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SEQUESTROMY OF TUBERCULOUS OSTEOMYELITIS IN PERIODONTITIS PATIENT: A UNIQUE REPORT

Periodontics

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ABSTRACT

Tuberculous osteomyelitis of mandible is an extremely rare condition and forgotten entity. Tuberculosis is a specific granulomatous infectious disease and a major cause of death in developing countries. Involvement of the periodontium has seldomly been reported in the recent literature. The diagnosis was based on patient's medical and dental history, clinical and radiographic examination, blood investigation, histopathologic examination of the tissue specimen. Antitubercular chemotherapy along with sequestrectomy and decortication are the treatment of choice for tuberculous osteomyelitic lesions affecting periodontium. We report a case of tuberculous osteomyelitis of mandible affecting periodontium leading to attachment loss and bone defect.

KEYWORDS

Osteomyelitis, Sequestrectomy, Tuberculosis

INTRODUCTION

Tuberculosis is a chronic infectious granulomatous disease caused by Mycobacterium tuberculosis which is an aerobic, slender, non-motile, non-encapsulated, non-sporing, rod shaped organism ranging from 2 to 5 μm^2 [1] and; it can affect any part of the body including oral cavity and periodontium [2]. A report says that every year, about 20 million prevalent cases and 8 million new cases are reported. WHO has documented that approximately 3 million people die annually of TB, and this is more common in developing countries [3].

Oral tuberculosis can be primary or secondary. Primary oral tuberculosis lesions are extremely rare and often occur in younger patients with associated caseation of the dependent lymph nodes; the lesion itself remains painless in most cases [4]. It usually involves gingiva and is associated with regional lymphadenopathy. Secondary oral tuberculosis lesions are common and usually involve the tongue, followed by the palate, lips, buccal mucosa, gingiva and frenulum [5]. Tuberculous lesion may present as superficial ulcers, patches, indurated soft tissue lesions, or even lesions within the jaw in form of osteomyelitis. [6] The sites of predilection are dorsal and lumbar vertebrae and epiphysis and diaphysis of long bones. Flat bones, including those of the skull and mandible, are rarely affected. Due to the rarity of tuberculosis of mandible, it seldom arouses clinical suspicion, especially when a positive history of a systemic infection or therapy is denied. The diagnosis, therefore, is variable and established after biopsy.[7]

Case Report

A 16-year-old male patient reported to the Department of Periodontics and Oral Implantology, Rama Dental College and Hospital, Kanpur, India with the chief complaint of dull pain, receded gums & pus discharge in lower left back tooth region since 6 month. Patient has thin built with moderately nourished. Patient gave a history of swelling in submandibular region from the past 5 months, along with the history of evening rise in temperature and weakness. He also had history of loss of appetite and weight loss, slight cough without expectoration. Patient had undergone root canal treatment 3-4 months back. Extra oral examination reveals palpable lymph node, slightly tender, measuring 1.5x 1.5 sizes in relation to left mandibular area. On clinical examination his oral hygiene was poor with deposits. Moderate periodontal pockets were present in the lower posterior teeth except with respective lower left 6, were on probing the pocket depth was more than 15 mm on distal surface along with recession and pus discharge **(Fig 1).** Intraoral x-ray revealed deep intrabony defect irt 36 **(Fig 2).**

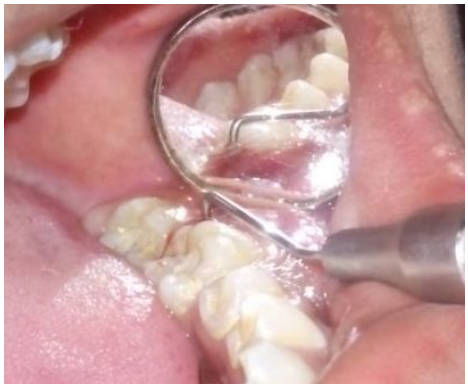

Fig: 1- Pocket depth measured with UNC-15 probe

Fig: 2- Pre-Op Radiograph irt 36 showing bone loss (distal)

INVESTIGATIONS

Routine laboratory tests and investigations for tuberculosis were simultaneously carried out. All haematological values were within the normal limits. The tuberculin (Montoux) test was positive, suggesting tubercular infection. An immunologic test to detect antibodies against *Mycobacterium* in patient's serum (ELISA) test was positive. Based on these investigation results patient was referred to the physician.

Case management

After completion of 6 months regimen of anti-tubercular therapy, patient reported back with a similar complaint. Before proceeding with the case, physician's consent was obtained and noted that there was no

abnormality in chest x-ray. Scaling and root planning were performed as a part of phase I therapy. Surgical intervention was planned after 3 weeks of the phase I therapy. Under local anesthesia, mucoperiosteal flap was reflected, as in the process of removal of granulation tissue sequestrum measuring 1.5x1.0cm surrounded by granulation tissue was observed. Sequestrectomy was performed with Schluger's bone file and curettes **(Fig 3).** Difference between healthy bone and sequestrum was clearly appreciated as sequestrum doesn't bleed on injury. Bone was excised until normal bleeding bone was encountered. All granulation tissue was removed, and root planning was performed **(Fig 4).** Bone graft was placed on the bone defect **(Fig 5).** Interrupted sutures were placed, and periodontal pack was applied. On 7th day sutures were removed and healing was uneventful **(Fig 6).** Patient was recalled after 1month. Post op IOPAR showed bone formation after 1 year **(Fig 7).**

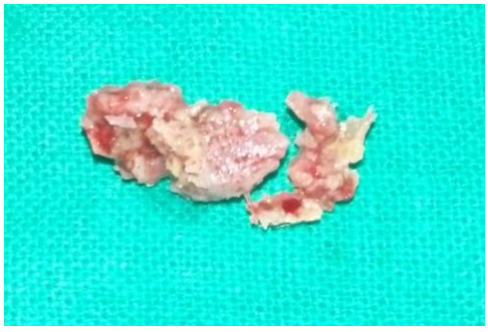

Fig:3- Sequestrum removed

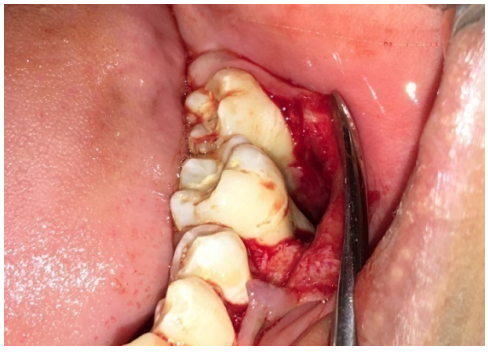

Fig:4- Defect between 36 & 37

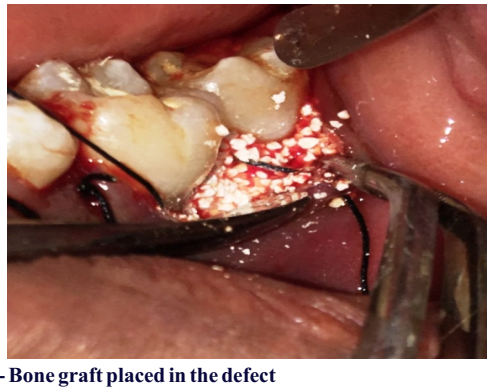

Fig: 5- Bone graft placed in the defect

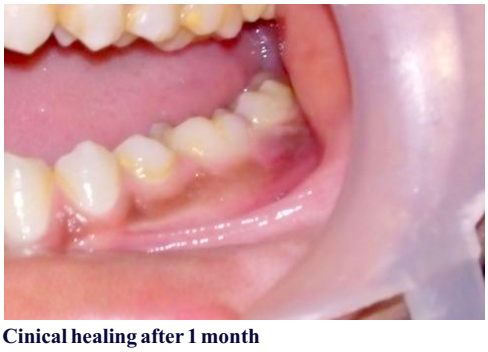

Fig:6- Cinical healing after 1 month

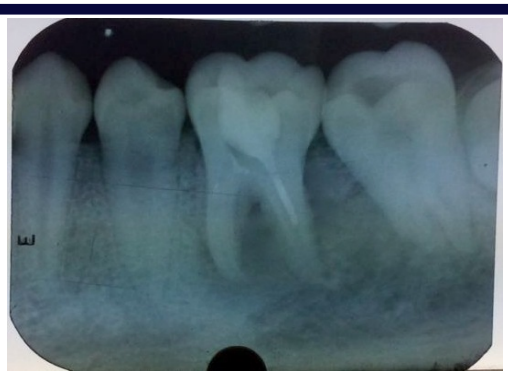

Fig 7: Post op IOPAR showing bone formation after 1 year

Histological Findings

Sequestrated bony and granulation tissue was sent for histopathological examination. Histological examination revealed presence of bone structure showing necrotic features showing osteocytes within the lacunae with the presence of ragged or irregular borders representing sequestrum & absence of peripheral osteoblastic rimming. Focal granulomatous process with Langerhan's giant cells and epitheloid cells were found in adjacent marrow cavities. This histological examination was suggestive of tuberculous osteomyelitis.

DISCUSSION

Tuberculosis is a chronic granulomatous disease that potentially affects various systems of the human body.[8,9] Tuberculosis is very common in India and South-East Asia, where the prevalence rate is about four in every 1,000 people.15% of tuberculous population of the world resides in India.[10] Primary oral tuberculosis is rare, as an intact oral mucosa, cleansing action of saliva, salivary enzymes, tissue antibodies and oral saprophytes act as barriers to infection. Any breach in these defense mechanisms, such as abrasions, tears, chronic inflammation, poor oral hygiene, tooth eruption, extraction sockets, periodontal disease, and carious teeth with pulp exposure may lead to infection by, tubercle bacilli. Poor socio-economic conditions with inadequate nutrition and lack of hygiene are predisposing factors to infection.[10,11]

Sixty percent of all cases of tuberculosis of jaw occur in children below the age of 16 years.[13] The incidence of TB in the jaw bones is very low, with mandible having a higher frequency than maxilla.[7] With the rarity of such lesions in periodontium, its diagnosis may be difficult and often missed in cases of delayed or absence of pulmonary and other systemic signs.

Tuberculosis of the jaw causes slow necrosis of the bone and may involve the entire mandible.[14] The destruction of the bone in radiographs appears as blurring of trabecular details with irregular areas of radiolucency. There is erosion of the cortex with little tendency to repair. Gradually the bone is replaced by soft tuberculous granulation tissue. Caseation appears at places followed by softening and liquefaction. A sub- periosteal abscess then forms presenting as a painless, soft swelling. This cold abscess may burst either intra or extra orally forming single or multiple sinuses. Pathological fracture of mandible and sequestration may also occur.[7]

The present case had no history and clinical evidence of pulmonary tuberculosis or osseous tubercular lesion in any part of body. Hence, the possibility of spread of bacterial infection through the open pulp carious tooth or gingival marginal inflammation due to poor oral condition. This regional extension of tubucular bacilli transferred into the soft tissue and underlined bone causing tuberculous osteomyelitis of the alveolar bone. The diagnosis of the case is extremely difficult as the radiographic changes were not very pathognomonic. The occurrence of this type of case is rare as the osteomyelitic changes in the alveolar bone continue to occur even after completion of anti tuberculin drug therapy. Better healing was observed only after sequestration and decorterization of the affected underlying bone, thereby complete removal of osteomyelitic lesion. There were no signs of recurrence and complete cure noticed.

CONCLUSION

The differential diagnosis of tubercular osteomyelitic need to be considered when the routine therapy fails to bring improvement in the

involved area within the alveolar bone, since the osteomyelitic changes occur in later stages of disease. When this lesion is primary and detected early, the disease is completely curable resulting in reversal of all destructive bone changes. The clinician should be involved in the effort to control oral tuberculosis through early detection and referral of patients to a physician for proper management by developing and implementing appropriate infection control programmes in the dental surgery.

Conflict of Interest: NIL

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