**Orofacial tuberculosis: Clinical manifestations, diagnosis and management**

Ramta Bansal¹, Aditya Jain², Sunandan Mittal³

¹Department of Conservative Dentistry and Endodontics, Institute of Dental Sciences, Sehora, Jammu, Jammu and Kashmir, ²Department of Physiology, Government Medical College, Patiala, ³Dashmesh Institute of Research and Health sciences, Faridkot, Punjab, India

**Abstract**

Orofacial tuberculosis (TB) is an uncommon form of extrapulmonary TB and is nonspecific in its clinical presentation. It can be misdiagnosed especially when oral lesions are present before systemic symptoms become apparent. Doctors especially attending dentist who generally is the first among clinicians to come across such pathological entity should be aware of the orofacial lesions of TB and consider them in the differential diagnosis of suspicious oral lesions to ensure early diagnosis of TB and its treatment. In this review, we have discussed in detail the clinical presentation of various forms of orofacial TB, diagnosis, and management of patients. Also, an update is provided about recent anti-TB drug development.

**Keywords:** Dentist, diagnosis, mycobacterium, oral tuberculosis, tuberculosis

**Introduction**

Tuberculosis (TB) is second only to HIV/AIDS as the greatest killer worldwide especially in developing countries where its prevalence is very high. The probable reasons for this are poverty, overcrowded shelters, lack in public health efforts to control TB, HIV infection epidemic and development of multidrug-resistant bacteria. The BRICS group (Brazil, Russian Federation, India, China and South Africa) accounts for 46% of all incident cases of TB and 40% of all TB-related mortality. India accounts for nearly one third of global burden of the disease. Caused by *Mycobacterium tuberculosis* (MBT), the disease most commonly affects lungs but in 10‑15% cases other parts of the body can also get affected, i.e. extrapulmonary TB. Orofacial TB is an uncommon form of extrapulmonary TB and is nonspecific in its clinical presentation. It can be misdiagnosed especially when oral lesions are present before systemic symptoms become apparent. Doctors especially attending dentist who generally is the first among clinicians to come across such pathological entity should be aware of the orofacial lesions of TB and consider them in the differential diagnosis of suspicious oral lesions to ensure early diagnosis of TB and its treatment. In this review, we have discussed in detail the clinical presentation of various forms of orofacial TB, diagnosis, and management of patients. Also, an update is provided about recent anti-TB drug development.

**Discussion**

Orofacial TB is a rare manifestation of extrapulmonary TB, occurring in approximately 0.1–5% of all TB infections. It can be primary or secondary. Primary form is rare and more commonly found in children and adolescents. In contrast, the secondary form is more common (0.005% to 1.5% of cases) and is usually seen in middle-aged and elderly patients. Orofacial TB can involve any site of the oral cavity and associated structures such as tongue, palate, lips, oral mucosa, jaw bones, sinuses, temporomandibular joint (TMJ), etc. Recently, Andrade *et al.* proposed a classification of orofacial TB [Table 1] based on the site involved.

The clinical presentation of various forms of Orofacial TB is discussed below in detail

**Tuberculous ulcer**

May present as single or multiple, superficial, or deep, painful or painless ulcers with an irregular border which tends to increase slowly in size. They usually develop as a small tubercle which then softens to form a shallow, ovoid ulcer with undermined margins and is lined with pale granulation tissue. Tiny single or multiple nodules called “sentinel tubercles” may be seen surrounding the
Table 1: Andrade’s classification of orofacial tuberculosis

| Type | Description |
|------|-------------|
| I    | Lumpy jaw; mandible or maxilla is involved and extraoral swelling is present without intraoral or extraoral draining sinuses |
| II   | Nonhealing extraction sockets with/without intraoral or extraoral draining sinus/sinuses |
| III  | Intraoral or extraoral draining sinus/sinuses in the orofacial region and an osteomyelitic bony lesion |
| IV   | TB lymphadenitis of the head face neck region without any features of type I, II, III, or V |
| V    | Lesion of other sites in and around the oral cavity, e.g., maxillary antrum, salivary glands, gingiva, orofacial muscles, tongue, etc. |

Tuberculous osteomyelitis of maxilla and mandible

Tuberculous osteomyelitis is rare and constitutes less than 2% of skeletal TB. Jaw involvement is even rarer.[25] The mandibular involvement is more frequent than maxilla and alveolar and angle regions have greater affinity.[26] Tuberculous osteomyelitis commonly affects the adults; however, in some cases children are also affected.[21] The spread of infection may be by direct transfer from infected sputum,[22] through an extraction socket or mucosal opening associated with an erupting tooth or by regional extensions of soft tissue lesions to underlying bone or by hematogenous spread.[26] Chapote[27] described four clinical forms of TB of the mandible. The first is the superficial or alveolar form that involves the alveolar process, second is the deep or central form in which the angle of the mandible is involved, third is the diffuse form characterized by progressive extensive necrosis of mandible that might involve the TMJ and the fourth one is acute osteomyelitis form.

TB of the jaw causes slow necrosis of the bone and formation of a sub-periosteal abscess (lumpy jaw) appearing as a painless, soft swelling. This sub-periosteal abscess may burst resulting in single or multiple sinuses intraorally or extraorally. Pathological fracture of mandible or sequestration may also occur.[29] Diagnosis of tuberculous osteomyelitis is a significant challenge as the smears for acid-fast bacilli usually do not yield positive results.[23] Polymerase chain reaction or nucleic amplification assays may be helpful in obtaining an earlier diagnosis; however, a negative result does not rule out TB.[24] In the early stages, when plain radiographs appear normal, MRI or CT may help to localize lesions. The radiographic picture of tuberculous osteomyelitis usually presents as a blurring of bone details leading to diffuse radiolucent picture and cortical plate erosion. It can also present as mixed radiopaque–radiolucent appearance or “worm-eaten” appearance of bony lesions with fistulae formation through which small sequestra are exuded. The findings are similar to that of the destructive disease if the periodontal tissues get involved.[29] More advanced lesions may appear as osteoporosis, bone lysis, sclerosis and periostitis that mimic chronic pyogenic osteomyelitis and it is often difficult to differentiate the two conditions. Joint involvement may be present but unlike pyogenic osteomyelitis, articular margins and cartilage space are spared. A solitary lytic lesion can also appear sometimes which can mimic neoplasia.[24] A biopsy is mandatory for the diagnosis, and anti-TB drugs along with surgical debridement if required are the main mode of treatment.

Tuberculous involvement of extraction sockets of teeth

Healing of the tooth extraction sockets is delayed and the socket gets filled with “tuberculous granulation tissue” consisting of many pink to red elevations.[17] Outbreak of TB following dental extractions at two community dental clinics has been reported where 15 patients developed primary TB lesions, out of them 8 patients had primary tooth socket involvement. The dentist who performed the extractions at both the clinics was found to have active bilateral pulmonary TB.[18]

Tuberculous gingivitis

Tuberculous gingivitis may appear as nodular or papillary proliferation of gingival tissues which is diffuse and hyperaemic.[13] There may be absence of any clinical attachment loss, alveolar bone loss or significant cervical lymphadenopathy. Such diffuse gingival enlargements fail to respond to initial usual therapy consisting of supragingival debridement.[14] Sometimes tuberculous gingivitis can be seen simultaneously with marginal periodontitis and enlarged cervical lymph nodes[15] or may present as periodontal loss of tooth support leading to loose teeth and gingival enlargement.[14] A biopsy of the lesion is mandatory for arriving at the diagnosis of TB.

Tuberculous dental periapical granuloma

Tuberculous involvement of the periapical tissue has often been reported. Three routes can be perceived to be entry portals for the tubercle bacilli to become implanted in the periapical tissues. The first is the invasion of the dental pulp through a deep carious lesion by the acid-fast bacilli in the saliva. Should the pulp degenerate and breakdown, a tuberculous periapical infection might result. A second route is the hematogenous and third is the deep periodontal pocket. Patients not responding to usual periodontal treatments may be harboring tuberculous infection of the paradental tissues even though its presence is not evident.[14] The lesions are usually painless and sometimes involve a considerable amount of bone by relatively rapid extension.[19]

Tuberculous of maxillary sinus

TB of maxillary sinus is usually a disease of adults and remains an under-diagnosed entity. It is usually secondary to pulmonary or extrapulmonary TB resulting from the bloodstream or by direct extension.[25] Primary sinonasal TB is rare probably due to bactericidal secretion, ciliary movement and mechanical filtering by vibrissae of nose.[26] Most commonly, it presents as nasal discharge, stuffiness of nose, crust formation and sometimes with epistaxis.[29] It can also present as fluctuant swelling, i.e. Pott’s puffy tumor and may resemble a malignant lesion. Three types
of sinonasal TB have been described: (i) Mucosal involvement leading to formation of polyps with minimal pus discharge, this type is more common; (ii) bony involvement and fistula formation with abundant discharge of acid-fast bacilli (AFB); this type can lead to midfacial defect; (iii) hyperplastic type has granuloma formation and mimics a malignancy. If not treated early, it can lead to complications like brain abscess and deterioration of vision. Antral lavage examination for AFB and culture for Mycobacterium tuberculosis can facilitate early diagnosis. The diagnosis of TB sinusitis is usually based on the absence of response to usual antibiotics, the presence of a caseous granulomatous inflammatory lesion and by bacteriological culture or polymerase chain reaction assay. Sinus surgery may be required for sinus drainage and specimen collection. CT or magnetic resonance imaging can be helpful to figure out the extent of disease. Appearance of calcification in sinuses on CT scans can be indicative of sinonasal TB, and imaging findings are mostly nonspecific. Anti-TB medication and/or surgical debridement is the mainstay of the treatment.

**Tuberculosis of temporomandibular joint**

TB of the TMJ is rare; only a few cases have been reported. The low frequency reported in the literature might be due to missed diagnosis than to its real prevalence. The clinical appearance of TB infection of the TMJ is un specific and can resemble osteomyelitis, arthritis, cancer or any kind of chronic joint diseases. Thus, TMJ TB should be considered in the differential diagnosis when the patients present with pain and stiffness of the joint or with chronic joint diseases. The onset of symptoms is insidious: Nocturnal muscular spasm, leading to soft and elastic joint tumefaction, characteristically without erythema, with edema and leading early to severe and localized periarticular muscle atrophy. Subsequently, necrotic destructive phenomena occur, which de-structure the joint. In the end stage of disease, fibrosis or bony ankylosis can develop. Diagnosis is usually made by culture, staining and imaging. MRI can detect intraarticular pus in the early stage of the disease when conventional radiological techniques are insensitive until bony changes develop. MRI findings also show bone marrow edema and extra-articular cystic collections in osteoarticular TB. In the advance stages, radiographic findings may show erosion of the condyle and glenoid fossa. Exploration and biopsy are necessary to establish the definitive diagnosis. The treatment of the TMJ TB consists of conventional drug therapy. Surgical excision and decortication is done when intense pharmacotherapy fails.

**Tuberculous sialadenitis**

TB of the salivary gland is a rare condition even in countries like India, where the disease is rampant. The most common salivary glands involved in primary TB are parotid glands, whereas in systemic TB submandibular glands are most commonly involved. There are two clinical forms of tuberculous parotitis. The first is the localized form which is common and involves intraglandular/periglandular lymph nodes, while the other diffuse parenchymatous form is very rare and is considered to be an acute pathology involving whole of the gland. It may be secondary to the nodal infection. Initially, mycobacterium manifests in the nodes of the preauricular area. It presents as slow growing, non-tender localized swelling in front and below the ear. The pain, abscess, fistula, and facial nerve involvement are the late features. The constitutional symptoms of TB like chronic cough, fever, weight loss may be present but are rare. The diagnosis of tubercular parotitis is very difficult because of the absence of symptoms and may often be misdiagnosed as a benign parotid tumor. A detailed history, examination and FNAC have been advocated for the diagnosis of tubercular parotitis. The Zielh-Nielsen staining and culture for the mycobacterium is usually found negative. If the patient is not already known to have TB or another mycobacteriosis, the diagnosis is made by microscopy after excision of the gland. Polymerase chain reaction (PCR) should always be considered before surgical intervention to enable differential diagnosis of a salivary gland tumor.

**Tuberculous lymphadenitis**

Tuberculous lymphadenitis in the cervical region, also known as scrofula, is the most common site of extra pulmonary TB and accounts up to 5% of the cervical lymphadenopathy. It often affects children and young adults in age range of 30–40 years and shows female predilection. It can present as a single or bilateral neck mass, affecting deep lymph nodes and may be associated with supraclavicular and axillary node involvement. Patients present with slowly enlarging asymptomatic lymph nodes in the neck which is persistent. The mass is referred to as a cold abscess, due to lack of local color or warmth and the overlying skin presents a violaceous color. Other symptoms of disease, such as fever, chills, malaise and weight loss, are present in about 43% of the patients. As the lesion progresses, skin becomes adhered to the mass and may rupture, forming a sinus and an open wound. Fine-needle aspiration cytology (FNAC) and direct microscopic screening for acid-fast bacilli (AFB) are recommended for the routine diagnosis of tuberculous lymphadenopathy along with culture that remains the gold standard for diagnosis. Diagnosis often requires biopsy. Therapy includes various types of anti-TB chemotherapy, surgical excision, or a combination of surgery and chemotherapy.

**Lupus vulgaris**

Lupus vulgaris is the most common form of cutaneous TB found in individuals with moderate immunity and high degree of tuberculin sensitivity. It is caused by M. tuberculosis and can involve the skin by hematogenous or lymphatic route. Eighty percent of the lesions are on the head and neck and most often on the face around the nose, eyelids, lips, cheeks, ears. Females are affected two to three times more often than males. Lupus vulgaris skin lesions are of five types – (a) plaque, (b) ulcerative or mutilating, (c) vegetating, (d) tumor-like and (e) papulonodular. The plaque form is the most common form of LV, accounting for 32% of cases, whereas ulcerative form is the most destructive and deforming of all lesions. A single or several, unilateral, reddish-brown papules first
appearing on face, neck or arms and then coalescing into erythematous plaques. The surface of the papules exfoliates and the centers scar. Papules recur on the scarred areas, gradually and repeatedly enlarging and coalescing. This leads to the formation of large, firm, elevated plaques. At the periphery are small reddish-yellow or brown nodules. The characteristic lesion is a reddish-brown plaque, composed of nodules which show an “apple-jelly” color when pressed with a glass spatula (diascopy). The lesions may ultimately develop into disfiguring skin ulcers if left untreated. In long-standing scarred lesion, squamous cell carcinoma can develop. Lupus vulgaris is diagnosed by the clinical features, pathology, and strong positive in tuberculin skin test. Identification of M. tuberculosis is made by PCR or culture. The disease treatment consists of systemic anti-TB drugs.

Apart from the above-mentioned oral manifestations, expectoration of the infected sputum may cause tuberculous tracheitis and laryngitis resulting in hoarseness, coughing, and pain, and tuberculous ulcers on the tonsils resulting in dysphagia.

Summary of the orofacial TB manifestations is given in Table 2.

Diagnosis of tuberculosis

Step by step approach is shown in Table 3.

Treatment of orofacial TB

The treatment of orofacial TB is the same as standard antimycobacterial treatment regimens used for treating pulmonary TB. The five basic or “first line” antibiotics that form the core of TB treatment are: Isoniazid (H), rifampicin (R), pyrazinamide (Z), ethambutol (E) and streptomycin. Second line or reserve drugs are used when first-line drugs are not effective and consist of Group I first-line oral agents like pyrazinamide, ethambutol, rifabutin; Group II injectable agents like kanamycin, amikacin, capreomycin, streptomycin; Group III fluoroquinolones like levofloxacin, moxifloxacin, ofloxacin; Group IV oral bacteriostatic second-line agents like para-aminosalicylic acid, cycloserine, terizidone, ethionamide, prothionamide; and Group V drugs with an unclear role in the treatment of drug-resistant TB like clofazimine, linezolid, thiactetazone, amoxicillin/clavulanate, high-dose isoniazid, imipenem/cilastatin, clarithromycin. WHO recommended treatment regimens for TB have several inherent problems, making new anti-TB drugs and treatment regimens a clinical and public health priority. New anti-TB drugs are being researched that should be affordable, have shorter treatment regimens, be more efficacious than existing drugs, should successfully treat MDR-TB, XDR-TB as well as latent TB and should also be compatible with antiretroviral drugs. After decades of quiescence in the development of anti-TB medications, two new drugs have been approved. The first novel drug bedaquiline for treatment of MDR-TB was approved in December 2012; it is also under clinical evaluation for the treatment of drug-susceptible TB, with drug regimens not containing rifamycins. The drug is still in Phase III trials and WHO urges caution in its use and strict adherence to conditions listed in the WHO interim policy guidance issued in June 2013. Delamanid is the second drug that has been was granted conditional approval by the European Medicine Agency in April 2014 for the treatment of drug resistant TB. Several other novel compounds are being evaluated and are in various phases of preclinical or clinical trials. Some of them are tabulated in Table 4.

| Table 2: Various manifestations of orofacial tuberculosis |
|---------------------------------------------------------|
| **Condition**                                           | **Salient Features**                                      |
| Tuberculous ulcer                                      | Shallow, ovoid ulcer with undermined margins and is lined with pale granulation tissue |
| Tuberculous gingivitis                                 | Nodular or papillary proliferation of gingival tissues which is diffuse and hyperemic |
| Tuberculous dental periapical granuloma                | Painless swelling and sometimes involve a considerable amount of bone by relatively rapid extension |
| Tuberculous involvement of extraction sockets of teeth  | Delayed healing, the socket gets filled with “tuberculous granulation tissue” consisting of pink to red elevations |
| Tuberculous osteomyelitis of jaws                       | Lumpy jaw, intraoral or extraoral single or multiple sinuses may be present. Pathological fracture of mandible or sequestration may occur |
| Tuberculosis of maxillary sinus                         | Nasal discharge, stuffiness of nose, crust formation and sometimes with epistaxis |
| Tuberculosis of temporomandibular joint                 | Nocturnal muscular spasm, soft and elastic joint tumefaction, without erythema, with edema and severe and localized periarticular muscle atrophy |
| Tuberculous sialadenitis                               | Slow growing, non-tender localized swelling is commonly present. Pain, abscess, fistula, and nerve involvement are the late features |
| Tuberculous lymphadenitis (Scrofula)                    | Slowly enlarging cold abscess in the neck may rupture forming a sinus and an open wound |
| Lupus vulgaris                                          | Single or several, unilateral, reddish-brown papules coalescing into erythematous plaques. Characteristic lesion is apple-jelly nodules |

Conclusion

The orofacial TB can occur anywhere in the oral cavity and its associated structure and presents a non-specific clinical picture. Doctors should be aware of the orofacial lesions of TB and consider them in the differential diagnosis to ensure early diagnosis and management of TB as any delay may lead to serious consequences. Also, the general public should be educated about TB and its extra pulmonary manifestations and made aware that the disease is completely curable if managed properly. This will reduce the social stigma attached with the disease which leads...
Table 3: Step by step approach to diagnose orofacial TB lesion

Medical history
Symptoms like cough of 3 or more weeks, chest pain, hemoptysis, low grade fever, night sweats, chills, loss of appetite and weight, mucoid sputum that changes to purulent are suggestive of TB. Prior TB exposure or TB treatment or disease such as HIV infection that increase risk for TB should be enquired

Physical examination
Patient’s general health is assessed and any abnormal local finding is observed

Screening tests: To determine if a person has been infected with TB bacteria: Latent TB infection (LTBI) or has progressed to active TB disease

Tuberculin skin test or Mantoux test
Is performed by injecting a small amount of PPD tuberculin into the skin of forearm. After 48 to 72 hours site of injection is checked for raised, hard area or swelling if present

Tuberculosis radiology
Pulmonary TB
Chest radiograph: In adolescents and adults upper lobe cavity consolidation with occasional mediastinal or hilar lymph node enlargement or pleural effusion. In infancy and childhood, intra-thoracic lymph node are enlarged, pleural effusion is seen, and lung lesions are present in lower lobes

Smear from 3 sputum samples obtained for acid-fast bacilli (in the absence of sputum sample, alternative sample sources are gastric washings, laryngeal swab, bronchoalveolar lavage, bronchial washings, fine-needle aspiration and tissue biopsy

Nucleic acid amplification testing (NAAT): Is a molecular technique used to directly detect the genetic material of the infecting organism or virus. NAAT may speed the diagnosis in smear-negative cases and may be helpful to differentiate non-tuberculous mycobacteria. NAAT may utilize polymerase chain reaction (PCR) technique or transcription-mediated amplification (TMA) or other forms of nucleic acid amplification methods to detect mycobacterial nucleic acid. The two most commonly commercially available tests are the amplified Mycobacterium tuberculosis direct test (AMTD, GenProbe) and ampiclon (Roche Diagnostics). The AMTD test appears to perform better than other currently available commercial tests.

Sputum culture:
It confirms diagnosis of TB, is more sensitive than smear staining, and evaluates drug sensitivity. Traditionally, cultures have used the Löwenstein-Jensen (LJ), Kirchner, or Middlebrook media (7H9, 7H10, and 7H11). New automated systems that are faster include the MB/BacT, BACTEC 9000, VersaTREK, mycobacterial growth indicator tube (MGIT) and the microscopic observation drug susceptibility assay culture

Blood Count
Lymphocytosis and raised erythrocyte sedimentation rate (ESR) is usually present

HIV Testing
TB patients should be tested for HIV within 2 months of diagnosis

Table 4: Recent anti-tuberculosis drugs in clinical trials

| Class         | Mechanism of Action                                                                 | Name of drug                           | Clinical trial phase |
|---------------|--------------------------------------------------------------------------------------|----------------------------------------|---------------------|
| Diaryquinoline| Inhibition of ATP synthesis and disruption of membrane potential                      | Bedaquiline                            | III                 |
| Nitroimidazoles| Inhibition of synthesis of mycolic acids; poisons the bacterial cell by releasing nitric oxide when metabolized | Delamanid                              | III                 |
| Fluoroquinolone| Inhibition of DNA biosynthesis                                                      | Gatifloxacin, Moxifloxacin             | III                 |
| Ethylenediamine| Inhibition of cell wall biosynthesis                                                 | SQ109                                  | II                  |
| Oxazolidinone  | Inhibition of protein biosynthesis                                                   | AZD5847, Linezolid, Sutezolid, tedizolid (for MRSA) | II                  |
| Rifamycin      | Blocks messenger RNA synthesis (transcription) by inhibiting the bacterial DNA-dependent RNA polymerase | Rifapentine                            | II/III              |
to delay in diagnosis and non compliance with the treatment. The main priority of the government should be prevention of development of drug resistance in TB and encourage research of new drugs to treat multidrug-resistant TB.

References

1. World Health Organization (WHO). Global tuberculosis report 2014. Geneva: WHO; 2014. Available from: http://www.who.int/tb/publications/global_report/gtbr14_main_text.pdf.

2. Mahajan S, Srikanth N, George T. Atypical Presentation of Oral Tuberculosis Ulcer. N Y State Dent J 2007;73:48–50.

3. Creswell J, Sahu S, Sachdeva KS, Ditti L, Barreira D, Mariandyshev A, et al. Tuberculosis in BRICS: Challenges and opportunities for leadership within the post-2015 agenda. Bull World Health Organ 2014;92:459–60.

4. Javali MA, Patil V, Ayesha H. Periodontal disease as the initial manifestation of abdominal tuberculosis. Dent Res J 2011;9:634–7.

5. Iype EM, Ramdas K, Pandey M, Jayasree K, Thomas G, Sebastian P, et al. Primary tuberculosis of the tongue: Report of three cases. J Oral Maxillofac Surg 2001;59:402–3.

6. Jain P, Jain I. Oral Manifestations of Tuberculosis: Step towards Early Diagnosis. J Clin Diagn Res 2014;8:ZE18–21.

7. Nagalakshmi V, Nagabhushana D, Aara A. Primary tuberculous lymphadenitis: A case report. Clin Cosmet Investig Dent 2010;2:21–5.

8. Kapoor S, Gandhi S, Gandhi N, Singh I. Oral manifestations of tuberculosis. CRHSMED J Health Res 2014;1:11–4.

9. Andrade NN, Mhatre TS. Orofacial Tuberculosis - A 16-year experience with 46 cases. J Oral Maxillofac Surg 2012;70:e12–e22.

10. Hasan S, Khan MA. Tuberculosis - A common disease with uncommon oral features: Report of two cases with a detailed review of literature. Proceedings of the World Medical Conference page 156–166. ISBN: 978-1-61804-036-7.

11. Kamala R, Sinha A, Srivastava A, Srivastava S. Primary tuberculosis of the oral cavity. Indian J Dent Res 2011;22:835–8.

12. Von Arx DP, Husain A. Oral tuberculosis. Br Dent J 2001;190:420–2.

13. Jain S, Vipin B, Khurana P. Gingival tuberculosis. J Indian Soc Periodontol 2009;13:106–8.

14. Gill JS, Sandhu S, Gill S. Primary tuberculosis masquerading as gingival enlargement. Br Dent J 2010;208:343–5.

15. Ebenezer J. Gingival tuberculosis. JIDA Tamil Nadu 2009;1:66–7.

16. Brodsky RH, Klattel JS. The tuberculous dental periapical granuloma. Am J Orthod Oral Surg 1943;29:B498–502.

17. Rodrigues G, Carnelio S. Oral tuberculosis: A Review. In: Focus on Tuberculosis Research. In: Smiteh Lt, editor. New York; Nova Science Publishers: 2005. p. 172.

18. Smith WH, Davies D, Mason KD, Onions JP. Intraoral and Pulmonary Tuberculosis following dental treatment. Lancet 1982;319:842–4.

19. Gadgil RM, Bhosreddy AR, Upadhyay BR. Osteomyelitis of the mandible leading to pathological fracture in a tuberculosis patient: A case report and review of literature. Ann Trop Med Public Health 2012;5:383–6.

20. Gupta KB, Manchanda M, Yadav SP, Mittal A. Tubercular osteomyelitis of mandible. Indian J Tuberc 2005;52:147–50.

21. Bhatt AP, Jayakrishnan A. Tuberculous osteomyelitis of the mandible: A case report. Int J Paediatr Dent 2001;11:304–8.

22. Chapotel S. Tuberculose mandibulaire. Rev Odont 1930;51:444–5.

23. Agarwal S, Caplivski D, Bottone EJ. Disseminated tuberculosis presenting with finger swelling in a Patient with tuberculous osteomyelitis: A case report. Ann Clin Microbiol Antimicrob 2005;4:18.

24. Manneppalli S, Mitchell-Samon L, Guzman N, Relan M, McCarter YS. Mycobacterium tuberculosis osteomyelitis in a patient with human immunodeficiency virus/acquired immunodeficiency syndrome (HIV/AIDS): A case report. Cases J 2010;3:67.

25. Sansare K, Gupta A, Khanna V, Karjodkar F. Oral tuberculosis: Unusual radiographic findings. Dentomaxillofac Radiol 2011;40:251–6.

26. dbeleahad IF, Bianchi S, Martinoli C, Klein M, Hermann G. Atypical extra spinal musculoskeletal tuberculosis in immunocompetent patients, a review. Part 1: Atypical osteoarticular tuberculosis and tuberculous osteomyelitis. Can Assoc Radiol J 2006;57:86–94.

27. Kumar S, Singh HP, Agarwal SP. Tuberculosis of the Maxilla and Reconstruction of Midfacial Defect using Temporals Muscle Flap. Int J Head Neck Surg 2012;3:49–52.

28. Goguen LA, Karmody CS. Nasal tuberculosis. Otolaryngol Head Neck Surg 1995;113:131–5.

29. Kant S, Srivastava R, Verma AK, Singh HP, Singh S, Ranganath TG et al. Maxillary sinus tuberculosis: Various presentations. Indian J Chest Dis Allied Sci 2013;55:175–7.

30. Jain MR, Chundawat HS, Batra V. Tuberculosis of the maxillary antrum and of the orbit. Indian J Ophthalmol 1979;27:18–20.

31. Kakeri AR, Patel AF, Walikar BN, Watve MV, Rashinkar SM. A case of Tuberculosis of maxillary sinus. AI Ameen J Med Sci 2008;1:139–41.

32. Sabayev V, Yein T, Khan R. A rare case of tuberculosis sinusitis in an immunocompetent patient. Chest 2004;126:973S–8S.

33. Moon WK, Han MH, Chang KH, Im JG, Kim HJ, Sung KJ, et al. CT and MR imaging of head and neck tuberculosis. Radiographics 1997;17:391–402.

34. Hellbing CA, Lieger O, Smolka W, Lizuka T, Kuttenberger J. Primary tuberculosis of the TMJ: Presentation of a case and literature review. Int J Oral Maxillofac Surg 2010;39:834–8.

35. Gandhi S, Ranganathan LK, Bither S, Koshy G. Tuberculosis of temporomandibular joint: A case report. J Oral Maxillofac Surg 2011;69:e128–30.

36. Ranganathan LK, Mathew GC, Gandhi S, Manohar M. Tuberculosis of Temporomandibular Joint Presenting as Swelling in the Preauricular region. J Oral Maxillofac Surg 2012;70:e28–31.

37. Kreiner M. Tuberculosis of the temporomandibular joint: Low prevalence or missed diagnosis? Cranio 2006;24:234–3.

38. Soman D, Davies SJ. A suspected case of tuberculosis of the temporomandibular joint. Br Dent J 2003;194:23–4.

39. Marotti M. Imaging of temporomandibular joint disorders. Rad 507. Med Sci 2010;54:133–48.

40. Choi JA, Koh SH, Hong SH, Koh YH, Choi JY, Kang HS. Rheumatoid arthritis and tuberculosis arthritis:...
Differentiating MRI features. AJR Am J Roentgenol 2009;193:1347-53.
41. Tauro LF, George C, Kamath A, Swethadri G, Gatty R. Primary Tuberculosis of Submandibular Salivary Gland. J Global Infect Dis 2011;3:82-85.
42. Thakur J, Thakur A, Mohindroo N, Mohindroo S, Sharma D. Bilateral Parotid Tuberculosis. J Glob Infect Dis 2011;3:296-9.
43. Handa U, Kumar S, Punia RS, Mohan H, Abrol R, Saini V. Tuberculous parotitis: A series of five cases diagnosed on fine needle aspiration cytology. J Laryngol Otol 2001;115:235-7.
44. Iseri M, Aydiner O, Celik L, Peker O. Tuberculosis of the parotid gland. J Laryngol Otol 2005;119:311-3.
45. Süoğlu Y, Erdamar B, Colhan I, Katircioğlu OS, Cevikbas U. Tuberculosis of the parotid gland. J Laryngol Otol 1998;112:588-91.
46. Birkent H, Karahatay S, Akcam T, Durmaz A, Ongoru O. Primary parotid tuberculosis mimicking parotid neoplasm: A case report. J Med Case Rep 2008;2:62.
47. Kim YH, Jeong WJ, Jung KY, Sung MW, Kim KH, Kim CS. Diagnosis of major salivary gland tuberculosis: Experience of eight cases and review of the literature. Acta Otolaryngol 2005;125:1318-22.
48. Fontanilla JM, Barnes A, von Reyn CF. Current diagnosis and management of peripheral tuberculous lymphadenitis. Clin Infect Dis 2011;53:555-62.
49. Prasad KC, Sreedharan S, Chakravarthy Y, Prasad SC. Tuberculosis in the head and neck: Experience in India. J Laryngol Otol 2007;121:979-85.
50. Vaid S, Lee YY, Rawat S, Luthra A, Shah D, Ahuja AT. Tuberculosis in the head and neck: A forgotten differential diagnosis. Clin Radiol 2010;65:73-81.
51. Fontanilla JM, Barnes A, von Reyn CF. Current Diagnosis and Management of Peripheral Tuberculous Lymphadenitis. Clin Infect Dis 2011;53:555-62.
52. Wang WC, Chen JY, Chen YK, Lin LM. Tuberculosis of the head and neck: A review of 20 cases. Oral Surg Oral Med Oral Pathol Radiol Endod 2009;107:381-6.
53. Girish KL, Isaac Joseph T, Krishna Prasad RS. Peripheral Tuberculous Lymphadenitis: A Case Report. JIDAT 2014;6:23-6.
54. Mukta V, Jayachandran K. Lung and lupus vulgaris. Lung India 2011;28:127-9.
55. Smoller BR, Horn TD. In Dermatopathology in systemic disease. Infectious disease and the skin. Mycobacterial infections-Tuberculosis. New York: Oxford: Oxford University Press; 2001. p. 215-7.
56. Kumar R, Agrawal S, Kanwar AJ. Perforation of nasal septum secondary to lupus vulgaris: A rare entity. Indian J Paediatr Dermatol 2012;13:41-3.
57. Rullán J, Seijo-Montes RE, Vaillant A, Sánchez NP. Cutaneous Manifestations of Pulmonary Disease. In, Sánchez NP, editor Atlas of Dermatology in Internal Medicine. New York: Springer Science and Business Media; 2012. p. 17-30. Available from: http://www.springer.com/978-1-4614-0687-7 [Last accessed on 2015 Jun 22].
58. Shimizu H. Shimizu’s Textbook of Dermatology. 2nd ed., Ch. 26. Mycobacterial Infections. Hokkaido: Hokkaido University Press/Nakayama Shoten publishers; 2007. p. 484-5.
59. Dhoat S, Rustin M. The skin in general medicine. Clin Med 2009;9:379-84.
60. Dermatology: An Illustrated Colour Text. 3rd ed. Elsevier Health Sciences. London: Churchill Livingstone; 2002. p. 46. ISBN 9780443071409.
61. Porous CE, Terezhalmy GT. Tuberculosis: Infection control/exposure control issues for oral healthcare workers. J Contemp Dent Pract 2008;9:1-13.
62. Kumar V, Abbas AK, Fausto N, Mitchell RN. Robbins Basic Pathology. 8th ed. United States: Saunders Elsevier; 2007. p. 516-22. ISBN 978-1-4160-2973-1.
63. Mazurek GH, Jereb J, Vernon A, LoBue P, Goldberg S, Castro K; IGRA Expert Committee; Centers for Disease Control and Prevention (CDC). Updated Guidelines for Using Interferon Gamma Release Assays to Detect Mycobacterium tuberculosis Infection. United States, 2010 MMWR Recomm Rep 2010;59:1-25.
64. Koh WJ, Jeong YJ, Kwon OJ, Kim HJ, Cho EH, Lew WJ, et al. Chest Radiographic Findings in Primary Pulmonary Tuberculosis: Observations from High School Outbreaks. Korean J Radiol 2010;11:612-7.
65. Centers for Disease Control and Prevention (CDC). Updated guidelines for the use of nucleic acid amplification tests in the diagnosis of tuberculosis. MMWR Morb Mortal Wkly Rep 2009;58:7-10.
66. Dinnes J, Deeks J, Kunst H, Gibson A, Cummins E, Waugh N, et al. A systematic review of rapid diagnostic tests for the detection of tuberculosis infection. Health Technol Assess 2007;11:1-196.
67. Moore DA, Evans CA, Gilman RH, Caviedes L, Coronel J, Vivar A, et al. Microscopic-observation drug-susceptibility assay for the diagnosis of TB. N Engl J Med 2006;355:1539-50.
68. Treatment of Tuberculosis: Guidelines. 4th ed. Geneva: World Health Organization; 2010. ISBN-13: 978-92-4-154783-3.
69. Zumla AI, Gillespie SH, Hoelscher M, Phillips PP, Cole ST, Abubakar I, et al. New antituberculosis drugs, regimens, and adjunct therapies: Needs, advances, and future prospects. Lancet Infect Dis 2014;14:327-40.
70. TB Alliance. Our Pipeline. Available from: http://www.tballiance.org/pipeline/pipeline.php. [Last accessed on 2015 Mar 01].

How to cite this article: Bansal R, Jain A, Mittal S. Orofacial tuberculosis: Clinical manifestations, diagnosis and management. J Family Med Prim Care 2015;4:335-41.

Source of Support: Nil. Conflict of Interest: None declared.