

The Change of Bone Erosion using Denosumab to Rheumatoid Arthritis

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Introduction

A 49-year-old woman presented to our hospital for treatment of rheumatoid arthritis (RA). Although she was taking methotrexate (8 mg/week), her symptoms had not resolved. SDAI was 26.2 at the first visit. We recommended biological agents, but she refused. Therefore, methotrexate was increased to 16 mg/week over a 1-year period, resulting in improvement of SDAI to 3.4. Bone mineral density (BMD) was measured during treatment, revealing low BMD at L2-L4 (0.834 g/cm²), so alendronate was started (35 mg/week). Because BMD did not improve and the serum level of tartrate-resistant acid phosphatase type 5b was 384 mU/dL, alendronate was switched to denosumab (60 mg/6 months; the drug concentration of denosumab was used as the treatment amount of the osteoporosis). Hand radiographs obtained before switching revealed generalized bone erosions and joint space narrowing, especially between the radius and scaphoid or lunate (Figure 1). After 9 months of denosumab therapy, the joint space between the radius and scaphoid improved (Figure 2).

Denosumab is a monoclonal antibody that inhibits receptor activator of nuclear factor kappa-B ligand, which induces osteoclast differentiation, and it is used to treat multiple myeloma and osteoporosis. Use of denosumab for RA was reported recently. In RA patients, denosumab provides protection against bone erosion, and not only prevents bone loss but increases BMD in the hand. This is the first imaging evidence that denosumab can inhibit osteoclast activity in a patient with controlled RA, suggesting that osteoclast suppression may be useful in RA.

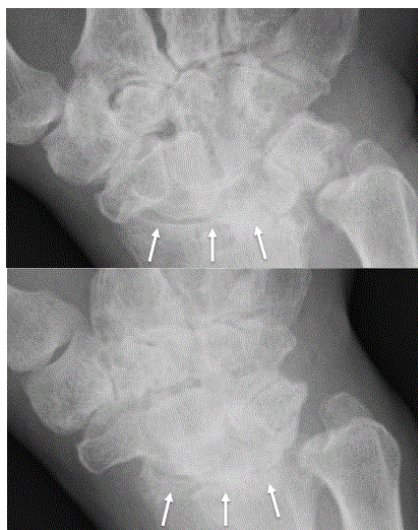


Figure 1: Bone erosions.

During this period, RA treatment and disease activity were unchanged. Although there was no difference of CRP (0.1 mg/dl, negative) before and after administration of denosumab, matrix metalloproteinase-3 decreased from 114.5 ng/ml to 78.0 ng/ml.

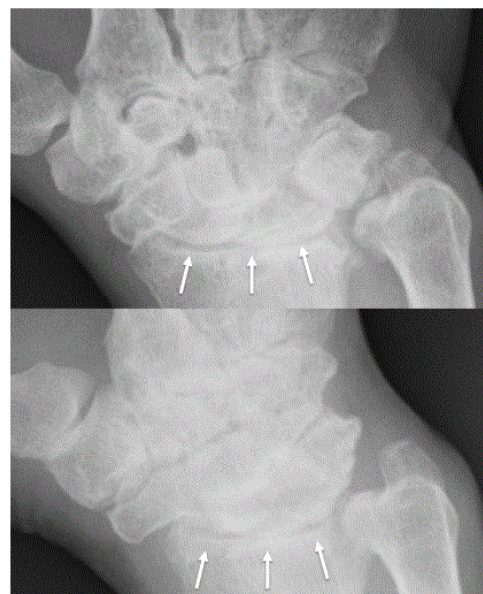


Figure 2: Denosumab therapy.

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