*IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)*
*e-ISSN: 2279-0853, p-ISSN: 2279-0861.Volume 18, Issue 5 Ser. 2 (May. 2019), PP 17-20*
*www.iosrjournals.org*

# Post Traumatic Osteonecrosis Leading to Maxillectomy & Mandibulectomy – A Review

[1]Dr George Skariah P, [2]Dr Georgie P Zachariah
*[1]MDS LLB MMM, Associate Professor, Dept of OMFS, Govt Dental College Trivandrum.*
*[2]PG Student, Dept of OMFS, PMS College of Dental Sciences and Research, Trivandrum.*
*Corresponding Author: Dr George Skariah P, MDS LLB MMM, Associate Professor, Dept of OMFS, Govt Dental College Trivandrum.*

***Abstract:*** *Osteonecrosis of the jaw is a site specific osseous pathology, characterized by exposed bone in the mouth that does not heal with 6 to 8 weeks of therapy. It is very likely that osteonecrosis of the jaw is a clinical entity with many possible aetiologies and its pathogenesis is not well understood. The risk factors for osteonecrosis of the jaw include bisphosphonates treatments, head and neck radiotherapy, periodontal disease, dental procedures involving bone surgery, edentulous regions, and trauma for poorly fitting dentures. These lesions typically become symptomatic in case of secondary infections, trauma to adjacent soft tissues, or other more rare complications such as pathologic bone fracture. We put forth a systematic review following athorough literature search of post traumatic necrosis leading to maxillectomy and mandibulectomy.*

Date of Submission: 26-04-2019 Date of acceptance: 11-05-2019

## I. Introduction

Literature search reveals that necrosis of large fragments of bone either in the maxilla or mandible following fracture of the jaws is an uncommon sequel. However, this could be an exception in clinical scenarios where there is presence of gross comminution and sepsis. The main vascular supply of the mandible is derived from the inferior alveolar artery on each side. Simultaneously periosteal blood supply also has a key role especially in elderly people.Literature also puts weight that there is anastomosis across the midline of the mandible. The survival of fragments of bone following fracture is not only dependent entirely on the arterial supply but also from the numerous muscular attachments to the bone and the anastomosis across the midline.[1]Post-traumatic necrosis of the maxilla and mandible are rare particularly the maxilla due to its rich vascular supply.[2] Osteonecrosis in the maxilla or mandible can be encountered when its vascularity is compromised. This may occur from a traumatic fracture.[3]Other notable etiological factors could be infections, tumours and radiation.[4]Recently, bisphosphonate-associated osteonecrosis has been reported as an emerging oral complication in cancer patients.[5]This paper deals with the systematic review of post traumatic necrosis leading to maxillectomy and mandibulectomy.

**Post traumatic necrosis**

Osteonecrosis of the jaw is defined as exposed, necrotic bone in the maxillofacial region for at least 8 weeks in patients receiving an antiresorptive medication for primary or metastatic bone cancer, osteoporosis, or Paget's disease, without history of radiation therapy to the jaws.[6]The staging of the osteonecrosis is based on severity of symptoms and extent of clinical and radiographic findings as shown in Table - 1.[7]

| | |
|---|---|
| Stage 0 | No frank bone exposure |
| Stage I | Chronic exposed, necrotic bone, inflammation, swelling, pain and radiographic changes |
| Stage II | Exposed bone with associated infection, pain, swelling |
| Stage III | Extensive disease that forms in large segments of the maxilla or mandible with extraoral fistulae, involvement of vital structures, or pathologic fracture |

Treatment strategies range from conservative local wound care to aggressive resective surgery of all necrotic bone. Conservative strategies include systemic antibiotics, oral antibacterial rinse, and debridement of loose necrotic bone that no longer has soft tissue coverage.[6] In the conservative management of patients with active osteonecrosis of the jaws, the treatment goal is focused on preventing disease progression rather than reversal of the process.[8] Any procedures that remove soft tissue and/or expose bone, including extractions, are generally avoided when a conservative treatment plan is followed. More invasive treatment strategies may include local curettage and debridement, en bloc resection, flap advancement and resective surgery.[9]

**Hypothesis**

Numerous hypotheses have been put forth for elaborating the physiological changes that happen in the jaws that undergo osteonecrosis. However, a few are widely accepted.There are two major theories regarding the pathophysiology of osteonecrosis of the jaw. The osteoclast-based, "inside-out" theory, in which the inhibition of osteoclastic activity and marked suppression of bone turnover, together with spread of physiologic microdamage and possibly local infection, leads to bone death within the jaw, with subsequent exposure. The "outside-in" theory suggesting a break in the oral mucosa leads to ingress of bacteria and local infection which, coupled with poor bone remodelling, conduce to bone death. Bisphosphonate-related ONJ may result from combination of these two mechanisms and hypovascularity also plays an important role.[10,11]

**Angiogenesis Inhibition**

Bone becomes necrotic without adequate blood supply. Anti-angiogenic therapies are now widely utilized to inhibit tumor invasion and metastases, targeting vascular signaling molecules such as vascular endothelial growth factor (VEGF).[12] In addition, all Bisphosphonates, especially nitrogen-containing Bisphosphonates, induce a statistically significant decrease in microvessel density in vivo. Hence, antiangiogenicsare considered as a contributing factor.[13]

**Inflammation & Infection**

Tooth extraction is generally the most common inciting event associated with ONJ, but teeth in adults are almost always extracted because they have periapical or periodontal infections or inflammation.[6,7] Inflammation/infection has been thought to play a role in ONJ, often occuring after extraction of teeth with advanced dental disease or around teeth with periodontal or periapical infection.[6,7] Recent studies have shown that the biofilm obtained from the samples taken from the infection and exposed bone revealed fungi and viruses in addition to the bacterial species. These multiorganism biofilms present challenges to therapy, and may require complicated strategies to eradicate the infection.[14]

**Bone Remodeling Inhibition**

Patients who are using Bisphosphonates are susceptible to osteonecrosis since bisphosphonates have direct effects on osteoclasts to significantly attenuate bone remodeling.[15] Osteoclast differentiation and function play vital roles in bone healing and remodeling at all skeletal sites, but osteonecrosis of the jaws only occurs in alveolar bone of the maxilla and mandible.[16]Discontinuing bisphosphonate therapy prior to surgical intervention offers faster recovery of normal bone homeostasis. Alveolar bone may demonstrate an increased remodeling rate as compared to other bones in the axial or appendicular skeleton, which may explain the ONJ predilection in the jaws. The increased bone resorption in the setting of dental disease, coupled with the thin overlying mucosa and a direct pathway through the periodontal ligament with the external environment, make the jaws a suitable breeding ground for ONJ to develop.[6]Another factor that points to the central role of osteoclastic bone resorption in ONJ pathophysiology is the effect of parathyroid hormone. Studies conducted in osteoporotic patients and animal studies simulating osteoporosis demonstrate the improved healing of extraction sockets and ONJ lesions with administration of parathyroid hormone, possibly due to its ability to improve bone homeostasis, by directly stimulating osteoblastic function and indirectly increasing osteoclastic bone resorption.[17]

**Osteonecrosis of the maxilla from traumatic fracture**

Generally, it is considered that the maxilla has a rich vascular supply. The vascular supply of the maxilla arises from the descending palatine artery and internal maxillary artery.[18]Hence,osteonecrosis of the maxilla can occur only when the vascularity is severely compromised. There are a few conditions that can affect this vascularity resulting in osteonecrosis of the maxilla. The causes can be broadly classified into septic and aseptic necrosis. Septic osteonecrosis can occur from a variety of infections particularly fungal infections such as mucormycosis and aspergillosis. Infections such as mucormycosis form thrombi within the blood vessels and result in decreased blood supply and necrosis.[4]Aseptic necrosis includes malignancy, radiation, trauma, surgery and bisphosphonates therapy.[4] Osteonecrosis of the maxilla from traumatic fracture is very rare.Literature suggests that medication-related osteonecrosis of the jaw is often preceded by dentoalveolar trauma. Even minor trauma, such as intubation and impression tray lesions, can precipitate osteonecrosis of the jaws[19]. Le Fort I osteotomy has become a routine procedure in elective orthognathic surgery. Although rare, ischaemia is one of the known complications of this procedure.[20] Bisphosphonates inhibit resorption by osteoclasts and cause induction of apoptosis which can lead to osteonecrosis. A positive association is seen between osteonecrosis and length of intravenous bisphosphonate therapy, oral hygiene and dental extraction.[21]Lesser-known causes of osteonecrosis include Wegener's granulomatosis, which is an inflammatory disease affecting blood vessels.[4] Acute necrotizing ulcerative gingivitis arising from the gingiva can involve the maxilla, while Gorham's disease

is another rare disorder of unknown aetiology that results in destruction and resorption of bone from proliferation of nonneolastic vascular or lymphamatous tissue.[4]

**Management of osteonecrosis**

Surgical reconstruction of the maxilla is a daunting task for the surgeon because it needs to include the restoration of the original form and function that has been lost.[22] The masticatory function, the resonance of the voice and the architecture of the face has to be restored so that the quality of life following the surgical intervention is optimal.[22] Many local flaps have been advocated for achieving soft-tissue closure of the defect. Local flaps of the palatal mucosa from the contralateral side or ipsilateral cheek mucosa can be used to close small defects. Pedical regional flaps can be raised for larger defects; the most common is the ipsilateral temporalis myofascial flap. Microvascular free-tissue transfer from the radical forearm flap and the latissimusdorsi flaps have also been advocated, but they rarely provide a functional solution. Osseous reconstruction of the maxilla has been attempted with little success.[23] Titanium mesh implants have also been tried as carriers for osseous tissue and the bone can then be used to place dental osseointegrated implants.[23] The reconstruction of the maxilla and the surrounding tissues using a prosthetic maxillary obturator device fabricated to include the lost dental arch still remains the method of choice because it achieves the best possible results compared with the other methods.[23]

## II. Conclusion

Trauma to the facial skeleton is very common now a days especially in road traffic accidents. High velocity injuries can cause fracture of the bone into multiple fragments. Care should be taken especially in clinical scenarios where there is soft tissue loss or patient has co-morbid conditions.Such injuries should be properly managed given that they can result in osteonecrosis of the jaws. Although rare, it should not be taken lightly as it can lead to significant anxiety for the patient and necessitate major surgery to fill the defect.

## References


[1]. Mac Gregor A. Arterial Supply of the Mandible. Proceedings of the Royal Society of Medicine. 1946; 39:347
[2]. Cornah J, O'Hare PM. Total maxillary necrosis following severe facial injury. A case report. Br J Oral Surg 1981; 19: 148–150
[3]. Lanigan DT, Hey JH, West RA. Aseptic necrosis following maxillary osteotomies: report of 36 cases. J Oral MaxillofacSurg 1990; 48: 142–156.
[4]. Auluck A. Maxillary necrosis by mucormycosis. A case report and literature review. Med Oral Patol Oral Cir Bucal 2007; 12: E360–4
[5]. Durie BG, Katz M, Crowley J. Osteonecrosis of the jaw and bisphosphonates. N Engl J Med 2005; 353: 99–102
[6]. Aghaloo T, Hazboun R and Tetradis S. Pathophysiology of Osteonecrosis of the Jaws. Oral MaxillofacSurgClin North Am. 2015; 27(4): 489–496
[7]. Ruggiero SL, Dodson TB, Fantasia J, Goodday R, Aghaloo T, Mehrotra B, O'Ryan F. American Association of Oral and Maxillofacial Surgeons Position Paper on Medication-Related Osteonecrosis of the Jaw-2014 Update. J Oral Maxillofac Surg. 2014; 72:1938.
[8]. McLeod NM, Brennan PA, Ruggiero SL. Bisphosphonate osteonecrosis of the jaw: a historical and contemporary review. Surgeon. 2012; 10:36.
[9]. Spinelli G, Torresetti M, Lazzeri D, Zhang YX, Arcuri F, Agostini T, Grassetti L. Microsurgical reconstruction after bisphosphonate-related osteonecrosis of the jaw: our experience with fibula free flap. J Craniofac Surg. 2014; 25:788.
[10]. Gacche RN, Meshram RJ. Angiogenic factors as potential drug target: efficacy and limitations of anti-angiogenic therapy. BiochimBiophysActa. 2014; 1846:161.
[11]. Pabst AM, Ziebart T, Ackermann M, Konerding MA, Walter C. Bisphosphonates' antiangiogenic potency in the development of bisphosphonate-associated osteonecrosis of the jaws: influence on microvessel sprouting in an in vivo 3D Matrigel assay. Clin Oral Investig. 2014; 18:1015.
[12]. Kumar SK, Gorur A, Schaudinn C, Shuler CF, Costerton JW, Sedghizadeh PP. The role of microbial biofilms in osteonecrosis of the jaw associated with bisphosphonate therapy. CurrOsteoporos Rep. 2010; 8:40
[13]. Allen MR, Burr DB. The pathogenesis of bisphosphonate-related osteonecrosis of the jaw: so many hypotheses, so few data. J Oral Maxillofac Surg. 2009; 67:61.
[14]. Aghaloo TL, Kang B, Sung EC, Shoff M, Ronconi M, Gotcher JE, Bezouglaia O, Dry SM, Tetradis S. Periodontal disease and bisphosphonates induce osteonecrosis of the jaws in the rat. J Bone Miner Res. 2011; 26:1871.
[15]. Dayisoylu EH, Senel FC, Ungor C, Tosun E, Cankaya M, Ersoz S, Taskesen F. The effects of adjunctive parathyroid hormone injection on bisphosphonate-related osteonecrosis of the jaws: an animal study. Int J Oral Maxillofac Surg. 2013; 42:1475
[16]. C. A. Migliorati, M. A. Siegel, and L. S. Elting, "Bisphosphonateassociated osteonecrosis: a long-term complication of bisphosphonate treatment," Lancet Oncology, vol. 7, no. 6, pp. 508–514, 2006
[17]. I. R. Reid, "Osteonecrosis of the jaw—who gets it, and why?" Bone, vol. 44, no. 1, pp. 4–10, 2009
[18]. Traxler H, Windisch A, Geyerhofer U, Surd R, Solar P, Firbas W. Arterial blood supply of the maxillary sinus. ClinAnat 1999; 12: 417–421
[19]. PouyaMasrooriYazdi, and Morten Schiodt, Dentoalveolar trauma and minor trauma as precipitating factors for medication-related osteonecrosis of the jaw (ONJ): A retrospective study of 149 consecutive patients from the Copenhagen ONJ Cohort. Oral Surg Oral Med Oral PathOral Rad 2015;19(4):416-422
[20]. Kramer FJ, Baethge C, Swennen G, Teltzrow T, Schulze A, Berten J, et al. Intra- and perioperative complications of the LeFort I osteotomy: a prospective evaluation of 1000 patients. J CraniofacSurg 2004; 15: 971–977.
[21]. Estilo CL, Van Poznak CH, Wiliams T, Bohle GC, Lwin PT, Zhou Q, et al. Osteonecrosis of the maxilla and mandible in patients with advanced cancer treated with bisphosphonate therapy. Oncologist 2008; 13: 911–920.

[22]. Nekora-Azak A, Evlioglu G, Ozdemir-Karatas¸ M, Keskin H. Use of biofunctional prosthetic system following partial maxillary resection: a clinical report. J Oral Rehabil 2005; 32: 693–695
[23]. Parr GR, Tharp GE, Rahn AO. Prosthodontic principles in the framework design of maxillary obturator prostheses. J Prosthet Dent 1989; 62: 205–212.

Dr George Skariah P. "Post Traumatic Osteonecrosis Leading to Maxillectomy & Mandibulectomy – A Review." IOSR Journal of Dental and Medical Sciences (IOSR-JDMS), vol. 18, no. 05, 2019, pp 17-20.