Dr. Vencovský, *et al* reply

To the Editor:

We thank Drs. Jearn and Kim for their comments[1] on our report regarding arthritis in idiopathic inflammatory myopathies (IIM)[2]. They point to a very high incidence of arthritis among our patients with anti-Jo1–positive myositis, and use their cohort of patients to show that there is a more intimate association with pulmonary involvement rather than arthritis in anti-Jo1–positive patients.

We reported 93% prevalence of arthritis in anti-Jo1–positive patients with IIM. The association between arthritis and the presence of anti-Jo1 antibodies is well accepted and has been described in many reports with variable frequencies, usually exceeding 50%[3,4,5,6,7,8,9,10]. Several landmark papers show a frequency that is remarkably similar to our data. Love, *et al*[3] reported 94% prevalence of arthritis in a group of antisynthetase antibody-positive patients, three-fourths of which had anti-Jo1. Marguerie, *et al*[4] found arthritis/arthralgias in 95% of patients with anti-Jo1 antibodies. Yoshida, *et al*[5] reported frequency of arthritis to be 100% among a group of anti-Jo1–positive patients, with significantly lower prevalence of 48% in patients with anti-Jo1–negative myositis. Interestingly, in Yoshida, *et al*'s paper, autoantibodies were detected by double immunodiffusion (DID), the same method used by Jearn and Kim in the study described in their commentary[1] on our publication.

Therefore, we see our observation as not considerably different from previous data. The main reason for the high prevalence of arthritis in our patients is the methodological approach used. We have actively searched for swelling and tenderness on physical examination of 66/68 joints or used a credible history of arthritis reported by the patient or treating rheumatologist, as explained in our paper. We point out that physical examination is still considered to be the standard method of joint involvement evaluation in arthritis, as illustrated in the current classification guidelines for rheumatoid arthritis. The main intention of our report was to describe various aspects of arthritis in patients with myositis, primarily because we frequently encounter this symptom in our patients and were not able to find a comprehensive evaluation of arthritis in a sufficiently sized cohort of patients with IIM in the literature. There is a large body of evidence that anti-Jo1, as well as other antisynthetase antibodies, are strongly associated with pulmonary disease in IIM, and numerous papers describe the association in various levels of detail. Therefore, we reported only on the basic data regarding other features of IIM, including lung involvement, and concentrated on the lesser-known aspects pertaining to joint involvement. Indeed, we were able to establish that 53% of patients had arthritis at any point during the disease course, even preceding the onset of myositis in some cases.

Jearn and Kim[1] analyzed 2 groups of patients with and without anti-Jo1 antibodies and were unable to find a statistical difference in arthritis presence. They suggest that ethnic background or anti-Jo1 antibody detection techniques might be responsible for the differences. The difference in methods used to detect arthritis is much more likely to explain the conflicting results. The use of radiographs to screen for joint involvement in IIM is clearly much less sensitive than physical examination and therefore may not be a reliable way to assess the prevalence of arthritis.

DID is probably less sensitive than the enzyme immunoassays and radioactive immunoprecipitation that we used for anti-Jo1 detection. However, that fact does not explain the different results because there was a significant difference in the prevalence of arthritis between groups defined by antibody presence identified by DID in Yoshida, *et al*[5]. We agree that other differences in the patient groups and their genetic background might have played some role.

Arthritis should be recognized as a frequent extramuscular manifestation in patients with IIM, which may pose a diagnostic dilemma at the early stages of the disease. Because of the strong association of arthritis with anti-Jo1 antibodies, their detection may be particularly helpful in establishing the correct diagnosis.

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