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.

Disclosure statement: The author has declared no conflicts of interest.

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Accepted 26 November 2008

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Rheumatology 2009;48:451–452
doi:10.1093/rheumatology/ken451
Advance Access publication 16 January 2009

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).

Secondly, we offer a possible explanation for the increased prevalence of critical peripheral ischaemia in our lupus patients. 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 prednisolone treatment. They suggested that immunosuppressive intravenous prednisolone 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 prednisolone 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.

Disclosure statement: The authors declare no conflicts of interest.

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Accepted 13 November 2008

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Rheumatology 2009;48:452–453
doi:10.1093/rheumatology/ken474
Advance Access publication 27 January 2009

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 their 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 prednisolone treatment. They suggested that immunosuppressive intravenous prednisolone 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 prednisolone 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.

Disclosure statement: The authors declare no conflicts of interest.

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Accepted 21 November 2008

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