Antiperinuclear factor in chronic juvenile arthritis

Sir: Nesher et al recently described the first comprehensive series concerning antiperinuclear factor in juvenile chronic arthritis (JCA). They found an overall positivity of 34%; in patients with the polyarticular type of the disease 16/28 patients were positive.1 Our results are at variance with these data: in a group of 313 patients only three were positive, all of them children with polyarticular onset (table). Thus our results are more in line with a recently reported Czechoslovakian series.2

Our material consisted of 49 fresh and 264 frozen serum samples. One of the former and two of the latter were positive. As antiperinuclear factor is predominantly, if not exclusively, of the IgG class, sample preservation would seem unlikely to have influenced the results. A more plausible explanation for the discrepancy lies in the immunofluorescence system. A major confounding factor causing variability in antiperinuclear factor results is the variation in substrate sensitivity, between different donors, of the buccal cells that contain the antigen. This drawback has barred antiperinuclear factor from coming into general use despite the long history of the test.2 We used a recently described improved technique that includes detergent treatment of the cells, which is reported to minimise, albeit not completely, donor differences.3 We tested the serum samples at a standard dilution of 1:5,7 which is also the titre we recorded for the WHO rheumatoid factor reference preparation that has been proposed by Feldkamp et al as the reference standard for antiperinuclear factor, too.6 Thirty per cent of serum samples from adult patients with rheumatoid arthritis tested in parallel by this technique were positive (unpublished data). In conclusion, antiperinuclear factor seems to be a specific but insensitive marker for the JCA subset with polyarticular onset that resembles adult rheumatoid arthritis. It contributes to the evidence for a basic difference between JCA in general and adult rheumatoid arthritis. The need for a common reference standard in future studies is obvious.

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Antiperinuclear factor

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<th>Antiperinuclear factor positive</th>
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<tr>
<td>Polyarticular</td>
<td>Rheumatoid factor positive</td>
<td>3/15</td>
</tr>
<tr>
<td>Polyarticular</td>
<td>Rheumatoid factor negative</td>
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</tr>
<tr>
<td>Oligoarticular</td>
<td>Systemic</td>
<td>0/0</td>
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<td>Total</td>
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Authors' reply: We thank Drs von Essen et al for their comments. We share their view that there is an obvious need for a reference standard for antiperinuclear factor studies.

Data from their study point to substantial differences in prevalence of antiperinuclear factor when compared with our results. Possibly, these variant results stem from the different methodologies which were applied: von Essen et al treated the buccal mucosa cells with detergents before the hybridisation with serum samples. This procedure did not decrease antiperinuclear factor antigen-antibody interactions in adult patients with rheumatoid arthritis (RA).1 It might do so in juvenile chronic arthritis (JCA), however, as characteristics of other autoantibodies in JCA, such as IgM rheumatoid factors, are different from those in adult RA.

Another possible explanation for the variant frequency of antiperinuclear factor might be a difference in its prevalence among various populations. As an example, preliminary results indicate antiperinuclear factor prevalence in 1:5 diluted serum samples of adult Israeli patients with RA is 40% (Nesher G, unpublished data), compared with 68-86% in European studies.1 It is possible that such differences exist between Scandinavian and American patients with JCA.

Several drawbacks, some of which are reported in these studies, prevent wider clinical use of antiperinuclear factor. Standardisation of the assay and evaluation of antiperinuclear factor prevalence in various populations might be two steps towards its common use.

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Antiperinuclear factor in chronic juvenile arthritis

Sir: We read with interest the article by Painelma et al1 on the diagnostic and prognostic value of antikeratin antibodies in rheumatoid arthritis (RA). We recently studied two groups with RA using indirect immunofluorescence for anti-bodies to the stratum corneum of the African ophagous. In the group of white patients with RA (n=30) we found a seroprevalence for antikeratin antibodies of 53%. In contrast, among the African rheumatoid group (n=54), which had significantly more RA, an antikeratin antibody seroprevalence of 6% was seen. Our findings suggest that there may be a wide variation in the incidence of antikeratin antibodies, and even when immunofluorescence is used there is a low sensitivity, low negative predictive value, and a moderate specificity. There is also evidence that immunofluorescence of serum with hetero- genous nuclear RNP core protein, in which the C-terminal domain shows partial homology with keratin, results in a significant reduction of antikeratin antibody titres.2

Our view is that although antikeratin antibodies may be useful in a subset of RA, these antibodies are of low discriminating ability when the disease is mild, as is often the case in early RA. Hence they are of limited value for routine diagnostic purposes.

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Sucralfate induced hepatitis in adult Still's disease

Sir: We were interested to read the report of sucralfate induced hepatitis in juvenile chronic arthritis, noting that one of the two patients had the systemic onset variety.1

We report an adverse reaction to sucralfate in a patient with adult Still's disease and comment on a potential mechanism for enhanced drug toxicity in this disorder. A 42 year old West Indian woman presented in November 1989 with malaise, weight loss, intermittent fever, and a symmetrical inflammatory polyarthropathy with diffuse, papulose, desquamating skin rash. Investigations showed a neutrophil leucocytosis (white cell count 15·8 x 10\(^3\) per µl) and a marked acute phase response (C reactive protein (CRP) 126 mg/l). Extensive screen for bacterial and viral infection was negative. A skin biopsy specimen showed perivascular polymorph infiltrates compatible with a small vessel vasculitis. Carpal erosions were seen on wrist radiographs.

We thank Drs van Essen et al for their comments. We share their view that there is an obvious need for a reference standard for antiperinuclear factor studies.

Data from their study point to substantial differences in prevalence of antiperinuclear factor when compared with our results. Possibly, these variant results stem from the different methodologies which were applied: von Essen et al treated the buccal mucosa cells with detergents before the hybridisation with serum samples. This procedure did not decrease antiperinuclear factor antigen-antibody interactions in adult patients with rheumatoid arthritis (RA).1 It might do so in juvenile chronic arthritis (JCA), however, as characteristics of other autoantibodies in JCA, such as IgM rheumatoid factors, are different from those in adult RA.

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Several drawbacks, some of which are reported in these studies, prevent wider clinical use of antiperinuclear factor. Standardisation of the assay and evaluation of antiperinuclear factor prevalence in various populations might be two steps towards its common use.

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Diagnostic role of antikeratin antibodies in RA

Sir: We read with interest the article by Painelma et al1 on the diagnostic and prognostic value of antikeratin antibodies in rheumatoid arthritis (RA).
Longstanding HLA-B27 associated Achilles tendinitis

Sirs: In 1987 we published the case of a B27 positive man who had presented for about four years with bilateral Achilles tendinitis as the only clinical manifestation of a B27 associated disease. We have followed up his case since his first visit in December 1986. The Achilles tendinitis went into remission in 1988. No other clinical manifestation of seronegative spondyloarthropathy has so far developed.

We have recently seen a similar case, which we report here. A 36 year old man was evaluated at our rheumatic disease unit in November 1990 for bilateral Achilles tendinitis which had persisted for four months. His medical history showed that he had had a similar episode in 1983, lasting for one month, which was attributed to 10 days' skiing. He denied any inflammatory spinal pain, peripheral arthritis, diarrhoea, uricosa, conjunctivitis, uveitis, psoriasis, cardiac symptoms, or physical injury. His family history was negative for spondyloarthropathy and other B27 associated syndromes.

Physical examination showed warmth, tenderness, and soft tissue swelling along both Achilles tendons and their calcaneal insertions. There was no limitation of spine movement or chest expansion. Laboratory evaluation showed only a C reactive protein of 12 mg/l (normal <5). HLA typing showed the B27 antigen.

Ultrasoundography by the technique of Fornage showed a diffuse thickening of both Achilles tendons, which was more severe on the right side (9 mm v 8 mm (figure)). Sacroiliac joint, lumbar spine, and foot radiographs were normal.

The Achilles tendinitis subsided after eight months from onset.

The bilateral Achilles tendinitis shown by our patient is consistent with a B27 associated disease process, and the clinical and echo-


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