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 symp- toms. 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 planar 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, Campylobacter jejuni 03 and 09 negative (despite negative enterocolitica (ESR) of 25 mm 1st h, HLA B27 positive, Campylobacter jejuni 03 and 09 negative (despite negative enterocolitica, (ESR) of 25 mm 1st h, HLA B27 positive, Campylobacter jejuni 03 and 09 negative (despite negative enterocolitica, (ESR) of 25 mm 1st h, HLA B27 positive, Campylobacter jejuni 03 and 09 negative (despite negative enterocolitica, (ESR) of 25 mm 1st h, HLA B27 positive, Campylobacter jejuni 03 and 09 negative (despite negative enterocolitica, (ESR) of 25 mm 1st h, HLA B27 positive, Campylobacter jejuni 03 and 09 negative (despite negative enterocolitica, (ESR) of 25 mm 1st h, HLA B27 positive, Campylobacter jejuni 03 and 09 negative). Serology had fallen (optical density 0.191), but stool cultures grew Campylobacter jejuni. Despite treatment with oral corticosteroids in patients with reactive arthritis were found. Most patients had received oral or peritendinous corticosteroids. There are a number of case reports where ruptured Achilles tendons have occurred with oral corticosteroids in patients with respiratory disease.14 There were no case reports of ruptured Achilles tendons with sulfasalazine or methotrexate without concomitant oral or percutaneous cortico- steroid therapy. The usual site of rupture of the Achilles tendon is 2-6 cm proximal to the calcaneal insertion. In this area there is a reduction in both the number and mean rela- tive area of vessels.14 In this case although the patient was taking prednisone, the rupture was at an unusual site—the tendocalcaneal insertion—suggesting that enthesitis had led to weakening of the tendon in this position. None of the previous reports have shown rupture at the tendocalcaneal insertion. It is interesting to speculate, that in this case suppressing inflammation by local corticosteroid infiltration around the Achilles tendon may have prevented rupture.

In summary we have reported a young man with a short history 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, sulfasalazine, 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 predispensing factor.1

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Dactylitis also involving the synovial sheaths of the palm of the hand: two more cases studied by magnetic resonance imaging

We previously reported on the case of a 37 year old woman suffering from B27 positive psoriatic arthritis and showing dactylitis of the middle finger also involving the uncommon synovial sheaths communicating with the ulnar palmar aponeurosis. The dactylitis was also present in the dorsum of the hand, which has been described in elderly patients with spondyloarthropathy (SpA).1

The first patient was a 65 year old woman with a 20 year history of asymmetric erosive peripheral arthritis of the metatarsophalangeal, midfoot, hindfoot, radiocarpal, mid-carpal, metacarpophalangeal (MTP), and proximal and distal hand interphalangeal joints and enthesitis of the quadriceps and patellar tendons on both patellae.

In October 1994 she was referred to us because of a reoccurrence of her peripheral arthritis in the right hand of two months’ duration.

Her family history was negative for SpA and other B27 related diseases.

Physical examination showed dactylitis of the fourth right finger together with synovitis of a large pitting oedema of the dorsum of the right hand. The painful swelling of the synovial sheaths of the right fourth finger extended as far as the palm of the hand. HLA typing showed A24, A29, B27, DR7, DR11, DQ2, DQ7 antigens. Tests for the rheumatoid factor and the antinuclear antibodies were negative. Spinal and pelvic radiographs were normal.

MRI performed according to the methods previously described showed flexor tenosynovitis of the right fourth finger extending without any interruption into the palm of the hand. On the dorsum of the hand oedema in the subcutaneous and peritendinous soft tissues and fluid in the extensor tendon synovial sheaths were observed.

The second patient, a 53 year old woman with a 12 year history of erosive asymmetric arthritis of the MTP joints, came to our unit in November 1995 showing dactylitis of the third right finger and planter fascitis 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, Cw, Cw2, DR3, DR12, DQ2, DQ7 HLA antigens.

MRI showed fluid collection in the synovial flexor sheath of the third left finger extending into the middle part of the hand (fig 2). Spinal and sacroiliac joint radiography were normal.

Both our patients have a B27 positive undifferentiated SpA.1 During the course of the disease they developed a destructive peripheral arthritis, peripheral enthesitis, and dactylitis.

SpA dactylitis results from flexor tenosynovitis.1 These two cases confirm our hypothesis that when flexor tenosynovitis involves a finger with synovial sheaths communicating with the ulnar palmar aponeurosis, the sausage swelling also extends into the palm of the hand.1

Patient 1 also showed synovitis with pitting oedema of the dorsum of the hand, which has recently been described in elderly patients with SpA.2 The aspect of the inflammatory oedema in late onset SpA is similar to that seen in the RS3PE (remitting seronegative symmetrical synovitis with pitting oedema) syndrome3 and in polymyalgia rheumatica.4 Unlike RS3PE syndrome, the pitting oedema of late onset undifferentiated SpA is usually unilateral and more frequent in the feet.5,6 Schaeverbeke et al have recently suggested that the oedema may be ascribed to an increase in capillary permeability as a result of focal inflammation.7 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.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 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 outcome in 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. 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?

What about IgA rheumatoid factor in rheumatoid arthritis?

We read with interest the editorial by Soltys 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 5 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. Table 1 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                              No</td>
</tr>
<tr>
<td>Tarkowski et al</td>
<td>IgA RF                          IgM RF, IgG RF</td>
</tr>
<tr>
<td>Teitsson et al</td>
<td>IgA RF                          IgM RF, IgG RF</td>
</tr>
<tr>
<td>Aronnson et al</td>
<td>IgA RF                          IgM RF, IgG RF</td>
</tr>
<tr>
<td>Gisond-Paquet et al</td>
<td>IgA RF                          IgM RF, IgG RF</td>
</tr>
<tr>
<td>Brik et al</td>
<td>IgA RF                          IgM RF, IgG RF</td>
</tr>
<tr>
<td>Wniska-Wilczok et al</td>
<td>IgA RF                          IgM RF, IgG RF</td>
</tr>
<tr>
<td>Eberhardt et al</td>
<td>IgA RF                          IgM RF, IgG RF</td>
</tr>
<tr>
<td>Eggelmeijer et al</td>
<td>IgA RF                          IgM RF, IgG RF</td>
</tr>
<tr>
<td>Van Zeben et al</td>
<td>IgA RF&gt; IgM RF&gt; IgG RF</td>
</tr>
<tr>
<td>Jorgensen et al</td>
<td>IgA RF                          IgM RF, IgG RF</td>
</tr>
<tr>
<td>Extra-articular manifestations</td>
<td>Yes                              No</td>
</tr>
<tr>
<td>Tarkowski et al</td>
<td>IgG RF                          IgA RF&gt; IgM RF&gt; IgG RF</td>
</tr>
<tr>
<td>Gisond-Paquet et al</td>
<td>IgG RF                          IgA RF&gt; IgM RF&gt; IgG RF</td>
</tr>
<tr>
<td>Elliot et al</td>
<td>IgG RF                          IgA RF&gt; IgM RF&gt; IgG RF</td>
</tr>
<tr>
<td>Lüödölksson et al</td>
<td>IgG RF                          IgA RF&gt; IgM RF&gt; IgG RF</td>
</tr>
<tr>
<td>Elston et al</td>
<td>IgG RF                          IgA RF&gt; IgM RF&gt; IgG RF</td>
</tr>
<tr>
<td>Jönsson et al</td>
<td>IgG RF                          IgA RF&gt; IgM RF&gt; IgG RF</td>
</tr>
<tr>
<td>Jorgensen et al</td>
<td>IgG RF                          IgA RF&gt; IgM RF&gt; IgG RF</td>
</tr>
</tbody>
</table>

*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.\(^1\)\(^2\)

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 RFs 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 Jonsson 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 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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