Letters to the Editor

Drs. Konsta and van der Horst-Bruinsma reply

To the Editor:

We thank Dr. Slouma et al1 for their earnest interest in our recent article, which examined the prevalence and radiographic progression of hip involvement in patients with ankylosing spondylitis (AS) treated with tumor necrosis factor inhibitors (TNFi).2

The prevalence of hip involvement in our study was very close to the authors’ previous one (60/165 patients, 36.4%).3 Moreover, the relation between disease activity (ie, C-reactive protein [CRP]), functional impairment (ie, Bath Ankylosing Spondylitis Functional Index [BASFI]), spinal radiographic damage (ie, presence of syndesmophytes), and hip involvement was confirmed by our study;2 similar to the authors’ previous work.3

In their correspondence, Slouma et al raise the question of potential structural repair of hip joints after treatment with TNFi. However, radiographic improvement of hip joints after TNFi initiation is reported in only 7 cases in literature review: the authors’ described case1 and in 6 patients with AS in the study of Song et al.4 The majority of observational studies that focus on radiographic progression of hip involvement after TNFi therapy report no deterioration of this manifestation.5,5,7 Our group demonstrated nonsignificant radiographic progression of hip involvement after 6 (SD 2.5) years of infliximab treatment in 23 patients with AS using a quantitative method of measurement of hip mean joint space width (MJSW) in addition to the Bath AS Radiographic Hip Index (BASRI-hip) score.1 Rocha et al1 described a case series of 4 patients with AS with hip involvement. Three of those patients, who remained on TNFi treatment, showed no radiographic deterioration of hips. Even Slouma et al, in their previous study4, reported that the MJSW of 23 patients who were treated with TNFi did not change significantly.

Our study3 included a large number of patients, with and without hip involvement, in a follow-up period of 7.0 (SD 2.3, range: 2.7-12.7) years. To derive more conclusions regarding hip radiographic progression, the radiographic assessment was conducted at 3 timepoints: before TNFi initiation, after 2.5 (SD 0.7) years, and after 7.0 (SD 2.3) years of TNFi treatment. The MJSW had a nonsignificant reduction at the aforementioned time intervals, in both patients with and without hip involvement, as displayed in Table 4 of our article.2 The slight increase in BASRI-hip score and decrease in MJSW at the end of follow-up could be attributed to age-related osteoarthritic changes. Indeed, no cases with reparative radiographic findings after TNFi treatment were demonstrated.

As far as the ultrasound or magnetic resonance imaging (MRI) is concerned, both can detect earlier stages of hip arthritis characterized by inflammation. Moreover, ultrasound seems to be superior to radiographs even in detecting structural, degenerative lesions.8 Thus, reparative findings in hip joints can be visualized by the aforementioned methods even after a relatively short period of TNFi treatment.8,10

Nevertheless, the radiographic improvement that Slouma et al1 and Song et al4 demonstrated mainly as widening of hip joint space could be attributed to a flexion contracture of hip due to arthritis, which resolved during TNFi treatment. We assume that after the resolution of hip arthritis (by TNFi), the leg can be extended and the joint space at the radiograph could be more visible and wider.

To conclude, TNF inhibition seems to stabilize the radiographic damage in the hip and even more data demonstrate this evidence. We sincerely thank Dr. Slouma and colleagues for their interest, time, and effort to bring forth this point of discussion through our study.

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REFERENCES