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Case Report

Perforation of the hard palate due to tuberculosis

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ABSTRACT

Tuberculosis (TB) of the hard palate is rare and usually secondary to pulmonary TB. We present a case of a 7-year-old boy who presented with difficulty in swallowing solid foods, low-grade fever and loss of weight. Oral cavity examination showed perforation of the hard palate with an irregular, undermined margin and a necrotic base. Chest X-ray showed homogeneous opacity in the right upper zone. Fine-needle aspiration of the cervical lymph nodes showed granular caseous necrosis and granuloma composed of epitheliod cells and histiocytes. In view of the clinical presentation and evidence of pulmonary and lymph node TB, the palatal perforation was most likely due to TB. Patient was started on antituberculous therapy and is on regular follow-up.

Key Words: Hard palate, perforation, pulmonary tuberculosis, tuberculosis

INTRODUCTION

Tuberculosis (TB) is a chronic granulomatous disease caused by *Mycobacterium tuberculosis*. The World Health Organisation estimates that each year more than 8 million new cases of TB occur and approximately 3 million people die of the disease worldwide.[1] India accounts for nearly one-fifth of the global burden of TB. Recently, the increase in the incidence of drug resistant TB has further worsened the problem. Oral TB is rare and accounts for less than 1% of all cases of TB.[2] With the increasing number of TB cases, unusual forms of the disease in the oral cavity are more likely to occur and be misdiagnosed. We herein present a rare case of palatal perforation secondary to TB.

CASE REPORT

A 7-year-old boy presented with difficulty in swallowing solid foods, low-grade fever and loss of weight for 11/2 month. His mother was on antituberculous therapy for pulmonary TB. There was no history of cough, abdominal pain, vomiting, diarrhea, or urinary complaints. On admission, his heart rate was 104/min, the respiratory rate was 28/min and blood pressure was 98/60 mmHg. His weight was 13 kg, and height was 104 cm (both below the fifth percentile for age and sex). Mild pallor was present. Oral cavity examination showed perforation of the hard palate, 3 × 3 cm with an irregular, undermined margin, and a necrotic base [Figure 1]. Bilateral matted cervical lymphadenopathy was present. Respiratory system examination showed crepitations on the right side anteriorly. Rest of the systemic examinations were normal. The Mantoux test was positive (28 mm). Investigations showed: Hemoglobin 8.8 g/dL, total leucocyte count 9000/cumm (neutrophils 40%, lymphocytes 60%), erythrocyte sedimentation rate 80 mm at the end of 1 h. His liver and renal functions tests were normal. Serology for human immunodeficiency virus was negative. His blood culture and urine culture did not show any growth. Computed tomography scan of the oral cavity showed erosion and scalloping of the posterior part of the hard palate on the right-side [Figure 2]. Chest X-ray showed homogeneous opacity in the upper-right zone. Gastric lavage for acid fast bacilli was positive. High power view of papanicolou stain of fine-needle aspiration of the lymph nodes showed granular caseous necrosis and granuloma

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composed of epitheliod cells and histiocytes [Figure 3]. Patient's relatives did not give consent for biopsy of the palatal lesion. However, in view of the clinical presentation and evidence of pulmonary and lymph node TB, the palatal perforation was most likely due to TB. He was started on antituberculous therapy (isoniazid, rifampicin, pyrazinamide, and ethambutol) and is on regular follow-up with the oral surgeons for repair of the palatal perforation.

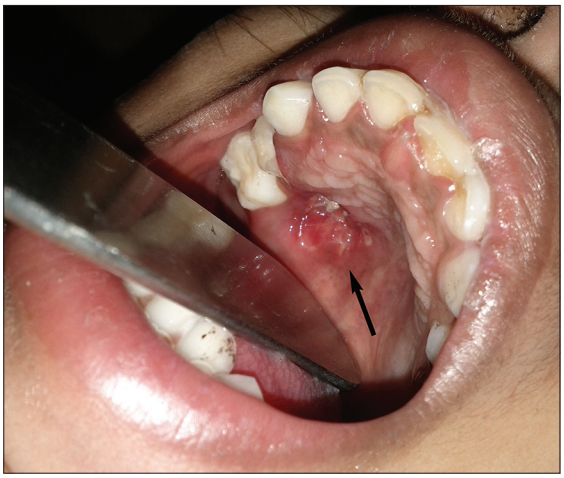

Figure 1: Oral cavity examination showing perforation of the hard palate, 3 × 3 cm in size with an irregular, undermined margin, and a necrotic base

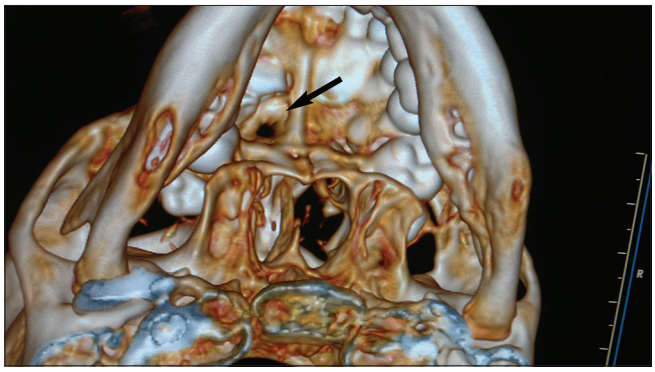

Figure 2: Plain axial computed tomography scan of midface with 3D reconstruction showing erosion and scalloping of the posterior part of the hard palate on the right side

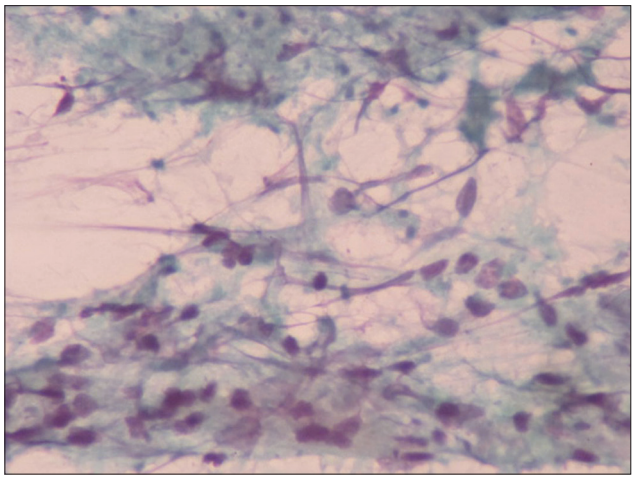

Figure 3: High power view of papanicolaou stain of fine needle aspiration of the cervical lymph nodes showing granular caseous necrosis and granuloma composed of epithelioid cells and histiocytes

DISCUSSION

Oral TB may either be primary, or more often, secondary to pulmonary TB.[2] In secondary oral TB, the bacilli reach the oral mucosa by hematogenous or lymphatic spread. In primary oral TB there is direct inoculation of the mycobacterium due to break or loss of the natural barrier resulting from trauma, inflammatory conditions, leukoplakia, tooth extraction, or poor oral hygiene.[2,3] Other local predisposing factors include dental cysts, periapical granulomas, dental abscess, periodontitis, and jaw fractures.[2-4] Abbot *et al.*[5] were able to isolate the tubercle bacilli from mouth washings of 44.9% of the patients with active pulmonary lesions and thus highlighting the importance of an intact mucosal epithelium in resisting oral TB infection. The systemic factors that favor the chances of oral infection in TB includes lowered host resistance and increased virulence of the organisms.[4] The prevalence of oral manifestations in pulmonary TB ranges from 0.8 to 3.5%.[4] Tuberculous involvement of oral cavity is rarely seen even in populations with high incidence of the pulmonary disease.[2] Factors that attribute to relative resistance of oral cavity to TB are protective effect of saliva, presence of saprophytes, resistance of striated muscles to bacterial invasion, and thickness of protective epithelial covering.[2] The most common site for oral TB is the tongue. Other sites include the soft palate, hard palate, lip, cheek, tonsils, gingiva, floor of mouth, uvula, and alveolar mucosa.[2] Table 1 highlights some of the reported cases of oral TB in the literature.[2,3,6-9] Oral TB lesions usually manifest as single, non-healing ulcers with an indurated, irregular, undermined margin, and a necrotic base. Oral lesions can also present as nodules, fissures, plaques, vesicles, tuberculomas, or granulomas.[6] The palatal lesion of TB may be seen as granulomas, ulceration, or perforation and are usually more common in the hard palate than in the soft palate.[9] According to Baruah, *et al.*[10] palatal involvement of TB resulting in perforation is usually seen in patients with strong immune responses and considered to be due to hypersensitivity to acid-fast bacilli that causes tissue destruction.

Table 1: Reported cases of oral tuberculosis

Site of tuberculosis	Authors
Gingival tuberculosis	Rodrigues, *et al.*[6]
Buccal mucosal tuberculosis	Nanda, *et al.*[3]
Tuberculous glossitis	Gupta, *et al.*[7]
Tuberculosis of soft palate	Gupta, *et al.*[8]
Tuberculosis of lip	Dixit, *et al.*[2]
Tuberculosis of hard palate	Suhail, *et al.*[9]
Tuberculosis of oral buccal mucosa	Nanda, *et al.*[3]

In addition to TB, the causes of palatal perforation include infection (syphilis, leprosy, leishmaniasis, or fungal infection), Wegener's granulomatosis, sarcoidosis, neoplasms (salivary or squamous cell), drug abuse (cocaine), and midline lethal granuloma.[10] In our case, the above differentials were unlikely in view of the clinical presentation, evidence of pulmonary and lymph node TB and the clinical improvement seen with antituberculous therapy on follow-up. The treatment of palatal TB should follow the general guidelines established for the treatment of extrapulmonary TB.[10]

CONCLUSION

TB of the palate is relatively rare and should be included in the differential diagnosis of palatal perforation. Also, a search for the primary site should be done extensively in all the cases of palatal TB.

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