Review article

Oral and maxillofacial tuberculosis: A review

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ABSTRACT

There are cases of oral tubercular lesions; however, they are scarce. Oral lesions were present in less than 0.1 percent of the tuberculous patients. the bulk of which mostly affected the tongue's base. The obscure location of the tuberculous lesions might account for the disparity in incidence of occurrence. Oral tuberculosis can occur as a primary or secondary infection. In younger individuals, primary lesions are rare, and they are frequently linked to enlarged cervical lymphadenopathy. Secondary oral Tuberculosis many a times exists together with pulmonary illness and can affect persons of all ages; however, those in their forties and fifties are more likely to be impacted. Organisms entering the sputum and subsequently entering the layers of mucosa via a tiny fissure on the surface of mucosa are the most likely mode of inoculation. The organisms were most likely transferred through the blood to the oral tissues and reached the submucosa before proliferating and ulcerating the overlying mucosa, while lesions of the mouth are less frequent, they are necessary for detecting and treating primary tuberculosis.

Keywords: Oral cavity; extrapulmonary; *Mycobacterium tuberculosis*; ulcer; HIV.

INTRODUCTION

Tuberculosis is a persistent granulomatous infection produced by several mycobacterial strains, most commonly Mycobacterium Tuberculosis in humans. In 1882, a German physician named Robert Koch identified the Tuberculosis bacillus (1). For centuries, it has been a huge public health issue around the world. *Mycobacterium tuberculosis*, causative agent of pulmonary tuberculosis, spreads person-to-person through airborne droplets, and *Mycobacterium bovis*, *which* causes intestinal tuberculosis, is transmitted through consumption of unpasteurized cow milk (2-4). One of the primary causes of disease and mortality worldwide is tuberculosis. Lower socioeconomic groups have a much higher risk of infection (5). Rare oral TB lesions are typically seen in children but can occasionally be seen in adults. The most likely carriers of tubercle bacilli are sputum or unpasteurized milk, which pass through a tiny gap in the oral mucosa and penetrate the mucosal tissue (6). The emergence of multidrug-resistant (MDR) organisms and the incidence of HIV infection are both factors in the rise in pulmonary TB cases (7). HIV and tuberculosis interact with one another. Each person with active TB will annually infect 10 to 15 other people if they are not treated, according to the World Health Organization (8). Therefore, early, and precise identification and diagnosis, in addition to therapy, are necessary for effective management of tuberculosis. Early tuberculosis identification is aided by cutting-edge diagnostic tools. Early detection and total eradication may be attainable if these tools are used in primary healthcare facilities. People with medical problems that weaken the immune system, substance misuse, silicosis, and diabetes mellitus are among the main risk factors for tuberculosis. head and neck cancer, severe kidney problems, being underweight, and organ transplants Clinically, it can take any shape, but as the incidence of tuberculosis lesions in the mouth has decreased, in the identification of oral lesions, they are frequently overlooked (9). Although oral tuberculosis is rare, it accounts for 0.5-5% of all Tuberculosis infections. This is due to the fact that infections are caused by systemic variables like a weak immune system or a bacterium with a high level of virulence. The existence or absence of local trauma in the oral cavity, prior dental and oral illnesses, and oral hygiene are additional factors that influence clinical symptoms in the oral cavity in people with TB infection, because of the drug-resistant TB outbreak, as well as the emergence of acquired immune deficiency syndrome, oral symptoms of tuberculosis are reappearing alongside numerous forgotten extrapulmonary illnesses (10).

Classification of oral tuberculosis

Temporomandibular joint lesions

The main tuberculosis of the Temporomandibular Joint (TMJ) is uncommon. When they first come, large numbers of the patients show preauricular swelling and spasm of local muscles. TMJ TB has a non-specific clinical and radiological presentation that may resemble osteomyelitis, arthritis, or other chronic joint illnesses. Preauricular swelling that hurts and is insensitive to antibiotic treatment is the most typical symptom (11).

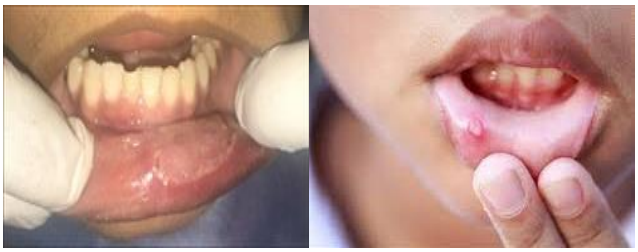

Fig. 1: Primary and secondary oral tuberculosis in HIV (PC-https://www.dailyexcelsior.com/insight-into-oral-ulcers)

Lesions of oral cavity and related soft tissue structures

The palate, mainly soft palate, floor of the mouth, gingiva, tongue and muco-buccal folds are common locations for oral tubercular lesions. The tongue is the most involved site, accounting for roughly half of all instances. Clinically there may be ulceration, swelling or irregular growth in the mouth cavity that can be solitary or many, ulcerated or non-ulcerated giving rise to the incorrect diagnosis of cancer. Salivary gland tuberculosis is frequently after oral cavity infection or primary TB of the lungs. The most prevalent site of involvement is the parotid gland clinical symptoms may be acute or persistent. Clinical differentiation can be difficult because acute tuberculosis can resemble acute sialadenitis. A stud collar-like pattern of many matted, hard swellings in the neck may be present in cervical tuberculous lymphadenitis. Histopathological investigations can be done by an incisional biopsy or Fine Needle Aspiration cytology of the lesion and microbiological diagnosis by culture of *Mycobacterium tuberculosis.* Histological anomalies can also point to granulomatous infection rather than TB. Therefore, to diagnose EPTB, doctors must rely on clinical and radiological appearance.

Lesions of the jaw bones

A patient with TB who has jaw involvement may present with a variety of clinical symptoms, including trismus and painless jaw swelling. Condyle destruction and soft tissue masses are seen on radiographs and computed tomography (CT) images. A TB infection is often diagnosed via radiological imaging, biopsy, and culture (12).

Transmission of *M. tuberculosis*

As a contagious illness, in pulmonary tuberculosis patients are the main carriers of the infection. Patients with active pulmonary TB (open tuberculosis) typically cough up droplet nuclei particles of around 2 to 5 um. Droplet nuclei can remain in the air for a long time due to their tiny size. The chance of infection is determined by various factors, such as infectiousness of the patient, the contact closeness, the number of bacteria breathed, and the prospective host's immunological status. The lungs are primary organs where the most infections start. *M. tuberculosis* is an acid-fast bacterium that causes tuberculosis. It is a non-spore-forming, rod-shaped, aerobic, thin, unencapsulated bacterium. These bacteria have lengths of 2–5 nm and a thickness of 0.2–0.5 nm. *M. tuberculosis*, an alcohol- and acid-resistant bacillus, is spread through the air by droplet nuclei and grows in pulmonary alveoli. Alveolar macrophages are the site of bacterial multiplication, which is disseminated via local lymph nodes. Inhaled droplet nuclei are so small that they can get past the bronchial barriers and enters the alveoli at the end, where phagocytic immune cells consume them such as dendritic cells and macrophages *M. tuberculosis* is first engulfed by phagocytic cells, then multiplies within the cells, and has the potential to cross the alveolar barrier and spread throughout the body (9). Saliva is produced in the human oral cavity and acts as a cleansing and protective agent thanks to its antibacterial qualities, which prevent the tuberculosis bacillus from penetrating the walls of the epithelium. Clinical studies, however, have demonstrated that the invasion of tuberculosis bacteria into the connective tissue below is caused by epithelial damage. Systemic factors, such as reduced human defenses and enhanced virulence of microbes, affect the chance of infection. The predisposing conditions in the oral cavity that cause oral TB include trauma in the oral cavity, bad mouth hygiene, and the presence of pre-existing lesions such as leukoplakia, abscesses, cysts granuloma of periapical region, periodontitis, and fractures of the jaw (13,14).

The host's immune reaction to *M. tuberculosis*

Beginning with phagocytosis of bacilli by antigen-presenting cells, such as histiocytes and dendritic cells in the lungs, *M. tuberculosis* infection occurs. PRRs that recognize pathogen-associated molecular patterns (PAMP) initiate and direct the host's innate immune response. (15). Toll-like receptors (TLRs), nucleotide-binding oligomerization domain (NOD) like receptors (NLRs), and C-type lectins recognize *M. tuberculosis* or its components (ligans). TLR contact with *M. tuberculosis* starts a signaling cascade within the cell that causes an inflammatory response. TLR contact, specifically between *M. tuberculosis* and TLR2 and TLR4, causes an innate immune response. The cell wall of *M. tuberculosis* is made up of high concentration of mycolic acids which is composed of lipids and polysaccharides. The important surface ligands of cell walls of *Mycobacterium tuberculosis* that react with TLR is Lipoarabinomannan (LAM). The interaction between LAM and TLR activates nuclear transcription factor (NFkB), resulting in the generation of cytokines like interleukin, tumor necrosis factor, interferons, chemokines, and nitric oxide. These cytokines help in detection of infection. Even though lymphocytes, neutrophils and monocytes

reach the infection site, they cannot kill the pathogens. To get beyond the macrophage's bacterial defense system (phagolysosome), bacilli grow in the phagosome to cause necrosis of macrophage (13). A separate macrophage, which is also unable to inhibit *M. tuberculosis* from developing, phagocytosis the discharged bacilli, which then kills them. T lymphocytes (CD4 and CD8) are prepared to recognize mycobacterial antigens when they reach the local lymph node. Primed T lymphocytes (CMI), which follow the cytokines generated by the TB infected cells back to the site of infection, which are produced because of the targeted immune response. Granulomas are caused by the increase of cells like T lymphocytes, macrophages, endothelial cells, fibroblasts, stromal cells, and dendritic cells (16,17).

In addition to inhibiting bacterial spread and providing a setting for interactions between macrophages and other immune system cells, and the cytokines produced by these cells, granuloma formation separates TB bacilli from the other lung tissue. M. tuberculosis-infected macrophage-presenting antigens are recognized and eliminated by CD4 T cells that release IFN-gamma. The death and survival of mycobacterial cells are balanced within the resultant granuloma. The granulomatous process prevents latent tuberculosis infection (LTBI), which is caused when some bacilli survive. This approach is sufficient to control the infection after an acute *mycobacterium tuberculosis* infection in 95% of patients, while the remaining 5% develop primary TB illness. The lifetime risk of getting TB in HIV infected patients is 50 to 70 % when compared to HIV-negative patients which are 10 % due to the deleterious effects of tuberculosis on the immune system. There is a link between immunosuppression due to the progression of HIV and development of oral lesions and other lesions such as hairy leukoplakia, oral candidiasis and viral load and impairment of immunological systems which can be used as a prognosticator. Several studies have connected the lesions of the oral cavity to other AIDS-defining disorders like pneumocystis and tuberculosis (18). Oral symptoms were present in 77.5% of patients who were co-infected with HIV and TB, demonstrating that oral lesions are a reliable indicator of co-infection. The best screening tool or clinical indicator for TB coinfection is patients with lesions of the oral cavity and if HIV is seropositive, they should be evaluated for TB (19).

Symptoms and signs in general

Patients with tuberculosis who have had it reactivated typically report weariness, weight loss, anorexia, a slight fever, and nocturnal sweats. Coughing is one of the symptoms of a lung infection; it begins dry but quickly turns into a torrent of purulent sputum and is frequently accompanied by blood (20).

Symptoms and signs in oral tuberculosis

Oral TB can cause either primary or secondary lesions. The first lesion is uncommon, occurring more frequently in young persons, and manifests as a solitary, painless ulcer with swelling of adjacent lymph nodes. Secondary lesions, which are widespread and frequently associated with pulmonary infection, typically manifest as a single, irregular, indurated, painful ulcer covered in an inflammatory exudate. Oral TB can infect any portion of the mucosa of the oral cavity; however, the most common site is the tongue. Other sites are the palatine tonsils, gingiva, palate, floor of the oral cavity. The uvula and salivary glands are also frequently affected. Primary oral TB might manifest as a long-lasting, painless ulcer and growth of the local lymph node. There are many different types of oral lesions that can develop, including ulcers, nodules, tuberculomas, and periapical granulomas. In addition to lesions in the jaws caused by tuberculous osteomyelitis or simple radiolucency of bone, oral TB symptoms can include swelling lesions of soft tissues or even superficial ulcers, patches (21). The most common form of these oral disorders is ulcerative. An ulcer, which commonly forms along the outer border of the tongue next to, a sharp broken tooth or similar irritating place, is the most common symptom of oral TB. Most patients with TB lesions of the oral cavity give a history of trauma. Any persistently inflamed or irritated location may encourage the localization of *M. tuberculosis*. The patient's ability to eat and get a good night's sleep is severely hampered by the extreme discomfort that is associated with this tongue lesion. The ventral, lateral border, dorsum, midline, and base of the tongue are the most common locations for tubercular ulcers. The lesions discovered often have an amorphous form. Oral TB frequently mimics malignant lesions as well as other lesions including Wegener's granulomas, traumatic ulcers, aphthous ulcers, actinomycosis, and syphilitic ulcers (22).

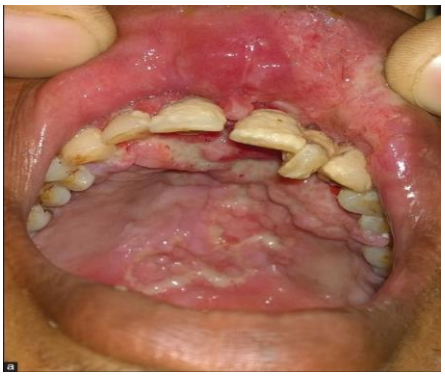

Fig. 2: Hard palate ulceration with necrotic slough, labial enlargement, and upper second incisor loosening (https://ijdvl.com/a-non-healing-oral-ulcer-as-a-manifestation -of-systemic-tuberculosis-in-an-immunocompetent-man/)

The palate had the highest prevalence of oral secondary tuberculosis lesions which is followed by other parts of the oral cavity like lips, tongue, gingiva

and alveolar bone. Although oral TB lesions are uncommon, multiple studies have found that 0.1-0.5% of pulmonary tuberculosis patients also acquire mouth secondary tuberculosis lesions. Nearly 97% of tuberculosis cases have secondary infections in the oral cavity that manifest as ulcers, 50% of which involve the tongue.

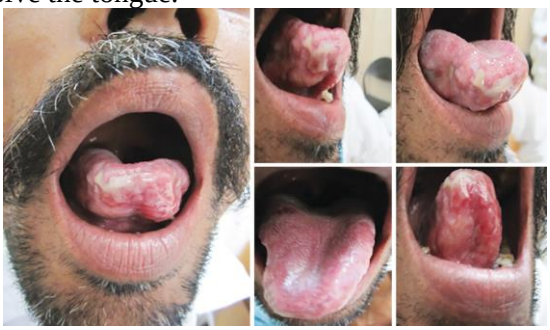

Fig. 3: Ulcers in the oral cavity in primary TB patients (14). (https://www.ijhns.com/doi/IJHNS/pdf/10.5005/jp-journals-10001-1356)

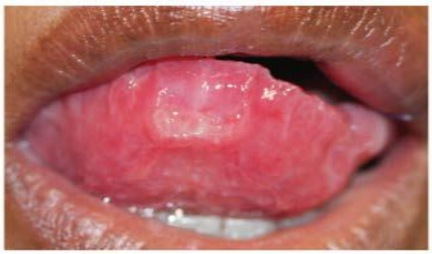

Fig. 4a: The ventrum of the tongue has an ulcer with slightly raised edges and a large zone of surrounding erythema. (https://www.semanticscholar.org/paper/Primary-Oral-TB-as-an-Indicator-of-HIV-Khammissa-Wood/cb0a910390eb21aeab445c23fbe96b32f6433216)

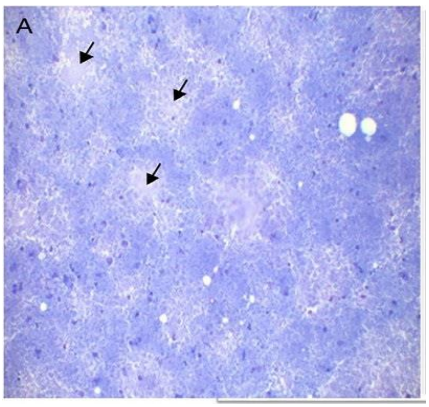

Fig. 4b: Tuberculosis of the cytomorphological type II.-Caseous necrosis (arrow)-Fine needle non-aspiration cytology for cervical lymph node tuberculosis diagnosis (Available from https://www.researchgate.net/figure/Cytomorphological-Type-II-tuberculosis-A-Caseous-necrosis-arrow)

When mycobacterium causes necrosis in infected tissue, lesions of the oral mucosa may appear as prismatic vesicles which might break open and form ulcers. Oral mucosa ulcers are painless and have a single, irregularly shaped, indurated, rough surface, a yellowish base, and an erythemal edge. Secondary tuberculosis develops as small, painless ulcers in most oral tuberculosis lesions most frequently observed on the hard palate than the soft palate. The primary tuberculosis lesions represent most of the gingival lesions. Many granules make up gingival lesions, which can also show up as erosion and marginal periodontitis. Tuberculosis patients have progressive, painless gingival enlargement that extends beyond the margin of gingiva and into the vestibule which may be seen along with enlarged lymph nodes. The squamous epithelial cell of gingiva which increases the thickness of the epithelial layers provides direct resistance to *M. tuberculosis*. Lip lesions often appear as small, granular ulcers (23).

Tuberculosis-induced osteomyelitis can potentially harm the maxilla and mandible. The maxilla is impacted less commonly than the mandible. Among the symptoms include trismus, lower lip paresthesia, and swelling of the local lymph nodes. removal of teeth damaged by tuberculous granulomas and pulmonary TB metastases that spread both hematogenous and lymphatically, resulting in maxillary and mandibular bone involvement (24). There have been reports of post-extraction socket infection and involvement of periapical tissue with tuberculosis. Basal tuberculosis can penetrate the periapical tissue through the exposed pulp, resulting in periapical granuloma tuberculosis or tuberculoma. When a tooth is extracted due to periapical lesions, the extraction sockets may be filled by a significant amount of granulated tissue (25).

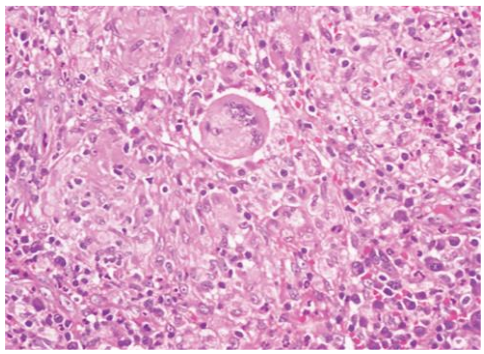

Fig. 5: The biopsy's histopathology revealed several confluent and distinct granulomas composed of epithelioid cells and multinucleated giant cells with central caseous necrosis. (PC-https://www.ijhns.com/abstractArticleContentBrowse/IJHNS/16881/JPJ/fullText)

Differential diagnosis

Malignancy is the most common reason for a non-healing ulcerative lesion in the oral cavity. The typical history is that of a quickly developing lesion that does not respond well to treatment. Usually, there is a history of long-term tobacco smoking. It is frequently accompanied with weight loss and chronic illness symptoms (26). The diagnosis of tuberculosis can be done by taking the patient's case history, physical examination of the patient, chest radiography and the Mantoux test. The tuberculin test, which is commonly used in TB screening, is the most reliable test for

determining whether children are currently infected with M. tuberculosis or have previously been infected.

Laboratory diagnosis of tuberculosis

Rapid technological improvements have made it easier to diagnose tuberculosis and its antitubercular susceptibility in clinical specimens. Improvements in microscopic methods, in vitro cultivation, and the use of molecular technologies are among them. The World Health Organization (WHO) has invested significantly in assessing the usefulness and viability of various diagnostic technologies, particularly in developing nations. The National Tuberculosis Elimination Programme (NTEP), which is now being implemented in India, aims to improve diagnosis services by establishing a national network of recognized intermediate reference laboratories and microscopy centres. Microscopy developments include fluorescence microscopy using fluorescence staining, sodium hypochlorite sedimentation smear microscopy of sputum smears. Recent advances *in vitro* culture, specifically Mycobacterium growth indicator tube (MGIT), has considerably reduced the amount of the time required for diagnosis, even in smear-negative samples, with the added benefit of simultaneously detecting resistance mutations. MGIT may perform first and second line drug susceptibility testing. The amount of time required has decreased because of the molecular identification of *Mycobacterium tuberculosis* and medication resistance. Truenat (MOLBIO, India) is a chip-based real-time micro-polymerase chain reaction test, and GeneXpert (Cepheid, USA) is a cartridge-based nucleic acid amplification assay. If the cost of testing is reduced, especially for use in private diagnostic laboratories, they appear to be on track to replace present methods for diagnosing tuberculosis (TB) in all situations. Line probe assay (LPA) is another molecular test endorsed by WHO to aid in diagnosing sputum positive cases for detection of INH and Rifampicin resistance (27).

Role of an oral pathologist

Differentiating oral tuberculosis from other illnesses based only on clinical signs and symptoms is frequently challenging. It is necessary for the clinicians to remember other infections like primary syphilis, deep fungal infections and other noninfectious conditions like squamous cell carcinoma and chronic traumatic ulcer while assessing a chronic indurated ulcer. The diagnosis of the tissues by excisional biopsy and culture of *Mycobacterium tuberculosis* can be done if there is no systemic involvement. Because tubercle bacilli are rare in oral biopsies, demonstrating AFB in biopsy specimens is ineffective. Numerous studies have found acid fast bacilli in just 7.8% of biopsy specimens. As a result, a negative test may not completely rule out tuberculosis. Another cause of anxiety is the emergence of tuberculosis as an AIDS consequence. Atypical non-caseating epithelioid granuloma may be observed on histological examination due to low counts of lymphocytes and an immunodeficient condition. In histology, an immunodeficient patient may not have caseation or granuloma which may make TB diagnosis more challenging. A radiographic evaluation of the chest and a Mantoux skin test are required to rule out systemic TB. Despite being a confirmatory test, an oral lesion biopsy may not be sufficient in most patients because granulomatous alterations may not be visible in early lesions. Repeat biopsies eventually reveal the lesion. A caseating granuloma with associated epithelioid cells and multinucleated giant cells can be used to make a differential diagnosis during histological analysis of biopsied tissue. For the ulcers of the tongue, deeper biopsies may be required because shallow biopsy may not show etiology because of epithelial hyperplasia. FNAC is an extremely sensitive and specific approach for diagnosing tuberculosis of parotid and or other major salivary glands (28).

Prognosis and treatment

An internationally recognized first-line therapy regimen should be given to all current patients. A 2-month course of Rifampicin(R), Ethambutol (E), Isoniazid (H), Pyrazinamide (Z) should be the first line of treatment. Rifampicin, ethambutol, and isoniazid should be administered for a minimum of four months because these 3 drugs comprise the continuous phase. The period of continuous phase can be stretched for four to six months in rare cases like bone and spinal or neural tuberculosis with involvement of nerves. Drug dosages for the patients should be determined by body weight as shown by weight bands. Every batch of the medicine should have its bioavailability guaranteed, especially if it has fixed dose combinations. By purchasing and prescribing from a supplier with a high level of assurance, (FDCs) are used. Every patient should be given a daily regimen and be constantly watched. However, depending on the available resources and practical conditions, the country programme may choose a daily or recurrent procedure for treating tuberculosis because both are effective if all doses are carefully adhered to. Every youngster with tuberculosis who is also HIV-positive should receive their daily regimen while being regularly supervised. Fixed dose combinations (FDCs) of two medications (Isoniazid and rifampicin), three medications (Rifampicin, ethambutol, and isoniazid), and four medications (Rifampicin, isoniazid, ethambutol and pyrazinamide) are recommended (29,30).

Drug resistant TB management

After microbiological confirmation with quality-assured testing, tailored regimens containing second-line anti-tuberculosis drugs are used to treat drug-resistant tuberculosis. The treatment plan for DR-TB

is based on patterns of drug susceptibility that have been microbiologically verified. The second line of treatment should consist of a combination of at least four medications that are susceptible. Above all, an injectable antibiotic like Kanamycin or Amikacin and a second-generation fluoroquinolone like Levofloxacin in high dose should be included in the treatment regimen. If cycloserine cannot be used, it may also contain Ethambutol, Pyrazinamide, Ethionamide, and either PAS (P- aminosalicylic acid) or cycloserine (30).

Treatment of HIV infected TB patients

HIV- TB co-infected patients should be treated like non-HIV- TB patients with the daily regime of anti TB drugs for the same duration. Only difference is that Rifampicin should be replaced with Rifabutin as the latter does not interfere with the bioavailability of boosted protease inhibitors regimen (like Atazanavir/ritonavir, Lopinavir/ ritonavir, Darunavir, ritonavir; reference – NTEP training module 1-4 2020). Regardless of CD4 cell level, antiretroviral therapy should be given to all coinfected patients with HIV and TB as soon as possible (after two weeks) after the start of anti-tuberculosis treatment. Prompt administration of antitubercular therapy followed by antiretroviral therapy is recommended at the earliest. In addition, Co-trimoxazole should also be added as prophylaxis for other opportunistic infections (31).

Modern molecular TB diagnostics have improved the speed and accuracy of TB testing compared to conventional microbiological tests during the past ten years, and emerging technologies look to continue this trend (32,33). NAATs are benefiting clinical practice in several ways. One such advantage is that frequent use of the Gene X-pert test shortens the time it takes to diagnose tuberculosis, enabling treatment to be started the same day rather than a few days later (34,35). Its usage has also enabled a rise in the proportion of patients who begin anti-TB treatment early. Gene X-pert can also be used to find rifampicin resistance.

Precautions for dental health care workers

The spread of several illnesses between patient and the dentist is a risk in clinical dental practice because of closeness of the patient's oral and nasal cavities. As a result, a barrier should be built to make clinical operations safe, preventing infection transmission and the incidence of cross infections. A full TB history will assist the dental practitioner in determining if the person is an active case receiving treatment, an active case not receiving treatment, or formerly infected but currently disease free. Non-treated active cases represent the greatest risk to dental healthcare staff. Dental healthcare personnel are constantly at danger of contracting tuberculosis from spatter, aerosols, or infected blood. Only active tuberculosis patients should receive quick and necessary dental care. It is not possible to determine the individuals who are sick because many severe infections are spread through air, blood or by contact with body fluids. So, it is necessary to prevent contact with blood, body fluids, and mucous membranes by using conventional measures. It is necessary to maintain a high level of disinfection and sterilization of instruments. Aerosol contacts can be avoided by using rubber dams; however, they should not be utilized if coughing is present. Hand washing, equipment worn to minimize exposure to the infections such as gloves, face masks, eye shields, head caps and surgical gowns should be worn, and strict sterilization protocols should be followed. The use of ordinary surgical face masks by dental healthcare professionals is discouraged since they do not offer any protection against the spread of tuberculosis (TB). Fresh mask should be used if it becomes moist at inter-appointments and during treatment of the patients. The equipment such as eyewear or face shields that can be reused should be cleansed and sterilized between patients. Regular cleaning and sterilization of handpieces and other oral equipment is recommended. The goal of the programme is to reduce the occurrence of both health care-associated infections in patients and occupational exposures in dental team members by providing a safe working environment.

CONCLUSION

In countries like India where tuberculosis is endemic, it is critical to know the possibility of Orofacial tuberculosis. A long-term lesion such as swelling, draining sinus or ulcer inside or surrounding the mouth cavity, may alert a doctor to the possibility of tuberculosis. EPTB presents clinically in an uncommon manner. EPTB may not even be on the differential diagnosis list at all, especially if the problem contains strange cryptic spots. Incisional biopsy for handy reachable site and Fine Needle Aspiration Cytology for deep-located lesions may aid in the diagnosis. It is important to start right away with a microbiological culture and histopathological analysis. Like this, TB may be considered when a standard course of treatment fails to improve the lesions. Chemotherapy will treat the lesion if it is discovered early, and mutilating surgery can be avoided. As a result, it is the dentist's responsibility to remember the possibility of tuberculosis (TB) in the suspected lesions of the oral cavity to prevent delay in treatment of the illness.

CONFLICT OF INTEREST

The authors declare no conflicts of interest.

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