# Tuberculosis - A common disease with uncommon oral features Report of two cases with a detailed review of literature

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Abstract— Tuberculosis is described as "king of diseases" in the Vedas and has been mentioned by Sushruta and Chakra in 600 B.C. Tuberculosis is a chronic infectious of worldwide prevalence, disease caused bv Mycobacterium Tuberculosis. The primary site of infection is usually the lungs, although it can affect any part of the body, including the oral cavity. Although oral manifestations of tuberculosis are rare, clinicians should be aware of their possible occurrence in their patient population in the form of ulcer, granulomas, involvement of the salivary glands and temporomandibular joints, osteomyelitis, desquamative gingivitis and tubeculous lymphadenitis. Such awareness can aid in diagnosing tuberculosis at an early stage, thereby preventing systemic complications and potential contaminations. This paper deals with two cases of tuberculosis diagnosed on the basis of oral manifestations, thereby emphasizing the importance of the dentist's role in diagnosis of a multisystemic disorder.

*Keywords*— Tuberculosis, Oral manifestations, chronic granulomatous disease

### I. INTRODUCTION

Tuberculosis is a chronic granulomatous multisystemic 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>. Tuberculosis is usually acquired by *Mycobacterium tuberculosis* and less frequently by the ingestion of unpasteurized cow's milk that is infected by *Mycobacterium bovis* or by

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other atypical Mycobacteria<sup>2</sup>. Depending upon the organ system involved, tuberculosis is classified clinically as Pulmonary or extra pulmonary. Pulmonary tuberculosis is the most common form of the disease. However, tuberculosis can also occur in the lymph nodes, meninges, kidneys, bone, skin and in the oral cavity<sup>3</sup>. Since the introduction of effective chemotherapy, tuberculous lesions of the oral cavity have become so infrequent that it is virtually a forgotten disease entity and pose a diagnostic problem. They account for less than one percent of cases of extra pulmonary tuberculosis, are usually associated with foci of disease elsewhere in the body and enlarged, palpable cervical lymph nodes are usually present<sup>4</sup>. Tuberculous oral lesions are relatively rare occurrence. Oral manifestations occur in approximately 3% of cases involving long standing pulmonary and / or systemic infection<sup>5</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 in most cases<sup>6</sup>. Primary lesions develop when tuberculosis bacilli are directly inoculated into the oral tissues of a person who has not acquired immunity to the disease and in fact, any area that is vulnerable to direct inoculation of bacilli from exogenous source can be a potential site. These frequently involve gingiva, tooth extraction sockets and buccal folds.7. In contrast, secondary oral tuberculosis is common (0.005% to 1.5% of cases) and is usually seen in older adults<sup>8</sup>. Secondary infection of oral tissues can result from either haematogenous or lymphatic spread or from auto inoculation by infected sputum and direct extensions from neighbouring structures. Intra oral sites frequently involved include the tongue, palate, lips, alveolar mucosa and jaw bones<sup>7</sup>. Tuberculosis in the oro-facial region may manifest in various forms: Tuberculous ulcer, Tuberculous gingivitis,

Tuberculous lymphadenitis, Tuberculoma, Tuberculous osteomyelitis, Tuberculous sialadenitis<sup>9,10</sup> and Tuberculous involvement of the Temporomandibular jaw. This paper deals with two cases of oro-facial tuberculosis and emphasizes the role of dentist in early diagnosis of tuberculosis based on the disease oral features, coupled with a detailed review of literature on the oro-facial manifestations of Tuberculosis.

### II. CASE REPORT 1

A 14 Year old female patient reported to oral medicine and radiology department, Faculty of Dentistry, Jamia Milia Islamia university, New Delhi with a complaint of chronic ulcerations and burning sensations in the mouth since past one year. History reveals that the patient developed a small ulcer in the gums in upper anterior region around a year back, which has progressively increased to attain the present size. The ulceration was accompanied by pain and burning sensations. There was an associated medical history of progressive weight loss (around 2.5 kgs) in past 3 months. The family history and personal history were non contributory. The patient had taken topical medications for the ulcer prescribed by the physicians, but there was no relief. On extra oral examination, the patient appeared malnourished. Single, non tender, moveable Submandibular lymph nodes, matted in consistency were palpable bilaterally. Intra oral examination revealed a single large, erythematous ulcerated area located in the labial gingiva involving the marginal, attached gingiva, interdental papillae and muco-gingival junction, extending from 13-23, ovoid in shape, 6x 2 cms, with undermined edges and irregular margins. Floor of the ulcer is erythematous with slight bluish discoloration of the mucosa in the periphery of the lesion (FIG. 1). Differential diagnosis included Pubertal gingivitis, Lichen planus, Pemphigus and tubercular gingivitis. Routine haematology showed raised WBC count and an increased Erythrocyte sedimentation rate (75 mm after Ist hour). Tuberculin test was positive, Saliva was positive for Acid Fast Bacilli and ELISA test was non reactive. Incisional biopsy was performed under local anaesthesia. Histopathological features revealed fibro collagenous tissue with multiple caseating granulomas composed of epitheloid cells, langerhans cells and foreign body type giant cells with lymphocytes, features consistent with the diagnosis of Tuberculosis (FIG. 2). Based on a history of progressive weight loss, chronic non healing ulcerated area extending upto the alveolar undermined edges, matted mucosa, with submandibular lymph nodes and characterstic histopathology showing caseating granulomas and epitheloid cells, the lesion was diagnosed as tuberculous gingivitis. The patient was treated with anti-tubercular therapy. Rifampicin 450 mg / tab before food x 3 months, Isoniazid 300 mg / tab before food x 3 months, Pyrazinamide 750 mg 2 tab before food in morning x 3 months and multivitamin capsules 2 capsules daily x 9 months. The patient reported after one month of therapy and showed complete resolution (FIG. 3). Regular follow up was done and there was no recurrence of the lesions.



FIG. 1 An ulcerated area on the labial gingiva extending upto the alveolar mucosa with respect to 13-23.



FIG. 2 Fibro collagenous stroma showing caseating granuloma with giant cells and lymphocytes.



FIG. 3 Healed lesions after anti-tubercular regimen.

### III. CASE REPORT 2

A 32 year old male patient reported to oral medicine and radiology department, Faculty of Dentistry, Jamia Milia Islamia university, New Delhi with a complaint of swellings below the chin region since three months. History revealed that the patient developed fever and cold three months back. The fever and cold subsided after medication. Subsequently the patient developed a small swelling below the lower jaw on the right side which was small in size and non-tender. A few days later he developed multiple swellings below the lower jaw on the right side and also on the left side. No pain was associated with the swelling. Since past two months, the swellings showed a progressive increase to attain the present size. No such similar swellings are seen anywhere else in the body. The medical history, personal history and family history were un remarkable, except a previous history of tuberculosis for which the patient had been treated 8 back. ON EXTRAORAL years EXAMINATION- Bimanual palpation of lymph nodes revealed enlarged lymph nodes in the right and left submandibular and submental region (FIG. 1 & 2). Multiple enlarged lymph nodes, four in number were seen in the right and left submandibular and submental region (FIG.3). The enlarged nodes measured about 1cm x 1cm in diameter. The nodes were non-tender on palpation, mildly firm and matted in consistency, no discharge was present from nodes on palpation. Inraoral examination was unremarkable. Deeply carious 36, 47 and horizontally impacted 48 were seen (FIG.4). As there are multiple, matted, enlarged , palpable submandibular and submental lymphnodes with a positive history of previously tuberculosis, this lymphnode treated enlargement is provisionally diagnosed as **TUBERCULOUS LYMPHADINITIS.** Investigative procedures were carried out-

### **1.ROUTINE HAEMATOLOGY-**

Haemoglobin content of 9.5 gm%, increased WBC count of  $7300 \text{ cells} / \text{mm}^3$ and an increased ESR of 65 mm in Ist hour.

### 2.CHEST RADIOGRAPH-

Radiograph of chest showed multiple radiopaque calcified spots in the lungs on both sides suggestive of previously healed lesions (FIG. 5).

3.PANORAMIC RADIOGRAPH- No bony changes were seen in the radiograph (FIG.6).

4.MONTOUX TEST- positive 30

mm in 48 hrs (FIG.7).

5.BIOSY- True cut biopsy specimen of submandibular lymph node was done. Photomicrograph shows granuloma like areas composed of central area of necrosis, epitheloid cells, macrophages, multinucleated giant cells (Langhans type) and few plasma cells in a lymphoid background. There is presence of moderate vascularity and areas of haemorrhage (FIG 8).

Correlating the history of previously treated tuberculosis, multiple enlarged, non tender, matted lymph nodes, supported by radiographic findings which showed patchy opacifications in the chest radiograph, positive Mantoux test and finally histopathological examination a diagnosis **"TUBERCULOUS** of LYMPHADENITIS" was made. The patient was treated with anti tubercular regimen and was periodically reviewed. There was a marked relief in the symptoms after treatment.





FIG. 2



FIG. 1, 2 & 3 Enlarged lymph nodes in right, left submandibular region and sub mental region.



FIG.4 Carious 36, 47 and horizontally impacted 48



FIG. 5 Calcified radio opaque in the lung parenchyma.



FIG. 6 Panoramic Radiograph shows no bony involvement.



FIG. 7 Induration on the forearm, showing Positive tuberculin test.



FIG. 8 Granulomatous lesion with epitheloid cells and lymphocytes.

### IV. DISCUSSION

Tuberculosis (TB) is a specific infectious granulomatous disease caused by *Mycobacterium tuberculosis*, a rod shaped, non spore forming, acid fast, aerobic bacilli. The disease also affects animals like cattle and this is known as *Bovine tuberculosis* sometimes transmitted to man<sup>9</sup>. Robert Koch first described M. tuberculosis, the causative agent of tuberculosis in 1882. M. tuberculosis is carried in airborne particles called droplet nuclei that are

generated when persons with infectious TB disease cough, sneeze, shout, sing or talk. Every year, approximately 2 million people in India develop tuberculosis, accounting for one fourth of the world's new tuberculosis cases<sup>11</sup>. Incidence of tuberculosis in india is 168 / 100, 000 population / year and prevalence is 312 / 100, 000 population / year<sup>12</sup>. In India tuberculosis is a major health hazard with a mortality rate of 30 deaths / 100,000 population / year, even after the National tuberculosis control programme [NTPC] has brought down the prevalence rate significantly<sup>12</sup>. TB has become the most common opportunistic infection in areas where the HIV infection is prevalent<sup>13</sup>.

Primary oral tuberculosis is rare, as an intact oral mucosa, cleansing action of saliva, salivary enzymes, tissue antibodies and oral saprophytes act as barriers to infection. Any breach in these defence mechanisms, such as abrasions, tears, chronic inflammation, poor oral hygiene, tooth eruption, extraction sockets, periodontal diseases, and carious teeth with pulp exposure may lead to infection by, tubercle bacilli<sup>5,14</sup>. Poor socio-economic conditions with inadequate nutrition and lack of hygiene are predisposing factors to infection<sup>14</sup>. Oral lesions of TB are non-specific in their clinical presentation and are often overlooked by the clinician<sup>7</sup>. Oral TB is common in 20-40 years of age group with a male-female ratio of 4:1<sup>15</sup>.

Various oro-facial manifestations seen in Tuberculosis are:

**1.TUBERCULOUS ULCER:** The common manifestation of oral tuberculosis is an ulcerative lesion of the mucosa. The lesion may be preceded by an opalescent vesicle or nodule which may breakdown as a result of caseation necrosis to form an ulcer. The typical tuberculous ulcer is an irregular lesion with ragged undermined edges, minimal induration and often with a yellowish granular base <sup>7</sup>. Although the tongue is the commonest site for oral tuberculous ulcers, they may also occur on the gingiva, floor of the mouth, palate, lips and buccal mucosa. Tuberculosis of the tongue has been presented as Macroglosia<sup>16</sup>. On the tongue, the common sites for a tuberculous ulcer are the lateral border, tip, anterior dorsum and the ventral surface <sup>1</sup> Tiny single or multiple nodules called ' sentinel tubercles' may also be seen surrounding the ulcer<sup>18</sup>. The tongue lesions are usually painful, grayishyellow, firm and well demarcated. The palatal lesions of tuberculosis may be seen as granulomas or ulcerations, and are more common in the hard palate than in the soft palate. Tuberculous ulcer affecting the unusual sites like the alveolus<sup>19</sup> and oro-pharynx, naso-pharynx and laryngo-pharynx<sup>20</sup> have also been mentioned in the literature.

**2.TUBERCULOUS GINGIVITIS:** Tubercular gingival lesions may present as exuberant and granulating or as mucosal erosions. Sometimes these

lesions may be seen simultaneously with marginal periodontitis<sup>21</sup>. Chronic desquamative gingivitis is associated with chronic infections affecting the gingiva, the most common being tuberculosis. Case reports of gingival tuberculosis appearing as diffuse gingival enlargement <sup>22</sup>, instead of the usual manifestation as an ulcer or localized granular mass, have also been been documented in the literature.

**3.TUBERCULOMA:** Tuberculosis may also involve the bone of the maxilla or mandible. One common mode of entry for the micro-organisms is into an area of peri-apical inflammation by way of the blood stream (Anachoresis ).These micro-organisms may enter the peri-apical tissues by direct immigration through the pulp chamber and root canal of a tooth with an open cavity. The lesion produced is essentially a tuberculous peri-apical granuloma or tuberculoma. These lesions were usually painless and sometimes involved a considerable amount of bone by relatively rapid extension<sup>9</sup>.

4.TUBERCULOUS LYMPHADENITIS: Tuberculosis of the lymphatic system is one of the most common of all extra-pulmonary tuberculosis, second only to tuberculous pleurisy.Its involvement of the cervical lymph nodes has been known for centuries as scrofula or the king's Evil<sup>23</sup>. Tuberculous lymphadenitis predominantly occurs in females and in the younger age groups <sup>23,24</sup>, in contrast to pulmonary tuberculosis which is more common in males and in older age group<sup>23</sup>. Tuberculous infection of cervical and submaxillary lymph nodes, or scrofula, a tuberculous lymphadenitis, may progress to the formation of an actual abscess or remain as a typical granulomatous lesion. In either case, swelling of the nodes is obvious clinically. They are tender or painful, often show inflammation of the overlying skin, and when an actual abscess exists, typically perforate and discharge pus9. Tuberculosis of the lymphatic system is largely confined to the cervical lymph nodes, mostly because the tonsils and adenoids provide an easy portal of entry for inhaled mycobacteria. FNAC is a well established diagnostic tool in the assessment of cervical masses. In developing countries like India, where tubercular infection is common and other granulomatous infections are rare, presence of granulomatous features on FNAC are highly suggestive of tuberculosis 25.

In the present case, the patient had a history of previously treated TB, chronic painless enlargement of lymph nodes, altered haematology, positive tuberculin test, presence of calcification in chest X Ray along with typical histopathology of a granulomatous disease. Also, there was no odontogenic source for the nodal enlargement.

**5.TUBERCULOUS OSTEOMYELITIS**: Diffuse involvement of the maxilla or mandible may also occur, usually by haematogenous spread of infection, but sometimes by direct extension or even after tooth

extraction<sup>9</sup>. The involvement of the mandible by TB infection is extremely rare as it contains less cancellous bone. But the mandibular involvement is more frequent than maxilla<sup>26</sup> and the alveolar and angle regions have greater affinity. Chapotel <sup>27</sup> described four clinical forms of tuberculosis of the mandible.

1. The superficial or alveolar form in which the alveolar process is involved either by direct extension of the tuberculous gingival tissues or by way of a deep carious tooth. The course is usually chronic, and necrosis of bone is progressive, with the formation of abscesses and fistulae.

2. The deep or central form, in which the lesion involves the angle of the mandible. It is found, according to Chapotel, almost exclusively in children during the period of eruption of the molar teeth.

3. The diffuse form, characterized by progressive extensive necrosis of mandible, which at times involves the tempromandibular articulation following a period of swelling and suppuration. Painless pathological fracture may occur. Severe general symptoms, accompanying a wide spread of tuberculosis affecting the liver, the lungs, the kidneys, and the meninges, are characteristic of the fatal aspect of this form.

4. The acute osteomyelitis form, in which, as the name implies, the sudden onset, the acute local and general manifestations, and the rapid course simulate those of an acute osteomyelitis of the mandible. This form is, however, very rarely observed<sup>27</sup>.

Tuberculosis of the jaw causes slow necrosis of the bone and may involve the entire mandible<sup>26</sup>. There is no characteristic radiographic appearance of TB of the jaws, or alveolar bone and most lesions are indistinguishable from those caused by pyogenic organisms. The destruction of the bone in radiographs appears as blurring of trabecular details with irregular areas of radiolucency.

There is an erosion of the cortex with little tendency to repair. Gradually the bone is replaced by soft tuberculous granulation tissue. Caseation appears at places followed by softening and liquefaction. A subperiosteal abscess forms presenting as a painless, soft swelling. This cold abscess may burst either intra or extraorally forming single or multiple sinuses. Pathological fracture of mandible and sequestration may also occur <sup>26</sup>.

**6.TUBERCULOUS SIALADENITIS:** Tuberculous parotitis was first described in 1981 by Kuruvilla. Tuberculous parotitis with pulmonary infection is seen more commonly, but primary type of isolated parotid tuberculosis is seen very rarely<sup>28</sup>. Tuberculous parotitis occurs in 2.5% - 10% of parotid gland lesion even in countries where the disease is endemic such as India <sup>29</sup>. It most commonly presents as a localized mass, resulting from infection of

intracapsular or pericapsular lymph nodes. It may also present as an acute sialadenitis with diffuse glandular enlargement. In this form the involvement is in the parenchyma of the salivary gland. It may also present as a periauricular fistula or as an abscess<sup>30</sup>. Another mode of involvement as stated by Carmody is from infected molar tooth. The most commonly implicated agent is mycobacterium bovis. Atypical mycobacterium rarely infects the parotid <sup>31</sup>. Primary tuberculosis of parotid gland presents in two forms: first acute inflammatory lesion mimicking sialadenitis which is more common, consisting of small and large abscesses, the parotid tissue is edematous, friable and indurated at places. second presentation is chronic tuberculous lesion which is circumscribed. The lesion presents as gradually increasing mass over months to years with no symptoms apart from swelling. On clinical examination it is impossible to distinguish them from parotid neoplasm <sup>31</sup>.

DIAGNOSTIC TOOL	METHOD /INFERENCE	ADVANTAGES	LIMITATIONS
1. TUBERCULIN SKIN TEST[TST] a.Heaf test	Multiple gun injects Multiple samples of testing serum over the flexor surface of the forearm in a circular pattern of six. Read at 3-7 days. Graded into 4 types.	Easier to interpret, with less inter observer variability. Less training required to administer and to read the test.	Multi puncture method 6 pricks-6 injections.
b. Mantoux test 2. RADIOGRAPHS	5 tuberculin units injected intradermally and read 48-72 hours later. Positive when induration of 5-15 mm is seen Areas of calcifications, cavities or radiolucency (darkened areas) Infiltrates or consolidation.	Used as screening tool. Helpful in diagnosis of active TB. More precise than radiographs Easy to perform.	Not recommended in; Infants less than 12 weeks Post montoux reaction ≥ 15 mm Previous TB disease. Exposure to X rays. Poor sensitivity. Cannot distinguish between active TB and healed TB in case of scar formation.
<ol> <li>STAINING         <ul> <li>Ziehl- Nelson staining</li> <li>Muramine fluorescence</li> </ul> </li> </ol>	Acid fast bacilli are seen as bright red rods against blue, green or yellow background. Visualises acid-fast bacilli as bright rods against dark background using fluorescent microscope.	Simple method, economical non invasive. Contrast bacilli can be readily seen under high dry objective. More sensitive Less tiring Quick results for large	Less than 10 <sup>4</sup> Mycobacteria / ml gives negative results. Similar appearance may be seen with saprophytic mycobacteria. Requires expensive equipment Used as a screening tool, not for final diagnosis
		number of slides.	

### **DIAGNOSTIC TECHNIQUES:**<sup>32</sup>

4.	Enzyme linked immunosorbent assays (ELISA)	Detects the presence of IgG and IgM antibodies when cultured with highly purified A 60 antigen extracted from mycobacteria.	More sensitive than staining Simple method Faster results	A60 antigen is common antigen to various species of mycobacteria leprae, tuberculosis and bovine.
In (I <sup>ı</sup> a.	terferon Release Assays GRAs) Quanti FERON TB Gold	Amount of Interferon gamma (IFN- Y) in response to contact with the TB antigens is measured.	Results within 24 hours Doesnot boost responses measured by subsequent tests, which can happen with tuberculin skin tests	Blood samples must be processed within 12 hours after collection while WBCs are still viable.
b.	T-SPOT. TB	Number of peripheral blood mononuclear cells used in the assay is quantified and enumerates individual T cells producing IFN-Y after antigenic stimulation, thus gives an overall measurement of antigen load on the immune system.	(TST) Not affected by prior BCG vaccination Faster results (within 24 hours) Allows the physicians to treat and control the disease much better	More data on the effectiveness of these tests in HIV-infected patients, young children, and other vulnerable groups are needed. To proceed within 6t hours of veni puncture.
5. a.Lo med	CULTURE wenstein – Jensen media ( LJ ia)	When grown on LJ media, M. tuberculosis appears as brown granular colonies ( buff, rough and tough)	Less expensive than BACTEC Less chances of contamination	Takes 4-6 weeks to get visual colonies on media. No differentiation between M. tuberculosis and other Mycobacterium species.
b.BACTEC		Detects the presence of oxygen in fluorescence by scanning it after every hour. Positive sample may contain $10^5 - 10^6$ CFU/ml.	Early detection Differentiates M. tuberculosis from other mycobacterium species. More sensitive than conventional LJ media	Expensive More medical technologist required. More risk of contamination

6. POLYMERIZED CHAIN REACTION (PCR)	Helps in detection of infectious agents and the discrimination of non- pathogenic from pathogenic strains by virtue of specific genes.	Very small size of DNA is amplified easily High sensitivity of PCR permits virus detection soon after infection and even before the onset of disease.	Localisation within tissues is not possible. Staging of mycobacterial disease is not possible.
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### TREATMENT:

### PREVENTION

Immunization with viable *Mycobacterium bovis* BCG is the most widely used preventive measure to control tuberculosis worldwide. Administered to newborns in a single dose, it prevents severe disease and reduces mortality among children from miliary and meningeal disease. However, BCG does not protect against pulmonary tuberculosis in children or adults<sup>33</sup> As mentioned earlier, optimal immune response

## to MBT infection appears to involve both CD4+ and CD8+ T-cells .

Standard antimycobacterial treatment regimens include antibiotics that target unique targets such as the synthesis of NAG-arabinogalactan and the early steps in mycolic acid synthesis.

Various Anti mycobacterial drugs used in treatment  $\operatorname{are:}^{\mathbf{34}}$ 

DRUGS	MECHANISM OF ACTION	ADVERSE DRUG EFFECTS
1.Ethambutol	Inhibits arabinosyl transferase	Optic neuritis
		loss of visual acuity
2.Pyrazinamide	Inhibits fatty acid synthetase	Morbiliform rash
		Arthralgias
		Hyperuricemia
3. Isoniazid	Inhibits fatty acid synthetase	Hepatitis
		Peripheral neuropathy
		Inhibits cytochrome P450 enzymes
		Hepatitis
4.Rifamycins:	Binds to RNA Polymerase and inhibits	Flu-like symptoms
Rifampin	transcription	Reddish urination
Rifabutin		GIT disturbances
Rifapentin		
SECOND LINE		
DRUGS:		Psychosis
1.Cycloserine	Inhibits monomer synthesis	Seizures
		Peripheral neuropathy
		Hepatitis
2.Ethionamine	Inhibits fatty acid synthetase	Hypothyroidism
		Ototoxicity
3.Aminoglycosides:	Binds to 30s ribosomal units and inhibit translation	Nephrotoxicity
Streptomycin		Neuromuscular blockade
Capreomycin		

### FIRST LINE DRUGS-

Kanamycin		
Amikacin		
	Inhibits topo-isomerase II (DNA Gyrase), thereby	Nausea
4.Fluoroquinolones:	releasing DNA with straggered double stranded	Abdominal rashes
Ciprofloxacin	breaks	Restlessness
Ofloxacin		Confusion
Gatifloxacin		
Levofloxacin		
Moxifloxacin		
	Competitive para-amino benzoic acid antagonist	GIT disturbances
5.Aminosalicylic acid	Isoniazid+Rifampin	
COMBINATION	Isoniazid+Rifampin+pyrazinamide	
DRUGS:		
Rifamate		
Rifater		
	•	•

**DOTS:** Daily observed treatment schedule is also being followed in TB cases.

### CONCLUSION

To conclude, mouth lesions of tuberculosis are rare. Nevertheless, the fact that tuberculosis may manifest in oral tissues, together with its non-specific clinical presentation and its infectious implications, demands an adequate acquaintance with its oral lesions. The dentist need to be aware that TB may occur in the oral cavity and should be included in the differential diagnosis of any ulcerated, indurated and non healing lesion of the oral cavity, especially in the lower socio-economic groups. Early diagnosis and treatment planning may help in preventing complications and death resulting due to this common infectious multi systemic disorder.

### REFERENCES

1.Memon GA, Khushk IA. Primary Tuberculosis of tongue. J Coll Physicians Surg Pak 2003; 13; 604-5

2.\_Mignogna MD, Muzio LL, Favia G, Ruoppo E, Sammartino G, Zarrelli C. Oral tuberculosis: a clinical evaluation of 42 cases. Oral disease 2000; 6: 25-30.

3.Topazian RG, Goldberg MH: Oral and maxillofacial Infections,  $2^{nd}$  edition, WB Saunders Co., pg 413, 1987

4. Eng H.L., Lu S.Y., Yang C.H., Chen W.J. Oral tuberculosis. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1996;81:415-20.

5. Hock-Liew E, Shin-Yu L, Chuang-HwaY, Wei-Jen C. oral tuberculosis. Oral surgery, Oral Medicine,

Oral Pathology, Oral Radiology and Endodontics 1996; 81:415-420

6.Nwoku LA, Kekere-Ekun TA, Sawyer DR, Olude OO. Primary tuberculous osteomyelitis of the mandible. J Maxillofacial Surg 1983; 11; 46-48

7. S.R.Prabhu and S.k.Sengupta. Bacterial infections due to mycobacteria A.Tuberculosis. Oral diseases in the tropics. Oxford university press, 1993: 195-202

8.Mani NJ. Tuberculosis initially diagnosed by asymptomatic oral lesions. Report of three cases. J Oral Med 1985; 40; 39-42

9.Shafer WG, Hine MK, Levy BM. A Textbook of Oral Pathology; 5<sup>th</sup> edition; WB Saunders Company, Philadelphia.

10. Martin S Greenberg, Micheal Glick. Burkitts Oral Medicine Diagnosis and Treatment 10<sup>th</sup> edition.

11. 1. Dye C, Scheele S, Dolin P et al. Global burden of tuberculosis: estimated incidence, prevalence, and mortality by country. JAMA 1999; 282: 677-- 86.

12. The world health report 2006. http:// www.int/GlobalAtlas/predefined Reports/ TB/ index.asp?strselect edCountry=ind

13.Miziara ID. Tuberculosis affecting the oral cavity in Brazilian HIV infected patients. Oral Surg Oral Med Oral Path Oral Rad Endo 2005; 100; 179-182

14 A.P.Bhatt and Amrita Jayakrishnan. Tuberculous osteomyelitis of the mandible: a case report. International journal of Paediatric Dentistry 2001; 11: 304-308..

15.Das P, Suri V, Arora R, Kulkarni K, Kumar K. Primary lingual tuberculosis mimicking malignancy: a report of two cases and review of literature. Th e Internet journal of Pathology 2007;6(2). available at: htt p://www.ispub.com/ostia/index. php?xmlfi lepath=journals/ijpa/vol6no2/lingual.xml

16. Ramesh V. Tuberculoma of the tongue presenting as macroglossia. *Cutis* 1997; **60**: 201-202.

17.Farber JE, Friedland E & Jacobs WF: Tuberculosis of the tongue, Am Rev Tuber, 766: 1940-42

18. Thilander H, Wennstrom A: Tuberculosis of the mouth and the surrounding tissues, Oral Surg Oral Med Oral Path 1956; 9; 858-70

19.Bipin Kumar : Tuberculosis of the oral cavity affecting Alveolus: A case report in Dentistry: 2011

20. Medhuri, Chander Mohan, Sharma ML. Posterior oropharyngeal wall tuberculosis. Indian J of Otolaryngol and Head and Neck Surg. 2002;54: 152-153.

21.Bjorlin G : Oral Tuberculosis: Odontologisk Revy, 18: 395-399

22. Bangalore Varadhan Karthikeyan, Avani Raju Pradeep, C.G. Dileep Sharma. Primary tuberculous Gingival Enlargement: A Rare Entity. Journal of Canadian Dental Association 2006; 72(7): 645-648.

23.Tan KK. Tuberculosis lymphadenitis in Singapore. Sing Med J 1988; 29; 441-416

24.Dandapat MC, Mishra BM, Dash SP, Kar PK. Peripheral lymph node Tuberculosis : A Review of 80 cases. Br J Surg 1990; 77; 911-912

25.Van de Schoot L, Aronson DC, Behrendt H, Bras J. The role of FNAC in children with persistent or suspicious lymphadenopathy. J Pediat Surg 2001; 36; 7-11

26. K.B.Gupta, M.Manchanda, S.P.S.Yadav and A.Mittal. Tubercular Osteomyelitis of Mandible. Indian journal of tuberculosis 2005; 52:147-150.

27. Chapotel S. Tuberculose mandibularie. Rev Odent 1930; 51:444-445

28.S elcuk A, Oruk V, Dere H, Boztepe F, Seckin S: Tuberculous parotitis: Can be left silent for a long time. KBB-Forum, 2006; 5(3):130-132.

29. Birkent H, Karahatay S, Akcam T, Durmaz A, Ongoru O: Primary parotid Tuberculosis mimicking parotid Neoplasm: A case report. *Journal of Medical Case Reports*, 2008;2:62-63.

30. Suleiman AM: Tuberculous parotitis: report of 3 cases. *British Journal of Oral and Maxillofacial Surgery*, 2001, 39(4):320-323.

31. Bakhshi GD, Shaikh AS, Borisa AD, Jamadar NM, Singh AG, Vora SH: Primary Tuberculosis of Paritid Gland Mimicking Parotid Tumour. *Bombay Hospital Journal*, (Special Issue), 2009;14-16.

32.Kanwar Deep Singh Nanda, Anurag Mehta, Mohita Marwaha, Manpreet Kalra, Jasmine Nanda: A Disguised Tuberculosis of the oral Buccal mucosa. Journal of Clinical and Diagnostic Research 2011: Apr, vol -5 (2); 357-360

33.Rodregues LC, Diwan VK, Wheeler JG. Protective effect of BCG against tuberculous meningitis and miliary tuberculosis: a meta-analysis. Int J Epidemiology 1993;22:1154-8. 34. Abramowicz M. (ed). Drugs for tuberculosis. Treat Guidel Med Lett. 2004 Dec;2(28):83-8.

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