with ankylosing spondylitis ATI developed within 24 weeks in association with undetectable serum trough infliximab levels, inefficacy of infliximab and infusion reactions. The number of patients, however, is too small to draw definite conclusions, but interestingly, these data point in the same direction as described previously in rheumatoid arthritis.³ Lower serum trough infliximab levels could be explained by enhanced clearance because of immune complex formation between anti-infliximab antibodies and infliximab. To prevent ATI formation that might inhibit the efficacy of infliximab, it might be helpful to increase the dosage of infliximab (as occurs in treatment of rheumatoid arthritis with infliximab), to shorten the interval between infliximab infusions (as is currently the strategy in Crohn's disease) or to provide coadministration of other immunosuppressives (such as methotrexate). These data should be confirmed in a larger group of patients with ankylosing spondylitis to develop a more patient-specific treatment, which might predict the inefficacy of infliximab at an early stage and might prevent adverse reactions.

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Magnetic resonance imaging in patients with rheumatoid arthritis with complete remission treated with disease-modifying antirheumatic drugs or anti-tumour necrosis factor α agents

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Biological treatment, mainly with anti-tumour necrosis factor (TNF) α agents, of patients with rheumatoid arthritis, has been shown to modify their clinical course and progression, and delay or avoid its radiological progression.^{1,2} Disease remission in rheumatoid arthritis has been traditionally considered when there is no clinical or biochemical evidence of disease activity.³

Magnetic resonance imaging (MRI) has been shown to be more sensitive than clinical and radiological parameters in evaluating the bone erosions and the inflammatory phenomenon that characterise disease activity in patients with rheumatoid arthritis.^{4–9} For this reason, the therapeutic response to disease modifying antirheumatic drugs (DMARDs) has been evaluated by MRI.^{4–9} However, there are no comparative studies on the therapeutic effectiveness of DMARDs and anti-TNF α agents using this type of imaging studies, mainly when patients have reached clinical remission.

We studied 10 patients with rheumatoid arthritis, who were induced to clinical and laboratory remission with DMARDs

alone (n = 5, table 1) or with an anti-TNF α agent with or without DMARDs (n = 5). In both groups, MRI of the hands was performed at least 3 months after disease remission in sagittal and coronal projections, and 24 joints were assessed in each patient. In the five patients receiving DMARDs alone, we found MRI evidence of synovitis, with a total count of 24 inflamed joints. By contrast, only two patients receiving anti-TNF α treatment showed MRI evidence of synovitis, with only two inflamed joints (p = 0.001, Fisher's exact test). In addition, we did not find a significant correlation between clinical or laboratory data and MRI results, as has been reported previously.^{9,10}

Our results corroborate the fact that MRI imaging is a sensitive parameter for the detection of joint inflammation and destruction in patients with rheumatoid arthritis.^{4–9} Our data also show that complete remission in this condition is easily achieved with the addition of anti-TNF α agents, and that treatment with DMARDs alone induces only an apparent remission, defined by clinical and laboratory parameters. We

Table 1 Clinical data of patients at the end of the study

Sex	Age (years)*	Evolution time (years)*	Functional	RF class	ESR	CRP	Drugs
Patients receiving DMARDs or anti-TNFα treatment							
F	50	8	I	+	11	12	Mtx, Etanercept
F	41	7	I	+	12	6	Etanercept
F	55	14	I	–	11	0	Mtx, Etanercept
F	48	5	I	–	6	0	Mtx, CE, Infliximab
M	49	6	I	+	0	0	Mtx, Etanercept
Patients receiving DMARDs alone							
F	29	2	I	–	14	0	Mtx, Aza, SZ, CE
F	61	2	I	–	12	0	Mtx, SZ, CE
F	63	3	I	+	15	0	Mtx, SZ, CQ
F	36	3	I	–	8	0	Mtx, SZ
F	45	3	I	–	11	0	Mtx, SZ, Lef, CE

Aza, azathioprine; CE, corticosteroids; CRP, C reactive protein; CQ, chloroquine; DMARD, disease-modifying antirheumatic drug; ESR, erythrocyte sedimentation rate; F, female; Lef, leflunomide; M, male; Mtx, methotrexate; RF, rheumatoid factor; SZ, sulfasalazine.

Figure 1 Bone erosions in a patient with rheumatoid arthritis receiving disease-modifying antirheumatic drugs without anti-tumour necrosis factor α , and with clinical and laboratory evidence of disease remission.

think that the routine use of MRI for the evaluation of therapeutic response in rheumatoid arthritis could modify current schemes of treatment in this disease, preventing the structural damage that characterises this condition.

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