EDITORIAL

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Unraveling the mystery of implant-related sequestration: a biomechanical breakthrough with far-reaching implications

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While simple implant surgery is safe in terms of the risk of medication-related osteonecrosis of the jaw (MRONJ), the number of late failures that might be related to long-term antiresorptive therapy is increasing. This unique pathologic entity frequently involves sequestrum formation attached to the failed implant[1]. This sequestrum formation with unclear pathophysiology has been widely reported for more than 10 years[1,2].

Regarding the terminology of this unique bone pathology, several different names have been used among clinicians, but there remains no dominant name. Several clinicians have proposed the term 'implant presence-triggered osteonecrosis'[3], while other clinicians have coined the term 'peri-implantitis-like MRONJ'[4]. During the last international team for implantology consensus conference, the term 'implant-related sequestration (IRS)' was agreed upon[5].

Surgery involving alveolar bone has been identified as a risk factor for MRONJ. Etiologies other than surgical trauma should be studied to determine the possible pathophysiology. The mechanical regulation of modeling and remodeling processes, collectively called bone remodeling, can be influenced by factors such as drugs, metabolic bone diseases, and dental implants integrated into the bone.

According to recent studies, peri-implantitis may play a role in the development of IRS[4,6,7]. The peri-implantitis theory is traditional outside-in pathophysiology, in which initial peri-implant bone lysis would increase the local concentration of bisphosphonates entrapped in the bone mineral in the vicinity of the implant[8]. However, those cases caused by denosumab could not be explained by the peri-implantitis theory due to the different mode of action.

The long-term occlusal stress on the alveolar bone by the implant could result in accumulated microdamage and may be a risk factor for MRONJ.

Reflecting Frost's mechanostat theory[9], microdamage caused by stimuli beyond physiologic tolerance should be eliminated, but the impaired bone remodeling process of patients on antiresorptive therapy failed to eliminate the microdamage, which may lead to the development of necrotic foci. Considering that the mechanotransduction theory is highly correlated with the role of osteocytes, empty lacunae, a histopathologic hallmark of MRONJ, can be explicative.

The compromised bony trabecular architecture described by Euler characteristics is often observed in osteoporotic bone, which is not optimal for withstanding mechanical loading. Research on MRONJ has mainly investigated from the context of biology. However, there is need for greater attention to the biomechanical pathophysiology of IRS. Although occlusal load as a risk of IRS is still premature and far-fetched, but it might be worth elaborating on some recent research related to it. Mine et al.[10] revealed that occlusal trauma alone could induce bone necrosis, and our research team demonstrated that occlusal load increased the risk of mechanical failure of compromised alveolar bone, which may be a possible link to the development of IRS based on finite element analysis (FEA) models[11]. However, FEA models involve disadvantages when emulating human organs, and some researchers are reluctant to accept their results. However, the rapid progress of computational biomechanics might allow better understanding of the interplay between biological elements and mechanical conditions.

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An FEA study also has limitations in validating the role of biomechanics as an etiology of IRS since various parameters from a living being cannot be incorporated. More sophisticated computerized models and more accurate modeling of biological events are expected to allow better analysis of human organs in the future.

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Conflict of Interest

No potential conflict of interest relevant to this article was reported.

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