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. 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 (Bardfield R, Inv Prague international pediatric rheumatology symposium, 1992).

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. We used a recently described improved technique that includes detergent treatment of the cells, which is reported to minimise, albeit not completely, donor differences. 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. Sixty per cent of serum samples from adult patients with rheumatoid arthritis tested in parallel by this technique were positive (manuscript in preparation).

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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Patients with juvenile chronic arthritis

<table>
<thead>
<tr>
<th>Onset type</th>
<th>Antiperinuclear factor positive</th>
<th>No of patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Polyarticular</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rheumatoid factor positive</td>
<td>3/15</td>
<td></td>
</tr>
<tr>
<td>Rheumatoid factor negative</td>
<td>0/73</td>
<td></td>
</tr>
<tr>
<td>Oligoarticular</td>
<td>0/195</td>
<td></td>
</tr>
<tr>
<td>Systemic</td>
<td>0/30</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>3/313</td>
<td></td>
</tr>
</tbody>
</table>

We recently studied two groups with RA using indirect immunofluorescence for antibodies to the stratum corneum of Helobas. In the group of white patients with RA (n=30) we found a serore prevalence of antikeratin antibodies of 53%. In contrast, among the African rheumatoid group (n=54), which had significantly more patients, an antikeratin antibody serore prevalence 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 immunosorption of serum with heterogeneous nuclear RNP core protein A1, in which the C-terminal domain shows partial homology with keratin, results in a significant reduction of antikeratin antibody titres.

Our view is that although antikeratin antibodies may be used as 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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 Sulphasalazine induced hepatitis in adult Still’s disease

Sir: We were interested to read the report of sulphasalazine induced hepatitis in juvenile chronic arthritis, noting that one of the two patients had the systemic onset variety. We report an adverse reaction to sulphasalazine 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 erythema of desquamating skin rash. Investigations showed a neutrophil leucocytosis (white cell count 15.8 x 10^9/l) 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.