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Change of bone mineral density and treatment of ankylosing spondylitis. Comment to the article of Kang KY et al. "The change of bone mineral density according to treatment agents in patients with ankylosing spondylitis" Joint Bone Spine 2011;78:188–93

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We read with great interest the paper by Kang et al. [1], analysing changes of bone mineral density (BMD) according to treatment regimen in ankylosing spondylitis (AS). They found that bone mass gained under treatment of AS was correlated with the reduction of biologic inflammation assessed by ESR and CRP, but disease activity (BASDAI, ASDAS) was not evaluated. As expected, increase in BMD was more important with an association of bisphosphonate and anti-TNF. Previous papers reported beneficial impact of TNF blockers upon BMD in AS. The mechanisms involved in this gain in BMD are the reduction of inflammation and subsequent reduction of bone resorption. Some other pathways may offer clues to explain this finding: the anabolic osteoblast signaling Wnt/DKK-1 pathway at one hand, and the adipokine network at the other hand.

Neutralisation of DKK-1 protects from systemic bone loss during inflammation and reduces sclerostin expression [2], but the results of TNF blockers upon DKK-1 levels or activity are discordant [3].

The other axis of interest is the role of adipokines. These mediators (adiponectin, resistin, leptin, and visfatin for the main), released from adipose tissue, have a close implication with systemic inflammation [4] and in AS [5,6], and bone metabolism [7]. TNF blockers have demonstrated an effect on body composition in several inflammatory conditions such as rheumatoid arthritis and in AS too. In AS, anti-TNF therapy is associated with increased body weight, and increased fat mass parallel to BMD gain [8,9].

In RA, the effect of treatment on adipokine expression gave variable results in several studies [4], and a recent study found that infliximab therapy increases body fat mass in early RA independently of changes in disease activity and levels of leptin and adiponectin. In AS, one study found no changes in adiponectin and leptin levels after 6 months of treatment with infliximab [10].

If adipokines may be a link between inflammation and bone metabolism, the results of the effect of the treatments, and in particular anti-TNF agents, upon this axis are not unequivocal, and more studies are requested in this field. Nevertheless, BMI variation at least, body composition and adipokine levels assessments should be taken into account when evaluating BMD in inflammatory diseases treated with TNF blockers, and biologic agents in general.

Disclosure of interest

The authors declare that they have no conflicts of interest concerning this article.

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