MRI of psoriatic nail disease pre- and post-TNF inhibitor therapy shows persistent subclinical inflammation after 6 months despite good clinical response

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Nail involvement is part of the clinical spectrum of psoriatic disease and is microanatomically associated with distal interphalangeal joint (DIP) entheses.1 Tumour necrosis factor (TNF) inhibitors have shown efficacy for psoriasis, arthritis, enthesitis, dactylitis and nail disease.2 Given the intimate links between psoriatic arthritis (PsA) and nail disease, it might be expected that nail disease improvement would be associated with resolution of the underlying arthropathic features.

All participants gave written consent. All cases fulfilled the Classification Criteria for Psoriatic Arthritis (CASPAR) criteria for PsA.3 All cases were due to start TNF inhibitor therapy for active PsA. A clinical and MRI assessment was performed at baseline and after 6 months of treatment.

High-resolution MRI was performed on one finger, with gadolinium contrast and Vaseline applied to the nail.4 The target finger for imaging was selected for current nail disease and active DIPJ arthritis. The MRIs were scored as previously reported.5 Seven patients were recruited. All patients had tenderness and swelling of the target finger’s DIPJ at baseline. TNF inhibitor prescription was made according to the National Institute for Health and Care Excellence guidelines. Four patients received etanercept and three adalimumab.

Marked improvements were seen in clinical parameters at 6 months. No patients had residual clinical swelling in the target DIPJ and one had persistent tenderness (table 1). Onycholysis and pitting were the most frequent abnormalities at baseline. Two patients had completely normal nails in the target finger at 6 months, with no difference in nail clearance between nail matrix features and nail-bed abnormalities.

Baseline MRI scans showed DIPJ enthesitis, bone marrow oedema (BMO) or synovitis in all patients (86%, 71% and 100%, respectively) (table 2). Collateral ligament entheseopathy was seen in 86%, flexor tendon entheseopathy in 71% and extensor tendon entheseopathy in 86%. Three patients with purely nail-bed nail disease at baseline also had marked underlying BMO, synovitis and enthesitis on MRI.

Follow-up MRI scans surprisingly showed persistent inflammatory changes in the DIPJ, distal phalanx and soft tissues around the nail (table 2, figure 1). No patient with baseline BMO showed complete resolution, and four of the five had no change in BMO score. All seven patients had synovitis at baseline; this resolved in two, improved in one, was unchanged in three and worsened in one patient. No patient...
had complete resolution of extensor tendon abnormalities. Collateral ligament abnormalities were largely unchanged. No relationship was seen between the MRI changes and clinical response to treatment.

Previous PsA MRI studies have shown conflicting data regarding the resolution of inflammation with TNF inhibitors, some reporting improvements or resolution, others finding persistent inflammation.6–10 In one study, greater reductions in BMO volume were seen at 18 months than 6 months.10 Our data are limited by the small number of patients and short duration of follow-up. In conclusion, our study demonstrated persistent subclinical musculoskeletal inflammation on MRI despite a good clinical response. Further imaging in larger cohorts with a longer duration of follow-up is needed to demonstrate the natural history of inflammatory lesions under therapy and the potential link to disease progression.

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REFERENCES


