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 calcanealcalcaneal insertion. A 22 year old male locksmith presented in December 1994 with bilateral metatarsalgia and right fourth toe dactylitis. Naproxen was started 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 tibial tendon, and bilateral plantar fasciitis. There was no history of rash, inflammatory eye disease, back pain, gout, 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. Cultured synovial fluid from the knee contained 10.9 \times 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 mas-
tive knee effusions prednisone 20 mg in the morning and methylprednisolone 7.5 mg weekly were added and most of his symptoms improved, although clinically his right Achil-
les 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 gastroen-
teritis. On this occasion the ESR was 8 mm 1st h and repeat Yersinia enterocolitica IgM serology had fallen (optical density 0.191), but stool cultures grew Campylobacter jejuni. He continued taking prednisone, sulphasal-
azine, and methotrexate and his joint symp-
toms improved. However clinically and symptomatically a low grade enthesitis con-
tinued in the right Achilles but local cortico-
steroid 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 explo-
ration and repair was undertaken. At opera-
tion rupture had occurred at the calcaneal insertion.

Despite the commonly recognised associa-
tion of Achilles tendonitis with inflammatory arthritis surprisingly few reports of rupture have been recorded. Associated conditions include rheumatoid arthritis,1 systemic lupus erythematosus,2,3 and gout,1 but no cases with reactive arthritis were found. Most patients had received oral or periarticular cortico-
steroids. There are a number of case reports where ruptured Achilles tendons have oc-
curred with oral corticosteroids in patients with respiratory disease.1,4 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 calcanealcalcaneal insertion. In this area there is a reduc-
tion in both the number and mean rela-
tive area of vessels.5 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 sup-
pressing 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 cortico-
steroid, 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 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 sup-
pressing inflammation by local corticosteroid infiltration around the Achilles tendon may have prevented rupture.

L STAFFORD
Rheumatology Department, Prince Henry Hospital, Sydney, Australia

Correspondence to: Dr JV Bertouch, Suite 6D, The Wakes Medical Centre, 66 High Street, Randwick NSW 2031, Australia.

7 Carr A, Norris S. The blood supply of the calca-

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 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.4 During the course of the disease they developed a destructive peripheral arthritis, peripheral enthesitis, and dactylitis. SpA dactylitis results from flexor tenosynovitis.5 These two cases confirm our hypothesis that when flexor tenosynovitis involves a finger with synovial sheaths communicat-
ing with the ulnar palmarcarpal 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.6 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) syndrome7 and in polymyalgia rheumatica.8 Unlike RS1PE syndrome, the pitting oedema of late onset undifferentiated SpA is usually unilateral and more frequent in the feet.9,10 Schaeverbeke et al have recently suggested that the oedema may be induced to an increase in capillary permeability as a result of local inflammation.11 The MRI findings in

References

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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

ANGELA PADULA
CARLO SALVARANI
Rheumatic Disease Unit, Arcoedale S Maria Nuova, Reggio Emilia, Italy

LIBERO BAROZZI
MASSIMO DE MATTEIS
PIETRO PAVLICA
Department of Diagnostic Radiology, S Orsola-Malpighi Hospital, Italy

FABRIZIO CANTINI
Rheumatic Disease Unit, Hospital of Prato, Italy

IGNAZIO OLIVIERI
Rheumatic Disease Unit, S Orsola-Malpighi Hospital, Italy

Correspondence to: Dr I Olivieri, Servizio di Reumatologia, Policlinico S Orsola-Malpighi, Via Massarenti 9, 40138 Bologna, Italy.


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 chronicity. Of course rheumatoid 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, disability scores among patients who do not express pain despite disease activity that require as much attention as the patients with active disease will also have developed 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 the rheumatologist to deal with, such pain.

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 years. 14

Furthermore, several reports have shown that RFs may be increased in serum many months or even years before clinical symptoms of RA appear. 11,12 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. 13 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. 15,16 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. 17 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. 18 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 IgG 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 IgG RF develop a more severe disease, with bone erosions or extra-articular manifestations, or both, than patients without IgA RF. 15,16,17,18 Table 1 summarises some published studies on the association between disease manifestations in rheumatoid arthritis? We read with interest the editorial by Sylots 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 years. 14

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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.

THORBJÖRN JONSSON
HEILGI VALDIMARSSON
Department of Immunology, National University Hospital, Landspítali, 101
Reykjavik, Iceland


10 Eggelmeijer F, Otten HG, de Rooy HH, Daha HE. LGMV DAIMARSSON.


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 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.

JOHN Axford
ANDY NOLITYS
Academic Rheumatology Unit, St George's Hospital Medical School, University of London, Cranmer Terrace, London SW17 ORE
Reactive arthritis and ruptured Achilles tendon

L STAFFORD and J BERTOUCH

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