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CASE STUDY

EFFECTS OF COMPROMISED HOST IMMUNITY IN MAXILLARY JAW INFECTION

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ABSTRACT

As in today's modern era of antibiotics where the incidence of osteomyelitis of jaws seems to be decreasing, it still remains a challenging clinical entity due to the substancial growth of graph of systemic diseases. Osteomyelitis is an inflammatory bone disease commonly related to complex microbiota, most commonly pyogenic staphylococcus, and occasionally, streptococci, pneumococci and enterobacteriae. It presents a varied clinical and radiographial manifestations depending upon the severity of its spread into the bony architecture. Based on the pathogenesis and nature of the disease, various classifications have been established for the proper understanding of its varied types. Several treatment options depending upon the severity of osteomyelitis are available.

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INTRODUCTION

Osteomyelitis is an inflammatory disease of bone marrow and adjacent cortex of bone which leads to decreased vascularity inducing bone necrosis. Osteomyelitis was first described by Frenchsurgeon, Edouard Chassaignac in 1852. Infection may be derived from the antrum, lacrimal apparatus, blood-borne or odontogenic, commoner being the oral infections, particularly endodontic infections (Brady *et al*., 2006), peri-implantitis, periodontitis and gingivitis (Coviello and Stevens, 2007). The most commonly detected oral microorganisms in osteomyelitis are Gram-negative anaerobic rods and cocci. Osteomyelitis can occur at any age however, it is more frequently to be seen between 4th to 6th decade of life. Among the facial bony architecture, mandible is more frequently affected than the maxilla. This is because maxilla is composed of thin compact bone and spongy medullary alveolar bone with abundant vascular supply, thus hinders bacterial colonization. Though maxillary osteomyelitis is a rare entity, there are various systemic conditions like diabetes mellitus, auto-immune diseases, agranulocytosis, leukemia, anemia, nutritional deficiencies, syphilis, cancer, which leads to decreased immune response thus predisposes an individual to such infections. The management of osteomyelitis ranges from simple curettage to resection of jaw with or without additional supportive pressured oxygen therapy. This paper presents a case of maxillary chronic osteomyelitis in an uncontrolled diabetic patient.

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Case report

A 45 years old male patient visited to the OPD of OMFS dept with a chief complaint of dull continuous pain in the left maxillary posterior region and heaviness over the left side of the face from last 6 months for which he had undergone extraction of left bicuspids and 1st& 2nd molars 5 months backs elsewhere. Mobility (grade II) was seen of right bicuspids to left cuspid. A 2 x 1 mm fistula in upper left molar region was seen with exposed necrotic maxillary alveolus. On palpation, soft necrotic bone was palpated over the left maxillary alveolus which was crossing the midline and extending upto right bicuspid region. Slight tenderness of both the maxillary sinus was present. This was accompanied by thick foul smelling nasal discharge from the left nostril, poor oral hygiene and severe halitosis. Radiograph of paranasal sinuses (Water's view) showed partial obliteration of left maxillary sinus and complete right obliteration of right maxillary sinus.

Orthopantamogram showed radiolucency extending from right 2nd premolar to left canine region. Computed tomography showed lytic areas with sclerotic borders, periosteal reaction and bone sequestration (Fig 1). He was diagnosed to have diabetes mellitus 8 years back, however his glycemic control was poor due to irregular medications. The clinical & radiographical picture of the lesion along with the associated systemic condition of the patient was suggestive of infection of maxillary bone involving both the sinuses. Ethical committee approval and an informed written consent was taken. The patient was treated with appropriate antibiotics; his diabetic status was brought under control and an incisional biopsy was done which was suggestive of chronic osteomyelitis. Pharmacological treatment was undertaken with Ceftriaxone, 1 g IV every 12 hours and metronidazole 400mg 8hourly IV. It was decided to perform subtotalmaxillectomy under general anesthesia. A gingiva-crevicular incision was made extending from right 1st molar region to left 3rd molar region. Exposing the foul smelling bony necrotic lesion (Fig 2). The entire necrotic mass was curetted out till the fresh bleeding was seen from the adjacent bony margins. The extraction of the multiple teeth which were involved was performed. The specimen was sent for the histopathological examination where the diagnosis was confirmed as presence of maxillary chronicosteomyelitis. Water tight closure was done with the help of 3-0 vicryl as a horizontal mattress suture. Patient was continued with the same antibiotics for a week which was the shifted to oral administration of the same drugs for another 5 days. Tablet Piroxicam 20 mg 12 hourly as an analgesic and anti-inflammmatory was given for a week. Nasivion nasal drops and karvol inhalation was encouraged twice daily for 2 weeks. The blood sugar was kept under control. A follow-up of 6 months showed no recurrence. The patient was then referred to the department of Prosthodontics for the rehabilitation.

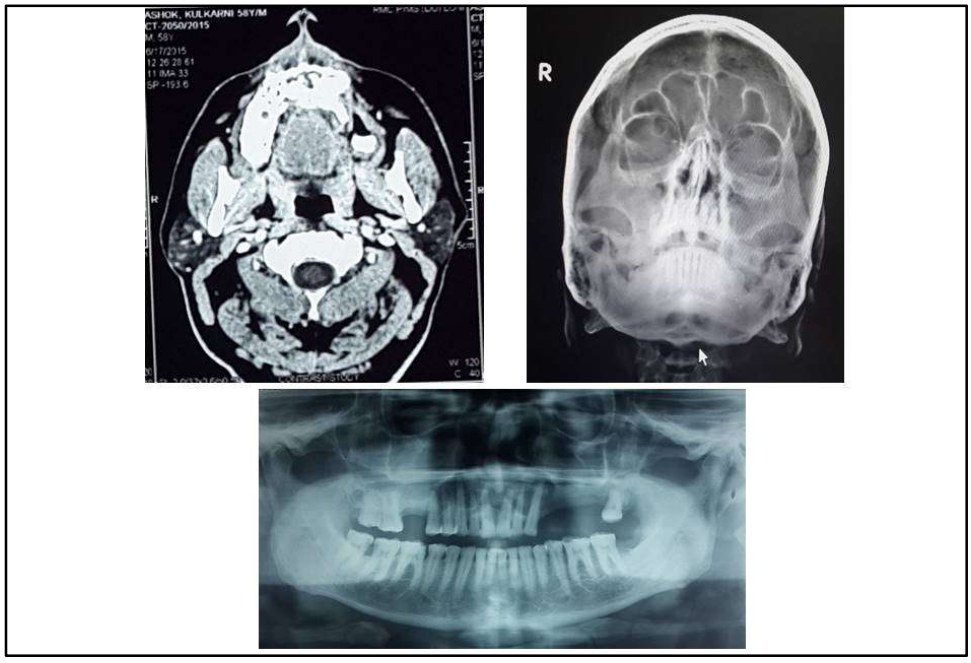

Fig. 1. Preoperative radiograph showing a radiolucent lesion with ill-defined orders in relation to upper jaw, resorption of left maxillary alveolus and maxillary sinus invasion

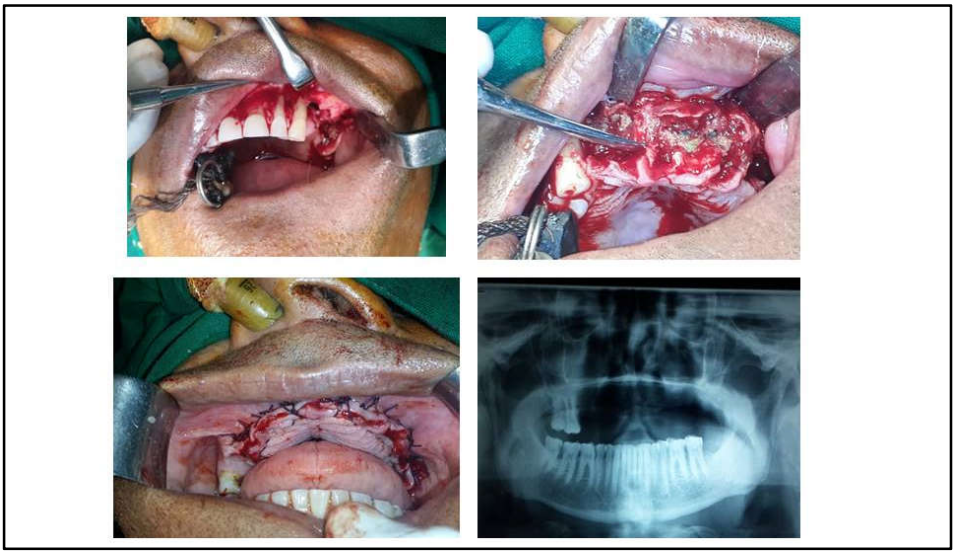

Fig. 2. Maxillary lesion exposition showing necrosed alveolar bone in relation to upper jaw and communication to maxillary sinus. Removal of the involved teeth

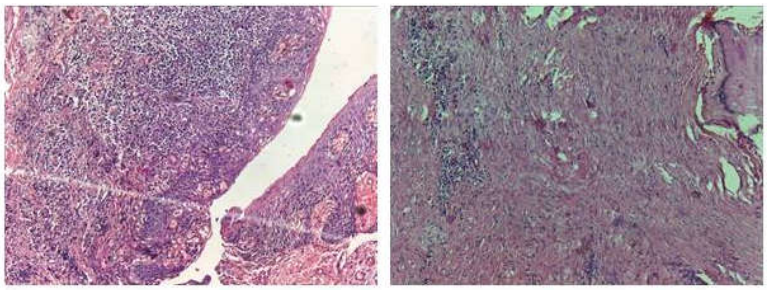

Fig 3: Histopathological examination of decalcified section showing bony trabeculae with ragged borders and absence of osteoblastic lining and osteocytes within the lacunae. Marrow spaces showed chronic inflammatory cells and endothelial lined blood vessels with red blood

Table 1. Classification based on clinical & radiographical features-

1.Suppurative osteomyelitis	2.Nonsuppurative osteomyelitis	3.Osteoradionecrosis
(i)Acute suppurative (ii) Chronic suppurative	(i)Chronic focal sclerosing (ii) Chronic diffuse sclerosing (iii)Garre's chronic sclerosing	

Table 2. Classification based on clinical, radiographical features, etiology & pathophysiology

1.Acute osteomyelitis	2.Chronic osteomyelitis
(i)Associated with hematogenous Spread (ii) Associated with intrinsic bone pathology or peripheral vascular disease (iii) Associated with odontogenic and non-odontogenic local processes	(i). Chronic recurrent multifocal osteomyelitis of children (ii). Garre's osteomyelitis (iii). Chronic suppurative osteomyelitis – Foreign body related – Systemic disease related – Related to persistent or resistant organisms (iv) True chronic diffuse sclerosing osteomyelitis

Table 3. Systemic conditions responsible for alteration of host defence mechanism

Diabetes mellitus	Malnutrition
Autoimmune disorders	Chemotherapy
AIDS	Alcohol and tobacco
Agranulocytosis	Drug abuse
Anemia	Corticosteroid and other immunosuppressive therapy
Leukemia	Syphilis

Table 4. An overview of conditions compromising blood supply of the jawbone

Diabetes mellitus	Osteopetrosis
Smoking	Osteoporosis
Florid osseous dysplasia	Radiation therapy
Fibrous dysplasia	Paget's disease

DISCUSSION

The word "osteomyelitis" originates from the ancient Greek words osteon (bone) and muelinos (marrow) and means infection of medullary portion of the bone. It beginns in the medullar cavityand havarian systems and extends to involve the cortex & periosteum of the affected area. The infection becomes established in bone when pus and edema in the medullary cavity and beneath the periosteum, compromises or obstructs the local blood supply. Following ischemia, the infected bone becomes necroticand leads to sequestrum formation, which is considered aclassical sign of osteomyelitis. Based on factors such as clinical, radiolographicaletiological and pathophysiological, osteomyelitis has been classified by Bernier *et al*. (1995), Marx and Mercuri (1991) (Table 1 & Table 2). Depending upon its origin, MacBeth classified maxillary osteomyelitis as traumatic (especially poorly consolidated fractures), rhinogenic, or odontogenic or hematogenic spread (Macbeth, 2001). The oral cavity harbors a large number of bacteria, among which many may be identified as possible pathogens to cause infection of the jawbone. The vast majority of cases of acute and secondary chronic osteomyelitis involving the jaws are usually caused by infection primarily spreading by a contagious focus. The most common foci are odontogenic, originating from infected pulp or periodontal tissue, especially periodontal disease, which leads to a breakdown of the periodontal barrier membrane, facilitating deep invasion pathogens, seems to be an important condition leading to osteomyelitis. In our case, origin of infection was odontogenic in nature as the patient had generalized periodontitis. The common vascular status between the maxillary teeth and the antral mucosa make spontaneous spread of infection between the two structures possible. Other than the beach in the local barrier facilitating the spread of infection, various systemic conditions with concomitant alterations in host defenses & local blood supply may influence profoundly the onset and course of disease& must be considered a critical factor in the establishment of osteomyelitis (Table 3 & Table 4). In these conditions immune cells and oxygen cannot reach the target area in an adequate manner. This facilitates the growth and spread of microorganisms, especially anaerobes, leading to establishment and progression of osteomyelitis. Osteomyelitis has been associated with a variety of systemic conditions which are responsible for alterations in the host defences. Our patient was a known case of diabetes mellitus with uncontrolled blood sugar levels. Out of the various debilitating conditions, diabetes mellitus plays a significant role in osteomyelitis of the jaw bones as it compromisesboth the hostdefence as well as the local blood supply. It plays its role in establishment of osteomyelitis as vascularity is compromised due to arthritis of the smaller vessels (Ranjit Kumar Peravali *et al*., 2012), thus reducing perfusion and the ability for an effective inflammatory response slowing healing rate due to reduced tissue perfusion and defective glucose utilization. Apart from this, it is also responsible for the diminished leukocyte chemotaxis and phagocytosis (Marc M. Baltensperger and Gerold K Eyrich).

Although until recently involvement of S. aureus, S. epidermidis, and Actinomyces were still discussed as the major pathogens in cases of osteomyelitis of the jaws, more recent studies favor the concept of a polymicrobic infection with several responsible pathogens. This shift in doctrine is explained mainly by modern, sophisticated culture methods, especially involving anaerobic media, which enable identification of possible pathogens more accurately. Consequently, many pathogens, which are mostly found in the healthy oral flora, have been associated with cases of jawbone osteomyelitis; however, prolonged antibiotic therapy prior to harvesting of the specimen and possible oral contamination complicate the interpretation of each result. Osteomyelitis of the maxilla is typically a polymicrobial infection that is caused by many types of odontogenic microbial flora. Both gram-positive and gram-negative microorganisms, including Staphylococcus aureus, epidermidis, streptococci and Bacteriodes are seen (Topazian *et al*., 2002). There is also an associated mixed flora, like hemolytic streptococci, pneumococci, Escheriquia coliand Actinomyces.

Imaging radiography required to determine the extent of disease & treatment planning is accomplished by conventional, computed tomography, magnetic resonance imaging and radionuclide bone scanning aids. The radiographic changes in osteomyelitis usually demonstrate a "moth-eaten" appearance due to enlargement of medullary spaces and widening of Volkmann's canals resulting from bone destruction. There is often presence of radio-opaque foci of sequestra (nonviable bone) and at times a sheath of new bone (involucrum) separated from the sequestra by a zone of radiolucencyis seen (Marx and Stern, 2003). Lesions are normally quite extended, often with undistinguishable borders (Regezi *et al*., 2007). These are the findings which can be appreciated in long standing case of osteomyelitis. In our case, radiolucency was seen extending from the maxillary right premolar region upto left canine region with regular boder. An obliteration of both the maxillary sinus were observed with sclerosed borders. Histopathologic appearance of OM shows necrotic bone with loss of osteocytes from their lacunae, peripheral resorption, and bacterial colonization. The periphery of the bone and the haversian canals contain necrotic debris and an acute inflammatory infiltrate consisting of polymorphonuclear leukocytes (Flygare *et al*., 1997; Shafer *et al*., 2005). The excisional specimen on histopathological examination showed bony trabeculae with ragged borders and absence of osteoblastic lining and osteocytes within the lacunae. Marrow spaces showed chronic inflammatory cells and endothelial lined blood vessels with red blood cells (Fig 3).

The management of osteomyelitis relies on a multidisciplinary approach, combining debridement, soft tissue coverage, and antimicrobial therapy to give the patient the bestchance of cure. The principles of treatment includes removal of the cause along with removal of the nonviable bone under the control of culture guided antibiotics. Apart from the definitive treatment of this infective disease, it is utmost important to evaluate the systemic condition of the patient and to rule out every possible cause responsible for compromised host defense and stabilizing it. Various treatment modalities are available which includes sequestrectation, debridement, decortication, saucerization, resection followed by reconstruction (Alberto *et al*., 2012). Adjunctive hyperbaric oxygen therapy (HBO) for non-radiation OM may be considered in refractory infections and among

medically compromised with no HBO contraindications. In our case, we followed the same strategy and thus removal of all the offending too thin the involved area were extracted followed by the subtotal hemi-maxillectomy was performed. To limit the spread of infection, patient was kept under the coverage of gram negative spectrum of antibiotics and the blood sugar levels were strictly made under control preoperatively and postoperatively.

Conclusion

Though the introduction of newer antimicrobials in todays' era plays an important role in minimizing the extent and progression of osteomyelitis, the increasing prevalence of immune-compromised conditions which act as predisposing factors to osteomyelitis seems to be rising. Infection of the maxilla can cause serious complications if it seeds into the cranial cavity. Thus, it is essential to achieve systemic stability, followed by antibiotic therapy along with surgical treatment. Any maxillary osteomyelitis be treated aggressively to avoid subsequent dreaded consequences. It is important for the treating physician to consider host compromise and treat any compromising condition, when feasible, concomitantly with the infection.

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