Reactive arthritis and ruptured Achilles tendon

It is widely accepted that reactive arthritis is associated with an 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 dactyliitis. 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, gastrointestinal or genitourinary symptoms. Investigations revealed an erythrocyte sedimentation rate (ESR) of 25 mm 1st h, HLA B27 positive, increased serum IgM antibodies to *Yersinia enterocolitica* 05 (optical density 0.581) (Yersinia 03 and 09 negative) despite negative stool cultures and synovial fluid from the knee contained 10.9 x 10^5 white blood cells (80% neutrophils, Gram stain negative, culture negative and no crystals). A diagnosis of reactive arthritis presumably secondary to *Yersinia enterocolitica* was made. Indomethacin 50 mg thrice daily and sulphasalazine 1 g thrice daily were initially given. Because of continuing active disease with recurrent massive knee effusions prednisone 20 mg in the morning and methotrexate 7.5 mg weekly were added and most of his symptoms improved, although clinically his right Achilles tendon was still troublesome. Prednisone was tapered. In October 1995 he presented with a flare up of disease (especially his right Achilles tendon) after an episode of gastroenteritis. On this occasion the ESR was 8 mm 1st h and repeat *Yersinia enterocolitica* 05 IgM serology had fallen (optical density 0.191), but stool cultures grew *Campylobacter jejuni*. He continued taking prednisone, sulphasalazine, and methotrexate and his joint symptoms improved. However clinically and synovially a low grade enthesitis continued in the right Achilles but local corticosteroid injections were not used.

In January 1996 when stepping out of a parked vehicle onto the right foot, he heard a 'snap' and suffered intense pain in the back of the heel. A clinical diagnosis of Achilles tendon rupture was made and surgical exploration and repair was undertaken. At operation rupture had occurred at the calcaneal insertion.

Despite the commonly recognised association of Achilles tendinitis with inflammatory arthritis surprisingly few reports of rupture have been recorded. Associated conditions include previous head injury, spasticity, and in polymyalgia rheumatica.
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.¹ 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 handedness 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 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.$^9$

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 the rheumatologist to deal with, such pain related behaviour may also be pathological. It is well recognised that a proportion of patients with rheumatoid arthritis battle on regardless and develop what has been called arthritis robustus with rapid aggressive joint destruction. Might these patients be found among Thompson and Carr’s pain free 10% with active joints?

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Table 1 Associations between individual RF isotypes and disease manifestations as reported in 14 studies on RA*$^{1–5}$

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<thead>
<tr>
<th>Disease manifestations</th>
<th>Association observed between RF isotypes and disease manifestations</th>
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<tr>
<td>Bone erosions</td>
<td><em>Yes</em></td>
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<tr>
<td>Tarkowski et al</td>
<td>IgA RF</td>
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<td>Teitsson et al</td>
<td>IgA RF</td>
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<td>Arrason et al</td>
<td>IgA RF</td>
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<tr>
<td>Gisond-Paquet et al</td>
<td>IgA RF</td>
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<td>Brik et al</td>
<td>IgA RF</td>
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<td>Winkska-Willocz et al</td>
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<td>Eberhardt et al</td>
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<td>Eggelmeijer et al</td>
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<td>Van Zeben et al</td>
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<td>Jorgensen et al</td>
<td>IgA RF</td>
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<th>Extra-articular manifestations</th>
<th>Association observed between RF isotypes and disease manifestations</th>
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<tr>
<td>Tarkowski et al</td>
<td>IgG RF</td>
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<td>Gisond-Paquet et al</td>
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<td>Ellson et al</td>
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<tr>
<td>Lüdewigsson et al</td>
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<td>Jorgensen et al</td>
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| *Not a complete literature survey.*
RA and different RF isotypes. Not all studies have agreed but different findings can at least in part be explained by technical differences in RF testing.

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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8 Winska Willoch HW, Thompson K, Young A, Corbett M, Shipley M, Hay F. IgA and IgM RF as markers of later erosive changes in rheumatoid arthritis. Scand J Rheumatol 1988; Suppl 75:238–43.

Authors’ reply

We are most grateful to Drs Jönsson and Valdimarsson for their additional comments with regard to rheumatoid factors and their definite pathogenic associations with disease mechanisms in rheumatoid arthritis. Their take 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), 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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