Editorial

Rheumatoid arthritis and osteoporosis

Rheumatoid arthritis is an inflammatory arthritis which changes the biomechanical properties of bones and leads to the alterations in bone components through the increased production of pro-inflammatory cytokines or by hormone mediated mechanisms (1-3).

In addition to traditional risk factors of osteoporosis, physical disability, inadequate treatment, and disease activity are also responsible factors of bone loss and osteoporosis in RA (4-6). Furtheremore, disease duration, seropositivity for anti-cyclic citrullinated peptide antibody (anti-CCP) and rheumatoid factor (RF) have been shown to be associated with bone loss in RA (7, 8).

In this issue of journal Mobini et al. (9), in a study of 121 patients with RA, with age of 55.7±10.1 years and mean disease duration of 10.1±9.2 years found prevalence rates of femoral neck osteoporosis at 16.5% and lumbar spine osteoporosis at 23.1%. The data reported in this study indicated that only the age and body mass index (BMI) were significant associated factors of osteoporosis in RA and the contribution of other factors of osteoporosis including disease activity, disease duration, physical disability, and seropositivity for anti-CCP and RF in the development of osteoporosis was not significant.

The authors found their data comparable with the rate of osteoporosis reported from some other geographic regions but lower than other studies.

The inconsistent results across various published studies may be attributed to several parameters including the characteristics of the study population, disease activity of the study population, quality of treatment, criteria used for definition of low bone mass, site of BMD measurement, and duration of RA (4-6, 10-13). In addition, age, severity of joint involvement, glucocoticoid therapy can differently affect the rate of bone loss during the course of RA and result in variations in results (11, 12, 14).

The status of bone mass in RA has been investigated in some case-control and longitudinal studies (8, 10, 15-17). Bone mass in RA was shown to be lower compared with non-RA controls. Low bone mass was more evident in untreated disease and in patients with prolonged disease duration particularly at the femoral neck region (8, 12, 14, 16). In some RA patients, even with active disease there was no reduction in bone mass and the patients' adequate bone mass was preserved particularly at the lumbar spine (14, 18).

It is known that low BMI and age are associated factors of osteoporosis in patients with RA as well as non –RA population. The main objective of studies which address osteoporosis in RA should be focused on the identification of reversible causes of osteoporosis such as disease activity, physical disability and elucidating their contribution in the development of osteoporosis for correction. These studies should include adequate number of patients for analysis, otherwise, the results may be underestimated. For example, the mentioned study required to include larger samples to confirm or deny a positive relationship between anti-CCP or RF with osteoporosis in RA. The most common causes of bone loss over the course of RA may be attributed to persistence of inflammation, adverse effects of cumulative dosages of glucocorticoid, physical disability due to joint destruction (4, 11). The appropriate treatment with anti-inflammatory drugs such as methotrexate with low-dose prednisolone can modulate the risk factors of osteoporosis and reduce the rate of bone loss and preserve further bone mass for later stages of RA (5, 6, 19). The improvement of hip synovitis with methotreate or glucocorticoids has been shown to prevent femoral neck osteoporosis (13). On the other hand, cumulative dose of glucocorticoids may result in bone loss and osteoporosis in lumbar spine. Conversely, glucocorticoids therapy by suppression of inflammatory process and joint pain can improve the physical activity and exert disease modifying effect and preserve bone mass (8, 12). The traditional factors of bone loss in RA and their relationship with osteoporosis may be recognized by conducting a case-control study. However, the contribution of RA associated factors like anti-CCP, RF, disease activity and functional disability in bone mass loss and osteoporosis require longitudinal studies (6, 14).

Despite the several studies in this context, this issue requires further studies particularly in regard to bone mass protective effect of new treatment in RA.

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