Acta Orthopaedica et Traumatologica Turcica 50 (2016) 708

Contents lists available at ScienceDirect

Acta Orthopaedica et Traumatologica Turcica

journal homepage: https://www.elsevier.com/locate/aott

Authors' reply

Serkan Sipahioglu

Harran University, Sanliurfa, Turkey

ARTICLE INFO

Article history:
Available online 10 November 2016

Thank you for your interest in our paper entitled, "Effect of salmon calcitonin treatment on serum and synovial fluid bone formation and resorption markers in osteoporosis patients".[1] In this study, we aimed to identify more specific parameters for osteoporosis diagnosis and osteoporosis treatment effectivity in relation to synovial fluid and to reveal the relationship between joint cartilage and osteoporosis. Apart from osteoporosis, as determined by bone resorption and formation products in synovial fluid, these results can also be interpreted for the evaluation of osteolytic activity in the joint which is significant for implant osteolysis. In osteoporosis, increased activity of receptor activator of nuclear factor kappa B ligand (RANKL) relative to its inhibitor osteoprotegerin in bone tissue which results in increased osteoclastic activity and bone resorption is a known issue.[2] Particle induced bone osteolysis which is a serious problem in implant loosening results from increased RANKL activity in the periimplant tissue which shows increased osteoclastic activity.[3] Therefore, increased bone resorption marker levels in synovial fluid can be expected in aseptic loosening caused by particle induced bone osteolysis, and our study can be a control group as for determination of bone resorption markers in the synovial fluid. Several studies have suggested that increased subchondral bone turnover is a determinant of progression of osteoarthritis similar to osteoporosis.[4] Disease progression can be followed by changes in the levels of bone resorption marker levels in the urine. More specifically bone resorption markers can be determined in the synovial fluid of the where osteoarthritis is progressively seen. A recent study has shown that high levels of bone resorption markers measured early in rheumatoid arthritis patients, predicted an increased risk of further articular damage.[5] However bone formation and resorption marker evaluation in these designated inflammatory diseases (osteoarthritis, rheumatoid arthritis, aseptic loosening) is more appropriate for following disease progression. Mechanisms that results in bone and cartilage metabolism should be detected as for preventing disease progression. It has been shown that bone resorption might related to A Disintegrin-like and Metalloproteinase with Thrombospondin type 1 Motifs (ADAMTS) enzyme activation/inhibition pathways.[6] In addition, while radiological measurements like bone mineral density provides local and static information only about the measured region, measurement of biochemical markers provides both systemic and dynamic results. So it determines the need for initiating treatment before radiological changes occur and also can be used for treatment adherence.[7] In addition, drugs targeting key molecules that are related to disease pathogenesis would be beneficial in improving symptoms or slowing disease progression.

References

1. Atbinici H, Sipahioglu S, Aksoy N, Baykara I, Isikan UE. Effects of salmon calcitonin treatment on serum and synovial fluid bone formation and resorption markers in osteoporosis patients. *Acta Orthop Traumatol Turc*. 2015;49:160–165.
2. Khosla S. Minireview: the OPG/RANKL/RANK system. *Endocrinology*. 2001;142:5050–5055.
3. Crotti TN, Smith MD, Findlay DM, et al. Factors regulating osteoclast formation in human tissues adjacent to peri-implant bone loss: expression of receptor activator NFkappaB, RANK ligand and osteoprotegerin. *Biomaterials*. 2004;25:565–573.
4. Bettica P, Cline G, Hart DJ, Meyer J, Spector TD. Evidence for increased bone resorption in patients with progressive knee osteoarthritis: longitudinal results from the Chingford study. *Arthritis Rheum*. 2002;46:3178–3184.
5. Straburzyńska-Lupa A, Nowak A, Romanowski W, Korman P, Pilaczyńska-Szcześniak Ł. A study of the link between bone turnover markers and bone mineral density with inflammation and body mass in postmenopausal women with active rheumatoid arthritis. *J Bone Miner Metab*. 2013;31:169–176.
6. Demircan K, Akyol S, Armutcu F. A multi-functional gene family from arthritis to cancer: a disintegrin-like metalloproteinase with thrombospondin type-1 motif (ADAMTS). *J Clin Anal Med*. 2013;4:429–434.
7. Lekkerkerker F, Kanis JA, Alsayed N, et al. Adherence to treatment of osteoporosis: a need for study. *Osteoporos Int*. 2007 Oct;18(10):1311–1317.

E-mail address: drserkans@gmail.com.

http://dx.doi.org/10.1016/j.aott.2015.11.002