CASE REPORT

Primary oral tuberculosis

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SUMMARY

Persistent oral ulcers and erosions can be the final common manifestation, sometimes clinically distinguishable, of a diverse spectrum of conditions ranging from traumatic lesions, infectious diseases, systemic and local immune-mediated lesions up to neoplasms. A case with oral tuberculosis and absence of any systemic manifestations is reported. The location and clinical presentation of the lesion is unusual and underlines the importance of considering tuberculosis in the differential diagnosis of oral lesions that affect the mucosa and the gingiva.

BACKGROUND

Tuberculosis (TB) has been a worldwide health problem for centuries. The primary form of disease is most often localised to the lung. In a minority of patients, progressive pulmonary disease spreads to other organ systems through self-inoculation via infected sputum, blood and lymphatic system, establishing the secondary form of TB.¹ It is a chronic infectious granulomatous disease caused by bacteria, transmitted through the respiratory tract by inhaling droplets contaminated by *Mycobacterium tuberculosis*, which is an acid-fast bacillus.² Less frequently, TB is caused by ingesting unpasteurised cow's milk that is infected by *Mycobacterium bovis* or by other atypical mycobacteria.¹

Oral manifestation of TB is rare with an incidence of 1.4% of total TB cases. Floor of the mouth, soft palate, gingiva, lips and hard palate can be involved; however, the tongue tip, dorsum, lateral borders, and base and palate are the most common sites of involvement for oral lesions.^{2,3}

Each year, about 8 million people develop TB, 2 million people have a latent form of the disease and 3 million people die from TB.² According to the literature oral lesions are seen in 0.05–5% of the patients with TB and chiefly affects the pulmonary system but it can also involve extrapulmonary sites including the head and neck region.² Oral lesions may be primary or secondary. In primary disease, the mouth can be the initial site of infection which is rare and is more likely to occur in younger rather than older adults. Secondary orofacial TB arises subsequent to TB from another site and thus may reflect oral inoculation with sputum or haematogenous spread of mycobacteria and usually occurs in all age groups.^{1,4,5}

We report a case of primary oral TB with two intraoral non-healing ulcers present in a 34-year-old individual without any systemic manifestations.

CASE PRESENTATION

A 34-year-old man was referred to the department of oral medicine and radiology for assessment of a non-healing oral ulcer on his left inner commissure and left labial vestibule. His initial clinical symptom was a burning sensation that started after a minor trauma from a toothbrush and cheek biting. The lesions have been first noticed 2 months back and remained unhealed even after conservative treatment. The patient had difficulty in eating and brushing. There was no history of cough, fever, haemoptysis and weight loss. He did not have any systemic symptoms, was not on any medications and had no history of allergy. General physical examination did not reveal any abnormality. The patient was a chronic smoker and occasionally consumed alcohol. There was no lymph nodal involvement.

Oral examination revealed two painful non-healing ulcers on left inner commissure and left labial vestibule in relation to 33 regions measuring 3 cm×2 cm and 3 cm×1 cm, respectively. The ulcers were roughly oval in shape with irregular crescentic borders. The edges were thin and undermined with slight induration at the base and tenderness was elicited on palpation (figure 1A,B). The radiographic study of that area did not reveal any abnormality. An incisional biopsy was performed under local anaesthesia. Histological examination of the excised specimen showed granulomatous inflammation containing Langhans' giant cells, epithelioid cells and lymphocytic infiltrate with areas of caseous necrosis (figures 2 and 3). On smear examination of the ulcer, several acid-fast bacilli were identified using a Ziehl-Neelsen stain (figure 4). The investigations were consistent with the diagnosis of TB. Blood tests were within normal limits except a raised erythrocyte sedimentation rate that was 35 mm in the first hour. ELISA for TB was positive. In order to rule out systemic involvement, chest radiograph was advised, which revealed absence of foci of infection (figure 5). Thus, the diagnosis of primary TB was confirmed.

INVESTIGATIONS

- Histological examination (granulomatous inflammation containing Langhans' giant cells, epithelioid cells and lymphocytic infiltrate with areas of caseous necrosis).
- Ziehl-Neelsen stain (identification of acid-fast bacilli).
- Blood test (raised erythrocyte sedimentation rate and positive ELISA for TB).

DIFFERENTIAL DIAGNOSIS

The differential diagnosis of a tuberculous ulcer of the oral cavity includes aphthous ulcers, traumatic

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Figure 1 Photograph showing the course of the tubercular lesions in a 34-year-old man. (A, B) Pretreatment photographs showing the lesions on the left inner commissure and left labial vestibule. (C, D) Photographs showing the regression of the lesions 1 week after the initiation of antitubercular treatment. (E, F) Photographs showing the regressed lesions 3 weeks after initiation of antitubercular treatment.

ulcers, syphilitic ulcers and malignancy, including primary squamous cell carcinoma, lymphoma and metastases. It is most likely that TB is only considered when the histological specimen reveals a granulomatous lesion. This would then lead to consideration of other oro-facial granulomatous conditions such as sarcoid, Crohn's disease, the deep mycoses, cat-scratch disease, foreign-body reactions, tertiary syphilis and Melkersson-Rosenthal syndrome.¹⁻⁵

TREATMENT

Antitubercular treatment regimen was initiated. According to the protocol used, the first phase of treatment included isoniazid

300 mg, rifampin 600 mg, pyrazinamide 1500 mg and ethambutol 800 mg daily for 2 months. The second phase of treatment consisted of isoniazid 300 mg and rifampin 600 mg daily for 4 months. Two weeks after initiation of the first phase of treatment tissue regeneration was evident in the area of the lesion (figure 1C,D) and in the following 2 weeks the lesion had almost resolved (figure 1E,F).

OUTCOME AND FOLLOW-UP

Lesions resolved after 2 weeks of antitubercular treatment (figure 1).

DISCUSSION

TB is a well-known entity encountered in our practices. On a national level, the incidence of TB is at an all-time low. Between 1993 and 2003, the incidence of TB has decreased by 44%, and it is currently at a historic low. These current statistics indicate the success of early TB management.¹ TB had resurfaced in

Figure 2 Photomicrograph showing granulomatous inflammation with Langhan's giant cells and focal caseous necrosis in H&E stain (original magnification $\times 10$).

Figure 3 Photomicrograph showing Langhan's giant cells in H&E stain (original magnification $\times 40$).

Figure 4 Photomicrograph showing acid-fast bacillus by Ziehl-Neelsen stain (original magnification $\times 10$).

inner-city due to the HIV epidemic and growing indigent population.^{1 4}

Transmission is through air droplets (ie, coughing, sneezing, speaking and even singing). These particles are 1–5 μm in diameter and contain *M tuberculosis*. A single cough can generate 3000 infective droplets. Fewer than 10 mycobacterial bacilli may initiate a pulmonary infection.¹

Four factors contribute to the likelihood of transmission: (1) number of organisms expelled, (2) concentration of organisms, (3) length of time of exposure breathing in contaminated air and (4) immune status of the exposed individual. Those who are immunocompromised, such as patients with HIV, are more likely to develop active TB. The organisms grow for 2–12 weeks until they reach 1000–10 000 in number, which is sufficient to elicit a cellular immune response.¹

Classic symptoms are often absent particularly in patients who are immunocompromised or the elderly. Up to 20% of patients with active TB may be asymptomatic. Classic features

Figure 5 Chest radiograph showing absence of foci of infection.

associated with active TB are cough, weight loss/anorexia, fever, night sweats, haemoptysis and lymphadenopathy. Signs and symptoms of extrapulmonary TB may be non-specific. They can include leucocytosis, anaemia and hyponatraemia due to the release of antidiuretic hormone release from affected lung tissue.^{1 2 5} All the above classical features were absent in our patient.

Although TB has a definite affinity for lungs, it can affect any part of the body including the mouth.⁶ The unusual presentation of the disease in the oral cavity are more likely to be misdiagnosed. The clinicians should be alert of this possibility and consider TB in the differential diagnosis of atypical lesions of the oral cavity, including those appearing as non-healing ulcers.^{3 5 6}

Oral manifestations of TB are quiet rare due to inability of *M tuberculosis* to invade the intact mucosa of oral cavity or pharynx. Cleansing action of saliva, the presence of salivary enzymes, tissue antibodies, oral saprophytes and the thickness of the protective epithelial covering have been proposed as the underlying mechanism. Any break or loss of these natural barriers, which may be the result of trauma, inflammatory conditions, tooth extraction or poor oral hygiene, may provide a route of entry for the mycobacterium in the primary forms of the disease while for the secondary form the pulmonary bacilli are transmitted to secondary lesions via the lymphatic and haematogenous route.⁷

Primary oral TB is observed more widely in children and adolescents. Its manifestations usually involves the gingiva, mucobuccal folds, inflammatory foci adjacent to teeth or extraction sites and are seen as superficial ulcers, patches, indurated soft-tissue lesions, nodules, fissures, plaques, granulomas, verrucous proliferations or even lesions within the jaw that may be in the form of TB osteomyelitis or simple bony radiolucencies.^{5 6} Our case presented as an indurated ulcerative lesion which was tender on palpation.

Secondary oral TB may occur in all age groups; however, middle-aged and older people are more likely to have oral manifestations of the disease, which are almost always painful. The most frequently occurring lesion is an ulcer, characterised by irregular edges with minimal induration. The base of an ulcer may be granular or covered with pseudomembrane. The dorsal surface of the tongue is affected most commonly, followed by the palate, buccal mucosa and lips. The salivary glands, tonsils and uvula also are involved frequently. Secondary lesions of the mandibular ridge (alveolar mucosa) are extremely rare.^{5 7 8}

The standard short course treatment for TB is isoniazid (along with pyridoxal phosphate to obviate peripheral neuropathy caused by isoniazid), rifampicin, pyrazinamide and ethambutol for 2 months, then isoniazid and rifampicin alone for a further 4 months. The patient is considered cured at 6 months (although there is still a relapse rate of 2–3%).⁹ For latent TB, the standard treatment is 6–9 months of isoniazid alone. If the organism is known to be fully sensitive, then treatment is with isoniazid, rifampicin and pyrazinamide for 2 months, followed by isoniazid and rifampicin for 4 months. Ethambutol need not be used. When situations like resistance to first-line therapy, extensively drug-resistant TB or multidrug-resistant TB arise, the second-line drugs are implemented for the treatment. There are six classes of second-line drugs which include aminoglycosides (amikacin, kanamycin), polypeptides (capreomycin, viomycin, enviomycin), fluoroquinolones (ciprofloxacin, levofloxacin, moxifloxacin), thioamides (ethionamide, prothionamide), cycloserine (closerin) and terizidone.¹⁰ A patient with multidrug resistant TB, who remains culture positive after many months of treatment, may be referred for lobectomy or pneumonectomy with the aim of

cutting out the infected tissue. Some complications of treated TB like recurrent haemoptysis, destroyed or bronchiectatic lungs and empyema are also amenable to surgical therapy.¹¹

Learning points

- ▶ Oral lesions may be primary or secondary. In primary disease, the mouth is the initial site of infection whereas secondary orofacial TB arises subsequent to TB from another site and thus may reflect oral inoculation with sputum or haematogenous spread of mycobacteria.
- ▶ Our case is unusual in that, the appearance of painful oral lesions caused the patient to seek the professional care that led to the diagnosis of tuberculosis.
- ▶ Clinicians should be aware of this rare entity which presents with oral lesions without any diagnostic sign and symptoms of systemic disease.

Contributors RPSM: material collection and manuscript preparation. SV: material collection and manuscript preparation. US: material collection and manuscript preparation. NA: material collection and manuscript preparation.

Competing interests None.

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