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Case Report

An unusual focus of extra pulmonary tuberculosis

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ABSTRACT

Extrapulmonary tuberculosis (EPTB) is an uncommon form of chronic infection, which does not present with the typical signs and symptoms of pulmonary tuberculosis. Most extrapulmonary forms of tuberculosis affect organs with suboptimal conditions for bacillary growth. Therefore, extrapulmonary tuberculosis generally has an insidious presentation, a slow evolution, and paucibacillary lesions and/or fluids. Access to the lesions through secretions and body fluids is not always possible; therefore, invasive techniques may be necessary to obtain material for diagnostic investigation. Oral tuberculosis lesions are infrequent and their occurrence is usually secondary to pulmonary involvement. Here is an unusual presentation of oral TB which was unraveled incidentally during a routine dental extraction.

Keywords: Extra pulmonary tuberculosis, Primary oral TB, Secondary oral TB, Dental pulp, Granuloma, Giant cells

INTRODUCTION

Orofacial tuberculosis is an uncommon form and presents at different sites such as the mandible (alveolar and basal bone); head; face and neck lymph nodes; salivary glands; maxilla and maxillary antrum; and soft tissues such as the gingiva, tongue, muscles of mastication, and buccal mucosa.[1]

TB also can infect the mandible secondarily by means of bacterial entry into the bone through an exposed dental pulp or a fresh extraction site.[2]

The dental pulp is very well protected in the centre of the tooth. Molecular techniques such as "suicide PCR" and Multiple Spacer Typing have identified and characterized micro-organisms in the ancient samples. The detection of bacterial DNA from ancient dental pulp provide the scientific evidences for diagnosis of the infectious diseases of the past.[3]

Mycobacterium tuberculosis and Mycobacterium leprae have also been detected in ancient teeth recovered from a 1st century tomb in Jerusalem suggesting the co-infection of these two mycobacteria in ancient human populations.[3]

CASE REPORT

A 35 year old female patient reported to the dental OPD OF HAH Centenary Hospital with the chief complaint of pain in relation to upper left back tooth region for 15 days. Patient had been on analgesics and antibiotics for the same. The patient's history and general examination

was non-contributory. No other systemic findings could be elicited from the history.

Patient presented with facial asymmetry with diffuse swelling over the left perioral region (Figure 1).

Figure 1: Bird's eye view showing diffuse swelling on the left side of face.

Intraoral examination revealed a firm mass extending from the upper left second premolar region to the canine region, this extended into vestibule onto the labial mucosa. The upper left second premolar was in a grossly decayed condition (Figure 2).

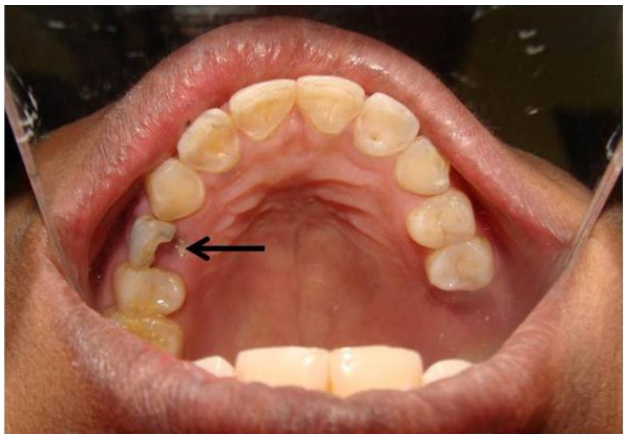

Figure 2: Grossly decayed upper left second premolar.

FNAC of the swelling was non-contributory. Beyond doubt the focus of infection was the upper left second premolar and the soft tissue mass was thought to be attributed to the prolonged intake of antibiotics resulting in a chronic reactive lesion.

Orthopantomogramradiograph was inconclusive as the swelling was a soft tissue encapsulation.

Excisional biopsy of the lesion along with extraction of the offending tooth was planned under local anesthesia. Intra operatively the soft tissue mass was found to be in continuity with apex of the decayed upper left second premolar. It was fibrotic in consistency without well-defined margins. A dehiscence on buccal cortical plate extending almost to the entire length of the root was present (Figure 3).

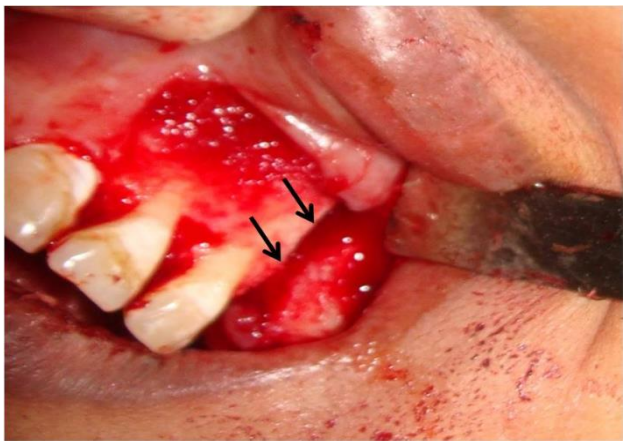

Figure 3: Bony dehiscence revealed after extraction.

The underlying bone however did not show any destruction. The mass was sent for histopathological examination (Figure 4).

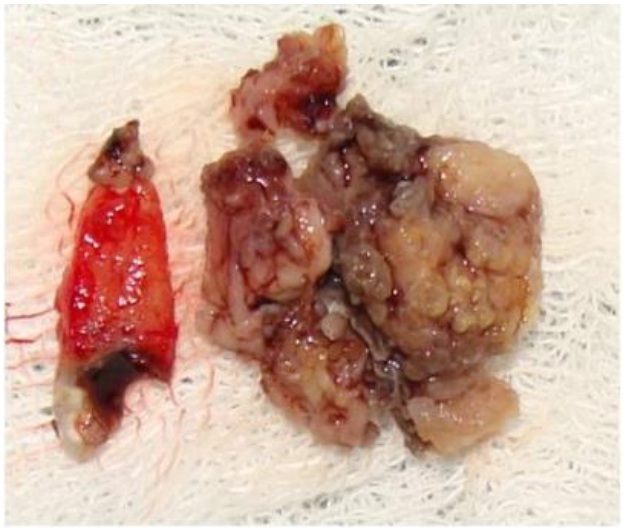

Figure 4: Extracted tooth and excised mass.

Microscopic findings showed fibroconnective tissue, fibroadipose tissue and skeletal muscle bundles along with numerous epitheloid cell granulomas with Langhan's giant cells and focal caseous necrosis. Extensive fibrosis with mild mononuclear inflammatory cells infiltrate was seen (Figure 5). However, Ziehl-Nielsen stain for acid fast bacilli was negative. Histologic features were consistent with granulomatous lesion, possibility of tuberculosis was suggested.

Despite the noncontributory history by the patient, on repeated questioning, she revealed a past history of

tuberculosis. At about the age of 19 years the patient suffered from tubercular pleural effusion and was cured.

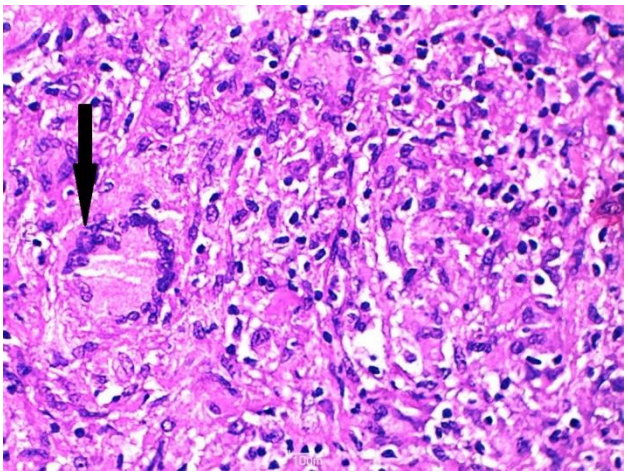

Figure 5: Photomicrograph showing epithelioid cell granuloma with characteristic Langhan's giant cell (black arrow).

In India where communicable diseases are still a taboo in some strata of the society, this explains the patient's reluctance to admit a history of TB. A medical consultation was sought for the patient. Ancillary tests were carried out which showed a high ESR of 27mm per hour (Westergreen method), positive Mantoux test (24x22 mm) and chest X-ray PA view which showed blunting of the right costophrenic angle and loss of volume suggesting pleural effusion (Figure 6).

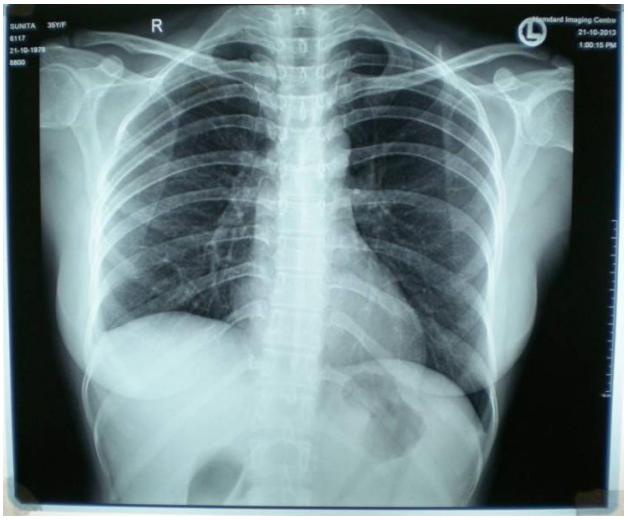

Figure 6: Chest X-ray PA view: Blunting of the right costophrenic angle and loss of volume.

To confirm, an ultrasonography of chest was done which was suggestive of localized pleural thickening due to an old resolved tubercular pleural effusion.

Anti-tubercular therapy was started and the patient is on a regular follow up (Figure 7).

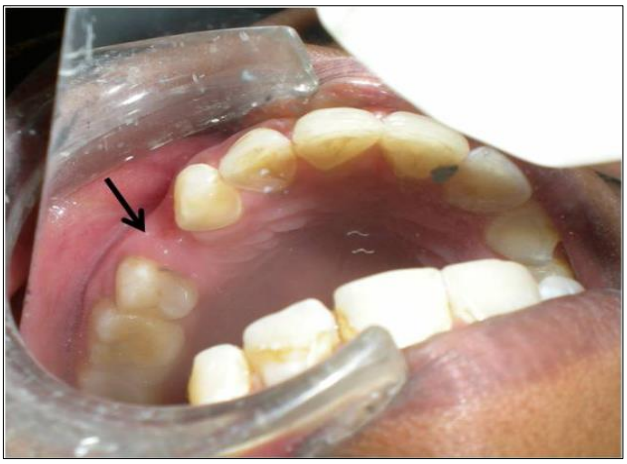

Figure 7: Surgical site after 2 months postoperative.

DISCUSSION

Primary pulmonary infection manifests with bacteremia, which has the potential to infect nearly all organs within the first weeks of infection. Symptoms are usually a mild respiratory illness with initial inoculation. In most cases, cell-mediated defenses contain the bacteria in a dormant lung focus of macrophages, and the patient becomes asymptomatic with a positive tuberculin skin test. In some cases, the primary TB infection becomes progressive with erosion of tuberculin granulomatous lesions beyond the lung parenchyma into nearby lymphatics or blood vessels. Head and neck manifestations of TB are caused by the hematogenous or lymphatic spread of the bacteria to affect the larynx, oropharynx, maxillofacial structures, ear, mastoid, and cervical spine.[4]

In extrapulmonary TB, symptoms may include an obvious mass or lesion with local pain, swelling, and occasionally fistula formation. Extrapulmonary lesions may be ulcerative, but granulomatous disease is more common.[5]

Only about 0.05% to 5% of patients with active TB present with oral lesions.[6]

TB of the head and neck has been documented extensively. A 10-year retrospective study in India 165 cases of TB of the head, neck, and oral cavity. Of these patients, 121 (73.3%) had isolated tubercular lymphadenitis; 24 (14.5%) had laryngeal TB, and four (2.4%) had tubercular otitis media. Three (1.8%) had cervical spine involvement; three (1.8%) had parotid gland involvement, and eight (5%) had oral cavity involvement. One patient had temporomandibular joint involvement, and one had TB of the nose.[7]

In the present case the patient suffered from TB at the age of 19 years and underwent treatment for the same. The current medical status confirms the patient to be suffering from TB. The confounding factor is whether the lesion is

an extrapulmonary focus of primary T.B. or secondary oral TB per se. The decayed upper left second premolar with an open access to the root canal could be a pathway of entry of bacilli. Organs with high oxygen tension are most susceptible to TB infection, including lung, kidney, and bone.[5]

Vascularization of dental pulp is important, proportionally comparable with the human brain. Pathogenic bacteria circulating in blood may occur and colonize the dental pulp.[8] Lipids such as mycolic acids of M.tuberculosis appear to be particularly robust in ancient remains.[9] Mass spectrometry has successfully identified specific bacterial proteins of M. tuberculosis from archeological bone samples.[10]

Though unusual, the possibility of primary oral tuberculosis per se exists. Since primary oral TB is more common in younger patients, occurs by direct inoculation of the oral mucosa from an infected individual and is exceptionally rare.[2] Also the integrity of the oral epithelium and the inhibitory effect of saliva are considered the reasons for the relative resistance to infection by M tuberculosis bacilli.[11] Considering these factors the diagnosis of primary oral TB could be ruled out in the present case.

In this case the dental pulp upper left second premolar could be an extra pulmonary focus of the previous episode of TB wherein the bacilli remained entombed and quiescent in the dental pulp. Reactivation occurred due to subsequent illnesses or immune suppression with the resultant periapical granuloma formation. The locally progressive nature of the lesion as evidenced by the bony dehiscence revealed during the extraction of the offending tooth confirms the chronicity of the lesion. Thus it could be diagnosed as secondary oral TB.

The secondary oral TB is far more prevalent and arises from mycobacterium-contaminated sputum self-inoculating the oral structures, or by means of hematogenous spread in miliary TB.[6] Any break in the continuity of the oral epithelium owing to an abrasion, ulceration, or an extraction socket facilitates inoculation of the bacilli present in the sputum, which is brought into intimate contact during coughing or speech.[11]

Oral TB may have a range of presentations. Typically ulcerative or granulomatous in form, these lesions are most common on the dorsum of the tongue or palate and may cause destruction of underlying bone in the maxilla or mandible, which may mimic squamous cell carcinoma.[11]

CONCLUSION

Although the rarity of orofacial tuberculosis is undisputable, it is imperative that a high index of suspicion be considered when dealing with lesions without a specific etiology. This is especially important in the present times, because the low incidence of orofacial tuberculosis (0.05% to 5% of all tuberculosis cases) can increase rapidly, if the lesion remains undiagnosed.[6] Also a detailed medical history with careful questioning is mandatory so as to avoid missing out on some vital aspect of the disease suffered in the past. Not only can it cause an epidemic, but because of improper treatment, the complications of tuberculosis can be manifolds, causing severe disfigurement, disability, and, at times, can prove to be life-threatening.

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