Correspondence

Atlantoaxial subluxation and spinal cord compression in psoriatic arthropathy

SIR, We read with interest the report by Pease and Pozo on atlantoaxial subluxation and spinal cord compression in psoriatic arthropathy.¹ It appears, however, that even in the age of computerised literature searches one can fail when looking for specialised data. The authors state quite correctly that the incidence of atlantoaxial subluxation (AAS) in psoriatic arthropathy is uncertain. The risk of AAS in psoriatic arthropathy, however, and in other forms of seronegative spondylarthritis has been reported, as has severe neurological dysfunction resulting from cord compression due to psoriatic AAS.²

The case presented by Pease and Pozo shows, in the lateral cervical radiography, good bone structures in the occipitocervical junction and, as there is no sign of vertical C2 subluxation one must assume that the atlantoaxial facet joints are intact.^{3,4} Thus the AAS must be caused by ligamentous laxity.⁵ It is known that involvement of ligamentous tissues is more frequent in psoriatic arthropathy than in seropositive rheumatoid polyarthritis.

The initial treatment with skull traction was certainly correct, but extension reduces AAS more effectively than traction.⁶

Further, the authors report a C5 motor defect on the left and present a neutral position lateral cervical radiography showing subaxial changes at C4–5 and C5–6 levels. In inflammatory diseases, subaxial instabilities associated with soft tissue proliferation inside the spinal canal are often the cause of radiculopathy.^{3,6} In this case the neurological problems were initiated by minor trauma to the skull. At least theoretically the transient spinal cord trauma could have been either at the atlantoaxial level or, alternatively, subaxially.

This article is valuable confirmation of previous reports.

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Serum cytidine deaminase as a laboratory test for acute inflammation in rheumatoid arthritis

SIR, The report by Thompson *et al* of raised levels of cytidine deaminase (CD) in the serum and synovial fluid of patients with rheumatoid arthritis (RA) raised the exciting prospect of a laboratory test more closely related to acute inflammation than any of those currently used in the study of inflammatory joint diseases.¹ We wish to report our experience in the use of serum CD measurement in RA.

We studied 19 patients with definite/classical RA with varying degrees of disease activity. Clinical measurements included count of total number of involved joints, number of swollen joints, Ritchie articular index, grip strength, duration of morning stiffness, time to onset of fatigue, fatigue analogue scale, pain analogue scale, and patient/physician global assessments of current status. Patients were ranked according to each measurement and a global ranking of disease severity was constructed on the basis of the sums of individual rankings. Routine laboratory tests included complete blood count, erythrocyte sedimentation rate (ESR), urine analysis, serum electrolytes, glucose, urea, creatinine, total protein, albumin, hepatic enzymes, rheumatoid factor, C reactive protein (CRP), antinuclear antibody, and quantitative immunoglobulins. A global ranking of severity of laboratory abnormalities was also constructed. CD was measured by the technique of Jones *et al*² at room temperature on serum samples previously frozen at -20°C .

We obtained a mean (SD) value for serum CD of 4.3 (2.2) U/ml for our 19 patients. This is slightly lower than the figure of 5.5 (2.3) U/ml reported by Thompson *et al*, but comparable. Our range of 1.3 to 10.7 U/ml appears almost identical to that illustrated in Fig. 2 of their paper. Eight patients had levels >4 U/ml (i.e., more than 2SD above the normal mean of Thompson *et al*).

Regrettably, we were able to show very little relation between the patients' clinical status and their CD levels.

Table Correlations between selected clinical and laboratory measurements

	Total joint count	Swollen joints	Ritchie index	Morning stiffness	Fatigue onset	Fatigue analogue scale	Pain analogue scale
CD	0	0	0	0	0	0	0
ESR	+	++	+	±	±	0	±
CRP	+	+	0	0	+	+	++
Platelets	±	+	0	0	0	0	0
Albumin	++	++	++	±	±	+	±
IgG	+	±	±	0	0	0	+

0=no correlation; ±=borderline correlation, $p<0.1$; +=significant correlation, $p<0.05$; ++=significant correlation, $p<0.01$. CD=cytidine deaminase, ESR=erythrocyte sedimentation rate, CRP=C reactive protein.

No correlations were evident between the CD figures and values for individual clinical measurements. In contrast, several of the routine laboratory tests showed significant or almost significant correlations. These are illustrated in the Table. Comparison of CD figures and overall ranking of disease severity showed that the two lowest figures (1.3 and 1.8 U/ml) were seen in the patients with the lowest disease severity ranking (1 and 2 respectively). Beyond that, the distribution of CD figures appeared to be quite random. The highest CD figures (10.7, 7.8, and 6.0 U/ml) were seen in patients with ranks 12, 6, and 7 respectively, whereas the most severely affected patients—ranks 16 (two patients), 18, and 19—had CD figures of 2.4, 5.8, 3.2, and 4.2 U/ml respectively.

CD correlated significantly with only one other laboratory test—namely, serum IgA ($r=0.556$; $p<0.05$). The two next highest correlation coefficients—for serum albumin ($r=-0.382$) and CRP ($r=0.356$)—were not significant. In contrast with the finding of Thompson *et al*, the correlation between CD and ESR was non-existent ($r=0.026$). Although the titre of rheumatoid factor did not correlate with CD level, we did find that seropositive patients had significantly higher CD levels than seronegative patients (4.8 (2.2) v 2.6 (1.4) U/ml; $p<0.05$).

In summary, although we have confirmed that patients with RA may have raised levels of serum CD, the latter do not appear to bear any relation to the clinical status of the patient or to commonly used routine laboratory tests. In our hands, at least, measurement of serum CD does not appear to be a useful contribution to the assessment of RA.

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SIR, We are pleased to learn that Dr Chalmers and colleagues have been comparing serum cytidine deaminase (CD) with other measures of joint inflammation in rheumatoid arthritis. Our suggestion that serum CD estimations may offer an integrated measure of acute (polymorph related) inflammation certainly requires 'further testing' at this stage.

Because of their small sample of 19 patients, however, the confidence intervals around the correlation coefficients are likely to be large and the power of the results correspondingly small.

Although the raw data for the patients are not provided the report suggests that results were normally distributed. This would allow the use of the more sensitive and reliable Pearson correlation coefficient¹ as in our paper.

Despite these shortcomings the study showed correlation between CD and albumin ($r_s=-0.382$) or C reactive protein ($r_s=0.356$) that approaches statistical significance and would be significant at the 1% level had 49 patients been studied as in our paper.

Under the circumstances the conclusions of Chalmers and colleagues may be inappropriate. Long term studies are underway which we hope will determine the usefulness of CD in the assessment of arthritis.

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