Case Report

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A Case Report of Palatal Ulcer: First Sign of Occult Tuberculosis

Dentistry Section

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ABSTRACT

Tuberculosis is a chronic granulomatous transmitting type of disease caused by the *Mycobacterium tuberculosis* complex. It can affect any part of the body, including the oral cavity. Oral tuberculosis can be primary or secondary, In oral cavity, tongue, buccal mucosa, lip, and palate may involve. Here, the authors present a case of a 50-year-old male patient of tuberculosis of palate, manifesting as a non healing ulcer. The ulcer was present in the middle part of the palate, having undermined edge and a non indurated margin. Though it was tender on palpation, there was no evidence of palatal perforation or bony erosion on radiographic examination. A chest radiograph revealed consolidation in the apex and right upper zone, and Cartridge Based Nucleic Acid Amplification Test (CBNAAT) of sputum was positive, but biopsy of the lesion could not be performed because of problem in patient's consent. The authors took a chance and started antitubercular drugs. They observed the changes of ulcer at regular intervals. No topical medication was given for the ulcer. After taking antitubercular drugs, the condition improved rapidly and The ulcer healed completely after completing the Intensive Phase (IP) only. Tuberculosis is a transmitting and fatal disease. Early diagnosis with proper treatment can prevent complications and the transmission of the disease to others.

Keywords: Extrapulmonary tuberculosis, *Mycobacterium tuberculosis* complex, Oral tuberculosis, Pulmonary tuberculosis

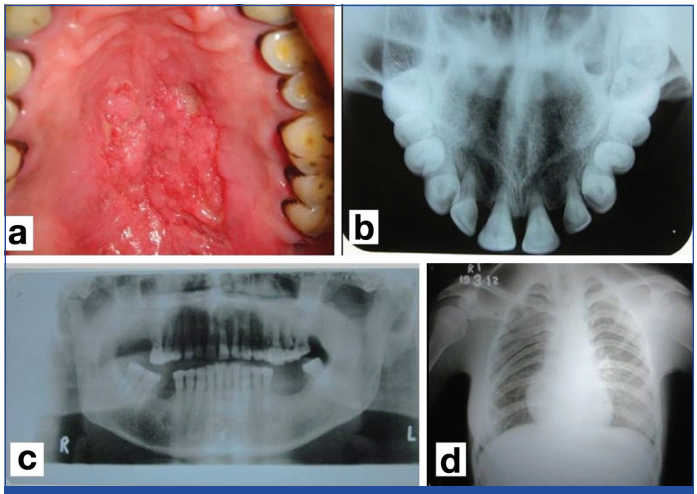
CASE REPORT

A 50-year-old male patient presented to the Outpatient Department with a complaint of an ulcer in the mid-palatal region since three months. The ulcer had gradually increased in size and had been painful for the last two months. The pain was dull in nature and aggravated during taking food. Patient felt difficulty in swallowing. Pain persisted for a few minutes to hours and then subsided on its own. The patient also gave history of weight loss (around 8-10 kg over the last one and a half months), productive mucopurulent cough, and malaise during the past two months. Prior to the visit to the department, the patient had taken topical anaesthetic ointment (ointment composition-Chlorhexidine Gluconate, Metronidazole, and Lignocaine Hydrochloride gel) till visit to the department and systemic antibiotics (Azithromycin for 14 days followed by Amoxicillin and Clavulanic Acid combination for 14 days, and Cefpodoxime Proxetil for 21 days) by a local doctor, with no relief. The patient's medical history and family history did not reveal any significant contributions. He had no history of diabetes, hypertension, endocrine disorders, liver disease, previous tuberculous infections, or current medication for other systemic illnesses. There was no history of genetic or communicable diseases in his family. The patient had a smoking habit of 5-6 bidis per day for the past 15-20 years.

During the examination, the patient weighed 40 kg. All vital signs were within normal ranges, except for a slightly elevated temperature of 99.0°F. No other abnormalities were detected upon general examination. A chest examination using a stethoscope was not done. On extraoral examination, the right and left submandibular, sublingual, submental, and cervical lymph nodes were not enlarged and were non palpable. No abnormality was detected during the examination of the Temporomandibular Joint (TMJ) and the muscles of mastication. There was no facial asymmetry present.

Intraorally, a well-defined erythematous trapezoidal ulcerated area was present in the middle portion of the palate on both sides of the midline, measuring about 2×2.5 cm. The border of the ulcer was irregular with an undermined edge, and yellowish slough was present over the ulcerated area [Table/Fig-1a]. On palpation, the base of the ulcer was irregular, and the border was not indurated. The ulcer was tender on palpation. No bleeding or pus discharge was present from the ulcerated area. A provisional diagnosis of granulomatous ulcer of the palate was made. Given the history of smoking, weight loss, and productive cough, the first differential diagnosis was a tuberculous ulcer. As the ulcer was painful and non healing type, other differential diagnosis included syphilitic ulcer, malignant ulcer, and fungal ulcer.

The maxillary cross-sectional occlusal radiograph showed no bony deformity in the palatal region [Table/Fig-1b]. The orthopantomograph showed no bony erosion or destruction in the palatal region [Table/Fig-1c]. Additionally, the orthopantomograph also revealed no bony pathology in the maxilla, mandible, or their supporting structures. Due to the patient's history of cough, a chest radiograph (posteroanterior view) was advised. It revealed non homogeneous opacity in both the apex and the right upper zone of the lung, with prominent bronchovascular markings in the rest of the lung fields. The chest radiograph was suggestive of both the apex and the right upper zone consolidation [Table/Fig-1d]. After getting chest radiograph the authors thought that palatal

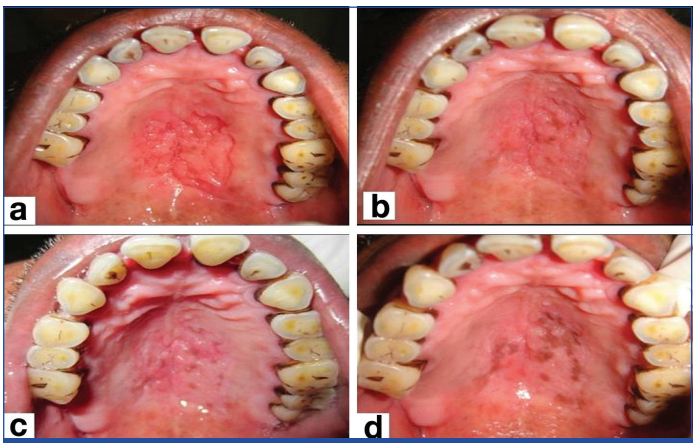


[Table/Fig-1]: Before treatment: a) Intraoral view showing ulceration on palate; b) Maxillary cross-sectional occlusal radiograph showing no bony deformity on palatal region; c) Orthopantomogram showing no bony deformity in maxilla and mandible; d) PA view of chest radiograph showing non homogeneous opacity seen in both apex and right upper zone of lung.

ulcer might be tuberculous ulcer and the tests for tuberculosis were then advised The blood reports were all within normal limits. The Erythrocyte Sedimentation Rate (ESR) (measured by the Wintrobe method) was 27 mm/1 hr. The Venereal Disease Research Laboratory (VDRL) test yielded a non reactive result. The Mauntoux test was positive, showing 20×18 mm of erythema and 19×17 mm of induration after injecting 0.1 mL of Purified Protein Derivative (PPD) (10 TU/5TU intracutaneously) 48 to 72 hours after injection. The sputum test for acid-fast bacilli showed negative result in the morning sample. Serology for the Human Immunodeficiency Virus (HIV) was negative. The Cartridge-Based Nucleic Acid Amplification Test (CBNAAT) of the sputum sample was positive. A biopsy was not done as the patient did not give consent. Based on all the findings, the diagnosis was given as secondary oral tuberculous ulcer in the palate, though there was a chance that the palatal ulcer may not be related to the chest and sputum findings.

Subsequently, a decision was made to monitor any changes in the ulcer will occur or not after starting Antitubercular Drugs (ATD). The patient was referred to a pulmonologist. According to the Revised National Tuberculosis Control Programme (RNTCP) guidelines from 2017, the Intensive Phase (IP) involved eight weeks of Isoniazid (INH), Rifampicin, Pyrazinamide, and Ethambutol administered in daily dosages as per four weight band categories. Follow-up was done. In the Continuation Phase (CP), Pyrazinamide and Ethambutol were stopped while the other drugs were continued for another 16 weeks at daily dosages. Additionally, Vitamin B6 (Pyridoxine) was also given once daily. No topical medication was advised for the ulcer.

After 20 days of starting the ATD, the size of the ulcer and tenderness reduced [Table/Fig-2a]. The patient did not complain of any fever or night sweats after that. One month into the treatment, lesion size was reduced by 90%, and there was no longer any tenderness [Table/Fig-2b]. Two months into the treatment, the lesion had almost completely healed [Table/Fig-2c]. Six months after completing the antitubercular drugs regimen, the ulcer had completely healed, and multiple small brown pigmentations had appeared [Table/Fig-2d]. This type of pigmentation may be attributed to the Addisonian effect, as tuberculous infection can destroy adrenal gland. The patient began gaining weight nearly after three months from starting the treatment. Regular follow-up was conducted for one year after completing the regimen, and no recurrence of the ulcer was seen.



[Table/Fig-2]: After starting antitubercular drugs: a) 20 days after the treatment showing reduction of ulcer size; b) 1 month after the treatment showing reduction of ulcer size almost 90%; c) 2 months after the treatment, at the end of IP, showing almost completely healed ulcer; d) 6 months after the treatment, at the end of CP, showing completely healed ulcer with pigmentation.

DISCUSSION

Tuberculosis, one of the oldest granulomatous diseases worldwide, is caused by *Mycobacterium tuberculosis* and, relatively less by *Mycobacterium bovis*, *Mycobacterium microti*, and *Mycobacterium africanum*. It is also called Koch's bacillus, named after the German physician Robert Koch who discovered this bacillus. In 1 5000-20000 years ago, archaeologists found spinal tuberculosis (Pott's disease) in Egyptian mummies, which was referred to as the "King's evil." In ancient times, tuberculosis was also known as kshay rog, phthisis, and white plaque [1].

The World Health Organisation (WHO) declared tuberculosis a global emergency in 1993 because of increasing prevalence of the disease, its association with HIV, and increased drug resistance. According to WHO reports in 2016, about 10.4 million new cases of tuberculosis and 1.8 million deaths occur worldwide each year due to tuberculosis. India is one of the six "high-burden countries," with about 60% of the total tuberculosis cases occurring there every year [1,2]. In India, tuberculosis claims the lives of two patients every five minutes [3].

Ingestion of unpasteurised cow's milk infected by *Mycobacterium bovis* or other atypical Mycobacteria can also cause tuberculosis [4,5]. These bacilli are acid-fast aerobic organisms that are generally transmitted through droplet inhalation. So, it may transmit during prolonged contact with an infected person [2]. The risk of contracting tuberculosis mainly depends on an individual's immunological status. HIV is a significant risk factor for tuberculosis as it suppresses cellular immunity. Other risk factors for tuberculosis include renal failure, the use of immunosuppressive drugs, vitamin D deficiency, smoking, alcohol consumption, patients on Tumour Necrosis Factor (TNF) antagonist therapy, severe malnutrition, diabetes, and corticosteroid use [6]. Individuals in low socio-economic status and healthcare workers, who frequently come into contact with tuberculosis patients, are at a higher risk of infection [2]. Smokers have nearly twice the risk of tuberculosis compared to non smokers. This may be due to ciliary dysfunction leading to a reduced immune response, increasing susceptibility to infection with *Mycobacterium tuberculosis* [3]. Passive smoking can also increase the risk of tuberculous infection, specially in children.

The lung is the most common site for tuberculosis. Extrapulmonary sites include the skin, kidneys, pharynx, lymph nodes, bones, joints, genitourinary tract, central nervous system, and oral cavity [4,5]. The prevalence of oral manifestations secondary to pulmonary tuberculosis may occurs from 0.8% to 3.5% [7]. Oral tuberculosis can be primary or secondary. Secondary oral tuberculosis mainly occurs in elderly persons, but primary variety mainly occurs in young individuals [1]. Oral manifestations may present in 0.05-5% of total tuberculosis cases [4]. The break or loss of the natural barrier can lead to the direct inoculation of mycobacteria in primary oral tuberculosis. Trauma, inflammatory conditions, extractions, and poor oral hygiene are the main predisposing factors for the primary type. In secondary oral or oropharyngeal tuberculosis, pulmonary involvement generally occurs first, and the route of spread can occur through haematogenous or lymphatic spread, from a healed primary focus, or due to invasion following the loss of the natural barrier [5]. Autoinoculation can also occur if infected pulmonary mucus comes into contact with susceptible areas of wounded mucosa [6]. Although a large number of bacilli may come into contact with various parts of the oral cavity in pulmonary tuberculosis, not all cases of pulmonary tuberculosis lead to secondary infection in the oral cavity because saliva plays an important protective role. Salivary enzymes, the cleansing action of saliva, tissue antibodies, oral saprophytes, and the thickness of the epithelial layer help prevent the invasion of bacilli into the oral mucosa [2,7]. Some authors have proposed that a certain Indian brushing habit, known as "Datoon" (brushing teeth with neem twigs), may cause trauma to the palatal region, thereby predisposing the wound to be seeded with the *Mycobacterium tuberculosis* complex [4].

Oral manifestations of tuberculosis are clinically non specific, which is why they are sometimes overlooked in the differential diagnosis, especially when systemic features are not clinically present. Oral tuberculosis can occur in any part of the oral cavity, with the tongue being the most commonly affected, followed by the floor of the mouth, gingiva, lips, soft and hard palate, and buccal

mucosa [4,5]. The primary variety is rare and generally manifests as a painless ulcer of long duration with superficial or deep tender lymph nodes. On the other hand, secondary lesions generally manifests as painful ulcers with irregular undermined margins, an indurated border, and the presence of slough. Difficulty in speech, deglutition and mastication are common symptoms in the secondary variety, and the lymph nodes are usually non tender on palpation [2,5]. Oral tuberculosis may also manifest as nodules, fissures, verrucous proliferation, tuberculoma, erythematous patches, lesions within the jaw in the form of osteomyelitis or periapical granuloma, and yellowish apple-jelly-like granuloma, apart from the non healing ulcerated form [4]. In the present case, the ulcer had an undermined edge with a non indurated margin. Microorganisms may reach the periapical tissue through the pulp chamber of a tooth with an open cavity, and then it may produce tuberculous periapical granuloma or tuberculoma, or diffuse involvement of the maxilla and mandible through haematogenous spread, causing tuberculous osteomyelitis. Perforation may occur in case of involvement of hard palate [8], but in our case, no perforation was present. Males are more commonly affected than females. Common symptoms of tuberculosis include a productive cough, night sweats, weight loss, and low-grade fever [1]. In the present case, all of these symptoms were not present except for a cough. Traumatic ulcer, aphthous ulcer, syphilitic ulcer, malignant ulcer, Wegener's granulomatosis, and actinomycosis are among the differential diagnosis for oral tubercular ulcer [5].

Histopathological findings include Langhan's type of giant cells, caseating granuloma with central necrosis surrounded by epithelioid cells, lymphocyte infiltration, and the presence of acid-fast bacilli on Ziehl-Neelsen staining. Non caseating granulomas may be found in cases of immunocompromised conditions. In the early stages of oral disease, granulomatous changes may not be present, and acid-fast bacilli may also not be found in the sample. In such cases, establishing a diagnosis through a biopsy of the oral lesion is often difficult to establish the diagnosis. According to various studies, only a small percentage of histopathology specimens stain positive for acid-fast bacilli. Therefore, a negative result does not completely rule out the possibility of tuberculosis. Since stained bacilli are not visualised in all cases, bacterial culture is required, and samples may be taken from sputum or any suspected body fluid or lesion surface. Sputum examination for acid-fast bacilli and chest radiographs should be done to rule out pulmonary tuberculosis when a tuberculous ulcer is present in the oral cavity [1,2,5,8]. The Mantoux test (tuberculin sensitivity assay) is also one of the procedures used to diagnose tuberculosis, involving the intradermal inoculation of purified protein derivative of *M. tuberculosis* on forearm to assess the immune response to the antigen. The Mantoux test is also used to detect latent tuberculosis. However, this test cannot differentiate between infection and active disease and is relatively less sensitive in immunocompromised individuals [1,6]. In the present case, the Mantoux test was positive, but it is not a confirmatory test for tuberculosis. Ultrasonography, Computerised Tomography (CT) Scan, and Magnetic Resonance Imaging (MRI) are other investigations that can be used as supporting tools for diagnosing tuberculosis [3].

According to the World Health Organisation (WHO) and the Revised National Tuberculosis Control Programme (now called as NTEP; National Tuberculosis Elimination Programme-NTEP), Cartridge-Based Nucleic Acid Amplification Test (CBNAAT) should be done in all cases of pulmonary and extrapulmonary tuberculosis to detect M. tuberculosis as well as rifampicin resistance. The test results comes within two hours from sampling. CBNAAT is a type of Real-time Reverse Transcriptase-polymerase Chain Reaction (RT-PCR) test that examines the specimen for genetic material specific to M. tuberculosis. It is a fully automated test conducted using the GeneXpert platform. Interferon-gamma Release Assays (IGRAs) can also be done to detect Mycobacterium [3,9,10]. Nowadays, sputum tests and CBNAAT are generally used for diagnosing tuberculosis in our country.

From 2017, according to RNTCP guidelines, the principle of tuberculosis treatment has shifted from Directly Observed Therapy (DOT) to a daily regimen involving the administration of a daily fixed-dose combination of first-line ATDs as per appropriate weight bands [Table/Fig-3] [9,10]. The number of tablets to be taken is determined by the patient's weight [Table/Fig-4] [3,9,10]. The treatment regimen may be extended in some cases for both new and previously treated cases. The drug regimen will differ in cases of multidrug resistance [10].

Types of cases	Treatment regimen for Intensive Phase (IP)	Treatment regimen for Continuation Phase (CP)
New cases	8 weeks HRZE (H=Isoniazid, R=Rifampicin, Z=Pyrazinamide and E=Ethambutol)	16 weeks HR
Previously treated cases	8 weeks HRZES + 4 weeks HRZE (S= Streptomycin injection)	20 weeks HRE

[Table/Fig-3]: RNTCP Guideline for treatment of tuberculosis from 2017 [9,10].

Drug regimen		Weight category			
		25-39 kg	40-54 kg	55-69 kg	>=70 kg
Intensive Phase (IP)	HRZE (75/150/400/275)	2 tablets	3 tablets	4 tablets	5 tablets
Continuation Phase (CP)	HRE (75/150/275)	2 tablets	3 tablets	4 tablets	5 tablets
Previously treated cases	Inj. Streptomycin	0.5 gm	0.75 gm	1 gm	1 gm

[Table/Fig-4]: Relationship between patient's weight and dose of antitubercular drugs [3,9,10].

Dentists should play a vital role in diagnosing tuberculosis from oral manifestations in cases of undiagnosed pulmonary tuberculosis. It can be a diagnostic challenge when an oral lesion is the sole manifestation of the disease. Oral healthcare workers are at a high-risk of tubercular infection because of close contact with the mouth and spreading of aerosols during dental procedures. A detailed case history should be taken before doing any dental procedure. Non-treated active cases are most vulnerable to healthcare workers. Oral procedures should be done to urgent and essential cases. Proper disinfection and sterilisation protocols should be followed. The use of a rubber dam can help reduce aerosol contact. For dental procedures involving known active tuberculosis patients, a well-equipped separate room with an effective air evacuation system and High Efficiency Particle Arresting (HEPA) filter, along with high-volume suction, are necessary to minimising aerosol generation and prevent the spread of tuberculosis [8].

CONCLUSION(S)

If oral tuberculosis is diagnosed, the primary site of the disease should be located before considering the oral lesion as primary tuberculosis. Medical personnel are always at risk when dealing with these types of cases. Mouth-to-mouth resuscitation is considered a high-risk factor. Caution should also be taken in outpatient settings when examining patients with chronic non healing ulcers in the oral cavity to prevent transmission. Oral tubercular lesions should be identified at an early stage not only for the benefit of the patient but also for the benefit of dentists and the community, as the patient can be a potential source for transmitting the disease. Tuberculosis should always keep in mind when diagnosing any non healing ulcer, erythematous patch, nodular growth in the oral cavity, or osteomyelitis of the jaws.

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