Methotrexate and Chronic Uveitis Associated with Juvenile Idiopathic Arthritis

To the Editor:

We read with interest the article by Dr. Foeldvari and colleagues1 about the efficacy of methotrexate (MTX) in the management of chronic uveitis in patients with juvenile idiopathic arthritis. It is to our understanding that ophthalmic data were derived from a review of the patients’ charts. The authors have solicited a thorough pool of information, but 2 areas of important information seem to be overlooked.

First, the standard treatment for chronic bilateral anterior uveitis usually comprises topical corticosteroids and mydriatics, followed by immunosuppressive agents in severe cases, and surgical intervention in the event of ophthalmic complications.2 It is important to note that even with the commencement of systemic immunosuppressive agents, concomitant topical corticosteroids are usually retained, with frequency of topical application adjusted with observation of anterior chamber inflammation. In the study in question, information on topical corticosteroid regimens during commencement of systemic immunosuppressive agents was largely lacking. The authors reported a good treatment remission as being achieved if we managed to reduce the topical corticosteroid regimen. This will undoubtedly incur significant bias and confounding influence.

Second, MTX is an immunosuppressive agent not without adverse reactions.3 For instance, it was found that as many as 20% of the pediatric patients taking regular MTX developed distressing side effects like nausea.4 Hence, inquiries into the potential untoward side effects and the associated precautionary measures for early detection of drug toxicity are almost mandatory in patients taking regular methotrexate.5 These important pieces of information were, however, not clearly stated in the article.

REFERENCES

Problems with Definitions of Disease Patterns in Psoriatic Arthritis

To the Editor:

The editorial by Taylor, et al1 was most interesting but deserves to be completed. Although the title refers to problems with the definition of axial and
peripheral disease patterns in psoriatic arthritis (PsA), only the axial patterns were really detailed. We think that the definitions of both axial and peripheral disease remain problematic. In addition, there is still confusion when distinguishing between true axial PsA and psoriasis with coincidental ankylosing spondylitis and between true peripheral PsA and psoriasis with coincidental rheumatoid arthritis (RA).

We do not believe the Moll and Wright criteria (an inflammatory arthritis associated with psoriasis in the absence of rheumatoid factor, RF) are sufficient because it is well known that patients with RA may be seronegative for RF at disease onset and as the disease progresses. For this reason we proposed that much of what is seen as very early inflammatory arthritis remains unclassifiable and possibly undifferentiated. This is not only true for the symmetric pattern of peripheral disease but also for asymmetrical oligoarthritis. In the literature, classification of patterns of PsA is complicated by the difficulty of assessing sacroiliitis on plain radiography. Assessment is now easier owing to performance characteristics of tomographic densitometry in cases of dubious radiographic sacroiliitis as well as magnetic resonance imaging (MRI) in cases of normal sacroiliac joints on plain radiography. Indeed it is well known that radiographic sacroiliitis can develop over time.

We agree with the authors that the European Spondylarthropathy Study Group (ESSG) criteria for spondylarthropathy (SpA) show poor sensitivity and specificity when applied to a case series of patients with PsA and RA. For example, a patient with knee monarthitis and psoriasis meets SpA criteria despite a possible coincidence between psoriasis and another cause of monarthritis including RA. It may now be time to revisit ESSG criteria because their concept of SpA is too broad. Only the axial patterns of PsA, enterocolopathies, reactive arthritides, and of course, ankylosing spondylitis should be classified as SpA based on objective data for axial disease. Finally we must be cautious about consensus exercises among experts in PsA as well as in early arthritis, as we have previously shown.

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REFERENCES

Dr. Helliwell, et al reply

To the Editor:

We thank Dr. Le Goff for his interest in our editorial and welcome his comments. The purpose of our editorial was to highlight current difficulties with subgroup definition for cases already diagnosed as psoriatic arthritis (PsA). Dr. Le Goff’s comments are primarily concerned with classification into PsA and non-PsA, particularly in early disease. We agree that most classification criteria only apply to established disease. This is also true for the newly developed classification criteria for PsA. Data from the aforementioned CASPAR study do not support the suggestion that cases of seronegative rheumatoid arthritis are misclassified as PsA. This suggests that although the Moll and Wright criteria are used by most investigators, other, unstated, clinical criteria (such as dactylitis and enthesitis) are employed. Early disease remains problematic and may well be undifferentiated initially, so that criteria specific for PsA (or any other inflammatory arthritis) may be impossible to develop without sophisticated imaging (such as magnetic resonance imaging). We also agree that “evidence” is generally preferable to “eminence”, but sometimes consensus opinion can offer a useful starting point.

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REFERENCES

Correction

Avćin T, Silverman ED, Forte V, Schneider R. Nasal septal perforation: a novel clinical manifestation of systemic juvenile idiopathic arthritis/adult onset Still’s disease. Table 1 was omitted and should appear as shown on the following page. We regret the error.
Table 1. Clinical characteristics of patients with systemic juvenile idiopathic arthritis/adult onset Still’s disease and nasal septal perforation.

<table>
<thead>
<tr>
<th>Case 1</th>
<th>Case 2</th>
<th>Case 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex</td>
<td>F</td>
<td>M</td>
</tr>
<tr>
<td>Age at onset, yrs</td>
<td>4.5</td>
<td>14.5</td>
</tr>
<tr>
<td>Disease duration at nasal septal perforation</td>
<td>6.5 yrs</td>
<td>9 mo</td>
</tr>
<tr>
<td>Disease course</td>
<td>Persistent</td>
<td>Persistent</td>
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<tr>
<td>Systemic features</td>
<td>Fever, rash, hepatosplenomegaly, lymphadenopathy</td>
<td>Fever, rash, serositis, hepatosplenomegaly</td>
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<tr>
<td>Arthritis</td>
<td>Polymyalgia</td>
<td>Polyarthritis</td>
</tr>
<tr>
<td>Complications</td>
<td>2 episodes of MAS</td>
<td>Myocarditis, myositis</td>
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<tr>
<td>Nasal symptoms</td>
<td>Obstruction, pain</td>
<td>Whistling</td>
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<tr>
<td>CT findings from sinuses</td>
<td>Minimal mucosal thickening</td>
<td>ND</td>
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<tr>
<td>Chest CT/radiograph</td>
<td>Normal</td>
<td>Serositis only</td>
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<tr>
<td>Autoantibodies</td>
<td>ANA neg, RF neg, ANCA neg.</td>
<td>ANA neg, RF neg, ANCA neg.</td>
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<td>Pathology</td>
<td>Skin biopsy: small vessel neutrophilic vasculitis</td>
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<td>Medications (*current)</td>
<td>NSAID, corticosteroids*, MTX, IVIG, cyclosporine A, infliximab, etanercept, anakinra, tacrolimus*</td>
<td>Indomethacin*, corticosteroids*, IVIG</td>
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