Comments on: The use of MRI in early RA: reply

Sir, I would like to thank Prof. McGonagle and Dr Tan [1] for their interest in the recently published editorial [2]. These authors argue that MRI in early RA does not have any ‘relevance’ as identical features can occur in other conditions, citing their own work in PMR (currently in press). It is true that MRI cannot be used to differentiate between the inflammatory arthritides in the early phase as synovitis, tenosynovitis, bone oedema and erosions have all been described in PsA, SLE and PMR as well as RA [3–6]. However, as alluded to in the article, it is often relevant to determine whether a patient with hand or wrist pain truly has evidence of inflammation and MRI can be most informative in this respect. Ejbjerg et al. compared MRI scans of the wrist from normal controls with RA patients and found that moderate–high grade synovitis and bone oedema (any grade) occurred exclusively in the RA group [7].

The finding of MRI erosions on the hands or feet must surely be regarded as highly significant because it indicates the onset of joint damage. The inexorable progression of the MRI erosion score in our own RA cohort from a median of 2.0 at baseline, increasing to 9.0 at 1 yr and 21.0 at 6 yrs [8], clearly reflects the destructive potential of the disease and shows exactly the same pattern as the progression of radiographic erosions [9] which follow. However, it is wise to be aware of the potential pitfalls of any imaging modality as the clinician could be misled by the finding of one or two questionable erosions. In a site-by-site analysis comparing MRI and CT scanning for the detection of erosions at the wrist, we found 87% concordance between modalities, but there were some examples of mismatch [10]. A small number of false-positive and -negative ‘MRI erosions’ were due to partial volume artefacts creating a blurred border between bone and adjacent soft tissue. This can occur at sites of ligamentous attachments where bony contours are irregular and where erosions frequently begin [11]. This highlights the importance of looking for a combination of features on MRI (see below) rather than relying solely on erosions, which can, as McGonagle et al. [1] point out, be confused with normal variants or degenerative bone cysts.

The most striking clinical relevance of MRI in early RA is its role in prognostication. Bone oedema has been shown repeatedly to be a poor prognostic sign both at the level of the individual bone (increasing the risk of erosion more than 6 times) and when scored from a joint region [8]. In a recent cohort study of 84 RA patients, a bone oedema score of >2 (using the RAMRIS system [12]) was an independent predictor of radiographic erosion progression after 1 yr, with an odds ratio of 2.7, whereas the synovitis score was not predictive [13]. Clearly, the issue of whether synovitis is the variable precursor of erosions remains contentious, with evidence for [14] and against [9, 13] in the literature. More secure is the evidence that high levels of ‘overall’ MRI joint inflammation (synovitis, tenosynovitis and bone oedema combined) indicate the potential for ongoing joint damage [8, 13]. When evidence of damage (in the form of MRI erosions) is also present, this should alert the clinician to the potential for a poor outcome and such a patient should be managed aggressively.

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F. M. McQueen

1Department of Molecular Medicine and Pathology, University of Auckland, Auckland, New Zealand

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Correspondence to: F. M. McQueen, Department of Molecular Medicine and Pathology, Faculty of Medicine and Health Sciences, University of Auckland, Private Bag 92019, Auckland, New Zealand. E-mail: fmqueen@auckland.ac.nz

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Comment on: Prevalence, serological features, response to treatment and outcome of critical peripheral ischaemia in a cohort of lupus patients

Sir, Jeffery et al. [1] have presented the data of seven patients with SLE drawn from a cohort of 487 patients followed over 28 yrs...
who had critical peripheral ischaemia. Interestingly, two of these patients had conditions (meningococcal sepsis and disseminated intravascular coagulation) which themselves can lead to digital ischaemia even without lupus as a comorbid condition. We present the data of 20 patients with gangrene of at least a finger or toe from a cohort of 344 patients of SLE seen over the last 10 yrs. This cohort consists predominantly of a female South Indian population attending a tertiary referral centre. We excluded the data of patients with reversible ischaemic changes.

The mean age of our cohort is 29.2 ± 10.7 yrs and mean duration of follow-up 38.56 ± 42.84 months. Of the 20 patients with gangrene of one or more digits, in 10 patients peripheral ischaemia was the presenting symptom leading to the diagnosis of SLE. The median duration of SLE in the other 10 patients was 31.5 months (range 7–78 months). The mean age of patients with gangrene was 37.3 ± 14.6 yrs. The observations on the clinical features of lupus in these 20 patients were as follows. Eight patients had malar rash, none had a discoid rash, four had photosensitivity, 17 patients had arthritis, four patients had lupus nephritis and six patients had haematological involvement. Only one patient reported RP. Antibodies to dsDNA were significantly elevated in 18 of the 19 patients in whom the data were available. Complement levels were reduced in 7 of 14 patients in whom they were measured. The aCLs (IgG and IgM) were assayed using Bio-Rad kits (Bio-Rad Laboratories, Hercules, CA, USA). Cut-off for negative value was considered as 23 GPL for IgG aCL and 11 MPL for IgM aCL. Three patients were positive for IgG aCL and three for IgM aCL, both being positive in one patient. Activated partial thromboplastin time was elevated in only two patients. Four patients were lost to follow-up after the initial admission. In the remaining 16 patients, the median follow-up after gangrene was 16 months with a range of 1–80 months. Four patients had recurrence of an ischaemic event, two in the same site and two in other sites, i.e. pulmonary thrombus and stroke.

According to our unit policy the initial therapy of digital ischaemia in SLE involves high dose steroids, intravenous (i.v.) heparin and oral anti-platelet drugs followed by oral anti-coagulation and monthly pulses of i.v. cyclophosphamide for 6 months. The active ischaemia responded to therapy in all patients. Intravenous prostaglandin (alprostidol) was used only for persistent pain or progressive gangrene. It was needed in two patients. Solone pulses, which have an early membrane stabilizing effect underlining cause.

The vasculitis in lupus is related to up-regulation of adhesion molecules on the endothelial cells probably as a result of interaction with circulating auto-antibodies and immune complexes [2]. The digital gangrene in our cohort is probably due to active vasculitis. This is probably the reason why methylprednisolone pulses, which have an early membrane stabilizing effect and later act on many mediators of inflammation [3], are successful in the therapy of digital gangrene of lupus. Prosta-glandins dilate blood vessels and have an anti-platelet effect and so may also be effective in digital ischaemia consistent with the policy of some units to use it as part of the initial therapeutic package.

To conclude, prevalence of digital gangrene in this Indian cohort is higher. The age of the patients with gangrene is higher than the mean age of the rest of the cohort. In 50% of the patients with digital ischaemia it occurred early in the course of lupus. The aCLs are negative in majority of our patients, in contrast to the suggestion of over-representation of aCLs in the index study. Almost all patients have active SLE with elevated dsDNA and low complements. The digital gangrene in our cohort is probably due to active vasculitis rather than a non-inflammatory vasculopathy. We believe that intravenous prostaglandin can be offered as therapy to patients who do not respond to initial therapy. **Disclosure statement:** The authors have declared no conflicts of interest.

L. RAJASEKHAR1, N. V. JAYACHANDRAN1, V. N. N. PRABU1, G. NARSIMULU1

1 Department of Rheumatology, Nizam’s Institute of Medical Sciences, Panjagutta, Hyderabad, Andhra Pradesh, India

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Correspondence to: L. Rajasekhar, Department of Rheumatology, Nizam’s Institute of Medical Sciences, Panjagutta, Hyderabad-500082, India. E-mail: lizarajasekhar@yahoo.com


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**Comment on: Prevalence, serological features, response to treatment and outcome of critical peripheral ischaemia in a cohort of lupus patients:** reply

Sir, We read with great interest the description of digital gangrene in lupus patients in an Indian population described by Rajasekhar et al. [1] in response to our recent publication in *Rheumatology* [2]. As the authors point out, the prevalence of aPLs was higher in our studies than in theirs and probably explained why they have managed to treat their patients relatively successfully using quite intense immunosuppression (monthly pulses of intravenous cyclophosphamide for 6 months) in contrast to our preference for intensive prostaglandin therapy. They suggested that immunosuppressive intravenous prostaglandins may not be necessary, as initial management of gangrene in lupus and if there are good grounds for believing that vasculitis alone in the absence of any accompanying thrombosis is thought to be the culprit, dispensing with prostaglandins may be an option. However, in reality, pre-gangrenous and, indeed, gangrenous lesions, due to digital ischaemia may develop with remarkable rapidity and certainly whilst waiting for results of blood tests, including aPLs, to come back from the laboratory, we would strongly advocate the use of intense intravenous vasodilatation if there is any doubt as to the underlying cause.

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C. NARSHI1, R. JEFFERY1, D. A. ISENBERG1

1 Centre for Rheumatology, Division of Medicine, University College London, London, UK

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Correspondence to: D. A. Isenberg, Centre for Rheumatology, UCL Division of Medicine, Room 331, 3rd Floor, Windyler Building, 46 Cleveland Street, London W1T 4JF, UK. E-mail: joan.perry@ucl.ac.uk