



## **Extensive Osteomyelitis of Midface in Adult Patients: Report of Two Cases**

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**Abstract**

*Osteomyelitis is a medullary infection spreading rapidly to cause significant bone volume loss. Midfacial bones are not as likely to be affected by osteomyelitis attributed to their porous and vascular architecture. However, fungal osteomyelitis of midface has been reported due to affinity of various fungi for sinonasal area. Periodontal infection extensive enough causing midfacial osteomyelitis is a quite rare condition. We describe here our clinical experience of extensive osteomyelitis of maxilla and other midfacial bones in two adult patients developed from direct extension of periodontal infection.*

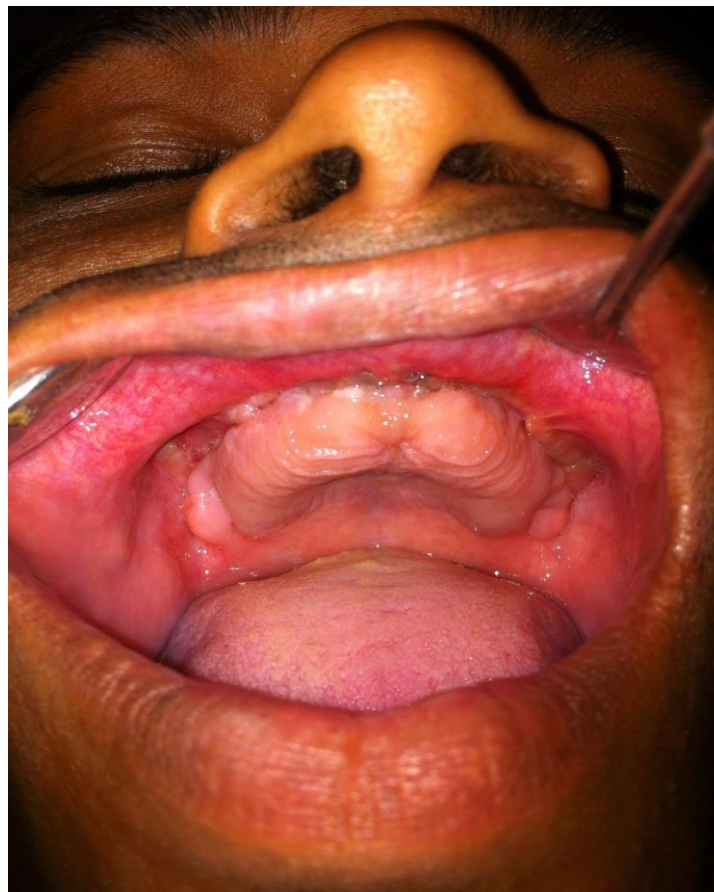
**Introduction**

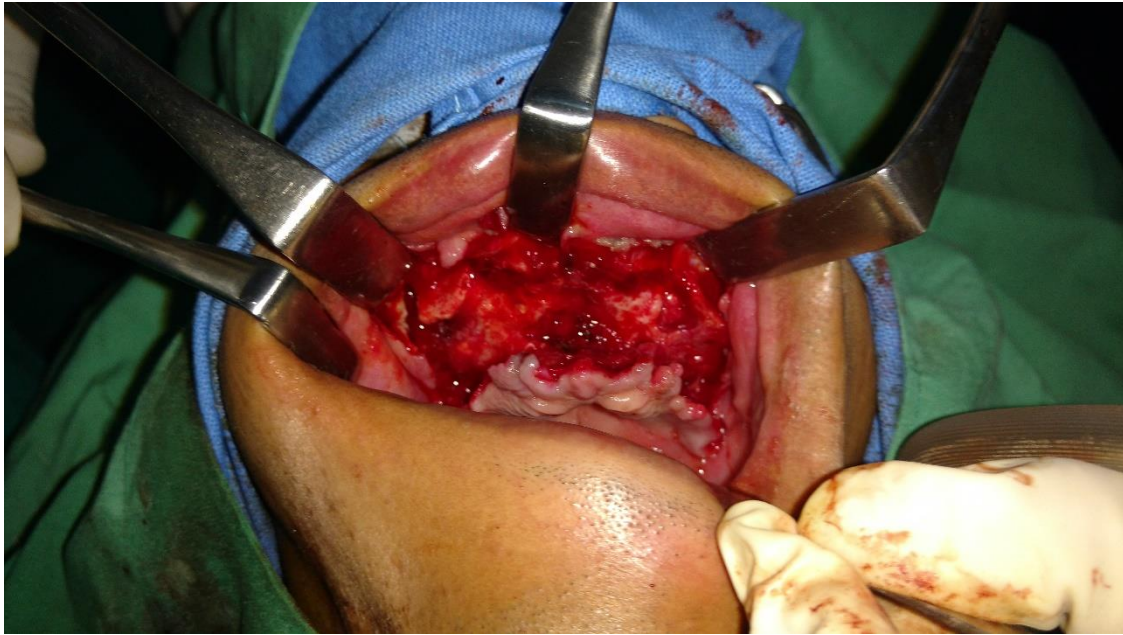
Osteomyelitis is a progressive inflammation of the bone which begins as an infection of medullary cavity and spreads rapidly to involve cortex and periosteum. Osteomyelitis has now become a rare bony infection in developed countries after the advent of antibiotics, improved nutrition, dental care, early diagnosis and intervention but in developing countries and in society with poor socio-economical class it is still commonly reported.[1] In the head and neck region, osteomyelitis of the mandible is very common compared to maxilla because of its bone architecture and vascularity. In maxilla, osteomyelitis is mainly reported in infants where infection arises from the nursing mother or obstetrician's finger or contaminated nipple in which the organisms enter through the mucosal defects in gingivae.[2] The predisposing factors for osteomyelitis of maxilla in adults chiefly include dental infections, maxillary sinusitis, trauma, or conditions which compromise the blood supply of the bone such as radiotherapy and malignancy.[3] Periodontal disease which leads to breakdown of the periodontal barrier membrane, facilitating deep invasion of pathogens, seems to be an important condition leading to osteomyelitis. Here we present two cases of osteomyelitis of maxilla and other midface bones in the adults which developed from direct extension of periodontal infection into the respective bones.

## Case 1

A 25 year old, male patient reported to our department with pain in entire upper jaw and mobile teeth of 4 months duration. History was suggestive of pain in gums of upper jaw associated with areas of ulcerations along with the fever and malaise 4-5 months ago which was resistant to medicinal treatment which further progressed to cause mobility of teeth in upper jaw and pus discharge from gums of associated teeth region resulting in exfoliation of anterior teeth. No significant medical history was present. Patient was nonsmoker. On intraoral examination patient had ulcerations over gingiva bilaterally in the premolar region of maxilla, exposed interdental bone from second premolar to opposite second premolar along with the pus discharge from the open socket, halitosis was also present along with very poor oral hygiene. CT face was advised which suggested a lytic lesion having moth eaten appearance with sclerotic foci diffusely involving bilateral maxillae, right zygomatic bone, inferior part of nasal septum and palatine bones with minimal periosteal reaction and irregularity with surrounding heterogeneity and loss of fat planes. Pus was sent for culture and sensitivity test. Amoxicillin- Clavulenic acid combination was given empirically for 6 days followed by 4 weeks course of Levofloxacin after receiving the sensitivity test report. Under GA the necrotic areas were curetted thoroughly till healthy bone was evident. The wound was packed with iodoform impregnated ribbon gauze which was regularly changed till good, healthy granulation tissue was formed. The tissues were sent for HPR which confirmed osteomyelitis. After 2 months, when satisfactory healing was ensured, an acrylic obturator was fabricated to cover the defect of zygomatic region.









## Case 2

A 35 year old young gentleman reported to our department with pain and sensitivity of teeth in right side of upper jaw since 3 months. There was no history of trauma, ENT infection or tooth extraction present. Patient was a smoker and tobacco-chewer. The patient was having type II diabetes mellitus and he was on oral hypoglycemics for the past 4 years. On intraoral examination, denuding ulceration of gingivae of right posterior teeth and left premolars were observed. All the maxillary teeth were mobile. Pus discharge was not present. CT was advised which suggested erosion and destruction of almost entire maxillary arch with buccal cortical breach associated with sequestrum formation. Amoxicillin- clavulenic acid combination was empirically started followed by amikacin after receiving the sensitivity test report.

The patient's blood sugar level was managed. Extraction of all the maxillary teeth and aggressive curettage of necrotic bone were done under general anesthesia. Regular iodoform impregnated ribbon gauze dressing was done till healthy healing was evident which took 3 months.





## Discussion

The term ‘osteomyelitis’ was introduced by Nelaton[4] in 1844 which implies an infection of the bone and marrow. Osteomyelitis can be defined as an inflammatory condition of the bone which begins as an infection of the medullary cavity which rapidly involves the haversian systems and progresses to involve the periosteum of the affected area.[3] Bacterial invasion into the cancellous bone causes inflammation and edema of the marrow space leading to compression of the blood vessels which eventually results in the development of ischemic and necrotic bone.[5] It may result from a local contiguous infectious focus and is further influenced by diseases that either affect the vascularity of bone or produces an alteration of host defenses.[6] Both the cases of this series had local odontogenic causes and along with that the second patient was having uncontrolled diabetes mellitus. Examples of systemic diseases that decrease host defenses include diabetes, anemia, and malnutrition. Radiation, malignancy, osteoporosis, osteopetrosis, and Paget’s disease are the conditions that decrease the vascularity of bone and therefore cause a predisposition to infection.[6] Local factors such as trauma and dental infection are very important as they are very frequently



encountered in the etiopathogenesis of osteomyelitis.[1] The most common foci are odontogenic in which infection originates from infected pulp or periodontal tissue or infected pericoronal tissue.[7] The presence of teeth creates a direct pathway to the bone by pulpal and periodontal disease.[1]

Periodontal disease, which leads to breakdown of the periodontal barrier membrane, facilitating deep invasion pathogens seems to be an important condition leading to osteomyelitis. Significant periodontal disease was found in 51% of the OM patients in a retrospective study [8]. In this series both the patient had history of periodontal disease progressing to cause osteomyelitis.

In the head and neck region, osteomyelitis of the mandible is very common compared to maxilla because of its bone architecture and vascularity. Maxilla may get involved by infection derived from the antrum, lacrimal apparatus, the dental germ or it may be blood-borne. Maxilla derives its arterial supply from the internal maxillary artery whose branches form anastomosing loops or arcades.

Hence sequestra may be localized, but when the internal maxillary artery is itself thrombosed, the whole maxilla sequestrates [2,9]. MacBeth classified maxillary osteomyelitis as traumatic (following injury or surgery, the primary site of infection may be antrum, teeth, or lacrimal sac), rhinogenic (spontaneous spread of infection from the antrum and postoperative rhinogenic cases) and odontogenic (dental-root sepsis may progress to osteomyelitis) [9]. Maxillary osteomyelitis has been commonly reported in infants because more bone is available due to the presence of smaller sinus. Infection in these cases arise from the nursing mother or attendant wherein the organisms enters through mucosal defects in gingivae [2]. But in our series both the patients were adult.

Incidence of maxillary osteomyelitis among poorly controlled diabetics in rural Indian population was 45.1% in contrast to 0% in other studies[10] In a review of 141 cases of jaw osteomyelitis in Nigeria, Adekeye and Cornah, it was found that odontogenic infections was the cause of 38% of mandibular, and 25% of maxillary involvement [3]. Unlike osteomyelitis in other regions of the body, which is predominantly due to *Staphylococcus aureus*, 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 [11]. The first patient's culture report showed presence of *S.aureus* and *E.coli* while in second patient causative microbe found was *E.coli*.

Clinical features of maxillary osteomyelitis are fever, rigors, pain and tenderness over the maxilla, halitosis, loosening of teeth, discharging sinuses, numbness of cheek, cellulitis of face, ocular symptoms like epiphora, proptosis, impaired eyeball movements, lymphadenopathy and occasionally blindness.

Swelling may be found additionally over the ascending process of the maxilla, in the canine fossa, over the hard palate, and the lateral nasal wall [9]. Both the patients of these series had pain, loose teeth, halitosis and gingival ulceration and along with that first patient had a history of fever too.

Imaging of the suspected osteomyelitis in the jaws is accomplished by conventional radiography, supplemented by computed tomography, magnetic resonance imaging and radionuclide bone scanning aids in determining the extent and degree of disease, location of sequestra, and in treatment planning. The radiographic image may demonstrate a “moth-eaten” appearance resulting from destruction by lysis and replacement with granulation tissue or may show bone destruction of varying extent in which there are “islands” of sequestra and at times a sheath of new bone (involucrum) separated from the sequestra by a zone of radiolucency, or it may demonstrate stippled or granular densification of bone caused by subperiosteal deposition of new bone [3]. CT imaging findings of our patients suggested lytic lesions with moth-eaten appearance and sequestrum formation.

Histopathologic appearance of the specimens showed necrotic bone with loss of osteocytes from their lacunae, peripheral resorption. The periphery of the bone and the haversian canals contained necrotic debris and an acute inflammatory infiltrate consisting of polymorphonuclear leukocytes. These findings were consistent with what has been described by the literature as osteomyelitis [11-13].

The primary goal for treatment of chronic osteomyelitis is to provide resolution of the infection by removing the source. It is well established in the literature that a longer antibiotic therapy is required for the management of chronic osteomyelitis; however, the exact duration remains controversial. Bamberger [14] suggested 4 weeks of antibiotic therapy after surgery, whereas Kim and Jang [15] reported favorable results in the management of chronic osteomyelitis with antibiotic application for 8 weeks after surgery. Management of necrotic bone include removal of loose teeth and sequestra, debridement, decortication, resection and reconstruction [3]. Adjunctive hyperbaric oxygen therapy (HBO) for nonradiation osteomyelitis may be considered in refractory infections and among medically compromised with no HBO contraindications [11-13]. After primary surgery both the patients responded well to the prescribed antibiotics which were given for 4 weeks.

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## Conclusion

Though osteomyelitis of maxilla is mainly seen in infants and children it may be seen in adults as well. In addition to carious teeth and sinus disease one may look for periodontal disease as a cause of maxillary osteomyelitis. Clinically mobile teeth, pus discharge, halitosis and numbness over cheek is seen. CT examination usually shows bony destruction with moth eaten appearance and sequestrum formation. Condition responds well to local debridement and long term antibiotic management without dreadful complications. Our paper highlights the negligent attitude of the patients towards dental care and lack of sufficiently trained staff at the primary care center which synergistically leads to worsening of a simple condition to a more complex case requiring aggressive management leading to increased morbidity associated with the disease. This paper again establishes the fact that the regular dental checkup is very necessary to lead a disease free life.

Apart from that the government and the other concerned authorities should make sure that every primary health care center has trained dental personnel to diagnosis and manage the oral diseases at the earlier stage so that a severely morbid condition can be prevented.

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