The Osteosarcoradionecrosis as an Unfavorable Result Following Head and Neck Tumor Ablation and Microsurgical Reconstruction

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KEYWORDS

• Osteoradionecrosis • Osteosarcoradionecrosis • Radiotherapy

KEY POINTS

- The soft tissue has a core role in the development and management of osteoradionecrosis, preferably called osteosarcoradionecrosis.
- Debridement and sequestrectomy may induce further destruction to the tissue already damaged by radiotherapy; it is better to be avoided.
- Necrosis is irreversible condition; once osteosarcoradionecrosis is diagnosed, only resection and reconstruction can provide decisive, cost-effective treatment.
- The reconstruction has a potential role to manipulate the postablative course, decreasing complications following radiotherapy.

INTRODUCTION

The osteoradionecrosis is commonly defined as bone necrosis following radiotherapy. The presence of chronic, nonhealing wound with bone necrosis for more than 3 months in the setting of previous radiotherapy and in the absence of tumor recurrence or metastasis is necessary for diagnosis.[1,2] Risk factors are well-identified and numerous and include the mandible, T3 and T4 tumors, alcohol and tobacco use, and radiotherapy dose of at least 60 Gy.[1,2] Three theories have been suggested since 1970 in an attempt to explain why and how osteoradionecrosis develops and progresses.[3–5] Although none of them is conclusive, Delanian's theory of radiation-induced fibroatrophy seems to have stronger evidence.[5,6] Classifications are plenty, and they all try to utilize different treatment modalities such as medical therapy, hyperbaric oxygen, debridement and sequestrectomy, and resection and flap reconstruction in a stepwise approach.[1,2,4,7–9]

However, scrutinizing what the literature has on osteoradionecrosis this far starting with the definition of osteoradionecrosis and ending up with treatment modalities, using logic, science, and experience to accept or reject findings bring to attention that osteoradionecrosis is not a

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bone-only disease entity. Mucositis, xerostomia, and soft tissue necrosis, fibrosis, and atrophy are all important features of osteoradionecrosis[10] that need not to be omitted from the definition or treatment plan. Furthermore, classifications, which in general aim at staging bone necrosis to guide therapy, may seem self-contradictory or illogical in the sense that necrosis is necrosis, which cannot be staged, but can be quantified, instead. This is particularly important, because if one agrees with the fact that necrosis is death of the tissue, an irreversible condition, then there can only be 1 treatment that works: resection. Moreover, although the role of radiotherapy in osteoradionecrosis is undisputable and at the core of pioneering invaluable refinements to decrease the incidence of osteoradionecrosis,[11,12] the preventive or predisposing role of ablation and reconstruction seems to be largely understated if not overlooked. The improved understanding of the underlying mechanisms in osteoradionecrosis may further warrant a review of the techniques utilized in treating osteoradionecrosis.

Based on this concise introduction, the authors aim at redefining osteoradionecrosis coining a novel term, the osteosarcoradionecrosis, to emphasize the importance of the soft tissue role played in the disease and treatment plan, revising treatment guidelines, taking aggressive approach given that necrosis is death, an irremediable condition, and providing an eye opener on how ablative–reconstructive surgeries may prevent or predispose to osteoradionecrosis.

OSTEOSARCORADIONECROSIS

Osteosarcoradionecrosis is a novel term refers to bicomponent, bone and soft tissue necrosis, following radiotherapy, in which necrosis is death of the tissue, an irreversible condition.

Following irradiation, a cascade of events such as endothelial cell destruction, vascular thrombosis, free radicals release, inflammation, altered osteoblast/osteocast activity, and proliferation of fibroblast into myoblast will ensue to an extent that depends on the intensity of irradiation and the tissue inherent characteristics.[1,5,13,14] It makes sense to speculate that when the bone is injured, other tissues of weaker resistance such as mucosa, skin, and muscles are already damaged, suggesting that the damage to soft tissue could proceed that occurring to the bone. As radiotherapy is applied at the same time, multitissue damage will result in a vicious cycle in which unhealthy skin and mucosa will not support a durable coverage over the bone, leading to soft tissue breakdown and bone exposure, and at the same time the unhealthy bone will not allow the tissue to heal, resulting in sinuses and fistulae. The absence of healthy barrier, mucosa or skin, and the presence of unhealthy bone, may provide a niche for infection, further complicating the situation. Furthermore, the symptoms many of the patients with the previously defined osteoradionecrosis have such as scarring and fibrosis, trismus, atrophy, xerostomia, mucositis, and pain highlight the involvement of the soft tissue. That being said, the chronic wound problem defining osteoradionecrosis is also the result of skin/mucosa necrosis or compromise. The soft tissue, therefore, is at the heart of the problem; this explains the importance of the suggested term "osteosarcoradionecrosis," rather than osteoradionecrosis alone, and allows one to approach the problem from all its aspects as a syndrome following tissue irradiation.

It is essential, however, to avoid liberal use of necrosis. Necrosis should only refer to tissue death,[15] which is irreversible and can only be managed by radical excision. The strict application of necrosis as a term in osteoradionecrosis will give rise the problem of what the condition in which the tissue is undergoing necrosis should be called?

The authors' best answer to this question is dividing the changes following irradiation into 2 major phases that may not be chronologically dependent on each other: the reversible damage phase and the necrosis, irreversible damage, phase. It is important to avoid stressing any chronologic relationship between these 2 phases because radiotherapy-induced damage, which begins with the delivery of radiotherapy, may vary in intensity and extent depending on tissue target, tissue tolerance, and preexisting conditions such as previous surgeries, explaining why some tissue may present with necrosis while irreversible damage could be the situation in some other tissue. The tissue in the reversible phase may reach a statuesque with lifelong risk of osteosarcoradionecrosis, or it becomes irreversibly damaged. The transformation process of phase 1 to 2 can be to a certain extent manipulated by some drugs such as pentoxifylline, tocopherol, clodronate, or steroids.[1,2,6,7]

The authors suggest that the reversible phase may correlate with the prefibrotic phase and the constitutive organized phase,[1,6,7] and it starts with the delivery of radiotherapy and ends once signs of bone and soft tissue necrosis develop clinically, on radiograph, or on histologic examination. The reversible phase could be thus asymptomatic or marked with clinical, radiographic, or histologic signs and symptoms that are not limited