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Distinctive radiological features of small hand joints in rheumatoid arthritis and seronegative spondyloarthritis demonstrated by contrast-enhanced (Gd-DTPA) magnetic resonance imaging

Vladimir Jevtic[1], Iain Watt[2], Blaz Rozman[3], Mojca Kos-Golja[3], Franci Demsar[4], Orest Jarh[4]

[1] Institute of Radiology, University Medical Centre, Ljubljana, Sovenia
[2] Department of Clinical Radiology, Bristol Royal Infirmary, Bristol, UK
[3] Department of Rheumatology, University Medical Centre, Ljubljana, Slovenia
[4] Institute Jozef Stefan, Ljubljana, Slovenia

Abstract. A series of patients with clinically early inflammatory joint disease due to rheumatoid arthritis, psoriatic arthritis and Reiter's syndrome were examined by plain film radiography and magnetic resonance imaging (MRI). The spin echo T1-weighted precontrast, T2-weighted, and, especially, T1-weighted postcontrast images demonstrated distinct differences in the distribution of inflamatory changes, both within and adjacent to involved small hand joints. Two major subtypes of inflammatory arthritis were shown, thus providing a specific differential diagnosis between rheumatoid arthritis and some patients with seronegative spondyloarthritis. In particular, all the patients with Reiter's syndrome who were studied, and half of those with psoriatic arthritis, had a distinctive pattern of extra-articular disease involvement. The need for a new classification of clinical subsets in psoriatic arthritis has been recently suggested. The present findings suggest that magnetic resonance imaging could be useful in such a reclassification of seronegative spondyloarthritis, as well as offering considerable potential for a reappraisal of pathogenesis and therapy. In this series, it was also noted that juxta-articular osteoporosis on plain film did not correlate with bone marrow oedema on MRI. Hence the aetiology of this common radiographic finding also merits further consideration.

Key words: Magnetic resonance imaging – Gd-DTPA – Contrast medium enhancement – Rheumatoid arthritis – Seronegative spondyloarthritis – Differential diagnosis

Early diagnosis of, and thus differentiation between, rheumatoid arthritis (RA) and seronegative spondyloarthritis (SNSA) is of considerable importance in the management of patients whose initial presentation is of inflammatory synovitis in small hand joints. It has been shown in animal models of arthritis that intravenously administered gadopentetic acid (Gd-DTPA) accumulates in actively inflamed joints [1]. However, this phenomenon is entirely non-specific, reflecting varying degrees of hypervascularity and abnormal vascular permeability, and has been described in several infectious and rheumatological inflammatory processes [2]. Thus, differential diagnosis between the various rheumatic diseases does not seem possible on the basis of contrast medium enhancement alone. Therefore, the final MRI diagnosis still depends largely on the morphological characteristics of the joint disease. However, whilst occasional reports have drawn attention to differing morphological subtypes within an RA group [3], very little attention has been paid to possible differences between RA and SNSA. The objective of this study was to evaluate possible differential diagnostic features in individual small hand joints of patients with clinically active RA and SNSA by comparing the early morphological intra- and extra-articular findings in spin echo T1-weighted (T1W) precontrast, T1W postcontrast and T2-weighted (T2W) MRI images.

Correspondence to: I. Watt, Department of Clinical Radiology, Bristol Royal Infirmary, Bristol, BS2 8HW, UK

Materials and methods

Two groups of patients were studied. In the first we studied individual small hand joints (10 proximal interphalangeal, 6 metacarpophalangeal) of 16 consecutive patients with RA (12 women, 4 men, mean age 49 years). The clinical diagnosis of RA was established using the revised ARA criteria [4]. The second group comprised 16 consecutive patients with SNSA (12 men, 4 women, mean age 39 years). The diagnosis was made using the criteria of Moll and Wright [5]. The group included 13 patients with a clinical diagnosis of psoriatic arthritis (PsA; 7 proximal interphalangeal, 6 metacarpophalangeal joints) and 3 with Reiter's syndrome (RS; all proximal interphalangeal joints). All the RS patients had the classical clinical triad of arthritis, non-gonococcal urethritis and conjunctivitis.

Disease duration in both study groups was less than 2 years. Only patients with clinical signs of active inflammatory disease were included. The selection of the individual joint for imaging was on the basis of the onset of acute pain and stiffness, as reported by the patient, the presence of active synovitis confirmed at

clinical examination and relatively modest changes on hand radiographs using the Larsen index (grades I and II) [6]. Laboratory evaluation of inflammatory activity included determination of erythrocyte sedimentation rate, rheumatoid factor and C-reactive protein. All the joints were imaged by conventional radiography and MRI. All posteroanterior hand and wrist radiographs were exposed the day prior to MRI. The latter was performed using a Bruker Biospec System (Bruker, Germany) with an Oxford Instruments (Oxford Instruments, UK) 2.35-T magnet having a bore diameter of 22.5 cm, so that the probe filling factor was optimal and no surface coils were needed. A spin echo sequence with T1W images (TR 603 ms, TE 30 ms, 2 averages, 5 min acquisition time) and T2W images (TR 2045 ms, TE 90 ms, 2 averages, 17 min acquisition time) was used, followed by a similar T1W examination immediately after Gd-DTPA (0.1 mmol/kg body weight, Magnevist, Schering) had been injected intravenously as a bolus through a cannula. In some patients a gradient echo technique was also used (TR 205 ms, TE 10 ms, flip angle 30°). The field of view (FOV) was 12 cm with a data acquisition matrix of 256×256. all joints were imaged in the coronal plane using consecutive 3-mm-thick slices. Short tau inversion recovery sequences (STIR) were not available.

Plain films and magnetic resonance images were interpreted independently in a qualitative fashion by two experienced skeletal radiologists, who were unaware of clinical findings. A consensus was reached in two cases where there was disagreement (one joint each in the RA and SNSA groups were classified as Larsen grade I by one radiologist, and grade II by the other. After consultation both cases were scored as grade I).

Results

RA group

Eleven patients were classified as having grade I disease and five as grade II according to the Larsen index [6]. MRI findings were consistent with the presence of active synovitis (11 joints) or early destructive pannus (5 joints) [3], revealing synovial proliferation in a curvilinear or ovoid distribution, with low to intermediate signal intensity on T1W precontrast images. T2W images showed synovial proliferation with high signal intensity with marked enhancement on T1W postcontrast images. In every case soft tissue inflammatory changes were localised within the joint capsule (Fig. 1) affecting both sides of the joint (Fig. 2), with no obvious abnormality in periarticular tissue.

SNSA group

Twelve cases were classified as grade I and four (all with psoriatic arthritis) as grade II according to the Larsen index [6]. All joints demonstrated high signal intensity on T2W images and intense contrast enhancement on T1W postcontrast images, MRI signs compatible with the presence of active inflammatory tissue. However, in nine cases of SNSA (six with PsA, three with RS) the distribution and extent of soft tissue involvement differed and was considerably greater than in the patients with rheumatoid arthritis. The inflamed tissue extended far beyond the joint capsule, involving neighbouring structures, for example thickened collateral ligaments and the surrounding periarticular soft tissue, the more proximal of the two bones comprising the joint usually being the more abnormal (Figs. 3, 4).

In seven cases of PsA – six affecting metacarpophalangeal joints and one a proximal interphalangeal joint – synovial proliferation was intra-articular and had a similar degree and extent of abnormality as the MRI findings in the RA group.

Gradient echo images did not demonstrate any distinctive features from those shown by the spin echo images. All the inflamed soft tissues demonstrated high signal intensity with the exception of the joint capsule, which was of low to intermediate signal intensity on both methods of imaging (Fig. 3C).

In only 2 of the 32 joints imaged, all with clinical MRI signs consistent with active inflammation, was

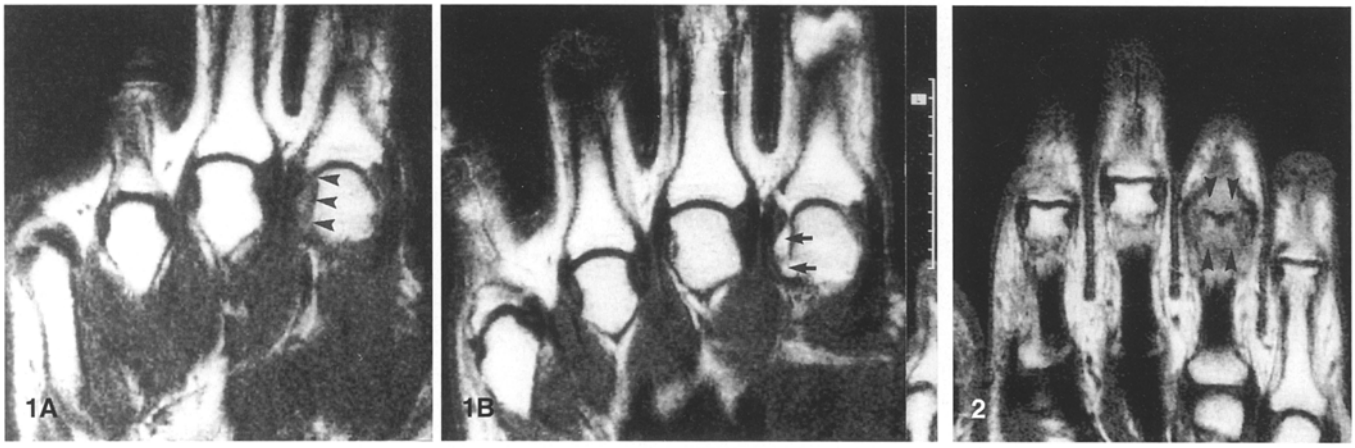

Fig. 1A, B. The index finger metacarpophalangeal joint in rheumatoid arthritis. MRI reveals soft tissue inflammatory changes within the joint cavity. **A** Coronal T1W spin-echo image. Synovial proliferation of intermediate signal intensity is shown without bone erosions (*arrowheads*). **B** T1W postcontrast spin-echo image. Marked contrast enhancement of inflamed synovium occurs (*arrows*)

Fig. 2. The ring finger proximal interphalangeal joint in rheumatoid arthritis. MRI image demonstrated findings consistent with bone marrow oedema on both sides of the joint. T1W spin echo image. Low signal juxta-articular bone marrow (*arrowheads*)

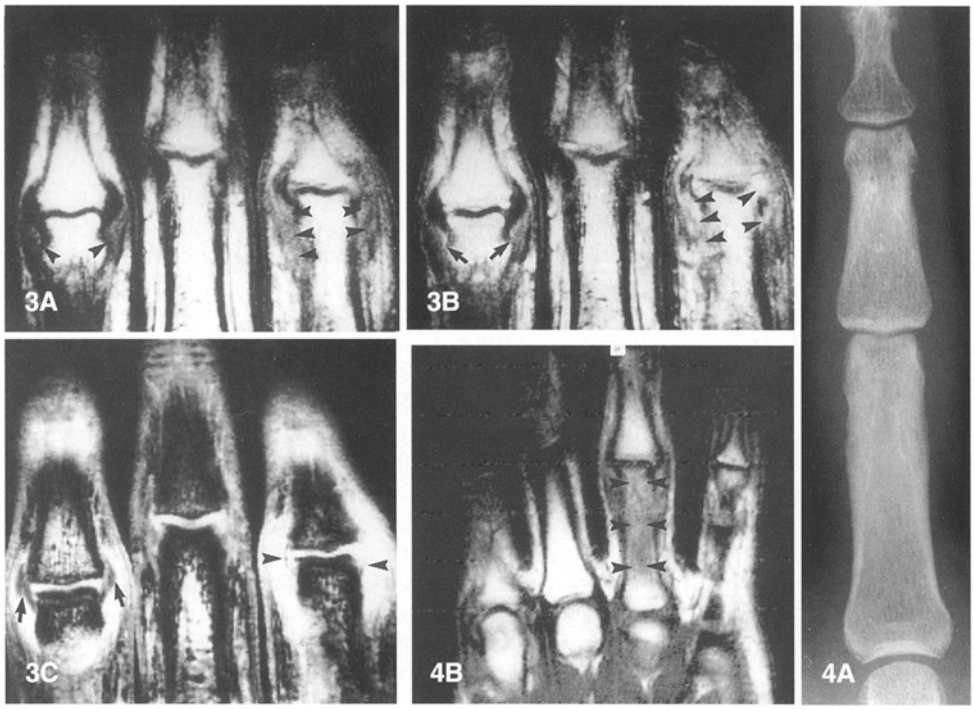

Fig. 3A–C. The ring and index finger proximal interphalangeal joints in psoriatic arthritis. MRI demonstrates extra-articular extension of soft tissue inflammatory changes with involvement of the collateral ligaments and the surrounding soft tissues of the proximal phalanx. Similar changes of signal intensity are seen in both proximal interphalangeal joints, but are more discrete in the index finger (*left*), affecting only the collateral ligaments. **A** Coronal T1W spin echo image. Inflammatory changes of intermediate signal intensity (*arrowheads*). **B** T1W postcontrast spin echo image confirms marked enhancement in the region of the ring finger proximal interphalangeal joint (*arrowheads*) and more discrete abnormality in the index finger proximal interphalangeal joint (*arrows*). **C** Gradient echo image demonstrates the joint capsule of intermediate (*arrowheads*) and low signal intensity (*arrows*) within the inflamed tissue of higher signal intensity

Fig. 4A, B. The ring finger proximal interphalangeal joint in Reiter's syndrome. MRI reveals bone marrow oedema only in the proximal phalanx. **A** Plain film. There is no juxta-articular demineralisation; the bone density of the proximal phalanx is slightly increased. **B** T1W spin echo image. Low signal bone marrow affecting only the proximal phalanx (*arrowheads*)

changed signal intensity of the juxta-articular bone marrow adjacent to a joint demonstrated. In these 2 joints T1W images confirmed low signal intensity bone marrow, with corresponding high signal intensity on T2W images and moderate contrast enhancement on T1W postcontrast images. In one of these patients, who had RA, bone marrow oedema affected subchondral bone marrow on both sides of a proximal interphalangeal joint (Fig. 2). The distribution was different in the other patient, with RS, in whom bone marrow oedema only affected the proximal phalanx adjacent to thickened inflamed collateral ligaments.

Discussion

A considerably body of evidence exists confirming the value of MRI in various rheumatic disorders, with and without the use of gadolinium based contrast agents. Although the inherent high soft tissue contrast resolution of MRI is further enhanced by the use of intravenously administered Gd-DTPA, the contrast enhancement is entirely non-specific and has been described in various infectious and rheumatological inflammatory diseases [2]. This is understandable, since contrast enhancement results from diffusion through the extracellular compartment due to increased vascular permeability [7]. Furthermore, histological examination of synovial membrane in such diverse rheumatic conditions as gout, septic arthritis, systemic lupus erythematosus, RS and osteoarthritis has demonstrated that vascular congestion, synovial membrane hypertrophy, mononuclear cell infiltration, fibrinoid deposits and even pannus commonly occur in disorders other than rheumatoid arthritis [8]. Thus, differential diagnosis between the various inflammatory rheumatic disorders does not yet seem possible on the basis of contrast medium enhancement alone.

Only a very limited number of studies are available [2, 9] in which histological specimens of synovium from RA have been correlated with Gd-DTPA uptake. These results support intuitive expectations of others that high contrast enhancement is due to hypervascularity, implying inflammatory synovial proliferation and enabling differentiation from non-vascular, non-inflammatory fibrous pannus. Based on these results, previous work by the present authors [3] has shown that demonstration and morphological categorisation of synovitis and pannus in small joint RA of the hand is possible using T1W precontrast, T2W, and, in particular, T1W postcontrast spin echo images.

In all 32 joints examined in the present study, predominantly soft tissue inflammtory changes were demonstrated. In 11 joints of patients with RA without bone erosion on plain films (all Larsen index grade I), MRI findings were consistent with simple synovitis (Fig. 1). However, in five synovial proliferation was associated with minor erosions representing early destructive pannus, and conformed with the Larsen index grade II changes on plain film. There was thus good correlation between MRI and plain film features in the RA group, as well as consistent findings with previously published work [3]. However, in all the RA cases soft tissue inflammatory changes were confined within the joint capsule, involving both sides of the joint. These features are entirely consistent with the anatomical distribution of synovium and reflect the basic concept of RA as an inflammatory disease characterised by chronic inflammatory synovitis.

In the patients with SNSA, the MRI findings fell into two distinct groups. In the first, comprising six patients with PsA and all three with RS, the extent and distribution of soft tissue inflammatory changes affecting proximal interphalangeal joints was similar but differed considerably from those shown in RA and its published subsets [4]. In two, with clinical findings consistent with psoriatic dactylitis, corresponding distal interphalangeal joints were abnormal also. In all these joints the degree and extent of soft tissue involvement was much greater, extending well beyond the joint capsule. Hence other anatomical structures were involved, comprising thickened collateral ligaments and neighbouring soft tissues, mainly in the region of the proximally located phalanx. In one case of PsA another abnormal joint (another proximal interphalangeal joint) was included within the same field of view as the joint under study. The abnormalities here were predominantly extracapsular (Fig. 3). Thus, whilst this case revealed not only the typical extra-articular location of soft tissue inflammatory changes in this group of SNSA patients, it also suggests the possibility that the joint capsule and synovium are not the primary target of the disease process, but may become involved later. Hence disease evolution may be of an inflammatory process initially within the collateral ligaments, spreading to surrounding soft tissues and only finally affecting the intracapsular structures. Although considerable soft tissue swelling is well recognised in patients with SNSA, sometimes producing a "sausage finger", it has usually been explained on the basis of adjacent tenosynovitis [10]. It is of interest to note that the two PsA patients with simultaneous involvement of the distal and proximal interphalangeal joints had considerable soft tissue swelling of the digits without tenosynovitis on MRI. Thus, in summary, this group of SNSA patients had MRI features which suggested that their inflammatory process primarily affects a number of tissue planes and is not predominantly a synovitis.

The second group of SNSA patients, all with PsA, in whom six metacarpophalangeal joints and one proximal interphalangeal joint were affected, all exhibited intra-articular distribution of soft tissue inflammatory changes identical to those in the RA group, and thus quite different from the other SNSA patients. There were no clinical, laboratory or radiographic differences between the two SNSA groups. This finding implies a heterogeneity in SNSA. The difficulty in distinguishing PsA from RA is well known; indeed, seronegative psoriatic polyarthritis simulating RA has been distinguished as a distinct entity by Moll and Wright [5]. Furthermore some authors have actually suggested that the clinical subsets of psoriatic arthritis are in need of review and revision [11]. The present MRI findings support that view, and indicate that MRI may be a vital tool in establishing the new subsets of psoriatic joint disease. Thus MRI may be crucial in making a differential diagnosis between patients with PsA and those with RA, and have major therapeutic implications. MRI distinguishes not only different phases of RA in small joints [3] but offers subdivision of patients with PsA into those who have a distinct pattern, also found in RS, and those whose disease resembles RA.

The final feature of note is the surprising scarcity of bone marrow oedema in these patients. Since only 2 of 32 joints, all having clinical and MRI signs consistent with active joint inflammation, exhibited this finding, it can hardly be described as a characteristic feature. The rarity of this finding was confirmed also in a different series of 65 small hand joints in patients with RA [12]. The findings, when present, of decreased bone marrow signal intensity on T1W images and increased signal intensity on T2W images are similar to those of transient osteoporosis described by Wilson et al. [13], suggesting that they represent bone marrow oedema. Gd-DTPA enhanced signal intensity in the same regions, demonstrated in both present patients, suggested hyperaemia. Furthermore, the distribution of abnormality in these two patients reflected the degree and extent of inflammatory changes in adjacent soft tissues. Thus in the patient with RA, bone marrow signal intensity changes were demonstrated on both sides of the joint (Fig. 2). The distribution was different in the case of RS, with widespread signal intensity changes only in the proximal phalanx, corresponding closely to the inflammatory changes in the local soft tissues (Fig. 4). It is of interest that plain films of these patients did not reveal juxta-articular demineralisation; indeed in the case of RS, bone density appeared slightly increased. Thus bone marrow oedema seems to be very rare in both RA and SNSA, but when it is present it reflects local distribution of inflammatory tissue.

This raises important considerations about the finding of juxta-articular osteoporosis on plain radiographs, a feature classically described as one of the early signs of RA and including in many widely accepted sets of diagnostic criteria [5, 7]. Its value for the early diagnosis of RA is rarely disputed 14]. This collateral phenomenon of arthritis generally has been attributed to the juxta-articular bone marrow hyperaemia provoked by local synovial inflammation [11, 15]. Thus, granted the rarity of MRI findings consistent with local bone marrow hyperaemia in this present study of early RA and SNSA, the relationship between juxta-articular osteoporosis and bone marrow hyperaemia in inflammatory rheumatic diseases would seem to need reappraisal. It is possible that mar-

row oedema could be detectable by other novel pulse sequences, such as STIR, which were unavailable in this study. However, conventional T1W and T2W sequences are unusually satisfactory to show marrow oedema, and there is thus no evidence to suggest that it may have been overlooked in this series.

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