Response to: ‘Denosumab, cortical bone and bone erosion in rheumatoid arthritis’ by Rossini et al

Dear Editor,

We thank Rossini et al for their comments on our recent study on inhibitory effect by denosumab on the progression of bone erosions in Japanese patients with rheumatoid arthritis (RA). They addressed their observation from point of view of bone in patient with RA. We think their view brings treatment of RA closer to treatment of osteoporosis.

Activated osteoclasts decrease bone mineral density (BMD) and stimulate bone erosion in patients with RA. Receptor activator of nuclear factor kappa-B ligand (RANKL) promotes osteoclast differentiation, maturation, and activation. Denosumab is a fully human monoclonal antibody against RANKL that inhibits osteoclast formation, function, and survival. Denosumab treatment increases BMD in cortical and trabecular bone. In addition, denosumab has been shown to improve cortical bone microstructure in subjects with osteoporosis or low bone mass.

Increases in lumbar spine and total hip BMD and inhibition of progression of bone erosion have been observed with denosumab treatment in subjects with RA, and beneficial effects on cortical bone similar to those described in subjects with osteoporosis/low bone mass might be expected also in those with RA. Denosumab improves bone microstructure of both cortical and trabecular bone, its effects on osteitis as well as bone erosion are expected in patients with RA.

Denosumab is a unique treatment for patients with RA and low bone mass.

Tsutomu Takeuchi, Yoshiya Tanaka, Naoki Ishiguro, Hisashi Yamanaka, Toshiyuki Yoneda, Takeshi Ohira, Naoki Okubo, Harry K Genant, Désirée van der Heijde

1Division of Rheumatology, Department of Internal Medicine, Keio University School of Medicine, Tokyo, Japan
2First Department of Internal Medicine, University of Occupational and Environmental Health, Kitakyushu, Japan
3Department of Orthopedic Surgery, Nagoya University Graduate School of Medicine, Aichi, Japan
4Institute of Rheumatology, Tokyo Women’s Medical University, Tokyo, Japan
5Division of Hematology/Oncology, Indiana University School of Medicine, Indianapolis, Indiana, USA
6Department of Radiology, Medicine and Orthopaedic Surgery, University of California, San Francisco, California, USA
7Departments of Radiology, Medicine and Orthopaedic Surgery, University of California, San Francisco, California, USA
8Department of Rheumatology, Leiden University Medical Center, Leiden, The Netherlands


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REFERENCES