Reactive arthritis and ruptured Achilles tendon

It is widely accepted that reactive arthritis is associated with enthesitis. We recently saw a patient with chronic Achilles tendinitis who suffered a rupture of this tendon at the tendocalcaneal insertion. A 22 year old male locksmith presented in December 1994 with bilateral metatarsalgia and right fourth toe dactylitis. Naproxyn was introduced with partial resolution of symptoms. In April 1995 he developed synovitis in the left knee, both ankles, and enthesitis in the right Achilles tendon, right posterior tibialis tendon, and bilateral plantar fascitis. There was no history of rash, inflammatory eye disease, back pain, gastroenteritis or genitourinary symptoms. Investigations revealed an erythrocyte sedimentation rate (ESR) of 25 mm 1st h, HLA B27 positive, revealed an erythrocyte sedimentation rate of reactive arthritis in which both yersinia and campylobacter have been implicated as triggering organisms. He required aggressive treatment with non-steroidal anti-inflammatory drugs, oral corticosteroid, sulphasalazine, and methotrexate to control disease with the main symptomatic area being enthesitis of the Achilles tendon. He sustained a rupture of the Achilles tendon at the tendocalcaneal insertion—suggesting enthesitis as the predisposing factor.

The first patient was a 65 year old woman with a 20 year history of asymmetric erosive arthritis of the MTP joints, came to our unit in November 1995 showing dactylitis of the third right finger and planter fasciitis of the left foot. Physical examination showed that the swelling and tenderness present along the flexor tendon course of the "sausage shaped" third right finger extended as far as the middle part of the palm of the hand. There was no limitation of spine movement and chest expansion. Laboratory investigation showed negative tests for rheumatoid factor and antinuclear antibodies and A1, B8, B27, Cdw, Cw2, DR3, DR12, DQ2, DQ7 HLA antigens. MRI showed fluid collection in the synovial sheaths of the flexor tendons communicating with the ulnar palmocarpal and carpometacarpal joints.

Both our patients had a B27 positive undifferentiated SpA. Dactylitis was a feature of the disease they developed a destructive peripheral arthritis, peripheral enthesitis, and dactylitis.

SpA dactylitis results from flexor tenosynovitis. These two cases confirm our hypothesis that when flexor tenosynovitis involves a finger with synovial sheaths communicating with the ulnar palmarcreal sheaths, the sausage swelling also extends into the palm of the hand. Patient 1 also showed synovitis with pitting oedema of the dorsum of the hand, which has recently been described in elderly patients with SpA. The aspect of the inflammatory oedema in late onset SpA is similar to that seen in the RS1PE (remitting seronegative symmetrical synovitis with pitting oedema) syndrome and in polymyalgia rheumatica. Unlike RS1PE syndrome, the pitting oedema of late onset undifferentiated SpA is usually unilateral and more frequent in the feet.

Schaeverbeke et al have recently suggested that the oedema may be a result of local inflammation. The MRI findings in
our patient confirm this hypothesis. The examination showed fluid in the subcutaneous and peritendinous soft tissues and inflammation of the extensor tendon synovial sheaths. Our cases suggest that the inflammatory oedema of the dorsum of hands and feet of elderly patients with SpA may result from extensor sheath synovitis rather than joint synovitis. The same might occur in hand inflammatory oedema of other rheumatic diseases of the elderly. 10

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MATTERS ARISING

Pain in the rheumatic diseases

In their correspondence about pain in the rheumatic diseases, Thompson and Carr report that some of their cohort of 100 patients with inflammatory arthritis show a disassociation between reported pain and objective measures of disease activity. In most patients there was a close linear relation between change of reported pain, the
number of swollen joints, and C reactive protein over two years. However 18 patients reported high pain scores despite no evidence of C reactive protein or swollen joint activity. On the other hand 10 patients reported no pain despite active disease. The difference could not be explained on the grounds of joint deformity. Thompson and Carr do not set out to explain their finding.

However their previous writings on subjects such as ‘handicap’ indicate they are well aware of the importance of psychosocial factors in the manifestations of disease and, by implication, the weakness of rigid application of the medical model to chronic disease. Only a small proportion of patients with the mechanical low back pain or tender fibromyalgic spots develops chronic pain syndromes and becomes severely disabled. Psychosocial factors rather than clinical findings or treatment prescribed are the strongest predictors of chronic pain in the low back pain. In patients with fibromyalgia Wolff et al have demonstrated that the number of tender spots is proportional to the degree of distress. They suggest that the tender point count could be considered as the erythrocyte sedimentation rate of distress.

Even in osteoarthritis, disease severity accounts for only a proportion of the individual variability in clinical outcome. After controlling for disease severity, psychosocial variables remain strong predictors of individual differences in functional impairment and pain.

Thus it is well established that psychosocial factors are important predictors of ongoing pain in non-inflammatory musculoskeletal conditions. There is no reason to anticipate that people will behave differently whether responding to the pain of an inflammatory or non-inflammatory nature. Thus it can be assumed that a proportion of those with rheumatoid arthritis will develop a chronic pain syndrome. This is almost certainly what has happened in the 18% of Thompson and Carr’s patients with high pain scores in the presence of inactive disease.

The appropriate treatment of these patients is not by first, second or third line drugs combined or otherwise but by paying attention to self management strategies, coping skills, etc. No doubt a proportion of those with active disease will also have developed chronic pain behaviours and associated disability that require as much attention as the raised C reactive protein and number of swollen joints.

And what are we to make of the 10% of RA patients who do not express pain despite active disease? Although they are a delight for patients who do not express pain despite active disease? Although they are a delight for active disease? Although they are a delight for people who express pain despite no active disease? Although they are a delight for people who express pain despite no active disease? Although they are a delight for people who express pain despite no active disease?

What about IgA rheumatoid factor in rheumatoid arthritis?

We read with interest the editorial by Syltys and colleagues about rheumatoid factors (RFs). They correctly stated that most naturally occurring RFs were of the IgM isotype while IgG RFs were thought to be associated with rheumatoid arthritis (RA). It should be pointed out in this context that it is very difficult to measure IgG RF and this RF isotype can only be detected in approximately 50% of RA patients whereas IgA RF, usually in combination with IgM RF, can be detected in most patients with seropositive RA. It should also be noted that increased IgA RF is associated with severe manifestations in RA and this has been extensively reported by several different groups in the last 10 years.

Furthermore, several reports have shown that RFs may be increased in serum many months or even years before clinical symptoms of RA appear, and it has also been reported that symptom free people with an increase in IgA RF or IgG RF have an increased risk of developing RA. This indicates that both IgA RF and IgG RF may have a primary role in the pathogenesis of RA.

Recent studies have shown that a combined increase in IgM and IgA RF, with or without IgG RF, is the most common RF pattern found in patients with RA. Thus, a combined increase in IgM and IgA RF is very specific for RA and rarely found in symptom free people or patients with other rheumatic disorders. It should also be noted that IgG RF and IgM RF are more frequently raised than IgA RF in symptom free members of families with multicase RA. This indicates that an increase in IgA RF is more specific for RA than an increase in IgM RF or IgG RF. Thus, switching from IgM RF to IgA RF may be at least as important in the pathogenesis of RA as switching to the IgG class.

Several studies have shown that RA patients with an increase in IgA RF develop a more severe disease, with bone erosions or extra-articular manifestations, or both, than patients without IgA RF. This summarises some published studies on the association between disease manifestations in RA and RF isotypes.

Table 1: Associations between individual RF isotypes and disease manifestations as reported in 14 studies on RA*

<table>
<thead>
<tr>
<th>Disease manifestations</th>
<th>Association observed between RF isotypes and disease manifestations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bone erosions</td>
<td>Yes</td>
</tr>
<tr>
<td>Tarkowski et al</td>
<td>IgA RF</td>
</tr>
<tr>
<td>Teitsson et al</td>
<td>IgA RF</td>
</tr>
<tr>
<td>Arnasson et al</td>
<td>IgA RF</td>
</tr>
<tr>
<td>Gousd-Paquet et al</td>
<td>IgA RF</td>
</tr>
<tr>
<td>Briks et al</td>
<td>IgA RF</td>
</tr>
<tr>
<td>Winkela-Wolch et al</td>
<td>IgA RF</td>
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<tr>
<td>Eberhardt et al</td>
<td>IgA RF</td>
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<tr>
<td>Eggelmeijer et al</td>
<td>IgA RF</td>
</tr>
<tr>
<td>Van Zeben et al</td>
<td>IgA RF</td>
</tr>
<tr>
<td>Jorgensen et al</td>
<td>IgA RF</td>
</tr>
<tr>
<td>Extra-articular manifestations</td>
<td>IgA RF&gt; IgM RF&gt; IgG RF</td>
</tr>
<tr>
<td>Tarkowski et al</td>
<td>IgG RF</td>
</tr>
<tr>
<td>Gousd-Paquet et al</td>
<td>IgG RF</td>
</tr>
<tr>
<td>Ellson et al</td>
<td>IgG RF</td>
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<tr>
<td>Ljulividdson et al</td>
<td>IgG RF</td>
</tr>
<tr>
<td>Ellson et al</td>
<td>IgG RF</td>
</tr>
<tr>
<td>Jorgensen et al</td>
<td>IgG RF</td>
</tr>
</tbody>
</table>

*Not a complete literature survey.
RA and different RF isotypes. Not all studies have agreed\textsuperscript{19,20} but different findings can at least in part be explained by technical differences in RF testing.\textsuperscript{19,21} Measurement of individual RF isotypes is clinically useful, both in terms of diagnostic and prognostic evaluation of patients with RA. Furthermore, it is probable that RF has a primary role in the pathogenesis of RA and this may apply even more to IgA RF than IgG RF.

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\textbf{Authors’ reply}

We are most grateful to Drs Jonsson and Valdimarsson for their additional comments with regard to rheumatoid factors and their definite pathogenic associations with disease mechanisms in rheumatoid arthritis. Their home message is that perhaps we should be measuring other rheumatoid factor isotypes, as well as IgM, as they may be more prognostically relevant. This may indeed be the case but, at present, IgM rheumatoid factor is the only isotype that can be precisely measured using techniques such as nephelometry where additionally there is an accepted primary (WHO) standard. IgG and IgA rheumatoid factors are often measured by enzyme linked immunosorbent assay and this is where problems may occur. IgM rheumatoid factors can interfere with the assay by binding to the antigen and then subsequently to the detection antibody to give false positive results. Use of F(ab)\textsubscript{2} gets over this to some extent, but IgM can still form complexes that may interfere. Currently there is no agreed international reference standard to make assays comparable between laboratories and in the UK there is no national quality assurance programme; other than for IgM. This means that there can be no independent assessment of laboratory performance of IgG and IgA rheumatoid factors if these were to be applied to clinical samples. The advice from Professor Pam Riches of the Protein Reference Unit at St George’s Hospital Medical School is that, at present, she would not recommend the use of non-standardised unvalidated assays other than for research.

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