

CURRENT UNDERSTANDING OF DRUG-ASSOCIATED OSTEONECROSIS OF THE JAW AND GENERAL INFORMATION ON OSTEONECROSIS

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Annotation: drug-associated osteonecrosis of the jaw is a serious complication of antiresorptive therapy, characterized by progressive necrosis of jaw bone tissue with bone exposure in the oral cavity. This paper examines current understanding of the etiology, clinical manifestations, and treatment approaches for this condition.

Keywords: osteonecrosis of the jaws, bisphosphonates, denosumab, antiresorptive therapy.

The link between bisphosphonate use and the development of osteonecrosis of the jaw was first described by Robert Marx in 2003. Since then, this problem has become one of the most pressing in maxillofacial surgery and dentistry.

The incidence of drug-associated osteonecrosis of the jaw (DAONJ) varies significantly: with oral bisphosphonates, the risk is less than 0.01%, while with intravenous administration, it can reach 15% in cancer patients. We believe this difference is explained not only by the drug dosage but also by the patients' underlying conditions.

The main group consists of bisphosphonates, drugs widely used in clinical practice for over 30 years. The nitrogen-containing compounds pose the greatest risk: zoledronic acid, pamidronate, and alendronate. It should be noted that zoledronic acid is 1,000 times more active than alendronate.

Denosumab (a monoclonal antibody to RANKL) and antiangiogenic agents such as bevacizumab and sunitinib are also considered high-risk medications. Our clinical experience shows that combination therapy significantly increases the risk of complications.

The central link in the pathogenesis is the suppression of osteoclast function, which leads to impaired bone remodeling. Bone becomes metabolically "frozen," losing its ability to regenerate after microtrauma.

The antiangiogenic effect of these drugs is equally important. Bisphosphonates are toxic to endothelial cells, disrupting neovascularization. This is critical for the jawbones with their intensive blood supply.

During the observation process, we identified several groups:

Medicinal factors: type of drug, cumulative dose, duration of treatment

Dental factors: traumatic interventions, inflammatory periodontal diseases, poor hygiene

Systemic factors: diabetes mellitus, rheumatoid arthritis, long-term use of corticosteroids

In the early stages, patients complain of numbness in the lower lip (Vincent's sign), associated with compression of the inferior alveolar nerve. Often, the first symptom is delayed healing of the socket after tooth extraction.

As the disease progresses, the classic triad develops: exposure of necrotic bone, pain, and secondary infection. In advanced cases, fistulas with purulent discharge and pathological fractures develop.

The diagnosis is established based on the following: A history of taking antiresorptive medications. Exposed bone tissue in the oral cavity for more than 8 weeks. No history of radiation therapy.

X-ray examination remains the basis for diagnosis. Orthopantomography can detect characteristic changes such as osteosclerosis, sequestration, and widening of the periodontal space.

The most informative is computed tomography, which makes it possible to accurately determine the boundaries of the lesion and plan treatment.

The classification of the American Association of Oral and Maxillofacial Surgeons is used:

Stage 0: non-specific symptoms without visible defects **Stage 1:** bone exposure without inflammation and pain

Stage 2: bone exposure with infection **Stage 3:** spread beyond the alveolar portion with fractures and fistulas

Conservative methods are the basis for early-stage treatment. We adhere to the principle of minimal intervention.

Antibiotics: doxycycline 100 mg 2 times a day, clindamycin 300 mg 4 times a day

Antiseptic rinses with chlorhexidine or miramistin

Elimination of traumatic factors

If conservative therapy is ineffective, surgical interventions are indicated. In the second stage, minimally invasive procedures such as curettage and sequestration are preferred.

At stage three, resection of the affected area with reconstruction may be necessary. Such surgeries are performed in specialized centers.

Prevention should begin before treatment: dental examination and oral hygiene. Extraction of teeth with a questionable prognosis. During treatment, regular checkups every 3-6 months.

Conclusions: drug-associated osteonecrosis of the jaw requires coordinated work between oncologists, dentists, and maxillofacial surgeons. Prevention remains the most effective approach.

Key principles of patient management: preliminary preparation of the oral cavity, early detection of signs of osteonecrosis, and predominantly conservative treatment of the initial stages.

It's important to understand that the development of MAONCH should not interfere with life-saving therapy for cancer patients. A reasonable balance is needed between the benefits of treatment and the risk of complications.

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