Bone marrow oedema on sacroiliac/spine MRI: is it really a sign of objective inflammation warranting treatment?

Sirs,
Baraliakos et al. reported on the fluctuation in sacroiliac and/or spine magnetic resonance imaging (MRI) among patients with non-radiographic axial spondyloarthritis (axSpA) treated with placebo for 12 weeks (1). The reason for choosing placebo-treated patients was to investigate the natural course of these patients. Among the 29 patients with a negative MRI at baseline, 30% developed inflammation on MRI. The authors concluded that clinically active patients may require retesting for objective inflammation to guide therapy.

There are some issues to be considered before making this conclusion. Firstly, while sacroiliac MRI is a very sensitive method to detect bone marrow edema (BME), these lesions can be present in other conditions such as mechanical stress (2). In a recent study, Varkas et al. studied 22 military recruits and showed that 41% of the recruits already had ≥1 BME lesion at baseline and BME lesions were detected in 50% of the recruits after 6 weeks of intense physical training. The frequencies of positive MRI according to ASAS definition at baseline and after physical training were 23% and 36%, respectively (3). Secondly, Rudwaleit et al. have reported a significant regression of spinal inflammation by MRI in patients with ankylosing spondylitis during etanercept therapy. However, this was not true for sacroiliac inflammation (4).

A similar example was observed during the previous efforts to use MRI to diagnose avascular necrosis of the hip. After the introduction of MRI in the 1980s, avascular necrosis not detected with plain radiographs was noted with MRI and orthopedic surgeons recommended core decompression procedure to all patients with an abnormal MRI. However, it was later observed that there was spontaneous improvement in MRI findings in many patients, especially in those who were asymptomatic (6). Most of the asymptomatic patients with an abnormal MRI developed no abnormality on plain radiographs after a mean follow-up of 16 years (6).

The other issue is the extent of contribution of MRI to clinical findings in making treatment decisions. A survey looking at the preferences of 600 physicians in treatment decisions for rheumatoid arthritis showed that nearly 50% made clinical decisions based on patient history and physical examination. We think this would hold true for all rheumatic diseases and using MRI as the only gold standard for treatment decisions would not be appropriate.

In the light of these issues and the fact that 21% of the 61 patients with a positive MRI at baseline had a negative MRI at week 12, we think that knowing the changes in inflammation on MRI among patients treated with adalimumab and the correlation between disease activity and inflammation on MRI would be more informative to make a conclusion whether inflammatory signs on MRI always indicate active disease or the transient fluctuation in MRI findings may be a non-specific finding in axSpA.

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