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*Correspondence to Dr. Joseph P. Michalski.

REFERENCES


Correspondence

Table 1 Facb-R+ cell characteristics

<table>
<thead>
<tr>
<th>Facb-R+ cells (%) in:</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patients with RA</td>
<td>Healthy controls</td>
</tr>
<tr>
<td>(n=8)</td>
<td>(n=6)</td>
</tr>
<tr>
<td>HLA-DR*</td>
<td>71 (19)*</td>
</tr>
<tr>
<td>Density &lt;1-062 g/ml</td>
<td>75 (12)</td>
</tr>
</tbody>
</table>

*Results are presented as mean (SD). Statistical analysis by Mann-Whitney U test.

Pulse steroid therapy in rheumatoid arthritis

Sir, Thank you for the opportunity to reply to the letter by Dr. Michalski and colleagues. We are uncertain whether the Fc receptor bearing cells isolated from the upper phase of their partition system are the same as those detected in our Facb rosette assay. We have shown previously that Facb-R+ cells share some surface characteristics with monocytes and appear not to show natural killer or antibody dependent cytotoxic activity. Thus enhanced expression of Fc receptors on rheumatoid mononuclear cells probably reflects changes in a number of distinct subpopulations. Our own recent evidence would support Dr. Michalski’s final comment that these changes could be associated with cell activation. We have found that Facb-R+ cells from rheumatoid patients express significantly more II major histocompatibility complex antigen by immunofluorescence and are of lower density on Percoll gradient centrifugation than equivalent cells from healthy control subjects (Table 1). Thus rheumatoid Facb-R+ cells appear to be identical to similar cells isolated from healthy individuals three days after secondary challenge with specific antigen. The maintenance of increased numbers of activated Facb-R+ cells in RA over long periods is probably due to repeated stimulation of the immune system in these patients.

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N J GOULDING L-J EALES N D HALL

References


Pulse steroid therapy in rheumatoid arthritis

Sir, After a decade of the use of intravenous 'pulse' megadoses of corticosteroids in rheumatoid arthritis we now have a controlled study that shows there is no difference in effect from a similar 'pulse' given orally. Although the authors pointed out that this obviates the need for hospital admission and thus makes fewer demands on medical and
nursing resources, they did not emphasise the financial savings, which are considerable. The average bed cost a day was $429-44 in 1987 at a large Australian teaching hospital similar to the authors' institution. The authors have shown that 'pulse' megadoses of corticosteroid is followed by improvement of rheumatoid arthritis, maintained for 24 weeks. Apart from mentioning that nine of the 24 patients were concurrently receiving new disease suppressive agents (presumably slow acting antirheumatic drugs or cytotoxic agents), however, they made no other comment on this aspect of their study. Perhaps they have refrained from doing so as there was no placebo treated control group for comparison.

I would think it worthwhile if the authors addressed this point in these columns lest we be left with the impression that the improvement related solely to the 'pulse' corticosteroid given. For instance: did the above mentioned nine patients achieve remission; were others already taking or subsequently to begin taking disease suppressive drugs; were any taking oral corticosteroids already, or was treatment started with them during the 24 week follow up period?

6 Waratah Avenue, Biggera Waters, Qld 4216, Australia

John Webb

References


Sir, The letter from Dr Webb correctly indicates the considerable economic advantage of administering 'pulse' therapy orally rather than intravenously. It should be noted, however, that many centres administer intravenous 'pulse' therapy on a day patient basis rather than admitting the patient to hospital, thus reducing the costs of intravenous 'pulse' therapy considerably. Nevertheless oral pulse therapy is more economical and appears to be as safe and effective as intravenous pulse therapy as our study indicated.

Dr Webb has also correctly highlighted the fact that nine out of the 24 patients had started treatment with a new disease suppressive agent at the same time as receiving pulse therapy. Although it is unlikely that the disease suppressive agent contributed to the initial clinical improvement of these patients, it is probable that their clinical improvement was sustained by the administration of these agents. The study was not designed to answer this question, which has been addressed in another publication and is the subject of a recently completed clinical trial by the authors. Finally, as shown in Table 1 of our paper seven patients in each group were already receiving disease suppressing agents at the time of study. All treatment was continued unaltered for the duration of the trial and no oral or intra-articular steroids were administered during the 24 week follow up period. No patient was receiving oral corticosteroids at the time of entry into the study.

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Malcolm D Smith

References


Note

Advances in systemic autoimmune diseases

Perugia and Cornell University Medical Colleges are the co-sponsors of this meeting to be held in Perugia, Italy from 6 to 9 November 1988. Further information (in USA) from Professor C L Christian, Hospital for Special Surgery, Cornell University Medical College, 535 East 70th Street, New York, NY 10021, USA and (in Europe) from Professor Fausto Orignani, Clinica Medica I, Perugia University, Policlinico Monteluce, 06100 Perugia, Italy.
Pulse steroid therapy in rheumatoid arthritis.

J Webb

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