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Tuberculosis

Joseph Rinaggio, DDS, MS

Department of Diagnostic Sciences, Room D-860,
University of Medicine and Dentistry of New Jersey–New Jersey Dental School,
110 Bergen Street, Post Office Box 1709, Newark, NJ 07103, USA

Although the impact of tuberculosis (TB) on humans has been most strongly felt in the last 2 centuries, the disease has likely coexisted with society for millennia. The disease seems to have originated in animals (possibly cattle) and crossed into human populations as animal domestication took place alongside the development of agriculture [1,2]. Descriptions of respiratory illnesses with symptoms resembling TB survive from ancient Greece and Assyria, and skeletal evidence of disease sequelae exists from ancient Egypt. With the rise of industrialization in the eighteenth and nineteenth centuries that resulted in urban concentrations of people living in destitution and squalor, TB, or *consumption* as it was commonly termed, became widespread within populations and was a frequent cause of death [2].

Because of many factors—colonialism [2], immigration, and the HIV epidemic among them—TB is now a global disease, with an estimated 1.86 billion people (32% of the world population) infected with the causative agent *Mycobacterium tuberculosis* [3]. In 1997, approximately 7.96 million individuals were diagnosed with TB; the regions with the highest incidence are the Indian subcontinent, Southeast Asia, and Africa [3].

In the United States, the incidence of TB had steadily declined from the mid-1950s to about 1985 [4], whereupon the trend reversed, showing a 20% increase in cases until 1992 [5]. Since then, however, cases have again gradually declined, with a 39% decrease in total cases in 2000 compared with the peak year of 1992 [6]. The increase in TB incidence in this country has been largely attributed to the spread of HIV disease [7,8] and to immigration of individuals from countries with high rates of indigenous infection [8]. The alarming reemergence of the disease led to an increased emphasis on control measures, the identification of infected individuals, and the development of an

E-mail address: rinaggjo@umdnj.edu

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effective treatment strategy coupled with efforts to ensure patient compliance, which has since resulted in an overall gradual decline in cases [6,9].

Despite the decreasing incidence of TB in the United States, health care workers remain at risk for contracting this disease, particularly those who work with the HIV-infected population or in areas with high concentrations of TB cases. The disease may be contracted by the inhalation of infectious respiratory droplets generated by patient activity such as coughing, sneezing, and talking; from aerosols created by the manipulation of the thoracic cavity during surgery or autopsy; or possibly during dental procedures.

Mycobacterium tuberculosis is an aerobic bacillus, approximately 0.5 μm long, [10] first isolated and described by the German bacteriologist Robert Koch in 1882 [2]. Related organisms include *Mycobacterium bovis*, acquired through the ingestion of unpasteurized milk [10], and the *Mycobacterium avium* complex, also known as the *atypical mycobacteria*, which are associated with opportunistic infections in the immunocompromised [11].

Transmission of TB

TB is spread by way of airborne droplets that contain bacilli, typically by coughing, sneezing, or talking [10]. These activities generate up to 3000 droplet nuclei of varying size [10]. Larger droplets settle within close proximity to the infected individual, whereas smaller droplets, with a diameter ranging from 1 μm to 5 μm, drift further away from their source and remaining suspended within the air for a minimum of 0.5 hours [12–14]. The suspension time of smaller droplets increases the likelihood that they will be inhaled, particularly in situations where individuals are living in crowded, poorly ventilated areas where exposure to infectious particles is maximized [10]. Although the individuals most susceptible to infection are those with continued close contact with an infectious source, the disease has been transmitted through brief, casual interaction [15].

The infectious droplets inhaled by a previously unexposed person are first routed through the trachea and primary bronchi where the larger droplets are deposited with little effect [13]. Smaller droplets, which may harbor one to several bacilli [13], are able to travel to the terminal bronchioles and alveoli where they settle, exposing the mycobacteria to host tissues. The tubercle bacilli are then engulfed by pulmonary macrophages, which depending on the quantity and virulence of the mycobacteria, may contain the infection or become overwhelmed by continuing bacillary replication and eventually succumb to the organisms, which are then liberated back into the lung parenchyma [10,16]. Additional macrophages also are recruited to the area: some may destroy those macrophages that contain replicating mycobacteria and others may directly participate in the ingestion and lysis of free mycobacteria [10]. Those macrophages able to control the organisms migrate to the regional lymphatic circulation, after which they may take up residence

within nearby lymph nodes or may disseminate throughout the body. Bacilli may also invade blood vessels, with subsequent systemic distribution [17].

The signature lesion of TB is the tubercle, a granuloma formed by the continuing ingress of macrophages and lymphocytes to the site of the infection [17]. The interaction between the mycobacteria and the host inflammatory cells creates a focal zone of necrosis that over time may undergo fibrosis and calcification [10,17].

The histologic hallmark of TB is granulomatous inflammation, often in the form of caseating granulomas. Granulomas are comprised of focal aggregates of chronic inflammatory cells: lymphocytes, epithelioid macrophages, and multinucleated giant cells of the Langhans type. Tuberculous granulomas are frequently associated with regions of tissue necrosis, termed *caseous necrosis* due to its gross appearance as a white or yellow friable, cheese like substance. The granuloma provides a mechanism of host containment of the mycobacteria, most likely by way of localizing the infection and inhibiting bacterial transit to other host tissues and by helping to prevent unrestrained proliferation through enhanced lymphocyte-macrophage interaction [18]. The granuloma is therefore instrumental in holding the organisms in a condition of latency [19].

Because granulomatous inflammation is not specific for bacterial infection and, in fact, can be seen in a variety of disease processes including foreign body reactions, autoimmune diseases, and fungal infections, histochemical stains have been used to enhance the detection of mycobacteria within tissue sections. The Zeihl-Neelsen stain is commonly used for routine tissue examination. Mycobacteria attain a red-to-magenta color but may still be difficult to identify unless present in clusters.

After the infection is established, symptomatic individuals will initially show pulmonary manifestations of the disease. At its early stages, primary TB is often limited to the periphery of the middle and lower regions of the lung [10,17], although in adults, reactivated disease is most commonly found in the lung apices [20]. The evolution of the pulmonary lesions is often associated with a focal pneumonia, with paratracheal and hilar lymphadenopathy due to lymphatic bacillary spread [10,17], which may cause partial or complete collapse of a lobe—a potentially fatal result [17]. Partial bronchial obstruction can result in emphysema and subsequent bronchiectasis [10,17]. Migration of mycobacteria into the pleural space may cause pleural effusion [10], although this can also be reactive in nature [21]. Fewer than 30% of patients with primary TB will exhibit radiographic abnormalities on chest films, often consisting of enlarged hilar lymph nodes or a well-defined, sometimes calcified zone within the lung itself [17]. Reactivation of the disease in adults, often referred to as *secondary TB*, tends to occur in the lung apices and shows a range of severity [10]. Early symptoms are nonspecific and include fever, malaise, weakness, and night sweats, with the eventual development of cough and dyspnea [10]. The pulmonary disease is spread locally by expulsion of mycobacteria-containing liquid from cavitated

lesions in the lung or lymph nodes to other parts of the lung, which if widespread, may result in tuberculous pneumonia [10,17]. The radiographic findings in secondary TB consist largely of cavitary lung lesions, inflammatory infiltration of the posterior and apical regions of the upper lobes, or sometimes the superior aspect of the lower lobes and, less commonly, pleural effusion and hilar and paratracheal lymphadenopathy [21].

Despite the initial establishment of TB within the lungs, the ability of the mycobacteria to gain access to the circulatory system widens the extent of the disease to encompass multiple organ systems. This multisystem spread often takes the form of gastrointestinal, genitourinary, and musculoskeletal involvement (Table 1), although virtually any tissue or organ system can be affected. The most common expression of extrapulmonary TB is tuberculous lymphadenitis (noted in more than one-fourth of cases), which usually presents as an asymptomatic enlargement of the cervical [10,22] or supraclavicular lymph nodes [10]. The involved lymph nodes may be single or multiple, mobile or nonmobile, and occasionally have draining cutaneous fistulae [10,22].

As previously stated, entrance of tubercle bacilli into the vasculature allows for systemic dissemination of the organisms. After they are distributed, the mycobacteria induce granuloma formation within their

Table 1
Manifestations of extrapulmonary tuberculosis

Organ system	Reference	Manifestation
Gastrointestinal tract	[68]	Pain
		Weight loss
		Mucosal ulceration
		Dysphagia
		Abscess/fistula formation
		Diarrhea
		Lymphadenitis
		Strictures/adhesions
		Bleeding
		Intestinal obstruction
		Peritonitis
Genitourinary tract	[10,69]	Inability/urgency to urinate
		Pain
		Pyuria
		Hematuria
		Mestrual irregularities
		Sterility
		Epididymitis
Musculoskeletal	[10,70,71]	Joint pain/swelling/limitation of movement
		Bone/cartilage destruction
		Ankylosis
		Pott's disease (spinal deformity, pain, limitation of movement)
		Osteomyelitis
		Paraplegia

resident tissues, producing lesions that at the gross and radiologic levels are reminiscent of millet seeds; hence, this type of TB is known as *miliary TB* [10,17]. Most clinical manifestations of miliary TB also tend toward nonspecificity and are similar to those seen in primary or secondary TB [10,17]. There may be additional symptoms, however, depending on the particular organs involved, such as lymphadenopathy, hepatomegaly, and splenomegaly, among others [10]. In the absence of timely and effective treatment, this form of TB also may be fatal [10].

TB of the head and neck

Excluding tuberculous lymphadenitis of the neck, which overall is present in up to 10% of persons suffering from extrapulmonary TB, disease occurrence in the head and neck is rare (seen in only 1% of patients) [11,22]. Between 80% and 90% of individuals presenting with head and neck manifestations of TB exhibit no sign of pulmonary disease [22]. Tuberculous lymphadenitis constitutes a component of head and neck disease in up to 90% of patients and typically presents as single or multiple enlarged lymph nodes that may be firm, fluctuant, or matted with fistula formation [11,22]. Reports differ as to whether there is a predilection for the anterior or posterior triangles [22]. It is important to rule out Hodgkin's lymphoma, which may present with similar regional and systemic symptoms [11]. Laryngeal involvement, rare nowadays, may arise from hematogenous seeding or direct contact with infected sputum [11]. Symptoms of laryngeal TB are nonspecific and consist largely of hoarseness, dysphagia, pain, and cough [11]. The physical signs of laryngeal TB resemble those of chronic laryngitis and squamous cell carcinoma [23]. Less common sites of involvement in the head and neck are the nasal cavity and tonsils where lesions may present as obstructive masses, septal lesions that may sometimes perforate, nasal discharge, or ulcerations [11,22]. Pott's disease may present in the cervical spine, leading to complaints of neck pain and stiffness [11].

Oral manifestations of TB

Oral tubercular infections are quite rare, being present in 0.05% to 5% of patients with TB [24]. Most cases of oral TB result from contact of the oral tissues with infected sputum or hematogenous dissemination in an older individual with pulmonary disease [24–26]. Cases of primary infection arising through direct mucosal invasion by mycobacteria, in contrast, are uncommon and typically seen in young patients who often present with cervical lymphadenopathy with or without cutaneous sinus formation [24,25,27]. The sites demonstrating the most frequent involvement with primary TB are the gingiva, vestibular mucosa, and extraction sockets, although the buccal

mucosa, tongue, palate, and floor of the mouth may be affected [24,25]. Mucosal lacerations, leukoplakia, poor oral hygiene, and dental extractions have been implicated as predisposing an individual to the development of oral TB [25]. Intact and healthy oral mucosa seems to provide a sufficient barrier to mycobacteria, with saliva also helping to control the organisms [24,28]. Secondary oral TB usually appears in the setting of pulmonary disease [25]. Patients may present with odynophagia and fever [25].

Lesions of the oral mucosa typically consist of ulcers, fissures, or swellings [24,25]. Tuberculous oral ulcerations may be solitary or multiple [24,25], occasionally painful [24], and usually involve the dorsum of the tongue [26]. Mature ulcers often have an irregular outline and a rough or granular surface [24–26], with the surrounding mucosa being erythematous and edematous [25]. Caseating necrosis is rare [26]. The physical characteristics of the ulcers are somewhat variable, which contributes to the wide differential diagnosis of these lesions that consists of traumatic and aphthous ulcers, bacterial and fungal ulcerations, sarcoidosis, and malignancies of epithelial, salivary gland, and lymphoid origin, as well as metastases [24,25]. Granulomatous inflammation deep within the tongue can produce macroglossia [29]. Tuberculous osteomyelitis has been reported in the craniofacial skeleton [30] and is associated with mucosal disruption due to tooth eruption or extraction, communication between a tuberculous lesion and the underlying bone, or hematogenous seeding. Lesions may be radiolucent or mixed, with features ranging from horizontal bone loss mimicking periodontal disease to apical radiolucencies simulating pulpoperiapical disease to extensive osteolytic lesions [30]. Mycobacterial infection of the intraglandular or periglandular lymph nodes produces a long-standing diffuse swelling of the parotid gland [31], as well as possible pain, facial nerve dysfunction, and overlying cutaneous erythema [32]. Fistulization of the lymph nodes may cause bacillary infiltration of the gland parenchyma [33]. In addition to TB, other diseases that may cause parotid gland enlargement include, in part, bacterial and viral infections such as actinomycosis and mumps, sarcoidosis, Sjögren's syndrome, salivary gland neoplasms [34], nutritional disorders, heavy metal poisoning, and HIV infection.

TB and HIV infection

The increase in the incidence of TB that occurred in the United States beginning in the late 1980s corresponded to the rise and maturation of the AIDS epidemic [5–7]. It has been estimated that as of 1995, HIV infection contributed to as many as 8800 new cases of TB in this country each year or up to 38% of the total number of annual new diagnoses [35]. HIV infection is considered the most prominent risk factor in acquiring active TB [35]. Worldwide, as of 1997, approximately 8% of persons with TB are coinfected with HIV [3]. Between 3.5% and 16.2% of coinfected individuals will

progress to active TB each year [9]. The development of TB in persons infected with HIV may be the result of newly acquired mycobacterial infection or the reactivation of latent disease [36]. It has been suggested that this susceptibility to mycobacterial infection in patients with HIV is due to a decreased response of a subset of T lymphocytes responsible for the production of interferon-gamma, which has an antimycobacterial action [37,38]. Studies have shown that coinfected patients with active TB and CD4 lymphocyte counts over 500/μL have an increased HIV load [39,40] compared with symptomatic HIV-positive individuals without TB. Additional laboratory studies suggest that *Mycobacterium tuberculosis* infection should be particularly ominous for individuals coinfected with HIV because active TB results in a heightened production of cytokines by macrophages, thereby encouraging viral replication [41]. Examination of the epidemiologic literature regarding *Mycobacterium tuberculosis* and HIV coinfection, however, has not substantiated this contention because it is still unclear whether the increase in mortality seen in coinfected individuals is the direct result of mycobacterial activity on the immune system [37,41]. HIV is also thought to impede the formation and function of the granuloma, allowing the bacilli to avoid localization, escape into the circulation, and reactivate from a latent condition [19]. Indeed, patients with AIDS and TB will show extensive extrapulmonary and miliary spread of the disease [42] and limited granuloma formation [43].

Management of TB

Vaccination

Soon following the identification of the tubercle bacillus, an attempt at vaccine development was made by Koch through the concentration of sterile bacterial products into a solution now known as *old tuberculin*, which underwent administration starting in 1890 and was ultimately discarded due to lack of efficacy [44].

Of the prospective vaccines that have since undergone trial, the bacille Calmette-Guérin (BCG) vaccine has gained widespread acceptance, and has been provided to approximately 85% of the global population [44]. There has, however, been a great deal of controversy regarding its effectiveness, with estimates of protection ranging from 0% to 80%, although a meta-analysis of prospective studies of the BCG vaccine determined a reduced risk of TB infection of approximately 50% [45]. Still, persistent uncertainties regarding its true efficacy have restricted the use of the BCG vaccine in the United States to infants and children likely to be exposed to an infectious case of TB [45] or to health care workers with a high level of exposure to patients with active multidrug-resistant TB [10]. HIV-positive individuals who are given the vaccine will develop TB infection involving multiple organ systems [46].

Testing

A widely used method for identifying individuals with previous exposure to *Mycobacterium tuberculosis* is through the subdermal injection of five tuberculin units of purified protein derivative (PPD) [10], known as the *tuberculin skin test* [9]. This test is recommended for health care workers, immunocompromised individuals, people at high risk for exposure to active TB, and recent immigrants from countries with high rates of infection [9]. This is typically introduced to the volar surface of the forearm (Mantoux method), with the patient returning in 48 to 72 hours to examine the injection site and measure the diameter of any resultant induration [9,10]. The amount of induration indicating infection ranges from 5 mm to 15 mm and varies according to the status of the patient's immune system, presence of clinical or radiographic signs of infection, and general risk for acquiring the disease [9]. There are some diagnostic pitfalls with tuberculin skin testing, however. False-positive results can emerge in people who have been previously immunized with the BCG vaccine, and false-negative results may be obtained in immunocompromised patients [9] and patients with sarcoidosis, measles, or Hodgkin's disease [47]. Also, patients with long-standing latent TB infection may not react with an initial tuberculin test; however, this will cause immune stimulation that will result in a positive reaction with a subsequent test, simulating a true conversion. To prevent confusion about time of exposure, people who are required to have periodic tuberculin testing (eg, health care workers), should initially undergo a two-step test in which an additional tuberculin test is administered 2 weeks after the first. Reaction to the second test indicates exposure at a much earlier date [9,10].

Laboratory determination of *Mycobacterium tuberculosis* infection may be accomplished through histochemistry, mycobacterial culture, or the detection and amplification of bacterial DNA. The simplest method, as previously mentioned, is by searching for acid-fast bacilli in tissue sections or sputum smears. In addition to being a sometimes difficult and time-consuming procedure, sensitivity is low and false-positive results may be obtained in patients who have clinical symptoms similar to TB but are, in fact, infected with *Mycobacterium avium*–complex bacilli [48]. The most specific method of diagnosis is through mycobacterial culture, which takes up to 8 weeks on conventional solid media [48]. Although inconvenient for obtaining a timely diagnosis, this technique is valuable in determining drug sensitivity [10]. The problem of obtaining a timely diagnosis can be circumvented by using rapid culture systems, which can shorten the time required for diagnosis to 2 to 3 weeks when growth in liquid media is combined with DNA amplification techniques [48].

Treatment

Central to the treatment of TB are antimicrobial drugs. These drugs are divided into first-line and second-line agents, based on bacteriocidal efficacy

and degree of toxicity. First-line agents, consisting of isoniazid, rifampin, pyrazinamide, ethambutol, and streptomycin, are preferred for their effectiveness and patient acceptability [9,10] and, therefore, are the most widely used. Chemotherapy for TB is a two-stage process, beginning with an initial stage of bacterial eradication, followed by a second stage devoted to the elimination of more resistant organisms [10]. The therapeutic regimen, which may be used in children and adults, typically consists of an 8-week course of isoniazid, rifampin, and pyrazinamide, followed by an additional 16 weeks of isoniazid and rifampin only [9,10]. Ethambutol or streptomycin are often included until drug susceptibility is confirmed [9]. The regimen may be modified on the basis of patient adherence, tolerance, the presence of drug interactions, and the nature of subsequent sputum cultures [9,49]. A patient is considered cured when three consecutive negative sputum cultures for *Mycobacterium tuberculosis* are obtained in persons who tested positive before the initiation of therapy [50]. The management of latent TB consists of a 9-month course of isoniazid [9]. Despite a full course of antibiotics, treatment is unsuccessful in as many as 5% of patients [51].

Treatment failure is recognized if patient sputum continues to culture *Mycobacterium tuberculosis* after 3 months of antimicrobial therapy [9] or acid-fast bacillus staining of sputum smears detects mycobacteria after 5 months of therapy [10]. The inability to successfully treat the disease is usually attributed to poor patient compliance [10], but other factors such as poor patient management by health care providers, malabsorption, and drug resistance may also contribute [51]. To counteract issues adversely affecting patient compliance, a method to help ensure patient adherence, designated *directly observed therapy*, has been developed [20]. This strategy is as simple as observing patients taking their medication and will sometimes incorporate incentives such as food, money, and clothing to encourage compliance [52]. Although an effective directly observed therapy program that ensures patient compliance is likely to improve control of the disease, its success has been difficult to quantify [51]. Initial infection with a strain of *Mycobacterium tuberculosis* resistant to antibiotics, poor patient compliance, poor medical management, and an inadequate health care infrastructure have been implicated in the rise in cases of drug-resistant TB [10,53]. Drug-resistant TB has become increasingly problematic in some parts of the world [53], particularly in eastern Europe, China, and Iran. Relatively low rates of drug resistance are currently found in the Americas and some African countries, likely due to effective treatment programs, the prescribing of single-pill antibiotic combinations [10], or unavailability of adequate medical treatment and antitubercular drugs [53]. Approximately 3.2% of newly diagnosed cases of TB worldwide are thought to represent multidrug-resistant TB in which treatment with isoniazid and rifampin (and possibly additional antibiotics) is unable to control the disease [54]. Multidrug-resistant TB likely occurs from a new infection with a previously resistant organism or from an established infection that becomes resistant through

inadequate drug formulations or poor therapeutic compliance [55]. Drug-resistant TB can be overcome by extending the duration of chemotherapy, the inclusion of additional antibiotics to which the organism is susceptible, and close patient surveillance [9,10,20].

Surgery is an infrequent component in the management of TB but may be indicated in cases of bronchial or tracheal compression due to mediastinal tuberculous lymphadenitis, bronchiectasis, severe lung destruction, and the presence of resectable pulmonary neoplasms [56].

Management of the dental patient with TB

Despite the currently declining incidence of TB in the United States, health care workers including dentists and their staff still remain at high risk for contracting the disease. The nature of the dental setting—close patient contact, the generation of potentially infectious spray through routine operative procedures, and the fact that many of the sequelae of this disease occur in the head and neck—places the dental office in a unique position of acting as a possible source of disease transmission, identification, and control. The role of the dentist in TB management is to determine the level of risk of transmission in his or her practice, to educate office staff about TB prevention and recognition of the symptoms and oral manifestations of the disease, to protect the staff and other patients from becoming infected should an individual with active TB be present, and to refer patients suspected of active TB infection for appropriate medical treatment (Box 1).

The Centers for Disease Control assigns the possibility of transmission of *Mycobacterium tuberculosis* in a dental setting according to a five-stage risk hierarchy. This hierarchy ranges from minimal risk to high risk and is based, in part, on incidence of TB infection in the surrounding community, the number of dental patients with active TB treated over the previous year, and any evidence of TB transmission within the practice (Table 2) [57]. The determination of risk for the dental facility should be performed by the dentist or the member of the office staff who oversees infection control procedures [58]. Independent of the risk category of a given practice, all dentists and staff should receive annual PPD testing.

A comprehensive medical history with detailed descriptions of past systemic illnesses and relevant social factors should be recorded for all individuals seeking dental treatment. Special attention should be given to any indication by the patient of a previous abnormal chest radiograph, persistent cough, or positive PPD test. Patients should be asked whether they have ever received a diagnosis of TB [58]. Country of origin, place of residence, and BCG vaccination status should also be noted. It is important to differentiate between individuals who are infected with *Mycobacterium tuberculosis* and those who are infectious. Infected but noncontagious people will demonstrate a positive PPD test and may report a previous physical and radiographic work-up for the disease and antitubercular

Box 1. Tuberculosis: dental management considerations

Patients with active tuberculosis
- Urgent dental treatment only
  - Engineering controls and personal respiratory protection needed

Patients with signs or symptoms suggestive of tuberculosis
- Urgent dental treatment only
  - Refer patient to physician for evaluation
  - Engineering controls and personal respiratory protection needed until active tuberculosis has been ruled out

Patients with a history of tuberculosis
- Routine dental treatment appropriate after it is established that patient has been adequately treated and followed and there are no signs and symptoms of active disease
  - Establish history of treatment and medical follow-up
  - Postpone routine treatment and consult with physician if adequacy of treatment or follow-up evaluation is questionable or if signs and symptoms of active disease are present

Patients with a positive tuberculin skin test with no history of tuberculosis and no signs or symptoms of active disease
- Routine dental treatment appropriate after it is established that the patient does not have active disease
  - Establish history of medical evaluation and prophylactic therapy
  - Consult with physician if there is any question of the presence of active disease

From Phelan JA, Jiminez V, Tompkins DC. Tuberculosis. Dent Clin N Am 1996;40:327–41; with permission.

chemotherapy [59]. Signs of active pulmonary TB should be absent. Such patients are eligible for routine dental treatment [59]. Infectious patients are those with active pulmonary and laryngeal disease [60] who will typically present with cough, hemoptysis, and fever, and who report fatigue, anorexia, weight loss, and night sweats [10,47].

Because the transmission of TB is dependent on the release of mycobacteria-laden respiratory droplets, people working within enclosed settings such as dental offices are theoretically at an increased risk for infection. The intimacy of the dental operatory coupled with the dispersion of oral and respiratory fluid that routinely occurs during dental procedures suggests

Table 2
Risk categories for the transmission of tuberculosis (TB) in a health care facility

Risk category	Description
Minimal risk	Located in a community with no reported TB cases within the previous year Patients with active TB are not admitted
Very low risk	Known cases of TB within the community Screening for active TB is a component of the medical assessment prior to examination and treatment Patients with active TB are isolated from inpatient areas Potential cases of active TB are referred to an associated facility for treatment
Low risk	No detection of intrafacility transmission of TB Less than six patients with active TB examined or treated per year
Intermediate risk	No detection of intrafacility transmission of TB Six or more patients with active TB examined or treated per year
High risk	Detection of intrafacility TB transmission Documented cluster of purified protein derivative conversions (two or more conversions in a span of 3 mo)

that transmission of the disease would be commonplace in this setting. So far, however, there has been no documented instance of TB infection passing from patient to dental practitioner, although there have been cases of patients acquiring the disease from their dentist. A dentist in the United Kingdom with active pulmonary TB was found to have infected 15 of his patients for whom he had extracted teeth [61]. It was presumed that the infection was established through the entrance of bacilli through the mucosal breach at the extraction site. Because at the time, gloves and masks were rarely used during routine dental procedures, it was speculated that the bacilli were transferred to the patients by aerosolized respiratory droplets or through direct contact with the dentist's ungloved hands when the socket was compressed. A possible case of multidrug-resistant TB transmission between two HIV-positive dental workers at a hospital dental clinic has also been reported [62].

Despite the lack of verifiable cases of patient–to–dental worker TB transmission in the literature, the documentation of TB spread in other health care settings [63–65] necessitates that measures be undertaken to protect all individuals in the facility who may be potentially exposed. Any elective dental procedures on a patient with established or suspected active TB should be delayed until the individual can be treated and subsequently proved non-infectious [47,58,62]. Patients who are suspected of having active pulmonary TB should spend as little time in the facility as possible [55]. To reduce the

potential exposure of uninfected individuals, care should be taken to prevent the release of respiratory droplets, which can be accomplished through the use of barrier techniques. If the patient in question is coughing or sneezing, he or she may be asked to wear a mask [55]. If the patient presents with emergent dental needs, a rubber dam, when applicable, should be employed. It is preferable that a low-speed handpiece be used for operative procedures, with minimal air syringe use [55]. If the use of a high-speed handpiece is unavoidable, any water spray generated should be minimized and suctioned properly.

Over the past 2 decades, the use of masks has become universal in dental practice. Unfortunately, standard half-face disposable surgical masks do not adequately adapt to facial contour and offer little protection from inhaling the minute droplet nuclei that transmit mycobacteria through the resultant gaps [66]. Masks that are effective in blocking tubercle bacilli tend to be large, uncomfortable, and expensive. Also, the greater resistance when drawing air through these masks may be especially taxing to those individuals who suffer from respiratory or cardiovascular diseases [13,66].

Protection against inhaling aerosolized infectious droplets may also be gained by renewing the air within the office through ventilation combined with high-efficiency particle air filtration [13]. It is recommended that ventilation systems completely renew the air in a room six times per hour. Despite this substantial air flow, it does not guarantee the prevention of TB transmission [13]. Also, these units tend to be costly to purchase, install, and maintain. An additional engineering control used to decrease the risk of TB infection is the installation of ultraviolet germicidal irradiation on the upper wall surface in examination rooms and operatories. Such units are relatively inexpensive to purchase and maintain, are effective in killing airborne bacteria, and are safe to patients and staff, provided they are properly installed [13].

Anyone suspected of having TB should be immediately referred to a physician for evaluation and a chest radiograph. People with extrapulmonary TB without respiratory or oral signs and symptoms are not at risk for spreading the disease [58]. Because interruptions in the oral mucosa can serve as sites of direct inoculation with tubercle bacilli, any sharp cusps, ridges, restorations, or denture wire should be smoothed or blunted and any parafunctional habits should be discouraged [47].

The dentist should be aware of potential drug interactions and adverse effects [67] when managing dental patients undergoing antitubercular therapy. Rifampin and isoniazid are both associated with hepatotoxicity, which may increase in severity when the drugs are taken together. Acetominophen should be avoided due to the potential for liver damage and its diminished effect when used in conjunction with rifampin. At high doses, rifampin is associated with flulike illness, leukopenia, and thrombocytopenia, increasing the likelihood of infections, excessive bleeding, shortness of breath, skin rash, and a brown-to-orange discoloration of urine and saliva. Rifampin will increase the rate of metabolism of anticonvulsants, antifungals

(fluconazole, ketoconozole, and itraconazole), anticoagulants, corticosteroids, and clarithromycin. Isoniazid may cause gastrointestinal disturbances, lymphadenopathy, skin rash, peripheral neuropathy, and a potentially fatal nonviral hepatitis. Simultaneous corticosteroid use can decrease the serum concentration of isoniazid. Reduced metabolism of anticonvulsants, benzodiazepines, ketoconazole, and warfarin may occur. Pyrazinamide is hepatotoxic at high doses. It may also induce gastrointestinal disturbances, thrombocytopenia, arthralgia, and myalgia. Ethambutol therapy is associated with visual and gastrointestinal disturbances, skin rash, headache, arthralgia, peripheral neuropathy, and fever. The use of acetylsalicylic acid and muscle relaxants is discouraged in those individuals taking streptomycin due to a heightened risk of ototoxicity [59] and respiratory paralysis [67], respectively. Streptomycin is also known to cause facial paresthesia, pancytopenia, and nausea, vomiting, and vertigo due to vestibulotoxicity.

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