Bilateral bisphosphonate-related osteonecrosis of the jaw with left chronic infection in an 82-year-old woman

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DESCRIPTION

An 82-year-old woman, treated for severe rheumatoid polyarthritis by tumour necrosis factor inhibitors (2003–2008), rituximab (2008), abatacept (2008–2011) and long-term steroid therapy, also received prolonged bisphosphonate therapy to prevent bone mass loss. In 2009, a bilateral bisphosphonate severe osteonecrosis of the jaw (BONJ) was discovered along with evidence of left chronic mandible infection in early 2012, as mucosal disruption and suppurative sinus tract occurred (figure 1A,B). Bisphosphonate was stopped and mild surgical debridement was performed. Despite administration of antimicrobial therapy including high dose of amoxicillin and fluconazole, the suppurative sinus tract persisted. As a left mandibular fracture with significant mouth opening reduction was spontaneously carried out, a radical surgery, consisting of complete hemi-mandibular resection without reconstruction, was performed. Surgical bone sampling revealed Actinomyces naeslundii in cultures, and prolonged amoxicillin therapy was administered. During the follow-up, the outcome was favourable without recurrence of the suppuration (figure 1C,D).

The pathogenesis of BONJ is not well known.1 Some risk factors have been identified, such as the route of administration or the duration use of bisphosphonate, the concomitant administration of corticosteroids and performance of dental invasive procedures.1 BONJ may be owing to: (1) osteoclast inhibition and antiangiogenic effect of bisphosphonate, resulting in oversuppression of bone turnover and avascular necrosis; (2) bisphosphonate mucosal toxicity leading to mucosal disruption and (3) involvement of Actinomyces species in biofilm in bone.1–3 BONJ with evidence of chronic infection is associated with severe morbidity, required adequate surgery (debridement or more aggressive surgery) and prolonged antimicrobial treatment targeting Actinomyces.1–3

Learning points

Bilateral bisphosphonate severe osteonecrosis of the jaw:

▸ Occur mainly in patients with several risk factors, such as age and coadministration of corticosteroid therapy.
▸ Is due to bisphosphonate toxicity, but there is some evidence that bone colonisation by Actinomyces species in biofilm participate to the pathogenesis.
▸ Could be associated with evidence of infection, requiring surgery and prolonged antimicrobial therapy targeting Actinomyces species.

Figure 1 Bilateral bisphosphonate severe osteonecrosis of the jaw with suppurative sinus tract (A). The orthopantomographic view revealed bilateral severe osteonecrosis (B, arrows). After radical surgery and prolonged amoxicillin therapy, the outcome of the infection was favourable without recurrence of the suppuration (C). The orthopantomographic view after complete hemi-mandibular resection with dental extractions and without reconstruction (D, arrow).

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REFERENCES