Reply

Sirs,

We are very grateful that you find our article about epidemiological characteristics of juvenile spondyloarthropathies (JSpA) interesting (1), and you gave us a brief report of successfully treated recurrent episodes of peripheral enthesitis with short courses of anti-TNF-α therapy (2). As we mentioned in our article, enthesitis was present in a great number of patients with JSpA at disease onset, which is in contrast to adults. In our study we showed only the history, clinical, laboratory and imaging data of patients, but we did not discuss the therapy. The majority of our patients received nonsteroidal anti-inflammatory drugs alone or combined with disease modifying antirheumatic drugs, and in severe cases corticosteroids. Till now, only two patients have been treated with anti-TNF therapy.

The first patient is a 15-year-old girl in whom JSpA started six years ago. The first symptoms were peripheral arthritis and ethesitis of the Achilles tendon with bilateral anterior uveitis. Because she was refractory to standard therapy, anti-TNF-α (infliximab 3 mg/kg at week 0, 2, and 6 followed by infusions every eight weeks) was introduced. She responded very well to this therapy (infliximab and methotrexate) and within six months she gained remission. Anti-TNF-α was continued and stopped after two years, but in only four months joint disease was reactivated without any signs of eye disease. Re-treatment with anti-TNF-α was started, and now after three months there has been a significant clinical and laboratory (C-reactive protein and erythrocyte sedimentation rate) improvement.

The second patient is a 14-year-old girl who was first misdiagnosed as having juvenile idiopathic arthritis – oligoarticular form. After two years she developed back pain, and standard radiographs showed bilateral sacroiliitis. She received combination therapy with methotrexate and/or sulfasalazine and non-steroidal anti-inflammatory drugs, but without any improvement. Only high doses of corticosteroids (1 to 1.5 mg/kg) achieved good clinical and laboratory response, but when we, several times, started with a gradually tapering of steroids there was an immediate worsening of all symptoms. We decided to introduce anti-TNF-α (etanercept 0.4 mg/kg twice weekly) with methotrexate and low doses of corticosteroids. In one month she responded remarkably, with an immediate clinical improvement (reduction of pain and disease activity on a visual analogue scale) and normalisation of C-reactive protein and erythrocyte sedimentation rate. After three months of receiving of anti-TNF-α, significant radiologic improvement was noticed. Now, after seven months, she still receives a combination of anti-TNF-α and methotrexate without any clinical and laboratory signs of disease.

According to your letter we found that you re-treated your patient with anti-TNF-α when he again started to complain of pain at the insertion of both plantar fasciae and the left Achilles tendon with worsening on MRI, and normal acute phase reactants. The main reason that until now we have introduced anti-TNF-α in only two JSpA patients (among 136 JSpA patients diagnosed in the last 14 years) is that we cannot introduce anti-TNF-α if we do not have elevated laboratory parameters (erythrocyte sedimentation rate higher than 28 mm/h and/or C-reactive protein higher than 12 mg/L) although we have significant radiological and clinical progression.

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