Oral tuberculosis in an asymptomatic pulmonary tuberculosis

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Tuberculosis (TB) has been a worldwide health problem for centuries. It most commonly affects the lungs, but rare oral manifestations of TB have been reported. The aim of this paper is to present a case of painful erythematous lesion of oral cavity in undiagnosed asymptomatic pulmonary tuberculosis. This case is unusual in that the appearance of the painful oral lesion caused the patient to seek professional care and was concurrent with quiescent pulmonary disease. (Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2011;111:e8-e10)

Tuberculosis (TB) is an infectious disease caused by bacteria, usually Mycobacterium tuberculosis. The disease is transmitted by inhalation of infective droplets that have been expelled into the air by a person with active TB. Less commonly, tuberculosis is caused by exposure to M. bovis through ingestion of unpasteurized infected cow milk. TB most commonly affects the lungs, but it may involve any organ of the body, including the mouth and oropharynx.1,2

A case of secondary tuberculous lesion in the oral cavity is presented here in an undiagnosed asymptomatic pulmonary tuberculosis patient. This case is unusual in that: 1) the appearance of the painful oral lesion caused the patient to seek professional care that led to the diagnosis of his asymptomatic pulmonary disease; 2) the lesion involved the posterior part of the oral mucosa, i.e., the posterior alveolus and junction of hard and soft palate, in contrast to earlier reports that most TB lesions have been located in the anterior portion of the oral cavity, involving the tongue, buccal mucosa, vestibular area near the corner of the mouth, or lower lip2-5; and 3) clinically it presented as a granular erythematous patch although most of oral tuberculous lesions are ulcerative.

CASE REPORT

A 70-year-old male patient came to the Department of Oral Pathology and Microbiology, Aurangabad, with the complaint of painful erythematous lesion over the upper right alveolus for 2 months. He also complained of difficulty in deglutition which caused him to seek professional care. His personal history revealed a habit of smoking 25 bidis per day for 30 years. A thorough medical history was taken that did not reveal any evidence of weight loss, chills, night sweat, or cough.

The intraoral examination revealed an erythematous lesion with granular surface (Fig. 1) involving the maxillary right alveolus and posterior palate. The lesion was 4×3 cm, irregular in shape, and firm in consistency. Extraoral examination revealed no evidence of lymph node involvement. A posteroanterior Water radiograph was taken which showed no evidence of bone involvement. His blood biochemistry was within normal limits, and serologic test for human immunodeficiency virus was nonreactive. The lesion was suspicious, and with a possible diagnosis of a premalignant lesion, a biopsy was advised.

Histopathologic examination of the incisional biopsy showed widespread coalesced tuberculous granulomas (Fig. 2) composed of epitheloid cells, lymphocytes, caseation necrosis, and multinucleated Langhan giant cells. In these multinucleated Langhan giant cells, the nuclei were arranged (Fig. 3) at the periphery of the cell in a horseshoe or ring shape and clustering at the 2 poles. This typical picture was suggestive of the diagnosis of tuberculosis, sarcoidosis, or fungal infection. Periodic acid–Schiff (PAS) and Ziehl-Nielsen (ZN) staining was performed. The PAS staining was negative for fungal hyphae, whereas the ZN staining showed acid-fast bacilli and confirmed the diagnosis of tuberculosis. However, it was unclear whether the lesion was primary or secondary. There was no history of TB, nor were there signs or symptoms present indicative of a pulmonary infection. A sputum examination was performed that was negative, and a chest radiograph was

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taken. That film (Fig. 4) showed calcified Koch calcification in the apical region of the upper right lung along with emphysematous changes.

The overall radiologic and histologic picture confirmed the diagnosis of quiescent primary pulmonary tuberculosis with secondary tuberculous lesion involving the oral mucosa. The patient was referred to the Department of Medicine for further management, where he was immediately started on isoniazid (300 mg), rifampin (600 mg), and pyrazinamide (1,500 mg) for 2 months. His condition improved rapidly, and within 3 weeks the oral lesion resolved (Fig. 5). After 2 months, the patient was asked to discontinue pyrazinamide and to continue with the rest of medicines for the next 4 months. Follow-up after that time showed complete resolution of the oral lesion.

**DISCUSSION**

Tuberculosis is an infectious disease caused by bacteria, usually *M. tuberculosis*. The disease is transmitted by inhalation of infective droplets that have been expelled into the air by a person with active TB. These initial infections causes small parenchymatous lesions
in the lungs. The primary lesions may heal by fibrosis and calcification. However, even after clinical healing, the bacilli may remain dormant for several years and may reactivate at an opportune moment. Therefore, secondary lesions can develop by reactivation of dormant primary complex or reinfection by a fresh dose of the tubercle bacilli.

Although oral manifestations of TB have been reported, they are rare, affecting ~0.05%-5.00% of infected patients. It has been proposed that the bacilli may reach the oral mucosa by hematogenous or lymphatic spread. Additionally, direct inoculation has been reported. Small tears in the mucosa because of chronic irritation or inflammation favor localization of the organisms, even with hematogenous spread.

In the present case report, the pulmonary focus was calcified and the sputum examination was negative, indicating quiescent primary lesion. The oral mucosal lesion presented with multinucleated Langhan giant cells and caseous necrosis along with the positive ZN staining for acid-fast bacilli. Therefore, this lesion may have resulted from reactivation of dormant primary bacteria that lodged in oral mucosa from either infected sputum or the hematogenous route. The patient had a history of exfoliation of his posterior teeth, and that inflamed and irritated mucosa could have favored the localization of the organisms, leading to the secondary mucosal lesion from quiescent primary pulmonary focus.

This is particularly interesting when one considers that in pulmonary tuberculosis, sputum laden with tubercle bacilli constantly bathe the oral mucosa. Yet active lesions of tuberculosis in the mouth are unusual. It is believed that intact squamous epithelium of the oral mucosa serves as a barrier to the penetration of TB bacilli along with the mechanical cleansing action of saliva and food. Also, the oral cavity possesses natural resistance to infection owing to the presence of salivary enzymes, circulating antibodies, and epithelial and connective tissue architecture which provides protective covering. It has also been proposed that *M. tuberculosis* has a strong predilection for invading only 1 tissue or organ in a given person.

In the present case, an immediate antitubercular therapeutic regimen was administered for 2 months, including isoniazid, rifampin, and pyrazinamide. Within 1 month, the patient reported with lesion regression. Only isoniazid and rifampin were continued for the next 4 months. No separate drug regimen was prescribed for the pulmonary focus, because it appeared to be quiescent. Follow-up at 6 months showed complete resolution of the oral lesion and symptoms, suggesting a successful outcome.

**CONCLUSIONS**

The identification of a tuberculous lesion in any location in the mouth is an unusual finding, and its discovery is usually indicative of underlying pulmonary disease. Oral tuberculous lesions may be either primary or secondary, and a secondary oral mucosal lesion can be the result of reactivation of dormant primary bacteria or reinfection from a fresh dose of tubercle bacilli. Therefore, even in the absence of any systemic signs and symptoms, in all cases of oral tuberculosis a search for the primary site of the disease should be considered to render optimal patient care.

**REFERENCES**


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