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Case Report

Post Traumatic Aseptic Necrosis of Maxilla and Its Surgical Excision: A Case Report

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Abstract

First thing which strikes in the mind after coming across with post traumatic necrosis of maxilla that is it possible? Looking back into literature we found only handful of incidences with traumatic maxillary necrosis. Osteonecrosis of the mandible and the maxilla is known; however, aseptic necrosis of the maxilla after traumatic fracture is hardly reported. Management of these cases can be quite cumbersome and require utmost care. We present a peculiar case report of post traumatic necrosis of maxilla and partial maxillectomy. This case aims to help clinicians realize the need to closely follow up and treat such patients with trauma as it can lead to osteonecrosis of the bone and cause difficulty in daily activities.

Keywords: post traumatic, maxilla, maxillectomy.

INTRODUCTION

Avascular necrosis is cellular death of bone components due to interruption of the blood supply. Without the vascular supply, the bone tissue dies and the bone collapses. It can affect any bone, but most commonly affects the epiphysis of the long bones. It is very rare to encounter maxillary necrosis after trauma. Only two case report has been published in past related to post traumatic maxillary necrosis [1, 2]. There are other incidences in which necrosis has been reported widely like maxillary osteotomy, infection, tumours and radiation [4-10]. Recently majority of cases with oral complication been reported for bisphosphonate-associated osteonecrosis in cancer patient [11-14]. Since maxillary aseptic necrosis after trauma was among the rarest to occur, we are drafting this literature.

Case Report

A 42-year-old male reported with the chief complaint of pain and mobility of anterior upper tooth region. Patient also had a complaint of foul odour and running nose in early morning.

On examination it was perceived that anterior maxillary segment was completely mobile. Pain was elicited while mobilizing the segment. Noticeable colour change was seen on anterior gingiva and on palatal mucosa.

Patient gave the history of road traffic accident 3 months back, where the major impact of the accident was on anterior face. He was them admitted to the primary health centre where only primary wound closure was done along with the medication.

Radiograph was not been taken and incidence of maxillary dentoalveolar went unnoticed. However, in last few weeks he started noticing the abnormal mobility of his upper jaw. Provisional diagnosis was made as non union of anterior maxillary segment (Figure-1). On radiographic examination radiolucency was appreciated extending up to the sinus.

No soft-tissue swelling or mass was present and no sclerosis; fracture or evidence of previous surgery was visible. He was prescribed prophylactic antibiotics and maxillary necrosectomy; (anterior maxillectomy).

Patient's pre-operative medical fitness was taken and he was prepared for the surgery. Under oro-endotrecheal intubation general anaesthesia was administered. Standard painting and draping were done. High vestibular incision was placed and entire mobile segment of maxilla was dissected with anterior teeth and soft tissue gingiva. Along with the necrotic segment few millimetres of normal surrounding tissues were also removed (Figure 2 & 3). Specimen were sent for biopsy and necrotic portions were sent for culture report. Skin graft was placed and healing was left for secondary intension. Patient's follow up was done for next four weeks and after that prosthesis was planned.

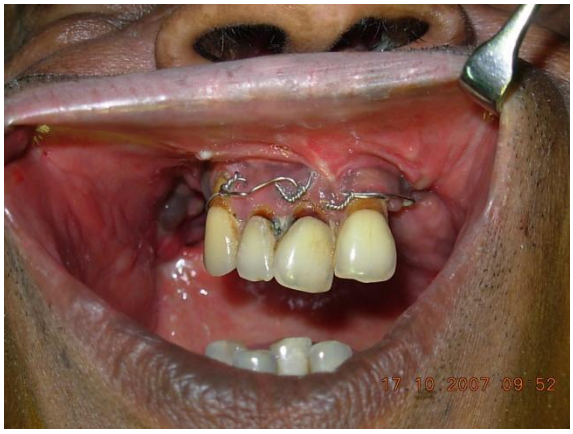

Fig-1: Mobile anterior maxillary segment

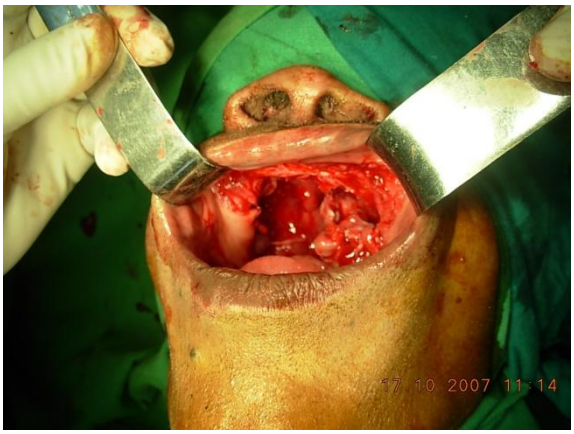

Fig-2: Surgical site showing denuded maxillary bone with vomer and maxillary sinus.

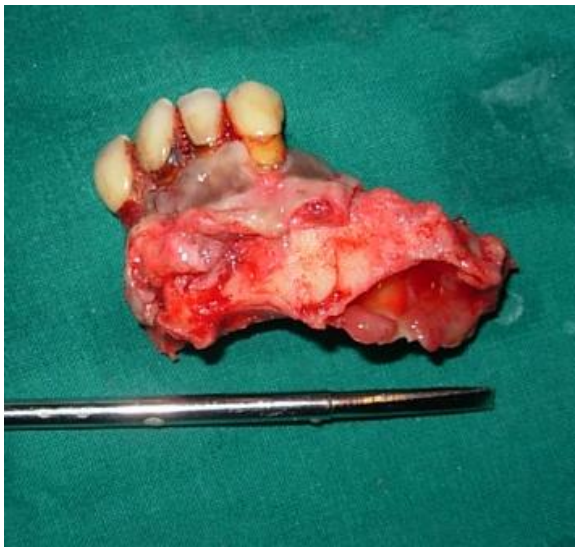

Fig-3: Excised segment of anterior maxilla

DISCUSSION

After going through previously documented literatures we got hold of only three case reports on traumatic maxillary necrosis. In one case reported by N Khan, patient had maxillary fracture and his recklessness towards the treatment lead into maxillary necrosis [1]. A case described by Cornah and O'Hare,1 the patient had extensive maxillary fractures associated with LeFort I and II-type fractures. This patient also developed maxillary infection. The maxilla was excised and appeared to have undergone complete necrosis [2]. In the other case, the patient sustained a Le Fort I-type fracture that was inadequately treated and resulted in a large maxillary defect [3].

Infections such as mucormycosis form thrombi within the blood vessels and result in decreased blood supply and necrosis [6]. Osteonecrosis from herpes occurs when the varicella zoster virus reactivates from latency and becomes an active viral replication. Our patient did not have any signs or symptoms of infections in his post-traumatic period and he was not immunocompromised. Malignancy of maxillary bone can also leads to a vascular necrosis, quite common is squamous cell carcinoma of maxillary sinus [15]. There are incidence where onco-radiation also resulted in aseptic necrosis of maxilla, which obliterates the arteriolar lumen and subsequent fibrosis of blood vessels. This ultimately results in avascular or aseptic necrosis [10, 13]. There were cases in past where lefort osteotomy for surgical correction skeletal deformities led to aseptic maxillary necrosis because of devoid of blood supply to the osteotomized site [5].

In recent times, there have been reports in medical literature supporting the association between bisphosphonate use and osteonecrosis [12]. This is most frequently reported in patients who are receiving bisphosphonates for metastatic disease. Bisphosphonates hamper resorption by osteoclasts and cause initiation of apoptosis [16]. The mandible is the most common bone affected followed by the maxilla. Osteonecrosis in patients taking bisphosphonate can show focal lytic changes on conventional radiographs and CT. A positive association is seen between osteonecrosis and length of intravenous bisphosphonate therapy, oral hygiene and dental extraction [12-14].

Treatment of aseptic necrosis initially involves good hygiene in the area, with frequent irrigation with saline. Ideally the patient should be treated with hyperbaric oxygen, and antibiotics should be considered to prevent secondary infection. A surgical debridement will be required to try to speed up the resolution of the necrotic process. Hyperbaric oxygen may hasten the delineation of the necrotic segments and allow a definitive debridement to be done at an earlier time. It will not, however, prevent or reverse the development of aseptic necrosis once it has started, but may limit its ultimate extent.

CONCLUSION

Trauma to the maxillofacial region is an unavoidable event; care must be taken for the early intervention of treatment. Complications can range from minor infection to complete necrosis of the affected site. Although necrosis of maxillae is one the rare entity but it is said that what is rare, that is hard to manage.

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