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Case Report

Medication-related Osteonecrosis of the Jaw: An Emerging Adverse Effect of Rituximab Therapy – A Case Report

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SUMMARY

Medication-related osteonecrosis of the jaw (MRONJ) is traditionally linked to antiresorptive and antiangiogenic therapies. However, recent evidence suggests that biologic agents, including monoclonal antibodies (mAbs), may also be associated with osteonecrosis. This study reports a case of MRONJ following Rituximab therapy in a patient after treating a non-Hodgkin lymphoma, emphasizing the emergence of this adverse effect in non-antiresorptive protocols. A female patient, following R-CHOP therapy, presented with a painless lesion characterized by bone exposure and suppuration in the periapical region of teeth 43 and 44. Initial management combined professional prophylaxis, antiseptic therapy, debridement, and endodontic treatment under antibiotic coverage. However, the lesion proved refractory, resulting in persistent suppuration and spontaneous bone sequestration after 5 months, which was subsequently treated surgically with adjuvant antibiotic therapy and photobiomodulation. Histopathological examination successfully ruled out malignancy. This clinical outcome highlights the increased susceptibility to bone necrosis in patients undergoing this specific immunotherapy regimen. Ultimately, the early identification of this non-antiresorptive-induced bone necrosis pathobiology is essential to facilitate conservative management and minimize the requirement for extensive surgical interventions.

Keywords

Medication-related osteonecrosis of the jaw; Rituximab; Lymphoma; Monoclonal

Antibody; Non-antiresorptive

Statement of Clinical Significance

This report highlights the risk of mandibular osteonecrosis in patients treated with Rituximab, independent of bisphosphonate use. We conclude that preventive dental screening and continuous oral monitoring should be integrated into standard oncology protocols. Such surveillance enables early diagnosis, minimizes disease progression, and averts radical surgical interventions.

INTRODUCTION

Osteonecrosis involves the death of bone tissue, ultimately leading to structural destruction, loss of function, and progressive pain, often exacerbated by secondary infection[1]. In the maxillofacial region, osteonecrosis is traditionally associated with antiresorptive therapies, either alone or combined with immunomodulators or

antiangiogenic agents. Clinically, the diagnosis is established by exposed bone or a fistulous tract probeable to bone that persists for more than 8 weeks[2]. Although medication-related osteonecrosis of the jaw (MRONJ) typically correlates with antiresorptive exposure, emerging evidence indicates that monoclonal antibodies, such as rituximab, may trigger bone necrosis independently [2–5].

Rituximab is a chimeric monoclonal antibody targeting the CD20 antigen on B cells, inducing cell death via complement-mediated lysis, antibody-dependent cytotoxicity, and apoptosis induction [6]. As part of the R-CHOP (rituximab, cyclophosphamide, doxorubicin, vincristine, and prednisone) protocol, rituximab is central to the gold-standard treatment for diffuse large B-cell lymphoma (DLBCL) [7].

Concurrently, a growing number of reports have linked its administration to jaw osteonecrosis. Therefore, this study aims to present a case report of MRONJ associated with rituximab therapy in a patient treated for DLBCL.

CASE REPORT

The study protocol was approved by the Ethics Committee of Pedro Ernesto University Hospital (CAAE: 93170325.7.0000.5259) and is in accordance with the declaration of Helsinki of 1975 and its later amendments.

A 45-year-old female patient was diagnosed with DLBCL in 2023 and underwent R-CHOP therapy from December 2023 to May 2024. Despite achieving complete remission, as confirmed by Positron Emission Tomography scan (PET-CT), she developed a spontaneous oral lesion with bone exposure five months post-immunotherapy. Her medical history is significant for hypertension, hepatitis A, and coronary artery disease requiring stent placement following a post-COVID myocardial infarction in 2022. The clinical presentation of exposed bone without prior radiotherapy or bisphosphonate use led to a suspected diagnosis of rituximab-related osteonecrosis of the jaw.

Clinical and radiographic examination revealed periodontal health in a reduced periodontium, negative pulp vitality in both teeth (43 and 44), and a painless area of exposed bone with active suppuration at the apical region of teeth 43 and 44 (Figure 1 and 2).

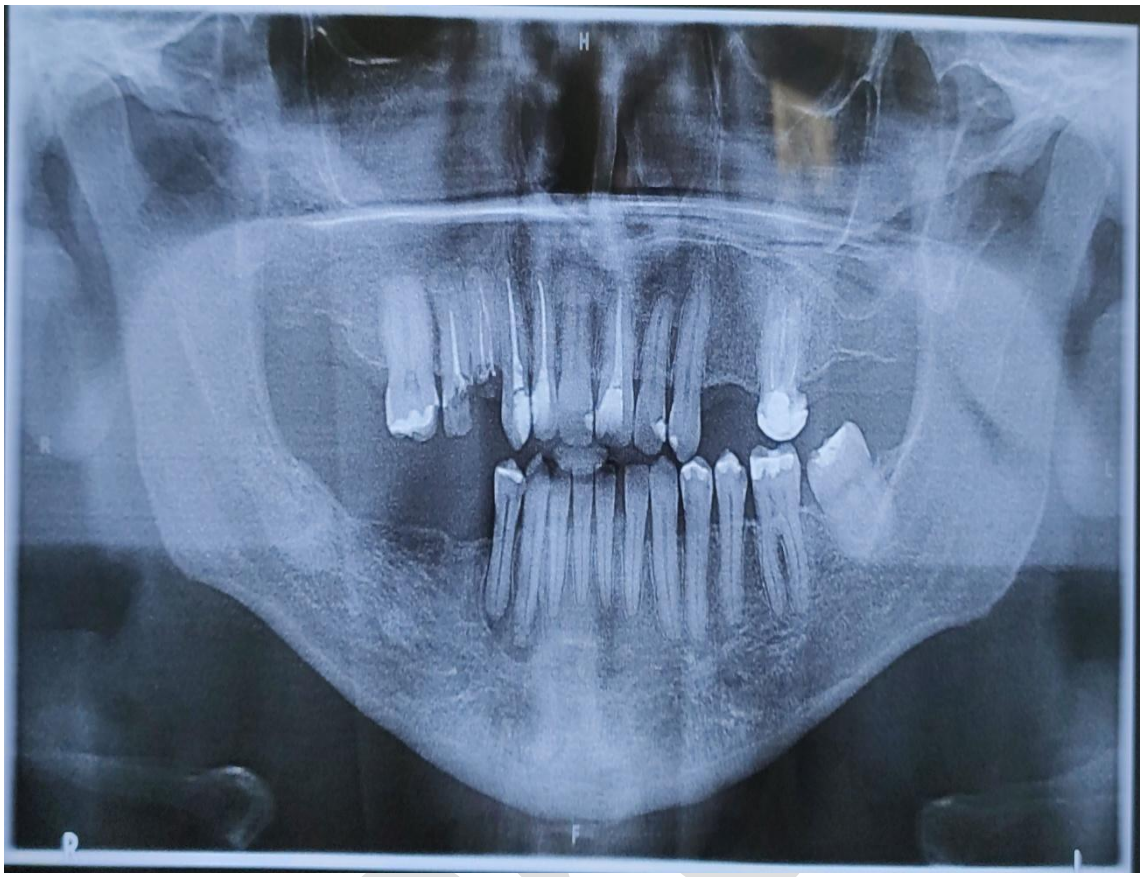

Image 1 - Initial panoramic radiograph showing periapical lesion affecting teeth 43 and 44.

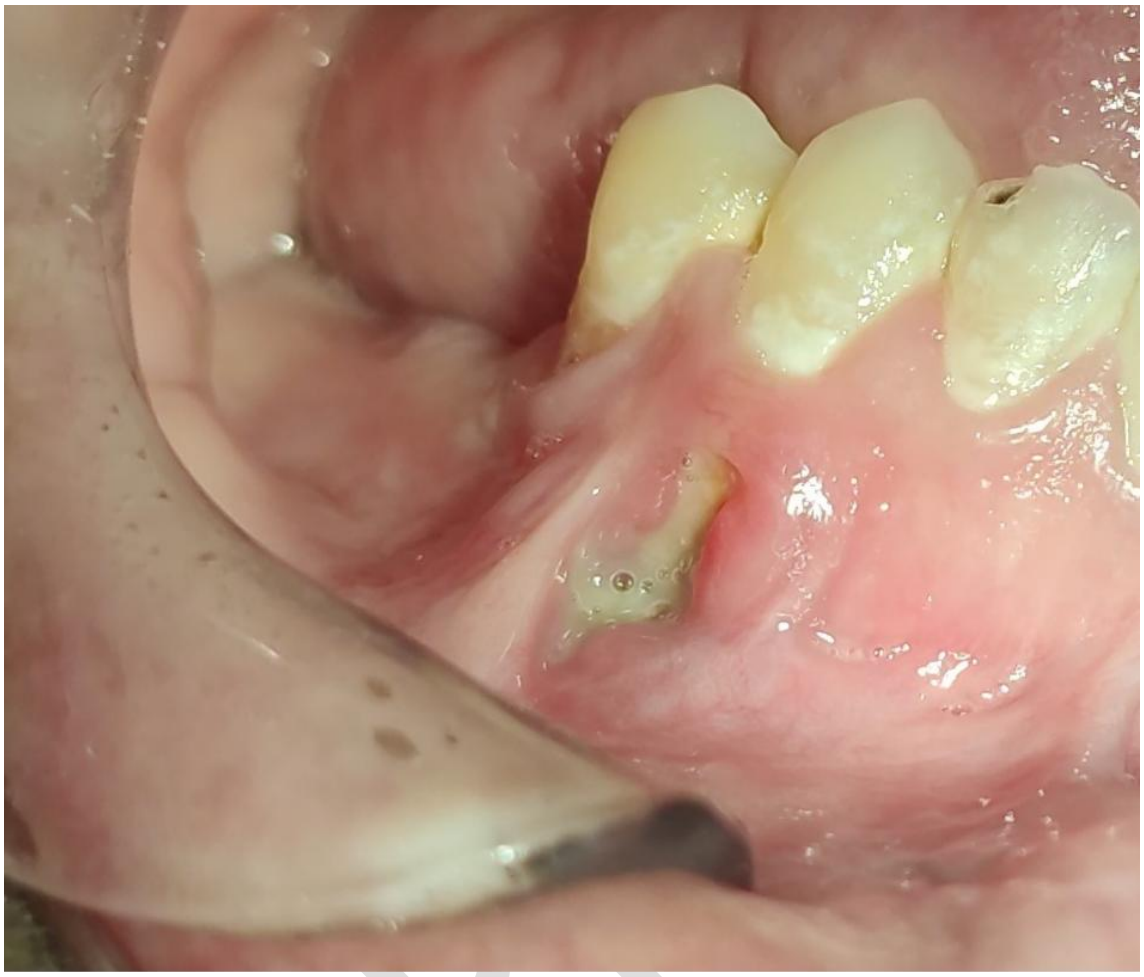

Image 2 – Clinical aspect of the initial osteonecrotic lesion in the region of 43 and 44.

Initial management consisted of supragingival scaling and a 14-day antiseptic protocol using 0.12% chlorhexidine mouthwash and 0.2% topical gel. Given the patient's immunocompromised status and the presence of active infection, surgical debridement and endodontic therapy were performed under antibiotic prophylaxis (2 g amoxicillin). Endodontic procedures utilized a reciprocating system (MK Life) with 2.5% sodium hypochlorite and 17% EDTA irrigation. Despite rigorous disinfection, lateral condensation obturation, and composite resin restoration, the lesion remained refractory. Cone-beam computed tomography (CBCT) revealed extensive periapical bone loss and buccal cortical plate destruction at teeth 43 and 44 (Figure 3).

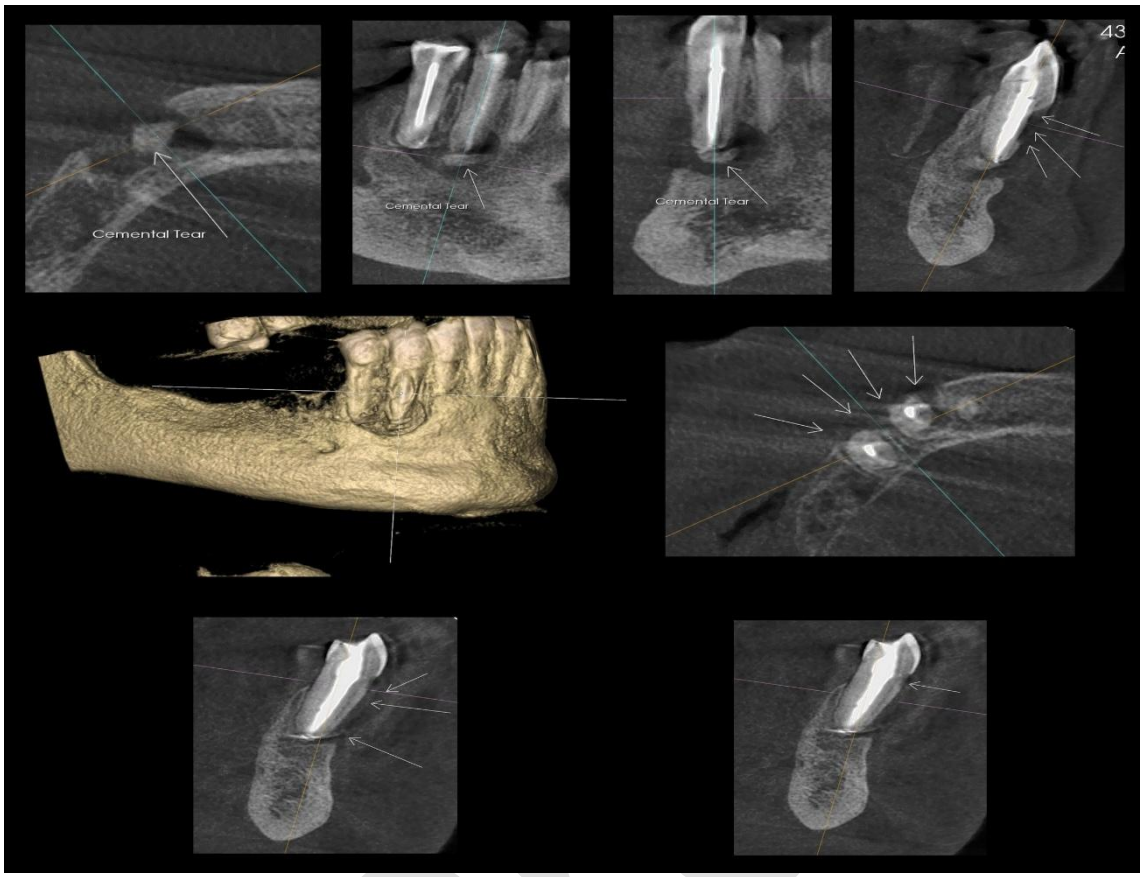

Image 3 – Cone beam computed tomography (CBCT) of the area showing alveolar bone loss in the periapical region of teeth 43 and 44.

A 5-month reassessment revealed persistent purulent exudate and the spontaneous expulsion of a bone sequestrum (Figure 4),

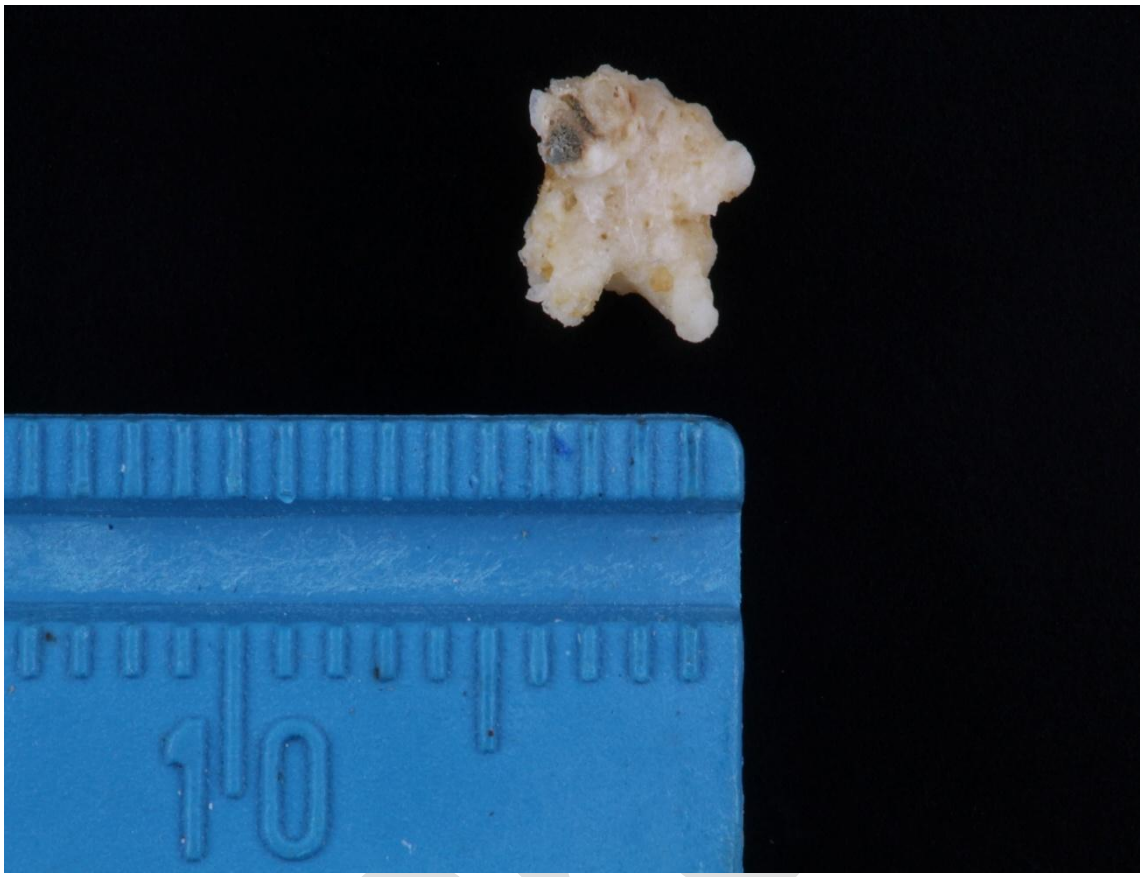

Image 4 - Bone sequestration that spontaneously expelled through the lesion.

confirming the progression of the necrotic process. Based on this clinical course and the patient's medical history, conventional osteomyelitis was ruled out, confirming the diagnosis of medication-related osteonecrosis of the jaw (MRONJ), in alignment with the American Association of Oral and Maxillofacial Surgeons (AAOMS) guidelines.

Due to the extent of the necrotic process, both teeth were extracted under antibiotic prophylaxis (2g amoxicillin), followed by thorough surgical debridement and primary closure. Histopathological analysis of the periradicular curettage and mucosal biopsy confirmed an inflammatory process and, crucially, ruled out lymphoma recurrence. Postoperative management included a 10-day course of amoxicillin (500mg tid), 3 days of dexamethasone (4mg bid), and topical chlorhexidine gel.

Five days post-extraction, the patient initiated red Photobiomodulation Therapy (PBMT) (660 nm, 100 mW, 2 J per point, 10 J/cm²) for 10 seconds at each point (Laser DMC Therapy XT – Brazil), nine additional sessions of PBMT were performed over the following 15 days. Sutures were removed on the seventh postoperative day. At the 30-day follow-up, clinical examination confirmed complete mucosal healing and total regression of the inflammatory process, with no signs of residual bone exposure (Figure 5).

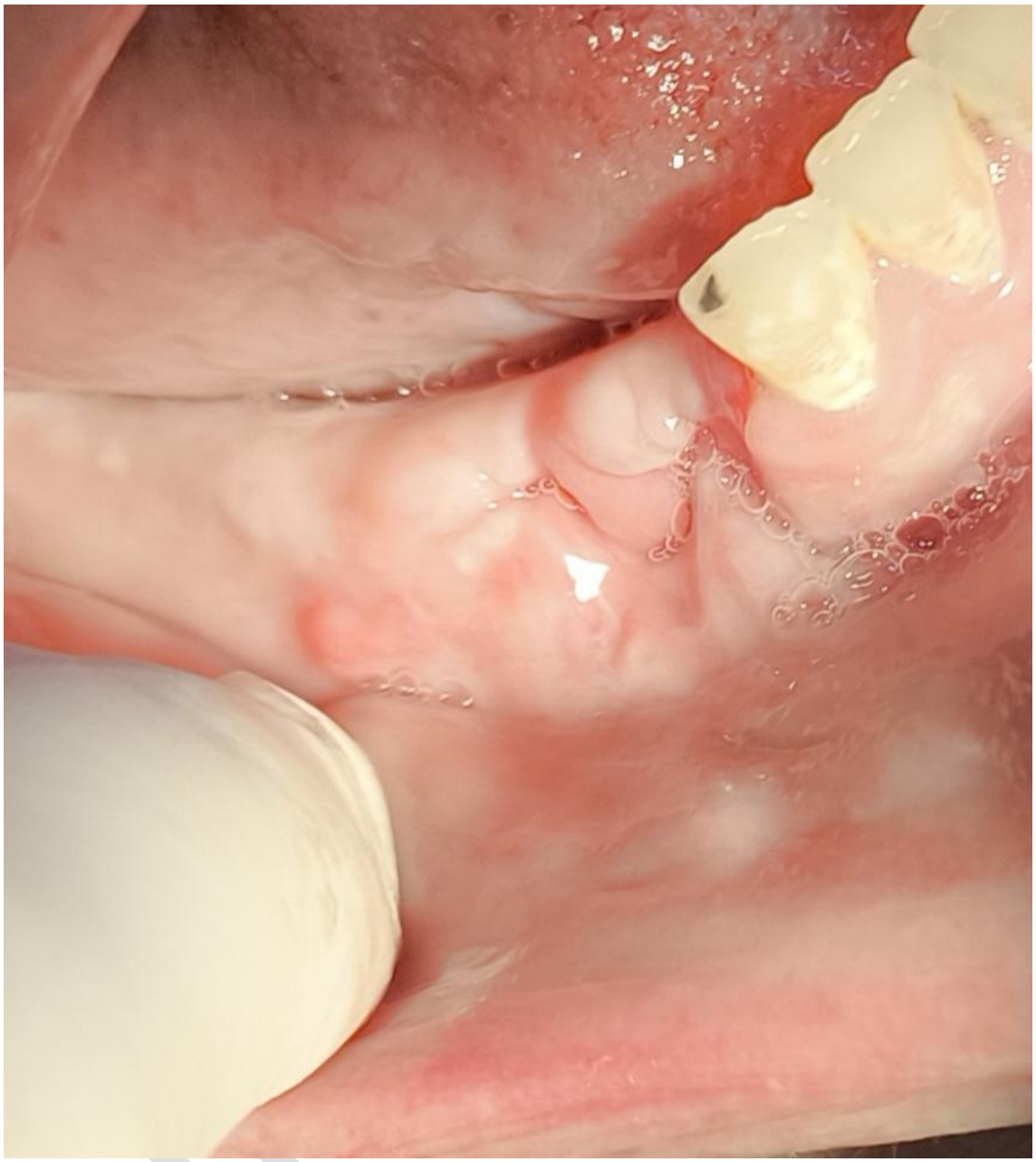

Image 5 - Clinical view 1 month after surgery showing mucosal healing of the area.

DISCUSSION

Several studies have reported an association between osteonecrosis of the jaw and various drugs, including humanized and chimeric monoclonal antibodies (mAbs), either with or without concurrent bisphosphonates use. Reported agents include denosumab, nivolumab, adalimumab, and rituximab[2–5]. Table 1

Table 1 – literature review of ostecrosis after treatment with Rituximab

Case Report	Age (in Years)	Sex	Context	Treatment	Symptoms
Marques et al, 2025 (present case)	45	Female	Diffuse large B-cell lymphoma	R-CHOP (rituximab, cyclophosphamide, doxorubicin, vincristine, and prednisone)	Oral lesion with exposed bone
Saran et al, 2024	52	Female	Diffuse large B-cell lymphoma	R-CVP (rituximab, cyclophosphamide, vincristine, and prednisolone)	Breathing difficulties and back pain
Javelot et al, 2019	69	Female	Rheumatoid arthritis	prednisolone, leflunomide, valsartan, vitamin D3, rituximab and history of residronate use 2 years prior (for 14 months from 2006 to 2008)	Oral lesions suggestive of acute herpetic gingivostomatitis
Ashtari et al, 2019	30	Female	Devic's syndrome and herpetic encephalitis	Prednisone, Azathioprine and Rituximab	Large necrotic palatal lesion
Gonzalez-Lugo et al, 2018	70	Female	Lymphoma stage IB_E	MIR protocol with a combination of rituximab and radiation therapy	Mild bleeding and pain in the right jaw, with gum ulceration and exposed bone
Aghaloo et al, 2017	52	Female	Non-Hodgkin lymphoma	Rituximab, bendamustine and prednisone	Foul taste with left cheek swelling.
Keribin et al, 2017	69	Male	Post-transplantation lymphoproliferative disorder – after kidney transplantation	Rituximab, loratadine and prednisone.	ONJ at site of initial oral ulcerations
Weigert et al, 2014	39	Male	B-cell non-Hodgkin's lymphoma	R-CHOP (rituximab, cyclophosphamide, doxorubicin, vincristine, and prednisone)	Lesions in the interdental papilla of the jaws.

Allegra et al, 2013	56	Not reported	Lymphoplasmacytic lymphoma	R-CVP (rituximab, cyclophosphamide, vincristine, and prednisolone)	Mucosal ulceration after tooth extraction
Baur et al, 2012	58	Male	Diffuse Large B-cell lymphoma.	R-CHOP (rituximab, cyclophosphamide, doxorubicin, vincristine, and prednisone)	Pain in the left mandible and paresthesia

provides a concise review of previously reported cases of osteonecrosis of the jaw following rituximab therapy.

The clinical trajectory reported by Baur et al. (2012)[8] closely parallels our findings, where a DLBCL patient developed mandibular MRONJ following the same therapeutic protocol. A critical divergence, however, remains the diagnostic latency; while their patient presented after one year with extensive mandibular involvement, our patient's early referral within months allowed for localized management at teeth 43-44. This correlation between early intervention and limited disease progression is further supported by Weigert [9] et al. and extends beyond DLBCL, as seen in Allegra[10] et al.'s report of rituximab-induced MRONJ in lymphoplasmacytic lymphoma.

Reports of rituximab-related MRONJ span multiple pathologies, including lymphoproliferative disorders and rheumatoid arthritis[3,11]. However, the interpretation of some cases is clouded by prior risedronate exposure or concomitant radiotherapy, which are established risk factors[3,12]. Our report aligns with the findings of Keribin et al.[11] and Aghaloo et al.[13], where MRONJ occurred independently of these factors. This suggests a distinct pathogenic pathway for rituximab, which in extreme cases, as documented by Saran et al.[14], can manifest as diffuse systemic osteonecrosis affecting the axial skeleton and long bones.

Rituximab is a monoclonal antibody (mAb) that targets the CD20 antigen on normal and malignant B cells, triggering cell death through complement-mediated lysis, antibody-dependent cell-mediated cytotoxicity (ADCC), and the direct induction of apoptosis [6]. Although infusion reactions and opportunistic infections are its main documented risks [15], a growing body of evidence suggests that rituximab-induced B-cell depletion may disrupt bone homeostasis [3,8,10,11,14]. This disruption occurs because B cells account for the largest production of osteoprotegerin (OPG) in the bone marrow microenvironment, which regulates bone resorption by binding to RANKL and inhibiting osteoclastogenesis [16]. Furthermore, studies indicate the occurrence of late-onset neutropenia weeks or months following rituximab therapy. Although its pathophysiology is not yet fully understood, evidence suggests that disturbances in the SDF-1/CXCL12 chemokine axis may interfere with the immune response [17]. Consequently, since neutrophils play a fundamental role in the oral cavity, episodes of late neutropenia can reduce the capacity to respond to local infectious foci [17]. This impairment may favor the persistence of inflammation and infection, creating an

environment conducive to the progression of osteonecrosis, particularly as pre-existing dental or periodontal infections increase the risk of MRONJ [18]. In addition, the concomitant use of corticosteroids in therapy for DLBCL contributes to the increased apoptosis of osteoblasts and osteocytes, which causes the collapse of the osteocyte-lacunar-canalicular system and vascular space causing local ischemia, thereby inducing bone necrosis[19]. Together, these combined factors impair bone remodeling capacity, leading to medication-related osteonecrosis of the jaw (MRONJ) emerging as a significant, albeit underrecognized, adverse effect of this immunotherapy.

The patient's treatment was tailored based on scientific evidence and her specific clinical status. While the use of antibiotic therapy and antimicrobial mouthwashes is well established for MRONJ prevention, their therapeutic employment during active osteonecrosis management is generally indicated from stage II of the disease onwards [2]., as demonstrated in the present study. There is, however, no consensus regarding the optimal duration of this antimicrobial regimen, with studies varying between one week [20], 10 days [21], 14 days [22], or until complete mucosal healing is achieved [23]. Regarding PBM, it represents a valuable therapeutic resource capable of modulating pain [24], stimulating cell proliferation [25], and enhancing angiogenesis [26]. In fact, two recent literature reviews indicated that complementary PBM therapy potentiated clinical outcomes, resulting in the complete resolution of MRONJ lesions [27,28].

The etiology of MRONJ is multifactorial, particularly in patients with malignancy-associated immunosuppression[2,29], and the condition is frequently precipitated by tooth extractions secundary to pre-existing periapical or periodontal infections[30]. Although a definitive causal link between rituximab and MRONJ has not been established to the same extent as with antiresorptive agents[2], the accumulating evidence suggests a potential drug-induced pathway that warrants further investigation.

CONCLUSION

This case report and brief literature review highlight a significant association between rituximab therapy and the development of osteonecrosis of the jaw, extending the scope of this pathology beyond conventional antiresorptive agents. The synergistic effect of targeted B-cell depletion combined with the anti-angiogenic effects driven by the corticosteroid component of the R-CHOP protocol impairs bone remodeling, thereby exacerbating susceptibility to bone necrosis. This critical adverse complication underscores the vital necessity for rigorous oral health monitoring in patients undergoing such immunotherapy regimens. Ultimately, the early identification of this non-antiresorptive-induced bone necrosis pathobiology is essential to facilitate conservative management and minimize the requirement for extensive surgical interventions.

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AUTHORS' CONTRIBUTIONS

Barbara Bruno Fagundes Marques was responsible for the conceptualization, clinical investigation, and methodology of the study, as well as the drafting and final revision of the manuscript. **Brenda Xavier dos Santos** contributed to the clinical investigation, endodontic procedures, and the writing and critical review of the case. **Raquel Richelieu Lima de Andrade Pontes** participated in the clinical investigation, methodology, and the final review of the text. **Renato Liess Krebs** provided the original conceptualization, supervised the clinical management, and conducted a thorough revision of the manuscript. **Eduardo Muniz Barretto Tinoco** was responsible for the project's conceptualization, general supervision, clinical coordination, and the final critical review of the scientific content.

CONFLICT OF INTEREST STATEMENT

Funding: This study was financed in part by the Coordenação de Aperfeiçoamento de Pessoal de Nível Superior – Brasil (CAPES) – Finance Code 001”

Competing interests: The authors declare that they have no conflict of interest.

Ethics approval: The study protocol was approved by the Ethics Committee of Pedro Ernesto University Hospital (CAAE: 93170325.7.0000.5259) and is in accordance with the declaration of Helsinki of 1975 and its later amendments.

Declaration of generative AI in scientific writing

During the preparation of this work the authors used Gemini (Google) in order to review text fluency and language, spelling, and grammar errors. This tool was not used to generate content. After using this tool, the authors reviewed and edited the content as needed and take full responsibility for the content of the published article

Consent to participate

The participant was informed and understood the objectives and the details of the study and signed a written informed consent document.

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