CASE REPORT

Primary tuberculosis of palate

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Abstract

Primary tuberculosis (TB) of the hard palate is very rare. A 74-year-old man was presented with 6-month history of dysphagia along with an irregular mass in the hard and soft palate. Magnetic resonance imaging (MRI) revealed thickened and increased signal intensity within hard and soft palate. Tissue biopsy showed focal caseating granulomatous-like lesion and the histochemical staining using Ziehl–Neelsen stain for acid-fast bacilli was positive. Positive histochemical studies provided evidences that the hard palate mass was most likely due to TB. Thus, the patient was started on antituberculous therapy.

Introduction

Tuberculosis (TB) is a life-threatening infectious disease with a high world incidence. It is mainly caused by *Mycobacterium tuberculosis* strain of acid- and alcohol-fast bacilli of Mycobacteriaceae family and relatively less by *Mycobacterium bovis*, *Mycobacterium microti* and *Mycobacterium africanum*.[1]

Pulmonary TB is the most common form of the disease. However, it can also occur in the lymph nodes, meninges, kidneys, bone, skin and oral cavity.[2] Both primary and secondary types of TB can cause lesion in the oral cavity. In secondary TB, lesions of the oral cavity may accompany lesions in the pharynx, lungs, lymph nodes and skin.[3]

TB with oral expression is considered rare. When oral lesions of TB are the sole manifestations of the disease, the clinician may face a diagnostic challenge. The importance of recognising this entity lies in its early diagnosis and treatment, as it can be easily confused with neoplastic or traumatic ulcers.

Case report

A 74-year-old man presented to us with a history of odynophagia along with dysphagia to solid food for 6 months. He had a history of lesion on the hard palate. It initially started like an ulcer and gradually increased in size. There was intermittent fever, loss of appetite and weight. He denied night sweats. He was a chronic smoker (50 pack years), there was no history of TB contact and on examination, BCG scar was absent. Intraorally, his oral hygiene was graded as poor with moderate deposits of stains throughout the dentition. Soft tissue examination revealed an irregular mass occupying the hard and soft palate crossing midline. On palpation, the mass was tender with indurated margin and it bled on touch. The mass was extending into the left alveolar ridge, left trigone and uvula (Figure 1). The right alveolar ridge and trigone were not involved and examination of the tongue and floor of the mouth were unremarkable. Correlating these features of a chronic ulcerative lesion of 6 months duration with an associated history of smoking and advanced age, a differential diagnosis of malignant neoplasm like squamous cell carcinoma and lymphoma, TB and mycotic ulcer was arrived at. Flexible nasopharyngolaryngoscopy of the nose and larynx were unremarkable. MRI of the neck and thorax revealed thickened and increased signal intensity within hard and soft palate with no evidence of cervical lymphadenopathy, focal lung nodule or metastases. Summary of blood investigations on admission of the patient is shown in Table 1.

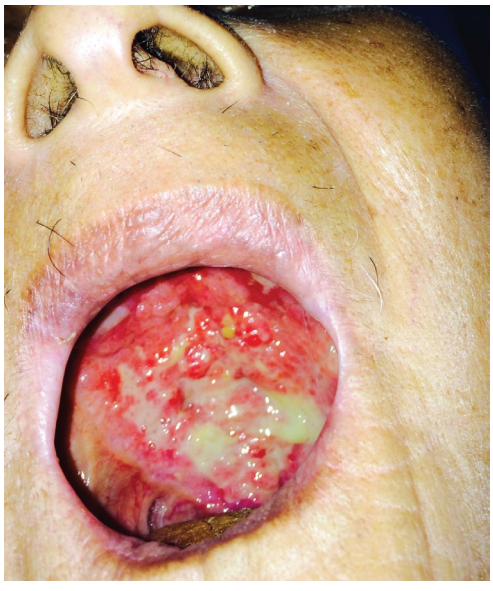

Figure 1. Oral cavity examination showing an irregular ulcerating lesion occupying the hard and soft palate crossing the midline with a sloughy surface

Table 1. Summary of blood investigations on admission

Investigations	
Haematology	
1. Full Blood Count • Total white blood cells count •Haemoglobin •Platelets	 4.5 x 10^3/UL (4.0 - 10.0) 9.7 g/L (13.0 - 17.0) 175 x 10^3/UL (150 - 400)
Biochemistry	
2. Renal profile • Sodium • Potassium • Urea • Creatinine	 137 mmol/L (136 - 145) 3.6 mmol/L (3.5 - 5.1) 8.8 mmol/L (2.76 - 8.07) 202 mmol/L (62 - 106)
3. Liver function test • Total protein • Albumin • Globulin • Total bilirubin • Alanine aminotransferase • Alkaline phosphatase	 66 g/L (66–87) 23 g/L (35–52) 42.8 g/L (20.0–36.0) 0.5 umol/L (0.8–2.0) 26 U/L (<41) 155 U/L (40–130)
Others	
Erythrocyte sedimentation rate (ESR)	63 mm/h (3–10)
Mantoux test	Negative
Human immunodeficiency virus serology	Negative

Biopsy was done from the lesion in the hard palate under topical anaesthesia. Histopathological examination showed benign ulcer edge with focal caseating granulomatous-like lesion formed by the epithelioid histiocytes, lymphocytes and scanty multinucleated giant cells of foreign body type of the hard palate and this was suggestive of tuberculosis (Figure 2).

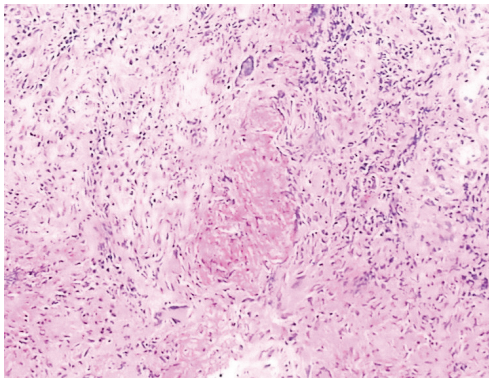

Figure 2. Focal caseating granulomatous-like lesion formed by the epithelioid histiocytes, lymphocytes and scanty multinucleated giant cells (10 × 10)

Histochemical staining using Ziehl–Neelsen stain for acid-fast bacilli was positive whereas periodic acid–Schiff stain and fungal body cultures were negative (Figure 3). There was no evidence of malignancy.

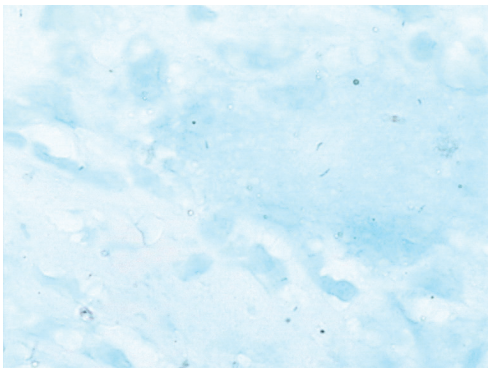

Figure 3. A multinucleated cell with engulfed bacilli (100 × 10)

In accordance with the existing guidelines, the patient was administered with antitubercular medication.

Discussion

Although TB has a definite affinity for the lungs, it can also affect any part of the body including the oral cavity. Oral manifestation of TB can be primary or secondary.[4] However, oral TB is rarely seen even in populations with high incidence of pulmonary disease.[4] Farber et al. reported that less than 0.1% of TB patients whom they had examined, exhibited oral lesions.[5] Few factors that attribute to relative resistance of oral cavity TB

are protection by the oral saliva, presence of saprophytes, resistance of the striated muscles to bacterial invasion and the thickness of the protective epithelial covering.[4]

The most common site for oral TB is the tongue. Other sites include the soft palate, hard palate, lip, cheek, tonsils, gingiva, floor of the mouth, uvula, and alveolar mucosa.[4] The pathogenesis of primary TB and secondary TB is different. In primary TB, there is direct inoculation of the mycobacterium due to loss of natural barrier resulting from inflammatory conditions, leukoplakia, tooth extraction, trauma and poor oral hygiene. However, in secondary TB, the bacilli reach the oral mucosa by haematogenous or lymphatic drainage.[4,6] Other predisposing factors include dental cyst, periapical granulomas, dental abscess, periodontitis and jaw fractures.[4,5-7]

Commonly, the manifestation of oral TB is an ulcerative lesion of the mucosa. The lesion may be preceded by an opalescent vesicle or a nodule that may break down as a result of caseation necrosis to form an ulcer. A typical TB lesion appears with ragged undermined edges, minimal induration and often with a yellowish apple jelly like granular base.[8] This lesion may ulcerate leaving radiating scars, which are quite characteristic.

In this case, no such gross pathology was observed. Instead, an irregular lesion with much indurated margin was seen. With no background history of TB contact and normal body parts, diagnosis was quite difficult. Interestingly, TB was diagnosed from the findings of tissue biopsy. We learned that in oral lesion, isolated TB diagnosis should be in list even though primary TB is extremely rare. Biopsy is an ultimate tool in the diagnosis of TB.

Unfortunately, this patient developed pancytopenia 2 months after treatment, cause of which unknown and thus a bone marrow aspirate and trephine biopsy (BMAT) was offered; however patient refused further diagnostic work-up and was subsequently lost to follow up.

Conclusion

Tuberculosis lesions of oral cavity can present with non-specific clinical appearance and it can mimic a malignant neoplasm. Primary lesions of TB manifest in the oral cavity as non-healing chronic ulcers. Hence, when diagnosing such lesions with non-healing tendencies, TB should be considered as differential diagnosis. A complete physical examination with diagnostic test, such as chest radiograph and tissue biopsy for histological studies must be included. An early diagnosis with prompt treatment will usually result in a complete cure.

How does this paper make a difference to general practice?

- TB with oral expression is extremely rare and when it is the sole manifestation, clinician may face a diagnosis challenge.
- Primary TB palate should be included in the differential diagnosis of chronic non-healing ulcer.
- Tissue biopsy is a must in all cases of chronic non-healing ulcer to obtain a diagnosis. In view of limited resources available in general practice setting, an early referral to tertiary center is recommended.
- TB shall not be excluded even if all other TB investigations were negative.
- TB is a curable disease; an early diagnosis is essential for prompt treatment.

References

1. Raviglione MC, O'Brien RJ, et al. Tuberculosis. *Harrison's Principles of Internal Medicine*, 15th edition. McGraw Hill;2001:1024–34.
2. Topazian RG, Goldberg MH. *Oral and Maxillofacial Infections*, 2nd edition. WB Saunders Co.;1987:413.
3. Prabbu SR, Wilson DF, Daftary DK, et al. *Oral Diseases in the Tropics*. Oxford University Press;1993.
4. Dixit R, Sharma S, Nuwal P. Tuberculosis of oral cavity. *Indian Journal Tuberculosis* 2008;55:51–3.
5. Farber JE, Friedland E, Jacobs WE. Tuberculosis of the tongue. *Am Rev Tuber*,766;1940–42.
6. Nanda KD, Mehta A, Marwaha M, et al. A disguised tuberculosis in oral buccal mucosa. *Dent Res J* (Isfahan). 2011;8:154–9.
7. Ebenezer J, Samuel R, Mathew GC, et al. Primary oral tuberculosis: Report of two cases. *Indian J Dent Res*. 2006;17:41–4.
8. Brennan TF, Vrabec DP. Tuberculosis of the oral mucosa – report of a case. *Ann Oto Rhinol Laryngol*. 1970;80:601–5.

CASE REPORT

Wellen's syndrome: Challenges in diagnosis

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Abstract

Wellen's syndrome is a pre-infarction stage of coronary artery disease characterised by predefined clinical and electrocardiographic (ECG) criteria of a subgroup of patients with myocardial ischaemia. Early recognition and appropriate intervention of this syndrome carry significant diagnostic and prognostic value. We report this unusual syndrome in an elderly man who presented with recurrent angina and characteristic ECG changes as T-waves inversion in the precordial leads, especially in V2–V6 during pain-free periods and ECG obtained during episodes of pain demonstrating upright T-waves with possible elevated ST segments from V1–V4.

Cardiac enzymes were positive and coronary angiography revealed critical stenosis in the proximal left anterior descending artery. It is important to timely identify this condition and intervene appropriately as these patients may develop extensive myocardial infarction that carries a significant morbidity and mortality.

Introduction

Wellen's syndrome was first postulated by de Zwaan et al. in 1982.[1] It is characterised as a disease state in which a patient with angina demonstrates typical electrocardiographic pattern of T-wave changes associated with critical stenosis (>90%) of proximal left anterior descending (LAD) coronary artery.[2] Discovering Wellen's syndrome is imperative, as these patients are at greater risk of developing anterior wall infarction within few weeks unless intervention is undertaken urgently.[3]

Case Summary

A 60-year-old elderly man with no previous comorbidities presented to our casualty with complaints of recurrent bouts of retrosternal chest pain, radiating to the left arm, which was mostly present on exertion and subsided on rest. It occurred mostly at morning and sometime in night during sleep. Each episode lasted for 15–25 min. Associated symptoms included profuse diaphoresis, dizziness, shortness of breath and palpitations. Patient had previous episodes of chest pain on exertion, which he overlooked. He had 27-pack year smoking history. He denied any illicit drug use including cocaine. On admission physical examination, patient was afebrile, his pulse rate was 90 beats per minute, blood pressure was 140/100 mmHg, respiratory rate was 18 breaths per minute and saturation on room air (SpO_2) was 97%. Systemic examination was unremarkable.

Basic blood parameters (complete blood cell count, electrolytes, liver and renal functions) and fasting lipid profile were normal. Initial electrocardiogram (ECG) at the time of admission revealed symmetrical and deeply inverted T-waves in precordial leads, especially in V2–V6 during pain-free periods (Figure 1A) and ECG obtained during episodes of pain that occurred after 24 h of admission; demonstrated sharpened upright T-waves with elevated ST segments from V1–V4 (Figure 1B). Cardiac biomarkers CPK-MB was 28 IU/L (normal range: 0–25 IU/L), Troponin T was 0.021 μg/L (normal range: 0.00–0.014 μg/L) and serum blood glucose level was 6.5 mmol/L. Transthoracic echocardiography showed that LAD territory was hypokinetic with moderate left ventricular systolic dysfunction and left ventricular ejection fraction (LVEF) of 40%. The patient was initially managed on anti-platelet, anti-thrombotic (subcutaneous low-molecular weight heparin), nitrates and 3-hydroxy-3-methylglutaryl-coenzyme A (HMG-CoA) reductase inhibitors (statins). A coronary angiogram (CAG) showed critical stenosis (90%) due to a thrombus in the proximal left anterior descending artery (Figure 2).

As the patient had recurrent bouts of retrosternal chest, characteristic precordial T-wave changes and critical stenosis of proximal LAD on CAG, we labeled him as having Wellen's syndrome. He was counseled for revascularisation procedure but he refused to do the same.