malignancy occurred. In both of these cases it is unlikely that there was a correlation between the adverse event and the study treatment, especially as the treatment was short.

DISCUSSION

Our data confirm the efficacy of infliximab in patients with PsA, suggesting that it may be the preferred treatment for patients resistant to conventional DMARDs.

In other reports the efficacy of treatment with anti-TNF α agents on PsA was less pronounced^{4 s} than in our study. This may be due to several factors, including the selection of the patients (11 of our 12 patients had a peripheral polyarthritis) and the therapeutic regimen (the association of MTX with infliximab might have enhanced its therapeutic effect). The maximum effect of this treatment on articular symptoms may not be apparent after the loading dose regimen of three infusions, but seen later on. Controlled studies and longer follow up are needed to optimise the selection of patients who can benefit from this form of treatment.

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REFERENCES

- Gladman DD, Stafford-Brady F, Chang C-H, Lewandoski K, Russell ML. Longitudinal study of clinical and radiological progression in psoriatic arthritis. J Rheumatol 1990;17:809–12.
- 2 Abu-Shakra M, Gladman DD, Thorne JC, Long J, Gough J, Farewell VT. Long-term methotrexate therapy in psoriatic arthritis: clinical and radiological outcome. J Rheumatol 1995;22:241–5.
- 3 Mease PJ. Tumour necrosis factor [TNF] in psoriatic arthritis: pathophysiology and treatment with TNF inhibitors. Ann Rheum Dis 2002;61:298–304.
- 4 Mease PJ, Gaffe BS, Metz J, Vanderstoep A, Finck B, Burge DJ. Etanercept in the treatment of psoriatic arthritis and psoriasis: a randomised trial. Lancet 2000;356:385–90.
- 5 Van den Bosch F, Kruithof E, Baeten D, De Keyser F, Mielants H, Veys Sond all bosch F, Kruino E, Balein D, De Keyser F, Mieldhis H, Veys EM, Effects of a loading dose of three infusions of chimeric monoclonal antibody to tumour necrosis factor (infliximab) in spondyloarthropathy: an open pilot study. Ann Rheum Dis 2000;59:428-33.
 Braun J, Brandt J, Listing A, Zink A, Alten R, Golder F, et al. Treatment of active ankylosing spondylitis with infliximab: a randomised controlled multicentre trial. Lancet 2002;357:1187-93.
 Gorman JD, Sack KE, Davis JC. Treatment of ankylosing spondylitis by inhibition of tumour necrosis factor a. N Engl J Med 2002;346:1349-56.
 Kruithof E, Van den Bosch E, Baaten D, Herssens A, De Keiser F.

- 8 Kruithof E, Van den Bosch F, Baeten D, Herssens A, De Keiser F, Mielants H, *et al.* Repeated infusions of infliximab, a chimeric anti-TNF monoclonal antibody, in patients with active spondyloarthropathy: one year follow up. Ann Rheum Dis 2002;61:207–12.
- 9 Dougados M, van der Linden S, Juhlin R, Huitfeldt B, Amor B, Calin A, et al. The European Spondyloarthropathy Study Group preliminary criteria for the classification of spondyloarthropathy. Arthritis Rheum 100124:1218 1991;34:1218-27.

Twenty eight joint count disease activity score in recent onset rheumatoid arthritis using C reactive protein instead of erythrocyte sedimentation rate

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reactive protein (CRP) and erythrocyte sedimentation rate (ESR) are traditional markers of disease activity in rheumatoid arthritis (RA). Although the two tests show a relatively high degree of correlation and follow the same reaction pattern, the variance of CRP is independent of ESR to some extent.¹ Apart from mirroring inflammation, ESR is affected by immunoglobulin levels, sex, and abnormal size or shape of red blood cells.23 CRP, which correlates more significantly than ESR with disease activity,¹³⁴ is a predictor of functional status/outcome and joint damage in RA.5-7 Although CRP is the better marker of inflammation, ESR adds information reflecting disease severity,4 8 and a combination of the tests may be worthwhile.

To create a simple instrument for assessment of disease activity in RA, Prevoo et al developed a disease activity score (DAS28) based on the number of tender and swollen joints (n=28), ESR, and the patient's self estimated general health.⁹ DAS28 has become an important tool for rheumatologists to monitor disease activity. However, considering its better indication of disease activity, it would be preferable if the CRP value could be used as an alternative to ESR for calculation of the DAS. Paulus et al constructed a nomogram to convert CRP values to ESR,10 which in turn could be successfully used to calculate the traditional DAS.

In our study we investigated whether it was possible simply to use the numerical value of CRP concentration (mg/l) instead of ESR (mm/1st h) for the calculation of DAS28. Seventy patients with recent onset RA (≤ 1 year) were included from the ongoing Swedish early RA cohort designated "TIRA". Each patient was seen by the same rheumatologist at inclusion and after 3, 6, 12, 18, and 24 months. The patients' assessments of general health assessed by a visual analogue scale (mm), the number of swollen and tender joints respectively (28 joint counts), ESR (mm/1st h), and plasma CRP (mg/l) were registered at all visits. CRP was analysed by turbidimetry at the hospital laboratory and the results presented as <10 or a value without decimals from 10 with no upper limit. CRP < 10 was given the value 5. In comparing ESR and CRP, 398 cases were analysed. The traditional DAS28 (DAS28/ESR) was calculated using the formula presented by Prevoo et al.9 A DAS28 based on CRP instead of ESR was also calculated by substituting the numerical value of ESR in the formula by plasma CRP (DAS28/CRP).

The mean (SD) ESR was 25.4 (2.73) mm/1st h (range 2-105). CRP ranged from 5 to 122 mg/l with a mean (SD) value of 15.65 (19.16). Figure 1A illustrates the correlation between individual CRP and ESR values (Spearman's correlation coefficient, $r_s=0.69$). Figure 1B illustrates the linear correlation between the two modes of DAS28 calculation $(r_s=0.93)$. DAS28 based on ESR ranged from 0.50 to 8.50 (mean (SD) 4.04 (1.48)) and DAS28/CRP ranged from 1.14 to 8.00 (mean (SD) 3.68 (1.46)). Thus the mean (SD) DAS28/ESR of 4.04 (1.48)was not statistically different from DAS28/CRP (3.68 (1.46)).

To conclude, the correlation between ESR and plasma CRP in this study was within the same magnitude as that reported



Figure 1 Comparison of ESR and plasma CRP (A), and DAS28 calculated with CRP and ESR respectively (B).

in previous studies on RA.¹⁴ Although the two values were by no means interchangeable, the correlation between the "orthodox" DAS28 and DAS28 based on plasma CRP was remarkably high, because the weight of the laboratory test is low and thus has only a small impact on the disease activity score compared with the other measures. These findings may be useful to all rheumatologists who prefer CRP as a laboratory marker of inflammation in RA, and who do not have access to ESR values for the calculation of the DAS28.

Letters

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REFERENCES

- Mallya R, de Beer FC, Berry H, Hamilton EDB, Mace BEW, Pepys MB. Correlation of clinical parameters of disease activity in rheumatoid arthritis with serum concentration of C-reactive protein and erythrocyte sedimentation rate. J Rheumatol 1982;9:224–8.
- 2 Talstad I, Scheie P, Dalen H, Roli J. Influence of plasma proteins on erythrocyte morphology and sedimentation. Scand J Haematol 1983;31:478–84.
- 3 Kushner I. C-reactive protein in rheumatology. Arthritis Rheum 1991;34:1065–8.
- 4 Wolfe, F. Comparative usefulness of C-reactive protein and erythrocyte sedimentation rate in patients with rheumatoid arthritis. J Rheumatol 1997;24:1477–85.
- 5 Jansen LM, van Schaardenburg D, van Der Horst-Bruinsma IE, Bezemer PD, Dijkmans BA. Predictors of functional status in patients with early rheumatoid arthritis. Ann Rheum Dis 2000;59:223–6.
- 6 Jansen LMA. Predictors of radiographic joint damage in patients with early rheumatoid arthritis. Ann Rheum Dis 2001;60:924–7.
 7 Devlin J, Gough A, Huissoon A, Perkins P, Holder R, Reece R, et al. The
- 7 Devlin J, Gough A, Huissoon A, Perkins P, Holder R, Reece R, et al. The acute phase and function in early rheumatoid arthritis. Creactive protein levels correlate with functional outcome. J Rheumatol 1997;24:9–13.
 8 Del Martin Martine JC, Schemat JC, Schemat
- 8 Bull BS, Westengard JC, Farr M, Bacon PA, Meyer PJ, Stuart J. Efficacy of tests used to monitor rheumatoid arthritis. Lancet 1989;ii:965–7.
- 9 Prevoo MLL, van't Hof MA, Kuper HH, van Leeuwen MA, van de Putte LBA, van Riel PLCM. Modified disease activity scores that include twenty-eight-joint counts. Arthritis Rheum 1995;38:44–8.
- 10 Paulús HE, Ramos B, Wong WK, Ahmed A, Bulpitt K, Park G, Sterz M, et al. Equivalence of the acute phase reactants C-reactive protein, plasma viscosity, and Westergren erythrocyte sedimentation rate when used to calculate American college of rheumatology 20% improvement criteria or the disease activity score in patients with early rheumatoid arthritis. J Rheumatol 1999;26:2324–31.

Horner's syndrome as an initial manifestation of Takayasu's arteritis

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akayasu's arteritis (TA) is a granulomatous vasculitis of the large arteries. It typically produces segmental arterial narrowing and occlusion, but aneurysm formation is uncommon.¹ Subclavian artery aneurysm presenting with Horner's syndrome is very rare.² We report on a patient with Horner's syndrome caused by subclavian artery aneurysm in TA.

CASE REPORT

A 37 year old Korean man was admitted because of right side ptosis and anhidrosis. He had visited the ophthalmology

department three weeks previously. Initially, his margin reflex distance 1 (MRD₁) was 1.5 mm (right) and 3.0 mm (left). The neostigmine test was performed, but the MRD₁ did not change. After administration of phenylephrine 1 drop, however, MRD₁ was improved to 3.0 mm in the right eye. Horner's syndrome was diagnosed. He had no trauma history. He did not complain of any claudication, pain, or dizziness. His blood pressure was 110/70 mm Hg in both arms and pulse rate 80 beats/min. Complete blood counts, serum protein, and urine analysis were normal. Antinuclear antibody and rheumatoid factor were