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EXPLANTATION BY OSTEONECROSIS AFTER WISDOM TOOTH EXTRACTION

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ABSTRACT

Defined as "death of bone tissue due to lack of blood supply", osteonecrosis or aseptic necrosis is a pathological condition associated with predisposing factors such as the use of medications, radiotherapy therapies, alcoholism, and idiopathic alterations [1,2]. Studies show that this pathology is often associated with long bones, affecting the femur with a high incidence, followed by gnathic bones, whose inflammatory processes are exacerbated by a wide microbiota presented in the oral cavity [2,3].

Keywords: Explantation. Osteonecrosis. Wisdom tooth extraction. Dental Complications.

INTRODUCTION

Defined as "death of bone tissue due to lack of blood supply", osteonecrosis or aseptic necrosis is a pathological condition associated with predisposing factors such as the use of medications, radiotherapy therapies, alcoholism, and idiopathic alterations [1,2].

Studies show that this pathology is often associated with long bones, affecting the femur with a high incidence, followed by gnathic bones, whose inflammatory processes are exacerbated by a wide microbiota presented in the oral cavity [2,3].

In gnathic bones, osteonecrosis is more commonly present in the mandible, since it has anatomical and physiological characteristics that give it a lower blood supply and greater bone density, being affected in 65% of cases in maxillary detention with 26% [4,5].

Osteonecrosis with continuous use drugs, such as antiresorptive drugs (bisphosphonates and denosumab) and antigens, are used in patients with skeletal disorders that induce bone loss or metastases associated with primary tumor [6].

Although they are drugs that inhibit osteoclast function and consequent decrease in bone remodeling, bisphosphonates and denosumab have different mechanisms of action, since the latter is an antibody and thus has a better distribution throughout the bone space, thus presenting advantages in treatment, especially in postmenopausal osteoporosis [6,7.]element.

Another difference observed is related to the potential for osteonecrosis formation, where studies show that patients submitted to extraction using bisphosphonates had poor bone remodeling, while denosumab, being an antibody, is transient, and as long as it is suspended, it is capable of allowing bone remodeling, since its half-life is short [8,9,] element.

The epidemiological profile of osteonecrosis of the jaws is uncertain, since its frequency is related to the duration of drug use, the molecule of origin, the dosage, and the route of administration. In relation to gender, it is more frequent in women, due to hormonal factors [10,11].

It can be stated that the highest prevalence is in patients who use bisphosphonates with intravenous administration (0.10%), when compared to oral use with 0.001% of the total cases [11,12].

The diagnosis in most cases is related to the signs and symptoms found and associated with the history of antiresorptive drug use [12]. Sometimes, it is necessary to use imaging tests such as radiographs or computed tomography, in order to measure the degree of extension and quality of remaining healthy bone [12,13].

The main clinical feature associated with osteonecrosis includes an area of ulcerated mucosa with exposed bone without vitality. It also presents signs of infection with local pain

and strong breath [14].

With the progression of the disease, an irregular bone surface and probable pathological fractures may be observed during chewing [15].

Radiographically, it presents with variations from radiolucent to radiopaque lesions, which are detectable only in more advanced stages, where it presents with an area of misshapen bone sequestration, surrounded by a radiolucent allus with well-defined boundaries [16,17,18]. As it is a pathology with infectious characteristics, professionals should be aware of possible differential diagnoses, among which alveolar osteitis, infectious osteomyelitis, periodontal diseases, and osteoradionecrosis can be highlighted18,19.

Specific alterations of the oral cavity can act as "triggers" for the development of drug osteonecrosis, such as the presence of active infectious processes such as carious lesions, periodontal diseases, or any alteration that may trigger an infectious process [2,3].

Preventive measures should be taken, whenever possible, before starting treatment with antiresorptive drugs, such as thorough evaluation and adequacy of the oral cavity medium [5,6].

The treatment of osteonecrosis differs as to the best therapy to be employed. Studies indicate that the combination of surgical techniques with adjuvant therapies is effective [7]. Surgical procedures must be well planned in advance, as they will generate new tissue trauma in regions with impaired healing [8,9]. Whenever possible, less invasive surgical procedures such as debridement and bone curettage associated with a broad-spectrum antimicrobial should be prioritized [6].

Alternative methods such as photodynamic therapy, hyperbaric chamber and the use of Platelet and Leukocytes-Rich Fibrin (L-PRF) have been shown to be effective in the treatment of early stages of the disease, as well as in association with surgical procedures [16,17].element.

LPRF is a concentrate of platelets and leukocytes that has a modulating capacity in the bone repair process by releasing growth factors. It acts to control inflammatory processes, stimulating osteogenesis and helping to repair compromised bone tissue [18,19].

CLINICAL CASE DESCRIPTION

Patient R.S.B.K., 78 years old, female, sought the private clinic for evaluation after the evolution of an abscess on the left side of the face. After clinical examination, a computed tomography scan was requested to aid the diagnosis, since of the relevant data, only one extraction of element 28 had been performed 8 months earlier. The patient reported that local discomfort persisted in the region of the third molar until the moment of

the evolution of the pathological condition. In the medical history, it was found that the patient had been affected and treated for breast cancer approximately 10 years ago. She used a chemotherapy drug, Aromasim, for 5 years. 3 years ago, she was affected by a pathological fracture of the rib due to osteoporosis, starting the use of Denusunab On intraoral clinical examination, a strong painful sensitivity was evidenced on palpation of the maxillary rim of the 28 and discretely on the buccal surface of the implant 27. After removal of the implant crown 27, a peri-implant probe confirmed marked exposure of the expirations and in a smaller number of the implant 26; and, communication with the alveolus of element 28 (Figures 1a. and 1b). On CT scans, the presence of the lamina dura and bone rarefaction on the alveolar border were observed (Figures 2 a and 2b). With the diagnosis of drug osteonecrosis due to denosumab confirmed, the surgical planning was organized with a previous prescription of amoxicillin with clavulanate and non-steroidal anti-inflammatory drugs. The implants were removed and the entire bone region involved was decorated (Figure 3. The suture was performed after the installation of L-PRF (Figure 4). After 2 weeks, the surgical wound evolved with suture dehiscence, exposing the yellowish face of the necrotic bone. There was no spontaneous discomfort. Only during palpation in the palatine and vestibular region (Figure 5).

Figure 1. Peri-implant and alveolar catheterization.

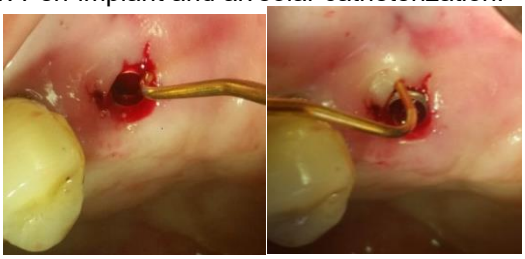


Figure 2. Panoramic image and tomographic sections.

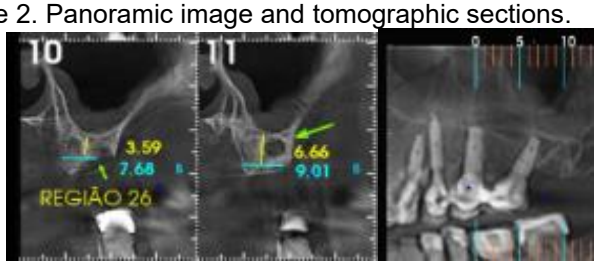


Figure 3. Explanting with decortication .a. LPRF installation.

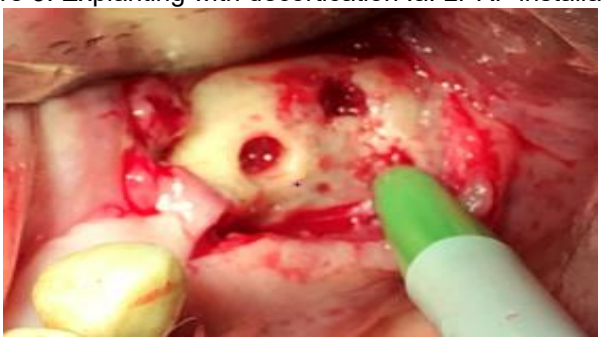
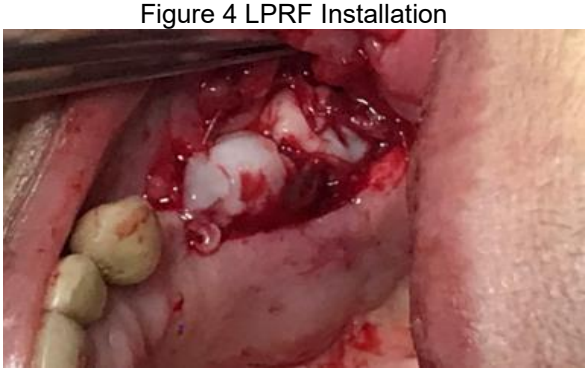
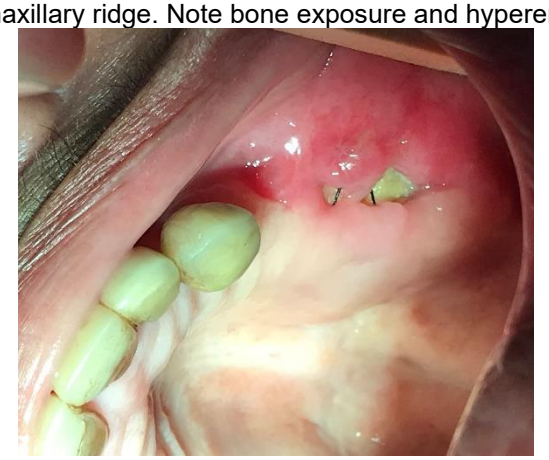


Figure 4 LPRF Installation



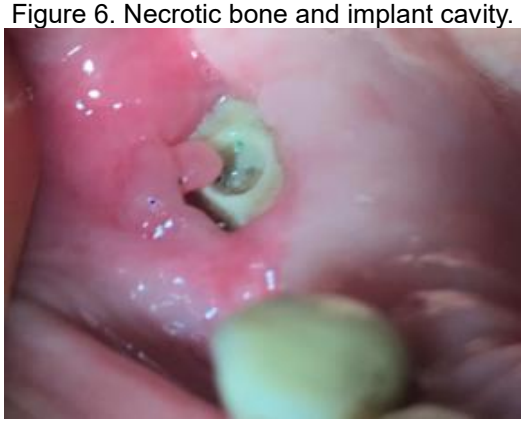
Due to the worsening of the patient's systemic condition with the appearance of liver metastasis, a surgical reapproach was postponed upon evaluation and release by the medical team. Palliative care through 0.9% SF irrigation, 0.12% chlorhexidine gloconate mouthwash, and 10 sessions of lasertheapia relieved symptoms. Thus, after 60 days, there was an increase in bone fenestration, evidencing the implant cavity 25 (Figure 6).

Figure 5. View of the maxillary ridge. Note bone exposure and hyperemia of the oral mucosa.



After palliative care was maintained, the evolution of fenestration remained stable despite the degenerative aspect of the exposed bone after 120 days (Figure 7).

Figure 6. Necrotic bone and implant cavity.



The recurrent and persistent use of mouthwashes such as 0.12% chlorhexidine gluconate and cepacol required a change due to the complaint of decreased taste. Therefore, the use of a mouthwash of Dutch origin called Blue M with a high concentration of active oxygen, Xylitol and lactoferric, whose joint action prevents bacterial growth, was instituted. Once the taste complaint was reestablished and without medical clearance for surgical intervention, the fenestration evolved, exposing a little more bone in the maxillary rim with the appearance of a worn necrotic 150 days after the operation. (Figure 7).

After 180 days of the initial intervention, a soft tissue repair without bone adhesion was observed during periodontal probing, allowing the observation of a granulation tissue isolating the bone segment, transforming it into a sequestration. It is worth noting that since denosumab has a half-life of 6 months, it is possible to suggest that its active ingredient declined, due to the increase in the osteoclatic activity of monocytes. Thus, it was possible to highlight bone sequestration without major sequelae and without the involvement of the maxillary sinus (Figures 8a and 8b)

Figure 5.Appearance of bone degeneration

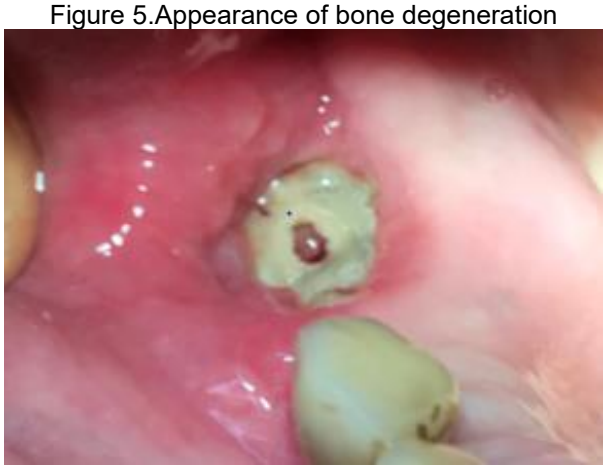


Figure 6.Bone exposure with decreased hyperemia

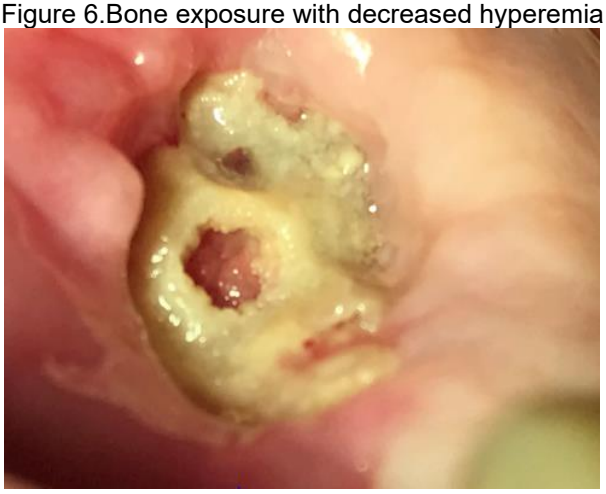


Figure 8a. Kidnapping removed. 8B after 10 days

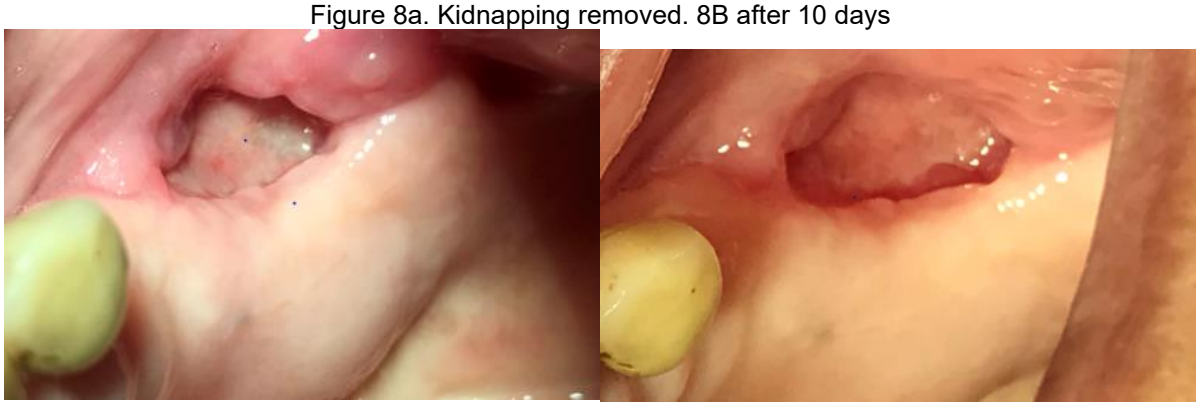
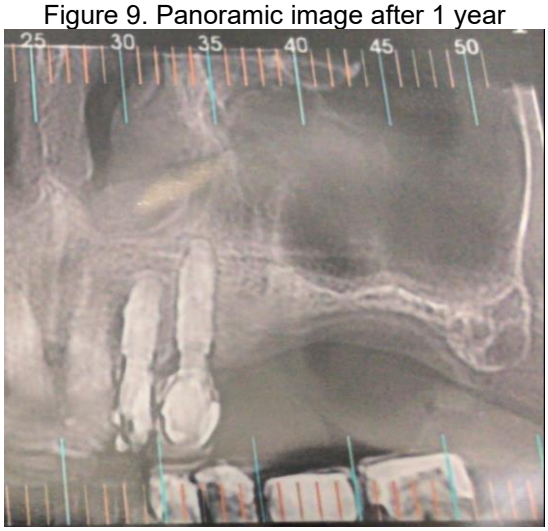
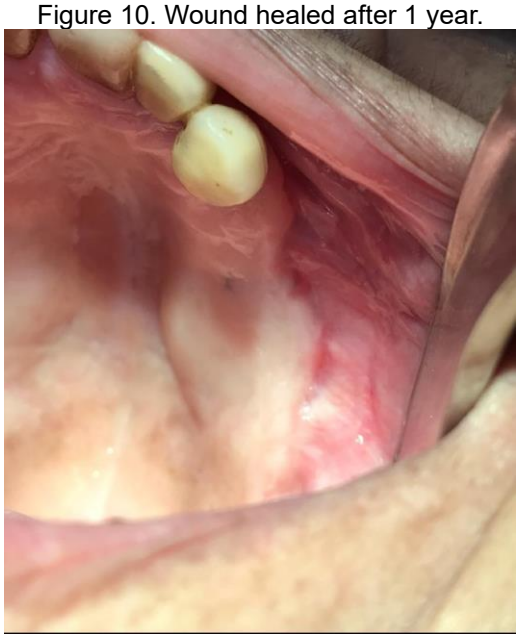


Figure 9. Panoramic image after 1 year



One year after the first surgical intervention, the operated region is in an excellent state of healing (Figure 9), with bone support on the floor of the maxillary sinus (Figure 10).

Figure 10. Wound healed after 1 year.



FINAL CONSIDERATIONS

Long- and short-term oral anti-resorptive drugs are currently a preventive alternative against osteoporosis, and are widely disseminated in the medical field. However, the lack of guidance regarding the adequacy of oral health is increasing the incidence of osteonecrotic lesions of odontogenic origin, and especially of traumatic dental interventions. The dental surgeon needs to perform an accurate clinical examination in order to detect patients using antiresorptive drugs, perform the appropriate technique or refer them to the most experienced professional.

REFERENCES

1. Aljohani, S., Troeltzsch, M., Hafner, S., Kaeppler, G., Mast, G., & Otto, S. (2019). Surgical treatment of medication-related osteonecrosis of the upper jaw: Case series. Oral Diseases, 25, 497–507.

2. Aljohani, S., Gaudin, R., Weiser, J., Tr, M., Ehrenfeld, M., Kaeppler, G., & Otto, S. (2018). Osteonecrosis of the jaw in patients treated with denosumab: A multicenter case series. Journal of Cranio-Maxillo-Facial Surgery, 46, 1515–1525.

3. Brozoski, M. A., Traina, A. A., Cristina, M., & Deboni, Z. (2012). Osteonecrose maxilar associada ao uso de bisfosfonatos. Revista Brasileira de Reumatologia, 52(2), 260–270.

4. Cano-Durán, J. A., Peña-Cardelles, J. F., Ortega-Concepción, D., Paredes-Rodríguez, V. M., García-Riart, M., & López-Quiles, J. (2017). The role of leucocyte-rich and platelet-rich fibrin (L-PRF) in the treatment of the medication-related osteonecrosis of the jaws (MRONJ). Journal of Clinical and Experimental Dentistry, 9(8), e1051.

5. Dahiya, N., Khadka, A., Sharma, A. K., Gupta, A. K., Singh, N., & Brashier, D. B. (2015). Denosumab: A bone antiresorptive drug. Med J Armed Forces India, 71(1), 71-75.

6. Grisar, K., Schol, M., Schoenaers, J., Dormaar, T., Coropciuc, R., & Vander, V. P. (2016). Osteoradionecrosis and osteonecrosis of the jaw: Similarities and differences. International Journal of Oral & Maxillofacial Surgery, 45(12), 1592–1599.

7. Khan, A. A., Morrison, A., Hanley, D. A., Felsenberg, D., Mccauley, L. K., Ryan, F. O., & Brown, J. P. (2015). Diagnosis and management of osteonecrosis of the jaw: A systematic review and international consensus. Journal of Bone and Mineral Research, 30(1), 3–23.

8. Lo-lin, T., Yu-feng, H., & Yu-Chao, C. (2016). Treatment of bisphosphonate-related osteonecrosis of the jaw with platelet-rich fibrin. Journal of the Formosan Medical Association, 115(7), 585–586.

9. Manzano-Moreno, F. J., Ramos-Torrecillas, J., De Luna-Bertos, E., Ruiz, C., & García-Martínez, O. (2015). High doses of bisphosphonates reduce osteoblast-like cell proliferation by arresting the cell cycle and inducing apoptosis. Journal of Craniomaxillofacial Surgery, 43, 396–401.

10. Maluf, G., Caldas, R. J., & Santos, P. S. S. (2017). The use of leukocyte- and platelet-rich fibrin (LPRF) in the treatment of medication-related osteonecrosis of the jaws (MRONJ). Journal of Oral and Maxillofacial Surgery, 75(9), 1795–1802.

11. Mcleod, N. M. H., Brennan, P. A., & Ruggiero, S. L. (2012). Bisphosphonate osteonecrosis of the jaw: A historical and contemporary review. The Surgeon, 10(1), 36–42.

12. Nicolatou-Galitis, O., Schiødt, M., Amaral Mendes, R., Ripamonti, C., Hope, S., Drudge Coates, L., & Van den Wyngaert, T. (2019). Medication-related osteonecrosis of the jaw: Definition and best practice for prevention, diagnosis, and treatment. Oral Surgery, Oral Medicine, Oral Pathology, and Oral Radiology, 127(2), 117–135.

13. Ramaglia, L., Guida, A., Iorio-Siciliano, V., Cuozzo, A., Blasi, A., & Sculean, A. (2018). Stage-specific therapeutic strategies of medication-related osteonecrosis of the jaws: A systematic review and meta-analysis of the drug suspension protocol. Clinical Oral Investigations, 22, 597–615.

14. Ribeiro, G. H., Chrun, E. S., Dutra, K. L., Daniel, F. I., Grando, L. J., & A. (2018). Osteonecrosis of the jaws: A review and update in etiology and treatment. Brazilian Journal of Otorhinolaryngology, 84(1), 102–108.

15. Rosella, D., Papi, P., Giardino, R., Cicalini, E., Piccoli, L., & Pompa, G. (2018). Medication-related osteonecrosis of the jaw: Clinical and practical guidelines. Journal of International Society of Preventive & Community Dentistry, 6(2), 97–104.

16. Sharma, D., Ivanovski, S., Slevin, M., Hamlet, S., Pop, T. S., Brinzaniuc, K., Petcu, E. B., & Miroiu, R. (2018). Bisphosphonate-related osteonecrosis of jaw (BRONJ): Diagnostic criteria and possible pathogenic mechanisms of an unexpected anti-angiogenic side effect. Vascular Cell, 5, 1–8.

17. Souza, L. N., De Cristina, A., Antunes, R., Flávia, V., Mari, A., Paula, A., & Alvarenga, R. L. (2009). Osteonecrose dos maxilares associada ao uso de bisfosfonatos: Revisão da literatura e apresentação de um caso clínico. Revista Portuguesa de Estomatologia, Medicina Dentária e Cirurgia Maxilofacial, 50(4), 229–236.

18. Viana, M. V. G., Carvalho, M. M. M., Fialho, P. V., Cardoso, L. C., Lasso, D. M. M., & Moreira, C. V. A. (2019). Considerações clínicas sobre o uso de L-PRF na terapêutica de osteonecrose medicamentosa dos maxilares: Relato de caso. Brazilian Journal of Health Review, 2(4), 3318–3327.

19. Woo, S., Hellstein, J. W., & Kalmar, J. R. (2006). Annals of internal medicine review systematic review: Bisphosphonates and osteonecrosis of the jaws. Annals of Internal Medicine, 144(10), 753–761.