



A RESEARCH STUDY OF MANDIBULAR OSTEONECROSIS RELATED TO THE PULPAL-PERIODONTAL SYNDROME

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ABSTRACT

The creation of sequestrum, a segment of avital bone detached from adjacent sound bone as a result of local bone necrosis, is a symptom of osteonecrosis of the jaw is frequently linked to ischemia. In the majority of cases, known jaw osteonecrosis is linked to both local and systemic causes are already present. Systemic osteonecrosis of the jaws is a possibility is induced i.e. As a result of radiation treatment for head and neck cancers, or as a result of bisphosphonate therapy (either for osteoporosis or for other reasons or the treatment of cancers such multiple myeloma, etc.) As a result of corticosteroid medication, cocaine misuse, coagulation disorders, and medication intake that affects clotting systems. Diabetes, leukemia, Paget's disease of the bones, fibrous dysplasia, malnutrition, and heavy metal poisoning are all possible causes. Local causes of jaw necrosis include past trauma from dental treatment (extraction of third molars, rubber dam implantation, or injuries due to sports) to the use of chemicals in dentistry, etc. The patient in this case report signed an informed consent form that was authorized by the Ethics Committee of the University of Delhi's School of Dental Medicine, India. A 38-year-old woman arrived with an 8-mm diameter exposed necrotic bone region on the lingual side of her right lower third molar. The patient appeared to be in good health and was not on any drugs. Local trauma, recent dental treatments, bulimia, or self-inflicted reasons of the lesion were all denied by the patient. The patient had never been given any bisphosphonates or corticosteroids, and had never been irradiated. This example demonstrates how pulp necrosis, a type of pulpal-periodontal syndrome, can cause bone exposure in some patients. The extraction of the diseased tooth, excision of the necrotic bone and granulation tissue, and bone remodeling are all necessary treatments for osteonecrosis caused by the pulpal-periodontal condition.

Key words:- Mandibular Osteonecrosis, Pulpal-Periodontal Syndrome, Case study.

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INTRODUCTION

The creation of sequestrum, a segment of avital bone detached from adjacent sound bone as a result of local bone necrosis, is a symptom of osteonecrosis of the jaw is frequently linked to ischemia.

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clotting systems. Diabetes, leukemia, Paget's disease of the bones, fibrous dysplasia, malnutrition, and heavy metal poisoning are all possible causes. Local causes of jaw necrosis include past trauma from dental treatment (extraction of third molars, rubber dam implantation, or injuries due to sports) to the use of chemicals in dentistry, etc. Bulimia, or self-inflicted eating disorders. Lesions in people who have psychiatric problem. Sixty cases of severe osteonecrosis and osteomyelitis of the mandible and maxilla were described by Khullar et al. By coagulopathies and osteopetrosis, Gorham's disease, infectious diseases (tuberculosis, herpes zoster, HIV, and osteomyelitis), toxicity (bisphosphonates, spider bites), and environmental impacts such as phossy jaw and radium jaw), and toxicity (bisphosphonates, spider bites). Over the years, the occurrence of osteonecrosis due to various causes has increased in the last 7 years. As a result, we present a unique instance of pulpal-periodontal syndrome-induced osteonecrosis and the following treatment for necrosis of the pulp.

Case Presentation:

The patient in this case report signed an informed consent form that was authorized by the Ethics Committee of the University of Delhi's School of Dental Medicine, India. A 38-year-old woman arrived with an 8-mm diameter exposed necrotic bone region on the lingual side of her right lower third molar. The patient appeared to be in good health and was not on any drugs. Local trauma, recent dental treatments, bulimia, or self-inflicted reasons of the lesion were all denied by the patient. The patient had never been given any bisphosphonates or corticosteroids, and had never been irradiated. The results of a full blood count were within normal limits. The ortho pan tomograph in the right molar area of the lower jaw was normal, with no pathological alterations. A deep periodontal pocket was seen on CBCT between the second and third molars, indicating a persistent inflammatory process. On the lingual cortical plate, an exophytic bone island with an 8-mm diameter and a bone sequestrum were discovered. Plasma therapy and low-level laser therapy for the lesion proved ineffective. For the next three days, an adhesive periodontal cellulose dressing and betamethasone ointment in Orabase which is made up of gelatin, pectin, and sodium carboxymethylcellulose in a plasticized hydrocarbon gel were used. Both therapies, on the other hand, yielded no results. The patient was next given the antibiotic amoxycillin as a peroral antibiotic for a week, but no improvement was seen. As a result, surgical treatment was carried out. For regional nerve block anesthesia, two ampoules of articaine were employed. A free bone sequestrum was excised with a hemostat after a sulcular incision and elevation of the mucoperiosteal flap. Using a surgical hammer and chisel, the remaining exophytic bone was removed. A round steel surgical bur was used to complete the final modulation of the bone surface. The third molar was surgically removed because

there was a periodontal pocket (7 mm detected with a periodontal probe) between the lower right second and third molar. A swab of this area was collected. Histopathological study was performed on the bone and granulation tissue removed during the surgical treatment. Antimicrobial photodynamic therapy (aPDT) was used after the photo sensitizer was applied to diminish the microbiota at the surgical site using a low-power diode laser. Silk sutures with a round needle were utilized. Chronic inflammation was discovered after histopathological examination. On the other side of the bone sample, a newly produced loose connective tissue was discovered in a bone fragment (close to the post extraction socket). Grassy granulation tissue with mixed inflammatory infiltration, including a large number of histiocytes, mononuclear cells, and eosinophils, as well as some granulocytes. Eight days following surgery, the sutures were removed. During the healing period, the patient did not report any postsurgical complications or subjective clinical signs and symptoms. The incision and area of previously necrotic alveolar bone were completely epithelized on clinical inspection.

Discussion:

Traditionally, osteonecrosis of the jaw has been linked to irradiation of patients with head and neck cancers, as well as the use of bisphosphonates for osteoporosis or malignant disorders such multiple myeloma. Variables that cause jaw osteonecrosis are usually known, although there have been a few case reports in the literature where no known factors for osteonecrosis development could be identified. Various chemicals used in dentistry, such as acid etchants, arsenic paste, and paraformaldehyde-containing pastes, might harm the gingiva and alveolar bone during endodontic therapy. Intraosseous anaesthetic and extended rotation of the perforator drills in the bone are two dental methods that might cause bone necrosis. Other medical procedures, such as laryngoscopy and the installation of an endotracheal tube, may also cause osteonecrosis. Other drugs, in addition to bisphosphonates, have been identified as potential causes of jaw osteonecrosis. Sunitinib (an antiangiogenic medication) had been used to treat the patient's renal cell carcinoma. In addition, the patient was treated with antibiotics and physiotherapy for a period of 12 weeks, after which he recovered completely. Antineoplastic medicines that act through an antiangiogenic mechanism may cause or exacerbate osteonecrosis, particularly in individuals who are taking bisphosphonates. A case of jaw bone necrosis one week following excision of the mandibular molar in a patient with lung cancer with metastasis was described by Serra et al in a patient with lung adenocarcinoma with metastasis. The sequestra were linked with ulceration of the overlying mucosa that lasted from a few days to three months, as documented in other similar case reports. It's possible that local damage from eating or brushing

induced ischemia, which led to jaw necrosis in our case. Surgical therapy is generally not recommended because it may promote additional ischemia of the dense cortical bone, which is likely owing to a compromised vascular supply. When possible, avital bone is allowed to sequester on its own. A bone fragment might be left to exfoliate naturally or carefully removed (1). Antibiotics, vasodilators, hyperbaric oxygen, and surgical debridement are commonly used to treat the patients, although the results are uneven.

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Conclusion:

This example demonstrates how pulp necrosis, a type of pulpal-periodontal syndrome, can cause bone exposure in some patients. The extraction of the diseased tooth, excision of the necrotic bone and granulation tissue, and bone remodeling are all necessary treatments for osteonecrosis caused by the pulpal-periodontal condition TABLE 4.

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