

REVIEW ARTICLE

Medication-related osteonecrosis of the jaw: Current concepts in epidemiology, pathophysiology, diagnosis, and management

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ABSTRACT

Aim: The aim of this review was to highlight the ongoing challenge of medication-related osteonecrosis of the jaw (MRONJ) in everyday clinical practice.

Materials and Methods: This review was conducted following a structured narrative approach, utilizing PubMed as the primary research database. The following keywords and their combinations with Boolean operators (AND, OR) were used: MRONJ, osteonecrosis, bisphosphonates, bone density conservation agents, denosumab. The literature search spanned publications from 2003 to April 2025, corresponding to the first description of bisphosphonate-related osteonecrosis of the jaw by Marx and subsequent evolution of the diagnostic and therapeutic concepts. The final set of 32 articles were reviewed and synthesized narratively.

Conclusions: Management strategies are examined across disease stages, from conservative measures and pharmacologic interventions to surgical resection and reconstruction. Emerging therapies, including teriparatide, hyperbaric oxygen, platelet-rich preparations, and cryosurgery, show promise as adjunctive treatments. Prevention remains paramount, emphasizing pre-treatment dental evaluation, patient education, and interprofessional collaboration.

KEY WORDS: bisphosphonates, osteonecrosis, denosumab, MRONJ, bone density conservation agents

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INTRODUCTION

Medication-Related Osteonecrosis of the Jaw (MRONJ) is a severe and multifaceted adverse drug reaction that predominantly affects patients undergoing treatment with antiresorptive agents—such as BP (bisphosphonates) and denosumab—and, increasingly, antiangiogenic drugs used in oncologic and antineoplastic regimens [1,2]. The jawbone's unique anatomy, vascularization, and constant exposure to microtrauma and microbial flora make it particularly susceptible to the effects of these agents [3,4].

First identified by Marx in 2003, the condition was originally classified as Bisphosphonate-Related Osteonecrosis of the Jaw (BRONJ) [5]. However, over time, reports of similar necrotic lesions associated with other agents prompted the American Association of Oral and Maxillofacial Surgeons (AAOMS) to adopt a more inclusive term—MRONJ—in its 2014 position paper. This term reflects a wider etiological scope and aligns with the evolving pharmacologic landscape of bone-modifying agents [6].

The AAOMS defines MRONJ by the following diagnostic criteria:

1. Current or previous treatment with antiresorptive or antiangiogenic agents;
2. Exposed bone or bone that can be probed through a fistula in the maxillofacial region persisting for more than eight weeks;
3. No history of radiation therapy to the jaws or metastatic disease involving the jawbones.

Under normal physiological conditions, mucosal healing following dental extraction or trauma is expected within approximately eight weeks. Persistence of bone exposure beyond this timeframe is therefore considered pathological [7].

The impact of MRONJ extends beyond localized bone necrosis. Patients frequently experience pain, impaired mastication, difficulties in speech, and compromised oral hygiene, all of which contribute to diminished quality of life [8,9]. In oncologic settings, the development of MRONJ may necessitate modification or discontinuation of essential systemic therapies, potentially affecting overall disease management.

Despite increasing awareness, MRONJ remains a diagnostic and therapeutic challenge. Its clinical course is often unpredictable, and management strategies vary depending on disease stage and patient-related factors. Early-stage disease may respond to conservative approaches, whereas advanced cases often require surgical intervention and long-term follow-up [10].

This review provides a comprehensive overview of MRONJ, focusing on epidemiology, risk factors, pathophysiology, clinical presentation, diagnostic modalities, and current treatment strategies, with particular emphasis on recent advances in imaging and adjunctive therapies.

AIM

The aim of this review was to highlight the ongoing challenge of medication-related osteonecrosis of the jaw (MRONJ) in everyday clinical practice. This remains an important issue for clinicians, as MRONJ is associated not only with bisphosphonates but also with denosumab, introduced in 2010 as a potentially safer alternative, which has likewise been linked to this condition. The review summarizes current knowledge on epidemiology, pathophysiology, diagnosis, and treatment, with a practical focus on prevention and on selecting appropriate management strategies based on disease severity in daily patient care.

MATERIALS AND METHODS

This review was conducted following a structured narrative approach, utilizing PubMed as the primary research database. The following keywords and their combinations with Boolean operators (AND, OR) were used: MRONJ, osteonecrosis, bisphosphonates, bone density conservation agents, denosumab. The literature search spanned publications from 2003 to April 2025, corresponding to the first description of bisphosphonate-related osteonecrosis of the jaw by Marx and subsequent evolution of the diagnostic and therapeutic concepts. The final set of 32 articles were reviewed and synthesized narratively.

ETHICS

All sources used in this literature review are publicly available.

AI STATEMENT

The author states that no AI tools were used in writing this article.

REVIEW AND DISCUSSION

EPIDEMIOLOGY

The true prevalence of MRONJ is difficult to determine due to variations in diagnostic criteria, underreporting, and heterogeneity among studies. A recent global scoping review by Clark and Glenny reported a pooled incidence rate of approximately 4.34%, though values ranged widely [11]. The incidence is markedly higher among cancer patients receiving high-potency intravenous bisphosphonates (such as zoledronic acid) or subcutaneous denosumab for skeletal metastases, with rates between 1% and 15%, and in some high-risk groups, even higher [12,13]. In contrast, MRONJ is less common in osteoporotic populations, with reported incidence typically between 0.01% and 0.3%. Nonetheless, given the vast number of patients treated for osteoporosis globally, the absolute number of MRONJ cases in this group remains clinically significant [14].

Risk factors include invasive dental procedures (particularly extractions), poor oral hygiene, use of corticosteroids, diabetes, smoking, and longer duration or higher cumulative doses of antiresorptive drugs. Females, especially postmenopausal women, appear more susceptible, likely due to the higher prevalence of osteoporosis and related treatments. MRONJ is more common in the mandible (up to 73% of cases) than in the maxilla, likely due to differences in vascularization and bone density. It is more frequently unilateral but may be bilateral in advanced cases or in patients with prolonged drug exposure [15,16].

PATHOPHYSIOLOGY

The pathogenesis of MRONJ is multifactorial and involves a combination of impaired bone remodeling, suppression of angiogenesis, microbial infection, immune dysregulation, and local trauma. Antiresorptive medications such as BP (bisphosphonates) bind avidly to hydroxyapatite and inhibit osteoclastic bone resorption, resulting in reduced turnover and accumulation of microdamage. Denosumab, a monoclonal antibody against RANKL, also suppresses osteoclast-mediated resorption but through a different mechanism [17].

The inhibition of bone turnover impairs the jaw's capacity to recover from physiological microtrauma and surgical insult. Antiangiogenic agents like bevacizumab further compromise vascular supply, leading to hypoxia and necrosis. Local infection, often by *Actinomyces* species, exacerbates tissue damage [18].

Table 1. Staging system for MRONJ based on „AAOMS American Association of Oral and Maxillofacial Surgeons’ Position Paper on MRONJ–2022 Update

Stage	Bone condition	Infection/ inflammation	Symptoms	Anomalies
Stage 0	No clinical evidence of necrotic bone	-	tooth pain without an identifiable dental (odontogenic) origin dull, aching jawbone pain, potentially radiating to the temporomandibular joint (TMJ) area maxillary sinus discomfort, possibly accompanied by mucosal thickening and inflammation changes in neurosensory perception	alveolar bone loss or resorption not attributable to chronic periodontal disease. altered trabecular bone pattern, with sclerotic changes and absence of new bone formation in extraction sockets. osteosclerotic areas involving the alveolar and/or adjacent basal bone. thickening or obscuration of the periodontal ligament, including lamina dura sclerosis and narrowing of the periodontal ligament space
Stage 1	Exposed necrotic bone or bone probeable through a fistula	-	- symptom-free	radiographic changes characteristic of Stage 0 may also be observed, limited to the alveolar bone
Stage 2	Exposed necrotic bone	+	symptoms characteristic of Stage 0 may also be observed	radiographic changes characteristic of Stage 0 may also be observed, limited to the alveolar bone.
Stage 3	Exposed necrotic bone	+	one or a combination of: necrotic bone exposure beyond the alveolar region (e.g., mandibular border/ramus, maxillary sinus, zygoma) pathologic fracture extraoral fistula oral antral or oral/nasal communication osteolysis reaching the mandibular border or sinus floor	radiographic findings are more advanced

Source: compiled by the authors based on [7]

Contributing systemic factors include diabetes, corticosteroid therapy, malnutrition, and genetic predisposition. Recent evidence suggests that mucosal integrity may be compromised in MRONJ patients, facilitating bacterial invasion and promoting chronic inflammation. The cumulative effect is progressive bone necrosis with delayed or absent healing [19].

CLINICAL PRESENTATION AND STAGING

MRONJ presents with a broad spectrum of clinical manifestations, ranging from asymptomatic bone exposure to severe pain, swelling, purulent discharge, halitosis, trismus, fistula formation, and pathological fractures. Although often triggered by dental extraction or trauma, spontaneous cases are also observed.

The American Association of Oral and Maxillofacial Surgeons (AAOMS) has established a clinical staging system for MRONJ in American Association of Oral and Maxillofacial Surgeons’ Position Paper on MRONJ–2022

Update encompassing Stages 0 through 3. This framework assists clinicians in assessing disease severity and provides standardized terminology for diagnosis and management. However, the treatment strategies associated with each stage require further evidence-based validation. This association identifies Stage 0 MRONJ as a potential precursor to more advanced stages of the disease in this Position paper, they emphasizes the importance of early identification and management of Stage 0 MRONJ to prevent progression (Table 1) [7].

IMAGING AND DIAGNOSTIC TOOLS

Accurate diagnosis relies on a combination of clinical examination and imaging. Panoramic radiography is commonly used as an initial assessment tool but lacks sensitivity for early disease.

Cone-beam computed tomography (CBCT) has become the preferred modality due to its ability to detect subtle osseous changes, including cortical disruption,

Table 2. Management strategies

Type of management	Indications	Management strategies
Conservative management	Stage 0 Stage 1 a selected cases	chlorhexidine rinses analgesics antimicrobial therapy (e.g., penicillin, clindamycin) routine follow-up emphasizes oral hygiene and non-surgical stabilization of dental conditions
Surgical management:	Stage 2 required for most Stage 3 cases	debridement of necrotic bone and surrounding granulation tissue sequestrectomy to remove loose necrotic fragments resection (marginal or segmental) in advanced cases reconstruction using local flaps (e.g., buccal fat pad), pedicled flaps, or vascularized free tissue transfer Perioperative antibiotics and sterile technique are vital to reduce recurrence. Adjunctive use of CBCT and intraoperative navigation improves precision.

Source: compiled by the authors based on [33]

sequestrum formation, and trabecular alterations. It is particularly valuable in early-stage disease.

Magnetic resonance imaging (MRI) provides superior soft tissue contrast and enables detection of bone marrow changes, often preceding visible bone exposure. T1-weighted images typically demonstrate hypointensity in necrotic regions, while T2-weighted sequences may reveal surrounding inflammation.

Additional modalities, such as bone scintigraphy, SPECT, and PET/CT, may be useful in selected cases but are limited by lower specificity.

The Composite Radiographic Index (CRI_m) has emerged as a quantitative tool for assessing radiological severity and correlates with clinical staging, offering potential value in monitoring disease progression [21].

HISTOPATHOLOGY

Histopathological evaluation, though not mandatory in all cases, provides important confirmation of diagnosis and differentiation from other osseous pathologies such as malignancy, osteoradionecrosis, or chronic osteomyelitis.

Typical histopathological findings in MRONJ include:

- non-vital lamellar bone with empty osteocytic lacunae
- prominent cement lines and bacterial colonization on bone surfaces
- chronic inflammatory infiltrates composed of lymphocytes and plasma cells
- surface biofilms containing Actinomyces-like colonies
- surrounding fibrosis and granulation tissue encasing necrotic bone

These findings support the hypothesis that MRONJ lesions are typically initiated by aseptic osteonecrosis, which becomes secondarily colonized by bacteria. Im-

muno-histochemistry may occasionally be required to exclude neoplastic processes [22].

MANAGEMENT STRATEGIES

Management strategies presented in Table 2.

Repeat imaging and clinical examination are recommended at regular intervals. Healing can range from complete mucosal coverage to partial response or relapse requiring further intervention [23].

ADJUNCTIVE THERAPIES

Recent advances have highlighted the role of several adjunctive therapies in enhancing healing or reducing MRONJ progression.

• **Teriparatide** is an anabolic drug based on a fragment of parathyroid hormone (PTH 1–34) that helps the body build new bone by activating bone-forming cells (osteoblasts). It appears to work especially well in osteoporotic patients with early stages (Stage 1 or 2) of MRONJ. Studies show that treatment with teriparatide can improve bone density and reduce the size of bone lesions.

• **Hyperbaric oxygen therapy (HBOT)** involves breathing pure oxygen in a pressurized chamber, which increases the amount of oxygen reaching poorly supplied tissues. This helps stimulate the formation of new blood vessels and supports healing of soft tissues. It's often used before or after surgery, especially in more difficult or resistant cases. However, its use is limited because it requires many treatment sessions and access to specialized, often expensive equipment.

• **Low-level laser therapy (LLLT)** helps cells work more efficiently by boosting energy production in mitochondria and reducing inflammation. When combined with standard treatments, it can speed up healing of the mucosa and help relieve pain.

• **Cryosurgery** uses liquid nitrogen applied after surgical cleaning of the area to destroy dead (necrotic) tissue while leaving healthy bone intact. This helps clearly separate the damaged tissue from the healthy part and supports new bone formation. Early reports suggest that this method may lower the risk of recurrence and speed up healing.

Other investigational therapies have also emerged, including the use of platelet-rich plasma (PRP) and platelet-rich fibrin (PRF), both of which are aimed at enhancing local tissue regeneration. Ozone therapy has been explored for its antimicrobial and regenerative properties, while probiotic therapy is being studied for its ability to modulate the oral microbiota and reduce pathogenic colonization. Although these approaches are not yet incorporated into standardized clinical protocols, they represent promising directions for future MRONJ research and integrative care strategies.

One substance that is worth mentioning is quercetin. It's a natural plant compound with anti-inflammatory and antioxidant effects that may support bone healing in MRONJ. It can influence osteoclasts and osteoblasts and reduce oxidative stress, but for now, the evidence comes mainly from lab and animal studies, with no strong clinical proof in patients yet [24–28].

PREVENTION

Prevention remains the cornerstone of MRONJ management. Pre-treatment dental evaluation is essential, particularly in oncology patients. Elimination of potential infection sources and completion of invasive procedures prior to therapy initiation significantly reduce risk. In patients already receiving antiresorptive treatment, invasive procedures should be minimized where possible. When unavoidable, careful surgical technique, antibiotic prophylaxis, and primary wound closure are recommended. The role of drug holidays remains controversial and should be considered on an individual basis, in collaboration with the prescribing physician. Effective prevention requires close interdisciplinary cooperation between dental and medical professionals [29,30].

DISCUSSION

MRONJ presents one of the most complex iatrogenic complications encountered in dental and medical practice, particularly due to its variable clinical course, multifactorial etiology, and challenges in early detection. As this review demonstrates, although substantial progress has been made in characterizing its pathophysiology and treatment options, gaps in knowledge

continue to obstruct consensus in clinical practice [1,6,7,11,16].

A major point of concern remains the disparity in incidence data between osteoporotic and oncologic populations, and even within those subgroups depending on drug type, route of administration, and treatment duration. This variation complicates universal risk assessment and calls for stratified prevention protocols tailored to patient type. Moreover, inconsistent use of diagnostic criteria, particularly in borderline or Stage 0 cases, complicates both clinical interpretation and scientific comparison across studies [11,31].

Surgical and conservative treatments continue to evolve, with many clinicians advocating for minimally invasive protocols, particularly in early stages. However, surgical resection remains indispensable for advanced MRONJ. The integration of digital planning, 3D prototyping, and guided resection marks a significant improvement in surgical precision and postoperative outcomes. Adjunctive therapies such as teriparatide and cryosurgery offer exciting possibilities but require further validation through randomized controlled trials and long-term follow-up [7, 10, 32, 33].

Furthermore, the ethical and clinical dilemma of drug discontinuation continues to divide practitioners. While a drug holiday might mitigate MRONJ risk, especially in low-risk osteoporotic patients, it carries potential harm in cancer patients with skeletal metastases. [7] Prospective studies and clinical guidelines must better define the thresholds at which temporary cessation becomes more beneficial than harmful.

In terms of imaging, the increasing use of CBCT and the development of radiographic indices such as CRIIm provide opportunities for earlier detection and better monitoring of treatment outcomes. Similarly, MRI's capability to detect bone marrow changes opens the door for preclinical identification of at-risk patients, though accessibility and cost remain barriers in many healthcare systems [21].

On a systemic level, there is a need for more robust educational efforts directed at both patients and non-dental healthcare providers. The oral manifestations of systemic medications are often underappreciated, leading to delayed referrals or inappropriate interventions. Interprofessional education and collaboration will be essential in improving outcomes [30].

Finally, the promise of personalized medicine—through genetic profiling, biochemical markers, and individualized imaging analytics—may revolutionize MRONJ risk prediction and therapy selection. Integration of such innovations into mainstream clinical practice will depend on multi-institutional collaboration and appropriate funding mechanisms. Until

then, meticulous preventive care, timely intervention, and coordinated management remain the bedrock of successful MRONJ care. The growing body of literature on MRONJ underscores the condition's complexity and the pressing need for evidence-based, multidisciplinary management. Despite increasing awareness, significant challenges remain regarding the timely identification of at-risk individuals, optimization of therapeutic strategies, and balancing the benefits of antiresorptive therapy with the risk of osteonecrosis [16,27,30].

The pathophysiology of MRONJ highlights the synergistic interplay between impaired bone remodeling, compromised vascularity, local trauma, and bacterial colonization. Understanding this interplay is critical for selecting the most appropriate interventions. For instance, early-stage MRONJ may benefit from conservative treatment, while advanced disease often necessitates surgical resection and reconstruction. However, the role of adjunctive therapies—ranging from teriparatide to hyperbaric oxygen—remains an active area of debate, with mixed outcomes reported in clinical trials [26,27,28].

Prevention is widely regarded as the cornerstone of MRONJ management. Yet, there is no consensus on the optimal strategy, particularly regarding drug holidays. This calls for more rigorous, prospective research to define risk-benefit thresholds. Similarly, variation in diagnostic criteria and staging systems hinders inter-study comparisons and evidence synthesis [7].

A shift toward personalized medicine, incorporating genetic, biochemical, and imaging markers, may enable risk-adapted therapy. Future guidelines should integrate these advances, offering dynamic, patient-specific protocols rather than static stage-based recommendations.

Ultimately, enhancing MRONJ outcomes will require better integration of dental and medical care. Dental professionals must be actively involved in treatment planning for patients prescribed antiresorptives, while physicians should be educated on oral health implications. Multidisciplinary collaboration and patient-centered care are pivotal in reducing the burden of MRONJ [29,30].

CONCLUSIONS

Medication-related osteonecrosis of the jaw is an increasingly recognized complication of antiresorptive and antiangiogenic therapies. Its multifactorial etiology, complex clinical course, and significant impact on patient quality of life make it a critical concern for both dental and medical professionals.

This review has outlined the current understanding of MRONJ, emphasizing the importance of early diagnosis, individualized treatment planning, and proactive prevention strategies. With innovations in imaging, surgical techniques, and adjunctive therapies, management outcomes are gradually improving.

Nonetheless, substantial gaps remain, particularly in identifying high-risk individuals, standardizing treatment protocols, and integrating care across disciplines. Addressing these gaps will require ongoing research, international cooperation, and continued investment in both clinical and translational science.

By fostering a multidisciplinary and evidence-driven approach, we can reduce the incidence of MRONJ, enhance therapeutic outcomes, and improve the overall well-being of patients undergoing vital skeletal therapies.

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CONFLICT OF INTEREST

The Authors declare no conflict of interest

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