

Medication Related Osteonecrosis of the Jaw: What is the Key Point?

Onur Şahin^{1,2*}

¹Department of Oral and Maxillofacial Surgery, Faculty of Dentistry, İzmir Katip Çelebi University, İzmir, Turkey

²Department of Stem Cell, Medicine Faculty, Ege University, İzmir, Turkey

***Corresponding Author:** Onur Şahin, Department of Oral and Maxillofacial Surgery, Faculty of Dentistry, İzmir Katip Çelebi University and Department of Stem Cell, Medicine Faculty, Ege University, İzmir, Turkey. **E-mail:** onur.sahin@ikc.edu.tr

Received: December 05, 2018; **Published:** December 12, 2018

Bisphosphonates (BP) are the most commonly used antiresorptive drugs in the prevention of skeletal complications of many diseases. It was described in 2003 that osteonecrosis of the jaw bones is caused [1]. The increase of localized osteonecrosis cases in jaw bones related to BPs; negative effects on quality of life and increased morbidity led researchers to identify early and to investigate the effective treatment strategies. It was understood that in addition to BPs, other antiresorptive (e.g. denosumab) and antiangiogenic drugs (e.g. bevacizumab and sunitinib) could cause osteonecrosis as well, so the "American Association of Oral and Maxillofacial Surgeons (AAOMS)" proposed to use "medication related osteonecrosis of jaw (MRONJ)" terminology instead of "bisphosphonate-related osteonecrosis of jaw (BRONJ)" [2]. IV BPs are frequently used to treatment of hypercalcemia associated with malignant tumors due to their antitumoral effects, in the treatment of cancers with bone metastases such as breast, prostate and lung, prevent skeletal complications in multiple myeloma, whereas oral BPs are frequently associated with osteoporosis and osteopenia and rarely Paget's disease and osteogenesis imperfecta treatment.

In 2014, three criteria were defined by AAOMS in order to be able to perform differential diagnosis of MRONJ with many different diseases that could cause clinical osteonecrosis. These criteria's are; the presence of antiresorptive and antiangiogenic drug use in the medical history of the patient, clinical exposure of the bone site for more than 8 weeks, with no radiotherapy story or metastasis localized in the jaw bone. Using bone exposure as a criterion in the diagnosis of MRONJ, causes delayed diagnosis and resistance to treatment. Stage 0 is described as an unexposed variant of MRONJ and presents non-specific clinical findings, radiographic changes and symptoms. Lack of specific clinical features often presents occurs a diagnostic difficulty for clinicians.

Although the first case of MRONJ is reported in 2003, pathophysiology is not fully understood, and potential mechanisms are still being discussed. Prevention and early diagnosis of MRONJ have been the target of MRONJ investigations. The possibility of disease occurrence without bone exposure, in turn, has been widely discussed in the literature, which should be considered during patient evaluation. Several studies have been reported investigating diagnostic strategies for predicting the onset of MRONJ such as analyzed of serum C-terminal telopeptide (CTX) and other bone turnover markers, using periapical radiographs standardized by an aluminium step wedge, measurement of thickness of the mandibular inferior cortical bone and detection of specific radiologic signs such as osteosclerosis, osteolysis, dense woven bone, a thickened

lamina dura, subperiosteal bone deposition and failure of postsurgical remodeling [3-5]. To correct and early diagnosis of patients with MRONJ and identification of parameters associated with the development of bone exposure are of importance in management.

The pathogenesis of MRONJ was attempted to explain with theories but the pathophysiology of this disease has not been exactly understood, despite the time since the first MRONJ case in the literature. Future studies should aim to explain the pathogenesis of MRONJ and finding effective early diagnostic strategies and treatment methods in larger groups of patients. The role of general dental practitioner, oral and maxillofacial surgeons, oncologists and orthopedists in this area must be regular checked for oral and dental health, and must be motivated to be trained and regularly controlled in patient oral care before starting antiresorptive and antiangiogenic drugs.

Bibliography

1. Marx RE. Pamidronate (Aredia) and zoledronate (Zometa) induced avascular necrosis of the jaws: a growing epidemic. *J Oral Maxillofac Surg.* 2003;61(9):1115-1117.
2. Ruggiero SL, Dodson TB, Fantasia J, Goodday R, Aghaloo T, Mehrotra B, O'Ryan F. American Association of Oral and Maxillofacial Surgeons. American Association of Oral and Maxillofacial Surgeons position paper on medication-related osteonecrosis of the jaw-2014 update. *J Oral Maxillofac Surg.* 2014;72(10):1938-1956.
3. Marx RE, Cillo CE Jr, Ulloa JJ. Oral bisphosphonate-induced osteonecrosis: risk factors, prediction of risk using serum CTX testing, prevention, and treatment. *J Oral Maxillofac Surg.* 2007;65(12):2397-2410.
4. Takaishi Y, Ikee T, Nakajima M, et al. A pilot case-control study on the alveolar bone density measurement in risk assessment for bisphosphonate-related osteonecrosis of the jaw. *Osteoporos Int.* 2010;21(5):815-825.
5. Torres SR, Chen CS, Leroux BG, et al. Mandibular inferior cortical bone thickness on panoramic radiographs in patients using bisphosphonates. *Oral Surg Oral Med Oral Pathol Oral Radiol.* 2015;119(5):584-592.

Volume 2 Issue 1 January 2019

© All rights are reserved by Onur Şahin.