IgA serum levels and disease activity in ankylosing spondylitis

Sir, I read with considerable interest the report by Franssen and associates of a prospective study of IgA serum levels and disease activity in ankylosing spondylitis (AS). In their comparative trial of phenylbutazone and diflunisal, serum IgA, erythrocyte sedimentation rate, and disease activity all decreased during regular drug treatment, suggesting a disease modifying effect of the non-steroidal anti-inflammatory drugs (NSAIDs) in AS.

That NSAIDs affect favourably the natural course of AS is not widely appreciated. In fact this concept first became clear to me after completion of a five year trial of indomethacin in 28 patients that dates back to 1968. As judged by several criteria, including articular pain, duration of morning stiffness, onset of fatigue, and joint mobility, the overall response to indomethacin was rated as good in 21 patients, fair in five, and poor in two. After receiving indomethacin for an average period of 33 months, 21 of the 28 patients were in the American Rheumatism Association functional class I. Only one patient had been so classified before the drug trial. The average Westergren erythrocyte sedimentation rate values for all 28 patients decreased during the drug trial from 39 to 26 mm/h (p<0.01, Student’s t test). Indomethacin was discontinued in three patients—in two because of a poor response to the drug and in one because of an adverse reaction. Clearly, the results of this trial suggest that indomethacin favourably alters the course of AS.

In 1981, 18 years after the first patients were entered into this trial, it was possible to locate and reassess 14 of the remaining 25 patients. Of the 14, four had achieved remission and required no further drug therapy. Indomethacin had been discontinued in four patients—in two because of gastrointestinal side effects and in two because of a more favourable effect from another NSAID. The remaining six patients continue to receive indomethacin at an average daily dosage of 100 mg (range 75–100 mg) and continue to benefit from its long term use.

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References

Idiopathic haemarthrosis with chondrocalcinosis

Sir, We read with interest the article by Woolf et al about the idiopathic haemorrhagic rupture of the shoulder in destructive disease of the elderly.

In our experience haemarthrosis may also be associated with articular chondrocalcinosis and joint destruction.

We have reviewed 28 patients with haemarthrosis in 32 joints (20 knees and 12 shoulders). Periarticular ecchymosis was found in only one case relating to the knee but in four cases relating to the shoulder. There were calcium pyrophosphate dihydrate microcrystals in the shoulder fluid in eight cases but typical calcification of the joint in only three cases. Degenerative radiological changes with rupture of the rotator cuff were common, but in three cases the presentation was that of a neuropathic joint.

Patients who did not improve with rest, aspiration of the effusion, and intra-articular injection of steroids were treated by radioisotopic synoviorthese. In these patients, after injection of 2–3 mCi of gold-198 (four cases) or rhenium-186 (nine cases), the shoulder effusions disappeared in 11 cases (mean follow up of four years). Persistence of pain after treatment in most cases was thought to be due to degenerative lesions.

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References

Connective tissue deposits in MRL/1 mice

Sir, The article on connective tissue abnormalities in MRL/1 mice published by Edwards et al in the Annals is very interesting. The description of morphological changes in the connective tissue confirms our observations on arthritis in the MRL/1 mouse that we published in 1985.

In our study of 58 animals of different ages and sex we...
found granular, globular, or spindle shaped deposits in the joint capsule, synovial membrane, and/or extra-articular tissue. The material was located extracellularly and/or in macrophages and the synovial lining cells. Occasional deposits were found within blood vessels and lymphatics, in the vessel wall, or perivascularly. The material showed negative reaction for amyloid, calcium, iron, and urate. Being often strongly basophilic, it resembled the haematoxylin bodies found in systemic lupus erythematosus. Although it showed a negative reaction with ethidium bromide, its nuclear origin cannot be excluded since the binding sites of DNA could be denatured or masked by the nuclear antibodies.

We found a positive correlation between the presence of these deposits and the intensity of the arthritis. Since the arthritis often began without deposits, and an association between them was found only in a proportion of joints, we concluded that the deposits do not have a causal role in the arthritis, but enhance it.

References


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Book reviews


Clinics in Rheumatic Diseases are known and read by many rheumatologists in practice and in training. They provide often useful subject reviews and are an excellent source of up to date reference material. This issue maintains the high standard and makes good reading. Walker Kean has marshalled a truly international cohort of authors to discuss certain topics under the title of 'Arthritis in the Elderly'. The advantage of such a volume is that it covers certain areas well and does not attempt to discuss everything, badly.

Some chapters I found exciting and illuminating—namely, those which approached areas of ignorance for this clinician; the immunology of aging and its relation to the pathogenesis of osteoarthritis was thought provoking and exciting, whereas the chapters on drug treatment perhaps missed an opportunity to develop a theme. The availability and usefulness of non-drug treatment, though examined in the final chapter, could have been expanded to give balance.

An epidemiologist approaching this volume would search in vain for detailed analysis of the problem and its sociological impact. Many authors pay homage to this overpowering fact and some of their opening paragraphs contain comments (and references) that are repetitive and platitudinous.

Some chapters, especially those on surgery and the connective tissue diseases, mention the approach necessary in relation to the elderly but provide a more general summary. Both these chapters are excellent and contain a wealth of material but seemed partly to miss the point. Some chapters could be predicted for their excellence, Resnick on x rays and Doherty and Dieppe on crystals can always be relied on, and their contributions here are no exception.

The highlight for me, however, and much to my surprise, was Healey's chapter on rheumatoid arthritis in the elderly. I found his sound commonsense approach and questioning attitude most refreshing.

Over the years the quality of the 'Clinics' has varied from moderate to excellent; this edition is definitely good and very good in parts. Some readers will find that areas they consider important are insufficiently covered, but all will find much that is worthwhile.

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In this latest addition to the 'Recent Advances' series there is something of interest for everyone. The subjects range from molecular genetics and its potential in rheumatic disease to the heterodox fringe in rheumatological management. I think those who found that previous editions in this series leaned too strongly towards immunology will find this edition more balanced. That is not to say that immunology is forgotten. There is an excellent chapter on the leucotrienes by J J Belch, clearly explaining their role in the inflammatory pathway, which is certainly easier reading than some articles I have delved into on this subject but is detailed enough to interest the specialist immunologist. Similarly, the chapter on iron, free radicals, and chronic inflammation by D R Blake et al should also appeal to the non-laboratory based clinician.

My main delight with this text was the emphasis on clinical problems. A discussion of antirheumatic agents in the elderly is tackled by J D Goode and delineates the vast number of problems involved in prescribing for the elderly and bravely tries to provide some of the answers, including a suggestion for 'geriatric rheumatologists'. Perhaps we have enough of these already.
Connective tissue deposits in MRL/1 mice.

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Ann Rheum Dis 1987 46: 85-86
doi: 10.1136/ard.46.1.85-c

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