

Chronic osteomyelitis of maxilla: a rare case report

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Abstract

Osteomyelitis of the jaws is more common in developing countries and their treatment may be prolonged and difficult. Osteomyelitis is an acute or chronic inflammation involving both the cortical and trabecular aspects of bone or bone marrow. Cranial bones are less commonly affected. Inflammation with involvement of surrounding structures causes great risk like cerebral abscess, encephalitis, or meningitis. Osteomyelitis is more commonly seen in mandible than maxilla. Here we present a rare case of chronic osteomyelitis in the maxilla in elderly male. The patient was treated with sequestrectomy and no recurrence was seen in the follow up.

Key words: Bone, Chronic, Inflammation, Maxilla, Osteomyelitis.

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Introduction

Osteomyelitis is an inflammatory condition of bone which involves the medullary cavity and the adjacent cortex. It can be seen more commonly in the mandible as compared to maxilla;¹ this occurs due to the fact that, in the maxilla, cancellous bone tissue is present with greater blood supply hindering bacterial colonization, as cellular response is enhanced and has increased blood flow counteracting invasion of the bone.^{2,3} Most cases of osteomyelitis of the jaws are odontogenic in origin, other sources of infection are also seen.⁴ Osteomyelitis appears during the 5th and 6th decade of life. It is associated with systemic diseases like autoimmune diseases, diabetes mellitus, leukemia, anemia, nutritional deficiencies, syphilis, agranulocytosis, cancer, chemotherapy and radiotherapy, as well as habits of alcohol or tobacco consumption.⁵ Diagnosis of osteomyelitis is based on the history of patient, clinical examinations, radiographic and surgical findings. Histopathological examination can aid in the diagnosis.^{6,7} The treatment of osteomyelitis depends on the sequestrectomy, debriding the surgical wound and removing the bone cortex associated with the systemic use of antimicrobials.^{8,9,10}

Case Report

A 50 years old male patient visited the department of oral medicine and radiology with the chief complaint of non-healing ulcer in front mid region of palate since 4 months and another ulcer in right back region of

palate since 2 months. The ulcer in anterior midline region of palate was gradual in onset, increased in size with time to attain the present size with no discharge. Palatal denudation started occurring 3 months after this which was previously small in size, increased in extent to attain the present size. Patient gave history of pain and decayed tooth in right upper back teeth region since 3 months which was gradual in onset, increased in severity with time. Pain was severe, intermittent and radiating to temporal region. Patient consulted the dentist for the same and pain subsided (on medication) but did not relieved completely. After 15 days, patient got his tooth extracted in the same region but the wound did not heal after extraction. There was history of ulcer in left back region of palate 1 month after extraction.

Patient was diabetic since 2 years and was under medication (oral hypoglycemics). He had altered taste sensation and foul smell in mouth. No history of similar ulcer, bone involvement elsewhere in the body. No history of limitation of mouth opening, recent weight loss, headaches, paresthesia, sneezing, hoarseness of voice, fever. There was no regurgitation of food and liquid from the nose with no episode of nasal bleeding. His past medical history was not relevant. Patient was married and was having three other members in his family (wife and 2 children). There was no known inherited disorder in his family. Patient brushes once daily with tooth paste in horizontal motion. No history of any other deleterious habit. On general examination, Patient was conscious, cooperative and well oriented to time, place and person. Patient was afebrile with pulse rate- 76beats/min, respiratory rate- 18 breaths/min and blood pressure- 130/80mm of Hg. No signs of pallor, icterus, cyanosis, edema, anemia and clubbing.

On extraoral examination, no gross facial asymmetry was present. Bilateral synchronous condylar movements with no evidence of crepitus, clicking or tenderness on both sides on temporomandibular joint examination. Maximum mouth opening was 45mm

interincisally with no chin deviation. Regional lymph nodes were not palpable. No abnormality was detected in eyes, skin, ear, nose, salivary glands and muscles.

On intraoral examination, two ulcers were present on palatal mucosa. First one was ill-defined denuded area in mid region of hard palate extending anteriorly from palatal gingiva of 11 involving rugae area to posterior boundary of hard palate posteriorly; laterally extending 1cm from both sides of palatal aspects of teeth. Its maximum dimensions were 4cm x 2cm with having everted edges and indurated borders exposing bony surface covered by necrotic slough surrounded by reddish area. Another one was well defined denuded area on palatal aspect of 17 region which was 0.5cm in maximum diameter. On palpation, inspeitory findings were confirmed regarding size, shape and extent. It was mildly tender, hard in consistency, rough surface texture. On probing the palatal area, bone was found to be intact.

No abnormality was detected in labial, buccal and vestibular mucosa, floor of mouth and tongue. Gingiva was reddish in colour, edematous, increased in size having blunt contours with loss of stippling and bleeding on probing was present. There was generalized gingival recession and gingival recession and periodontal pockets were present with respect to 11 and 21. On hard tissue examination, 17 and 47 were found missing, 37 was having proximal caries and generalized grade II calculus and stains were present.

On radiographical examination, IOPA showed extraction socket with respect to 17 region and horizontal bone loss with respect to 15 and 16. Maxillary cross-sectional occlusal view showed missing 17 with horizontal bone loss in maxillary anteriors and no other bony changes. Oral pantomograph showed missing teeth (17 and 47), proximal caries (with respect to 37) and generalised horizontal bone loss with well-defined radiolucencies with respect to periapical regions of 11,12,13,21,22,23,24. No abnormality was detected on paranasal sinus X-ray.

Biopsy was done at the site of lesion (mid palatal region) which was sent for histopathological examination (under H & E stain); and it revealed both epithelium and connective tissue components: epithelium was pseudostratified ciliated columnar in nature and connective tissue stroma showed dense irregular aggregates of bony trabeculae with empty lacunae i.e. without osteocytes in most of areas and with osteocytes in few areas. Interstitial tissue was loose, fibrous with marked chronic inflammatory infiltrate chiefly consisting of lymphocytes suggestive of chronic osteomyelitis. No fungal hyphae were seen.

Patient was treated with sequestrectomy under general anaesthesia. On first follow up (after 15 days), betadine dressing was given. On second follow up (after 1 month), betadine dressing was done and patient was advised obturator 2 months after 2nd follow up.



Fig. 1: clinical picture



Fig. 2: OPG

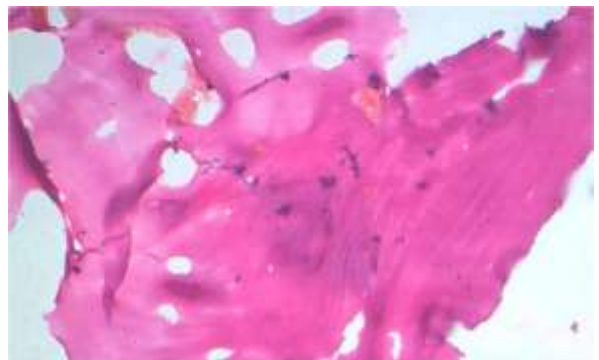


Fig. 3: Histopathological picture

Discussion

Osteomyelitis is an inflammatory disease of bone which affects bone marrow - frequently the cortical bone and periosteum. This condition was life threatening before antibiotics came into use.^{11,12,13} The very first report on osteomyelitis was written in 1832 by British physician Sir Benjamin Brodie. The first time he described a type of abscess known as the Brodie abscess which is a chronic features of osteomyelitis.¹⁴ Osteomyelitis can be caused by trauma, surgical procedures and infections such as endodontic and periapical infections.¹⁵ In our case, it was associated with odontogenic infection and may be aggravated by trauma. Oral microflora which are most frequently involved in osteomyelitis are gram negative

anaerobic rods and facultatively anaerobic cocci of staphylococcus and enterococcus.¹⁶ Systemic diseases are predisposing factors for osteomyelitis as in our case patient was diabetic. Clinical features may include local pain, fever, swelling, purulent discharge, intra-oral and skin fistula, unhealed soft tissue in the oral cavity, neuropalsy in the involved area, pathological fracture and trismus.¹⁷ In our case, pain, soft tissue induration and swelling over the hard palate was seen.

On radiograph, chronic osteomyelitis appears as a radio-transparent lesion. It can show opaque focal areas in some cases. Lesions are usually large with border that are undistinguishable.^{18,19} The most distinguishing feature of chronic osteomyelitis is sequestra and laminating new periosteal bone.

Histopathologically, osteomyelitis shows increase in number of osteoblasts, thick bony trabeculae and fibrous marrow. In chronic osteomyelitis, pathological bone remodelling is seen. Chronic inflammatory cells are also evident as seen in our case. Most cases show sequestrum formation.¹⁷ Osteomyelitis can be managed with a course of antibiotics combined with surgical treatment and regular follow up.²⁰

Conclusion

On one hand, occurrence of osteomyelitis is rarer with the advent of newer antibiotics, imaging techniques and better social conditions, but on the other hand with the increasing prevalence of immunocompromised conditions like diabetes mellitus, HIV infection and other predisposing factors osteomyelitis seems to be on the rise. Osteomyelitis is a multifactorial disease and its presentation varies. Infection of the maxilla can cause serious complications for the patient such as infection of cranial cavity and brain. It is essential that any maxillary osteomyelitis be treated aggressively to avoid subsequent dreaded consequences.

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