

Osteoradionecrosis of the Jaws: What Every Oral Physician Should Know

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Abstract: ***Objective:** Osteoradionecrosis (ORN) of the jaws is a severe late complication of radiotherapy in head and neck cancer patients. Despite advances in radiation delivery and preventive dental care, ORN continues to cause significant morbidity. This narrative review aims to summarize the current understanding of the epidemiology, pathogenesis, clinical features, diagnostic criteria, imaging characteristics, and management strategies for ORN. **Study Design:** A narrative review of the English-language literature was performed using published clinical studies, reviews, and classic articles related to osteoradionecrosis of the jaws. **Results:** ORN is characterized by devitalized irradiated bone that fails to heal in the absence of recurrent malignancy. The mandible is affected more frequently than the maxilla due to its limited vascularity. Pathogenesis is multifactorial and has evolved from infection-based concepts to contemporary hypoxic–hypocellular–hypovascular and radiation-induced fibroatrophic models. Diagnosis relies on clinical persistence of bone exposure, radiographic changes, and exclusion of tumor recurrence. Management ranges from conservative care and pharmacologic antifibrotic therapy to hyperbaric oxygen therapy and surgical resection in advanced disease. **Conclusions:** ORN remains a challenging clinical entity with no universally accepted gold-standard treatment. Emphasis should be placed on prevention, early diagnosis, and stage-appropriate multidisciplinary management to optimize outcomes.*

Keywords: Osteoradionecrosis; Radiotherapy complications; Mandible; Hyperbaric oxygen therapy; Pentoxifylline; Tocopherol

1. Introduction

Oral cancer represents a major global health burden, with approximately 500,000 new cases and 270,000 deaths reported annually worldwide. India alone accounts for nearly 40% of the global oral cancer burden, with oral malignancies responsible for approximately 7% of cancer-related deaths in males and 3% in females. Radiotherapy is a key component of multimodal management for head and neck cancers; however, it is associated with several long-term complications. Among these, osteoradionecrosis (ORN) of the jaws is the most serious, characterized by progressive bone necrosis, impaired wound healing, and potential pathological fracture.^{1–3}

Definition and Diagnostic Criteria

Osteoradionecrosis has been defined as a radiation-induced ischemic necrosis of bone associated with soft tissue breakdown, occurring in the absence of persistent or recurrent tumor.⁴ Marx further refined the definition by specifying exposed bone greater than 1 cm persisting for at least six months without healing.⁵ Current diagnostic criteria emphasize prior radiation exposure, persistent mucosal breakdown with exposed devitalized bone, and exclusion of malignancy recurrence.⁶

Historical Perspective

The first description of radiation-induced bone necrosis was reported by Regaud in 1922.⁷ Ewing later introduced the term “radiation osteitis” to describe the pathological changes.⁸ Early investigators, including Meyer and Titterton, classified ORN as a form of osteomyelitis of irradiated bone, a concept that dominated management strategies for several decades.^{9,10}

Pathogenesis

The understanding of ORN pathogenesis has evolved considerably over time.

Meyer’s radiation–trauma–infection theory proposed that radiation devitalizes bone, trauma provides a portal of entry, and secondary infection leads to necrosis.⁹ However, this theory failed to explain cases occurring without preceding trauma.

Marx introduced the hypoxic–hypocellular–hypovascular theory, demonstrating that irradiated tissues exhibit reduced cellularity, diminished vascularity, and chronic hypoxia, resulting in impaired healing and tissue breakdown.^{5,11} Microorganisms were shown to act primarily as surface contaminants rather than causative agents.

The radiation-induced fibroatrophic theory proposed by Delanian and Lefaix describes ORN as a progressive fibrotic process initiated by endothelial damage, chronic inflammation, and dysregulated fibroblast activity, ultimately leading to tissue fragility and necrosis.¹²

Epidemiology and Risk Factors

ORN most commonly affects patients in the fifth and sixth decades of life. Earlier studies reported incidence rates as high as 37%, whereas contemporary series report rates below 5%, reflecting improvements in radiotherapy techniques and preventive dental care.^{13–15} The mandible is affected more frequently than the maxilla, particularly the posterior body region. Major risk factors include radiation doses exceeding 60 Gy, poor fractionation schedules, large radiation fields, post-radiation dental extractions, poor oral hygiene, smoking, malnutrition, and trauma to irradiated bone.^{16,17}

Clinical Features

Clinically, ORN presents as persistent exposed bone for more than three months, often accompanied by pain, suppuration, halitosis, trismus, dysesthesia, and dysgeusia. Progressive disease may result in pathological fractures, intraoral or extraoral fistulae, and secondary infection.^{18,19}

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Imaging Features

Conventional radiographs typically demonstrate irregular radiolucencies, patchy sclerosis, ragged borders, and sequestration. Computed tomography and cone-beam computed tomography provide superior assessment of cortical erosion, heterogeneous bone density, and pathological fractures.²⁰ Magnetic resonance imaging reveals altered marrow signal intensity and extension of inflammation into adjacent soft tissues.²¹

Histopathologic Features

Histologic examination reveals avital bone with empty osteocytic lacunae, absence of osteoblastic rimming, thickened fibrotic vessel walls with endarteritis, and replacement of marrow spaces by collagen-rich fibrous tissue. There is typically no clear demarcation between viable and nonviable bone.²²

Differential Diagnosis

The differential diagnosis of ORN includes chronic osteomyelitis, medication-related osteonecrosis of the jaw, recurrent or metastatic malignancy, traumatic bone exposure, and systemic osteonecrotic conditions.²³

Management

Management of ORN is stage-dependent. Conservative therapy includes meticulous oral hygiene, saline irrigation, analgesics, and antibiotics for secondary infection. Pharmacologic antifibrotic therapy with pentoxifylline and tocopherol has shown promising results in early-stage disease.^{12, 24}

Hyperbaric oxygen therapy enhances tissue oxygenation, angiogenesis, and fibroblast proliferation and is most effective as an adjunct to surgery or in early disease.^{5, 25} Ultrasound therapy, electrotherapy, and ozone therapy have also been explored as adjunctive modalities with variable success.^{26–28} Advanced ORN requires surgical debridement or resection, often guided by Marx's staging system.⁵

Prevention

Preventive strategies include comprehensive dental evaluation before radiotherapy, extraction of teeth with poor prognosis, adequate healing intervals prior to irradiation, lifelong fluoride therapy, strict oral hygiene maintenance, and cautious post-radiation dental interventions.^{17, 29}

2. Conclusion

Osteoradionecrosis of the jaws remains a complex and challenging complication of head and neck radiotherapy. Prevention, early diagnosis, and individualized multidisciplinary management are essential to minimize morbidity and improve patient outcomes.

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Conflicts of Interest

The authors declare no conflicts of interest.

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References

- [1] Ferlay J, et al. *CA Cancer J Clin.* 2021; 71: 209–249.
- [2] Sankaranarayanan R, et al. *Lancet.* 2015; 385: 1687–1696.
- [3] Clayman L. *J Oral Maxillofac Surg.* 1997; 55: 233–234.
- [4] Wong JK, Wood RE, McLean M. *Oral Surg Oral Med Oral Pathol.* 1997; 84: 16–21.
- [5] Marx RE. *J Oral Maxillofac Surg.* 1983; 41: 283–288.
- [6] Chrcanovic BR, et al. *Oral Maxillofac Surg.* 2010;14:3–16.
- [7] Regaud C. *Compt Rend Soc Biol.* 1922; 87: 629–631.
- [8] Ewing J. *Acta Radiol.* 1926; 6: 399–412.
- [9] Meyer I. *J Oral Surg.* 1971; 29: 38–45.
- [10] Titterton PJ. *Br J Oral Surg.* 1972; 10: 94–102.
- [11] Marx RE, Johnson RP. *Oral Surg Oral Med Oral Pathol.* 1987; 64: 379–390.
- [12] Delanian S, Lefaix JL. *Radiother Oncol.* 2004; 73: 119–131.
- [13] Thorn JJ, et al. *Radiother Oncol.* 2000; 54: 187–193.
- [14] Reuther T, et al. *Int J Oral Maxillofac Surg.* 2003; 32: 289–295.
- [15] Nabil S, Samman N. *Int J Oral Maxillofac Surg.* 2011; 40: 229–243.
- [16] Beumer J, et al. *Oral Surg Oral Med Oral Pathol.* 1984; 57: 540–544.
- [17] Wahl MJ. *J Am Dent Assoc.* 2006; 137: 163–165.
- [18] Store G, Larheim TA. *Dentomaxillofac Radiol.* 1999; 28: 196–203.
- [19] Kanatas A, Rogers SN. *Br J Oral Maxillofac Surg.* 2014; 52: 115–120.
- [20] White SC, Pharoah MJ. *Oral Radiology.* Mosby; 2014.
- [21] He Y, et al. *Oral Surg Oral Med Oral Pathol Oral Radiol.* 2015; 119: 566–575.
- [22] Marx RE. *J Am Dent Assoc.* 1985; 110: 695–697.
- [23] Ruggiero SL, et al. *J Oral Maxillofac Surg.* 2014;72:1938–1956.
- [24] Delanian S, et al. *Int J Radiat Oncol Biol Phys.* 2011;80:832–839.
- [25] Hunt TK, Pai MP. *Surg Gynecol Obstet.* 1972; 135: 561–565.
- [26] Harris M. *Br J Oral Maxillofac Surg.* 1992; 30: 149–153.
- [27] Reher P, Harris M. *Br J Oral Maxillofac Surg.* 1998; 36: 46–48.
- [28] Nogales CG, et al. *J Craniofac Surg.* 2008; 19: 1465–1469.
- [29] Beumer J III, Curtis TA. *Maxillofacial Rehabilitation.* Mosby; 1979.