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CASE REPORT

Primary tuberculosis of the oral cavity

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Tuberculosis is one of the major causes of ill health and death worldwide. Nevertheless, tuberculous lesions of the oral cavity are rare and can be a diagnostic challenge, particularly in young immunocompetent patients. Most of the cases are secondary to pulmonary disease and the primary form is uncommon. In this paper, we present a case of primary oral tuberculosis, affecting the floor of mouth in a 13-year-old Brazilian male patient.
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Introduction

Tuberculosis is a re-emerging infectious granulomatous disease caused mainly by *Mycobacterium tuberculosis*, an acid-fast bacillus that is transmitted primarily via the respiratory route. Less frequently, tuberculosis may also be caused by other two species of bacteria, *M. bovis* and *M. africanum* (Samaranayake, 2002). According to the World Health Organization, tuberculosis is responsible for death of approximately 2 million people each year and it is estimated that between 2002 and 2020, approximately 1 billion people will be newly infected, over 150 million people will get sick, and 36 million will die because of tuberculosis.

Tuberculosis has a definitive affinity for the lungs causing primary disease. However, any part of the body can be affected, including the mouth and normally these lesions are secondary to lung disease (de Aguiar *et al*, 1997).

Reports have shown that oral lesions occur in 0.05–5% of the patients with tuberculosis and frequently are secondary affecting more usually elderly patients. On the other hand, the primary form is uncommon and more usually affects young patients (Mignogna *et al*, 2000).

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The aim of this paper was to report a case of primary oral tuberculosis in a 13-year-old patient affecting the floor of mouth.

Case report

A 13-year-old white male was referred to the Oral Diagnosis Clinic (Orocentro), Piracicaba Dental School, University of Campinas, São Paulo, Brazil, for evaluation. His main complaint was a painless oral ulcer in anterior region on the floor of mouth that he noticed 2 months before. According to him, the lesion had increased recently causing halitosis. The medical history was not significant for any serious disease. Extra-oral examination did not show any alteration.

Oral examination revealed a 2.5 × 1.5 cm ulcer on left anterior region of the floor of the mouth. The ulcer had irregular borders and the base was covered by a grayish-white slough (Figure 1).

Non-specific ulcer, bacterial, fungal and viral infections were included in the differential diagnosis. Incisional biopsy of the affected mucosa was performed and histopathological examination showed granulomatous inflammation containing Langhans-type giant cells, raising the possibility of granulomatous infection, including tuberculous or fungal infection, or a diagnosis of sarcoid (Figure 2a,b). Ziehl-Nielsen staining demonstrated acid-fast bacilli (Figure 3) and the definitive diagnose of oral tuberculosis was established. The patient was referred for a complete medical examination. Chest X-ray was taken without revealing abnormal findings (Figure 4) and primary oral tuberculosis had been diagnosed.

Anti-tuberculosis therapeutic regimen consisting of isoniazid, pyrazinamide and rifampicin was used and maintained for 6 months. One month after beginning of the treatment the lesion healed and showed complete resolution. The patient is in follow-up for 3 years without showing any recurrence (Figure 5).

Discussion

Tuberculous lesions of the mouth may be either primary or secondary to pulmonary tuberculosis with secondary lesions being more common (Eng *et al*, 1996). Eng *et al*

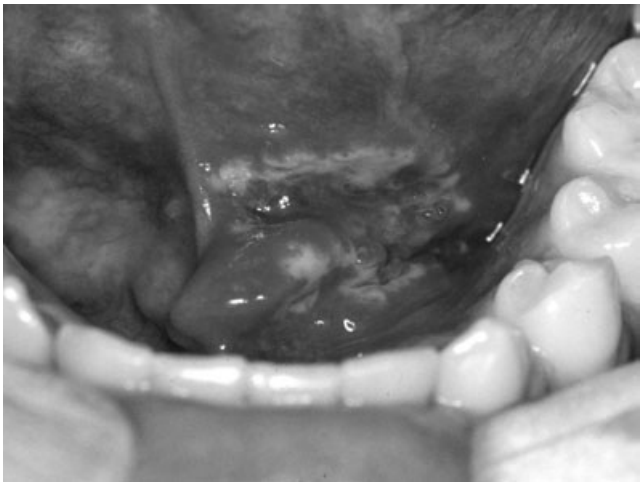

Figure 1 Clinical view showing an erythematous ulcer with undermined edges covered by a grayish-white slough on the floor of the mouth

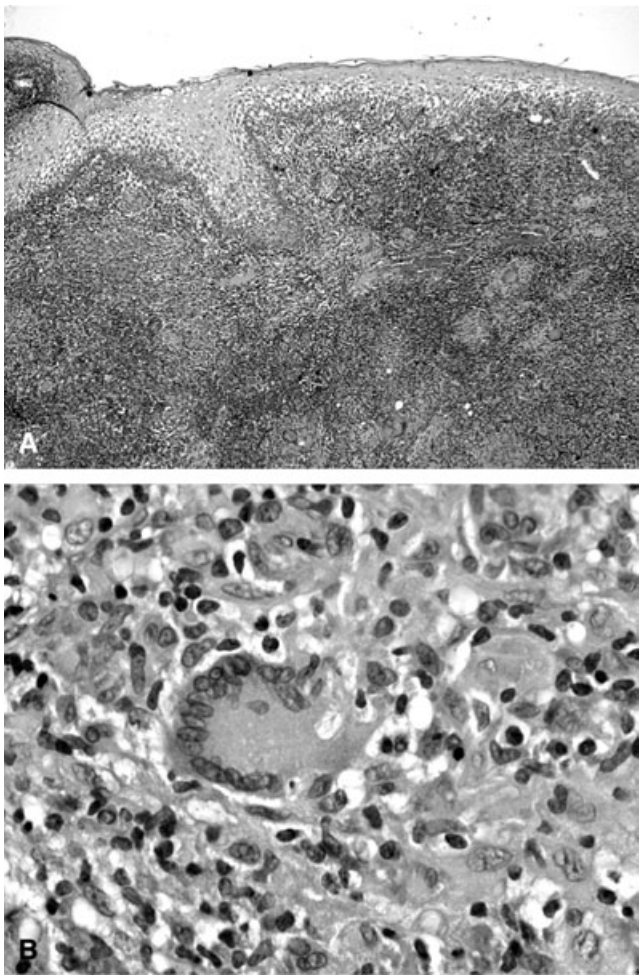

Figure 2 Histopathological examination showing: (A) oral mucosa with intense granulomatous inflammation (H&E, ×50), (B) numerous epithelioid cells and a Langhans giant cell (H&E, ×400)

(1996) demonstrated radiographic evidence of pulmonary tuberculosis in 93.3% of the patients with oral tuberculosis. In the case that we presented, no evidence

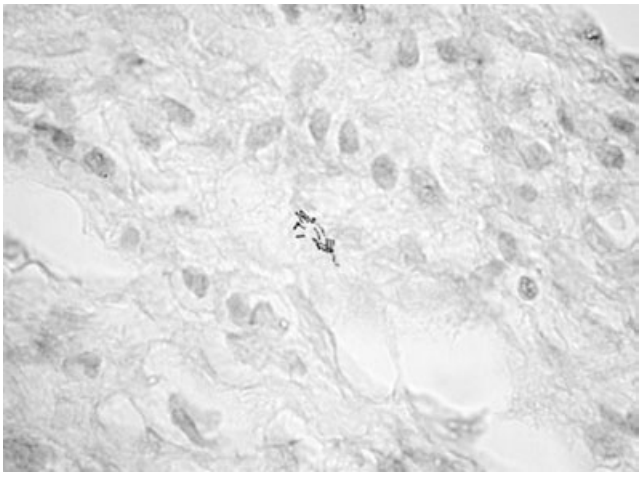

Figure 3 Ziehl–Nielsen staining demonstrating acid-fast bacilli (original magnification: ×1000)

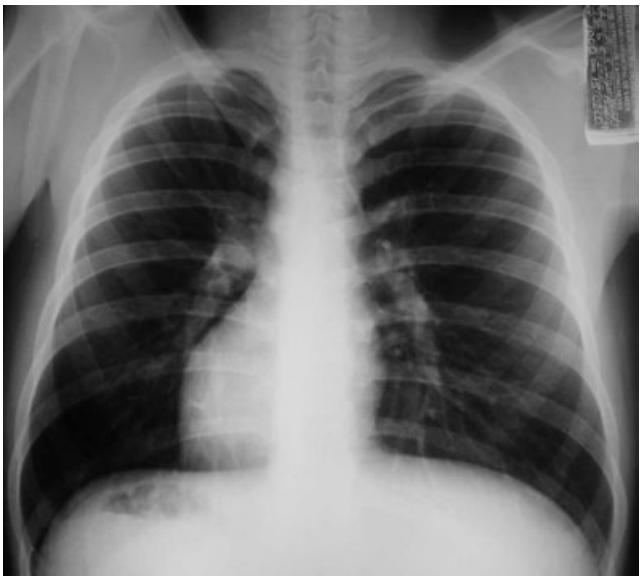

Figure 4 Chest radiograph without abnormal findings

of lung or other systemic involvement was found, supporting the diagnosis of primary oral tuberculosis.

Primary form of tuberculous oral lesions is more commonly found in children and adolescents than in adults, usually affecting the gingiva and mucobuccal folds (Mignogna *et al*, 2000). An inflammatory focus adjacent to teeth or teeth extraction sites has also been reported (Eng *et al*, 1996; Rivera *et al*, 2003). In addition, primary lesions are often associated with enlarged cervical lymph nodes (Eng *et al*, 1996; Mignogna *et al*, 2000). The secondary form is more frequent in middle-aged and older persons and involves mainly the tongue and the hard palate (Rauch and Friedman, 1978).

The mechanism of primary inoculation is unknown. However, it is thought that the mycobacterium is inoculated directly into the oral mucosa (Rauch and

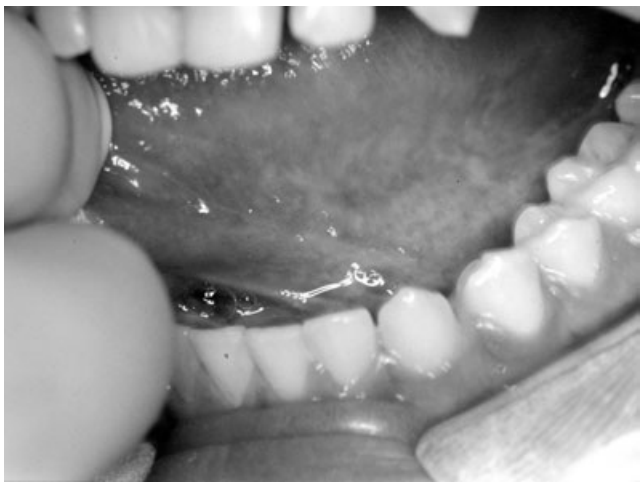

Figure 5 Clinical view 3 years after treatment

Friedman, 1978). The intact oral mucous membrane presents a natural resistance to mycobacterium invasion. This resistance has been attributed to the cleansing action of saliva, the presence of salivary enzymes, tissue antibodies, oral saprophytes, and the thickness of the protective epithelial covering. Any break or loss of this natural barrier, which may be result of trauma, inflammatory conditions, tooth extraction, or poor oral hygiene, may provide a route of entry for the mycobacterium (Iype *et al*, 2001).

Although the clinical picture is variable, oral lesions typically consist of a stellate ulcer with undermined edges and a granulating floor (von Arx and Husain, 2001). Nodules, fissures, tuberculomas or granulomas also can be found. The lesions may be single or multiple and painful or painless (Mignogna *et al*, 2000). Skin, cervical lymph nodes, and salivary glands are also frequently involved (Zheng and Zhang, 1995).

Clinical diagnosis can be difficult because tuberculosis can mimic a variety of other conditions, including malignancy, traumatic and aphthous ulcers, syphilis, sarcoidosis, and deep mycotic infection such as paracoccidiodomycosis and histoplasmosis (Mignogna *et al*, 2000). In the present case, a possible fungal etiology was strongly considered, given the high prevalence of paracoccidiodomycosis and other fungal infections in Brazil.

For confirmation and differential diagnosis, Mantoux reaction, biopsy for histologic examination, acid-fast stains, and culture should be obtained (Mignogna *et al*, 2000). Positive tuberculin skin test just indicates previous exposure to the *M. tuberculosis*. Although presumptive diagnosis of tuberculosis can be based on histopathological examination and identification of the bacilli in tissues using special stains, because of the relative scarcity of the bacilli within tissue, mycobacteria can be demonstrated by means of special stains only in 27–60% of cases (Rivera *et al*, 2003). Culture of microorganisms have shown good results, although it has technical difficulties, lack sensitivity and may last 4–6 weeks (Mignogna *et al*, 2000; Rivera *et al*, 2003). Sophisticated techniques such as PCR can be used alternatively, especially when the conventional methods of diagnosis render equivocal results (Goel *et al*, 2001; Vargas *et al*, 2001). If a tubercular lesion is suspected, a chest X-ray is indicated to investigate the possibility of pulmonary involvement, and if confirmed, should be followed by systemic examination to detect tubercular lesions in other areas, such as urinary tract and bone (MacFarlane and Samaranayake, 1989; Mignogna *et al*, 2000).

von Arx and Husain (2001) described granulomas with sarcoid-like and numerous giant cells, some of the Langhans type. They raised the possibility of granulomatous infection, including tuberculous or fungal infection, or a diagnosis of sarcoid. Subsequent stains for fungi (PAS and Grocott Silver) and bacteria (Gram stain) were negative. However several acid-fast bacilli were identified with a Ziehl–Nielsen stain situated within the granulomas and considered that the infection was consistent with tuberculous granulomatous infection. For our case the histopathological examination showed granulomatous inflammation containing Langhans-type giant cells and Ziehl–Nielsen stain was positive for bacilli.

Before the tuberculin testing of dairy herds, many cases arose from the consumption of milk infected with *M. bovis*. However, in those areas of the world where unpasteurised milk is consumed, bovine tubercle bacilli often cause human infection (Pande *et al*, 1995). Our patient lived on a farm where the consumption of unboiled or raw milk is common and, presumably he had consumed infected milk.

Chemotherapy if given regularly is effective but must be given for long periods. Agents most commonly used in triple therapy include rifampicin in combination with isoniazid and pyrazinamide, usually for the first 2 months of treatment. Ethanbutol can be added as a fourth drug when isoniazid resistance is considered likely. Continuation therapy with the two drugs rifampicin and isoniazid is usually given for the further 4 months, so that a total of 6 months therapy is given (Samaranayake, 2002). In the case that we presented the patient was treated with isoniazid, pyrazinamide and rifampicin for 6 months.

In conclusion, although tuberculosis of the oral cavity is relatively rare, with the increasing incidence of tuberculosis, it must be considered in the differential diagnosis of atypical ulcerative lesions of the mouth.

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