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Seronegative Spondyloarthritis and Magnetic Resonance Imaging

To the Editor:

Troppmann and Karsh report a study of 29 patients with spondyloarthritis (SpA) in which they conclude that the requirement for magnetic resonance imaging (MRI) could present an additional barrier to timely treatment with anti-tumor necrosis factor (anti-TNF) agents in 12 (41% of the cohort evaluated) patients with SpA in Canada

Of these 12 patients, 7 had a Bath Ankylosing Spondylitis Disease Activity Index (BASDAI) ≥ 4, with low acute-phase reactants, and 5 had low BASDAI (≤ 4) but elevated acute-phase reactants. The Spondyloarthritis Research Consortium of Canada treatment recommendations for access to anti-TNF require at least 2 out of 3 of the following: BASDAI ≥ 4, elevated acute-phase reactants, and MRI features of inflammation in sacroiliac joints or spine. So it is correct that these 12 patients would not meet the criteria for access to anti-TNF. However, several issues are not addressed in the discussion of these observations.

The most important issue is that it is not clear how many of these patients actually required treatment with anti-TNF therapy. For instance, the 5 patients in the group with a BASDAI ≤ 4 and elevated acute-phase reactants had a mean BASDAI of only 2.7! How many of these patients would actually be considered candidates for anti-TNF therapy on clinical grounds? Is the cost of such treatment justified, potentially for several decades, in the absence of any additional features of active disease? What other objective measures of disease would be more helpful than an MRI to verify the presence of active disease?

It is also unclear how many of the 7 patients with a BASDAI ≥ 4 but normal acute-phase reactants would actually be considered candidates for anti-TNF therapy. A previous report has shown that as many as 40% of patients with a BASDAI ≥ 4 reported being in a patient-acceptable symptom state (PASS)2. Moreover, the BASDAI does not discriminate between mechanical and inflammatory causes of back pain. Consequently, does the absence of any objective measure of active disease constitute justifiable grounds for long-term therapy with anti-TNF agents?

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