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Osteonecrosis of the jaw: lights and shadows in the knowledge of its pathophysiology

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Osteonecrosis of the jaw (ONJ) was described by Marx et al.[1] in 2005. In the following years, both isolated cases and series of patients were published which, over the years, was decreasing, on the one hand, due to the saturation of the journals and the low interest that the description of new cases may cause. Furthermore, knowledge of this disease has lead to the development of preventive measures that may have diminished its incidence.

Regarding ONJ, a whole range of "fears, risks and dangers" have been developed that are largely unjustified. ONJ was indicated as a complication of prolonged bisphosphonate treatment and in this sense it was equalized to the diaphyseal fractures[2], when both processes most certainly have different etiopathogenic mechanisms[3]. Fears concerning ONJ or diaphyseal fractures developed a whole doctrine about the need to suspend treatment with bisphosphonates or denosumab, the so-called "therapeutic vacations" that in reality what it was about was simply to suspend the antiresorptive treatment, before that the possible complications of its use appear[4-6]. This is especially common in the field of dentists, who, in many cases, concerned about the possible development of an ONJ do not perform virtually any dental intervention in patients receiving bisphosphonates or denosumab. With this, what has been observed is an increase in the abandonment of treatment with antiresorptive drugs which produces an increased risk of fragility fractures after discontinuation of bisphosphonate therapy, a risk that has an extreme severity in the case of suspension of denosumab treatment, with the appearance of multiple vertebral fractures[7-11].

ONJ occurs mainly in patients suffering from cancer (more than 90% of the cases described) and who have received potent bisphosphonates or denosumab at doses not used in osteoporosis treatment[3,12,13] and in which there has been a dental intervention[14]. Among patients receiving antiresorptives for the treatment of osteoporosis, the occurrence of cases, although it is true that it has been reported, is very scarce, almost exceptional[15,16].

In this issue of the Revista de Osteoporosis y Metabolismo Mineral, Quintana et al.[17] present the findings observed in a series of patients with ONJ in which they have carried out a complete study of both the amount of bone mass, determined by densitometry, and bone quality, estimated by the trabecular bone score (TBS) and Quantitative Ultrasound, an unfairly undervalued, harmless and simple technique that can assess bone quality and predict the risk of fracture as well as traditional densitometry[18,19]. The results obtained differ from the myths developed about ONJ and is that the excess suppression produced by these drugs would produce a "frozen" bone of poor quality and weakness. As can be seen in these results, it is most likely that the quantity and quality of the bone in ONJ does not show general alterations. Rather, involvement is local and influenced by multiple factors. All of this leads us to conclude that we still do not know many facts about the etiology, pathogenesis and pathophysiology of ONJ, and that we still have more shadows than lights on this matter[12,20,21].

Conflict of interests: The author declares no conflicts of interest.

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