susceptible to infection, such as immunocompromised adults. Investigation of contacts may include Heaf testing and chest radiography. All staff in regular contact with

patients were at potential risk of contracting tuberculosis. However, evidence from the 1980s suggested that the incidence of tuberculosis.<sup>1</sup> TB is a recognized occupational risk for dentists, as we work in close proximity to the nasal and oral cavities of patients, with generation of potentially infectious sprays during routine operative procedures. A history of TB should prompt the clinician to distinguish whether the person is an active case under treatment, active case without treatment or previously infected but currently disease free. The nontreated active cases pose maximum risk to the dental personnel. Only dental emergencies should be undertaken for treatment under controlled environment for active cases of TB, such as the one described here. The constant risk of contracting the disease should encourage the dental clinicians to follow basic precautions of using face masks, protective eye gear, and gloves. Also, high standards of operatory disinfection and instrument sterilization should be maintained.<sup>6,35</sup>

### **PREVENTION A NECESSITY**

Dental Practice has a great potential for transmission of various infections from patient to Dentist, patient to patient as well as a dentist to patient due to close proximity to the nasal and oral cavities of the patient. Dental healthcare professionals are at the constant risk of getting exposed to TB by the means of splatter, aerosols or infected blood. It is important to provide a safe working environment that reduces the risk of both healthcare-associated infections among patients and occupational exposures among dental team members. Dental treatment for those with active Tuberculosis should be limited

### **REFERENCES**

1. Von Arx D.P, Husain A. Oral Tuberculosis. British Dental Journal ,190(8):420-422,( 2001)
2. Kolokotronis A, Antoniadis D, Trigonidis G, Papanagiotou P. Oral tuberculosis: Oral Dis ,2:242-3,(1996)

to urgent and essential procedures. Maintenance of proper hand hygiene, personal protective equipment (eye shields, face masks, headcaps, gloves and surgical gowns) and proper sterilization procedures should be followed. Standard surgical face masks do not protect against TB transmission; dental healthcare personnel should use particulate face masks. Masks should be changed at regular intervals, inter-appointments (between patients) and intra-appointments (during patient treatment) if it becomes wet. Rubber dams can be used to minimize aerosol contact however, if coughing occurs rubber dam should not be used. Any contaminated item is a potential exposure source, so by taking care to limit contamination to the greatest extent possible. Reusable facial protective equipment (protective eyewear or face shields) should be cleaned and disinfected between patients. Handpieces and other oral instruments should be cleaned and autoclaved regularly. Gloves should be worn while working on the patients.<sup>41</sup>

### **CONCLUSION**

Tuberculosis remains a devastating disease throughout the world. Tuberculosis of the oral cavity is relatively rare, the unusual forms of the disease in the oral cavity are more likely to be misdiagnosed; with the increasing incidence of TB, it must be considered in the differential diagnosis of atypical ulcerative lesions of the mouth. Thus identification of tuberculosis is of significance not only to the patient himself, but also to the dental team that comes in contact and the community at large, where the patient can be a potential source for spread of infection.

3. Ananya Mandal MD. History of Tuberculosis. News Medical.net 2014.
4. Kapoor S, Gandhi S, Gandhi N, Singh I. Oral manifestations of tuberculosis. Chrismed J Health Res. ,1:11-14,(2014)
5. World health organization: Global Tuberculosis report 2013.

6. Eng HL, Lu SY, Yang CH, Chen WJ. Oral tuberculosis. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* ,81:415-20,(1996)
7. Goel MM, Ranjan V, Dhole TN, Srivastava AN, Mehrotra A, Kushwaha MR, *et al.* Polymerase chain reaction vs. conventional diagnosis in fine needle aspirates of tuberculous lymph nodes. *Acta Cytol* ,45: 333-40,(2001)
8. Pekiner FN, Erseven G, Borahan MO, Gumru B. Natural barrier in primary tuberculosis inoculation: oral mucous membrane. *Int J Tuberc Lung Dis* ,10(12):1418,(2006)
9. Emmanuelli JL. Infective granulomatous diseases of head and neck. *Am J Otolaryngol* ,14:155-67,(1993)
10. Ito FA, de Andrade CR, Vargas PA, Jorge J, Lopes MA. Primary tuberculosis of the oral cavity. *Oral Dis*,11:50-53,(2005)
11. Woods RG, Amerena V, David P, Fan PL, Heydt H, Marianos D. Additional precautions for tuberculosis and a self-assessment checklist: *FDI World* ,6(3):10-7,(1997)
12. Dye C, Scheele S, Dolin P, Pathania V, Raviglione MC. Global burden of tuberculosis: estimated incidence, prevalence, and mortality by country. *Journal of the American Medical Association* ,282: 677-686,(1999)
13. World Health Organization (WHO). Regional Consultation on Social Determinants of Health. A Report. New Delhi: WHO Regional Office for South-East Asia; 2005.
14. Sierra C, Fortún J, Barros C, Melcon E, Condes E, Cobo J, *et al.* Extra-laryngeal head and neck tuberculosis. *Clin Microbiol Infect* ,6:644-48,(2000)
15. Shafer WG, Hine MK, Levy MB. A Textbook of Oral Pathology. Philadelphia: WB Saunders,340-440,(1983)
16. Chisholm DM, Ferguson MM, Jones JH. Introduction to Oral Medicine. Philadelphia: WB Saunders, 59-60,(1978)
17. Mignogna MD, Muzio LL, Favia G, Ruoppo E, Sammartino G, Zarrelli C. Oral tuberculosis: A clinical evaluation of 42 cases. *Oral Dis* ,6:25-30(2000)
18. Gupta A, Shinde K J, Bhardwaj I. Primary lingual tuberculosis: a case report. *J Laryngol Otol* ,112: 86-87,(1998)
19. Hathiram B T, Grewal D S, Irani D K, Tankwal P M, Patankar M. Tuberculoma of the cheek: a case report. Primary lingual tuberculosis: a case report. *J Laryngol Otol* ,111: 872-873,(1997)
20. Ramesh V. Tuberculoma of the tongue presenting as macroglossia. *Cutis* ,60:201-202(1997)
21. Bhat N A, Stansbie J M. Tuberculous parotitis: a case report. *J Laryngol Otol* ,110: 976-977,(1996)
22. Suoglu Y, Erdamar B, Katircioglu OS, Cevikbas U. Tuberculosis of the parotid gland. *J Laryngol Otol*,112:588-591,(1998)
23. Kothari P, Bartella L, Carter J, Chan O, Piper K. Tuberculosis of the mandible in a child. *J Laryngol Otol*,112:585-587,(1998)
24. Gal G, Kaplan I, Calderon S, Carlson E R. Large perimandibular swelling. *J Oral Maxillofac Surg* , 55: 1134-1143,(1997)
25. Ruggiero S L, Hilton E, Braun T W. Trismus and preauricular swelling in a 20-year-old black woman. *J Oral Maxillofac Surg* ,54:1234-1239,(1996)
26. Tong F M, Chow S K. Primary tuberculous tracheitis. *J Laryngol Otol* ,112: 579-580,(1998)
27. Gupta N, Nuwal P, Gupta ML, Gupta RC, Dixit RK. Primary tuberculosis of soft palate. *Indian J Chest Dis Allied Sci* ,43:119-21,(2001)
28. Memon GA, Khushk IA. Primary tuberculosis of tongue: *J Coll Physicians Surg Park* ,13(110): 604-05,(2003)
29. Popowich L, Heydt S. Tuberculous cervical lymphadenitis. *Journal of Oral Maxillofacial Surgery*,40:522-524,(1982)
30. Mohapatra PR, Janmeja AK. Tuberculous lymphadenitis. *Journal of*

- the Association of Physicians of India., 57: 585-590,(2009)
31. Iqbal M, Subhan A, Aslam A. Frequency of tuberculosis in cervical lymphadenopathy. *Journal of Surgery Pakistan(International)*.,15:107-109,(2010)
  32. Thilander H, Wennestrom A. Tuberculosis of mouth and the surrounding tissues. *Oral surg Oral Med Oral Pathol.* ,9:858-70,(1956)
  33. Haddad NM, Zaytoun GM, Hadi U. Tuberculosis of the soft palate: An unusual presentation of oral tuberculosis. *Otolaryngol Head Neck Surg* ,97:91-9,(1987)
  34. Brennan TF, Vrabec DP. Tuberculosis of the oral mucosa. Report of a case. *Ann Otol Rhinol Laryngol* , 79:601-5,(1970)
  35. Turbiner S, Giunta J, Maloney PL. Orofacial tuberculosis of the lip. *J Oral Surg* , 33:343-7,(1975)
  36. Mani NJ. Tuberculosis initially diagnosed by symptomatic oral lesion. Report of three cases. *J Oral Med* ,40:39-42,(1985)
  37. Rauch MD, Friedman E. Systemic tuberculosis initially seen as an oral ulceration: Report of a case. *J Oral Surg* , 36:387-9,(1978)
  38. Pandit AA, Khilani PH, Prayag AS. Tuberculous lymphadenitis, extended cytomorphological features. *Diagnostic Cytopathology.* ,12: 23-27,(1995)
  39. Dimitrakopoulos I, Zouloumis L, Lazaridis N, Karakasis D, Trigonidis G, Sichletidis L. Primary tuberculosis of the oral cavity. *Oral Surgery, Oral Medicine, Oral Pathology, Oral Radiology, and Endodontics.* ,72:712-715,(1991)
  40. American Thoracic Society, Centers for Diseases Control and Prevention, Infectious Disease Society of America. Treatment of tuberculosis. *Am J Resp Crit Care Med.* ,167:603-62,(2003)