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 tendonitis 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. Naproxen 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 fasciitis. There was no history of rash, inflammatory eye disease, back pain, gastroenteritis or There was no history of rash, inflammatory eye disease, back pain, gastroenteritis or syphillis. 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, 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, implying enthesitis as the predisposing factor.

Dactylitis also involving the synovial sheaths in 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 uncom mon synovial sheaths communicating with the ulcer minor. 

Despite the commonly recognised association of Achilles tendonitis with inflammatory arthritis surprisingly few reports of rupture have been recorded. Associated conditions include reactive arthritis,1 systemic lupus erythematosus,1,2 and gout,1 but no cases 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.1,2 There were no case reports of ruptured Achilles tendons with sulphasalazine 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 relative area of vessels. 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, synovial sheath involvement was shown by ultrasonography. We have recently seen two cases confirming our hypothesis that when flexor tenosynovitis involves a finger with synovial sheaths communicating with the ulnar palmar sheaths, the sausage swelling also extends into the palm of the hand.1

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 palmar sheaths, 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. 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) syndrome1 and in polyarthritis rheumatica. Unlike RS3PE syndrome, the pitting oedema of late onset undifferentiated SpA is usually unilateral and more frequent in the feet.1,17 Schaeverbeke et al have recently suggested that the oedema may be ascribed to an increase in capillary permeability as a result of focal inflammation. The MRI findings in

References

our patient confirm this hypothesis. The examination showed fluid in the subcutaneous and tendinous 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 ‘handy men’ 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 pain in chronic musculoskeletal conditions. However, the available clinical data are controversial in this area. Current studies variously report an absent* or only weak correlation between disease activity and pain scores, that disease activity is a strong predictor of pain and that disease activity influences pain indirectly via depression.*

Therefore we feel that the cause and effect relation between psychosocial distress and self reported pain and disability remains an hypothesis that would explain our findings rather than a conclusion of the findings themselves.

We are currently studying these relations in more detail.

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Authors’ reply

We agree with Dr Jones that psychological factors are likely to be important in self reported pain and disability in chronic musculoskeletal conditions. However, the available clinical data are controversial in this area. Current studies variously report an absent* or only weak correlation between disease activity and pain scores, that disease activity is a strong predictor of pain and that disease activity influences pain indirectly via depression.*

Therefore we feel that the cause and effect relation between psychosocial distress and self reported pain and disability remains an hypothesis that would explain our findings rather than a conclusion of the findings themselves. We are currently studying these relations in more detail.

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

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<th>Disease manifestations</th>
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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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Authors’ reply
We are most grateful to Drs Jonsson and Valdimarsson for their additional comments with regard to rheumatoid factors and their different 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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