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The fatty Romanus lesion: a non-inflammatory spinal MRI lesion specific for axial spondyloarthritis

A N Bennett,1,2 A Rehman,3 E M A Hensor,1 H Marzo-Ortega,1 P Emery,1 D McGonagle1

ABSTRACT
Background Fatty changes at vertebral corners have been reported on MRI in ankylosing spondylitis but the distribution or specificity of these lesions to early axial spondyloarthritis (axial-SpA) has not been determined.

Objective To assess the diagnostic utility of fatty Romanus lesions (FRLs) for axial-SpA in a population with chronic back pain.

Methods Axial-skeleton T1 SE and fat-suppressed MRI were performed on 174 patients with back pain and 11 controls. MRI lesions including FRLs were scored blind. An imaging diagnosis was given on MRI findings alone and compared with the ‘gold standard’ treating doctor’s diagnosis.

Results Twenty-nine patients had FRLs: 31% (20/64) of patients with spondyloarthritis, 13% (6/45) with degenerative arthritis, 4% (2/45) with spinal malignancy, 5% (1/20) with ‘other’ diagnoses; none of 11 normal subjects had FRLs. The majority of the FRLs in SpA were present in the thoracic spine. The diagnostic utility of FRLs for SpA (likelihood ratio (LR) = 4.7) was significantly (p<0.05) greater than for other diagnoses and increased further (LR = 12.6, p<0.05) when more than five FRLs were present. Of note 5/20 (25%) patients with SpA with FRLs had no diagnostic bone-oedema lesions on fat-suppressed MRI, suggesting that FRLs may be useful diagnostically in axial-SpA.

Conclusion This study defines the FRL as a diagnostic imaging feature of axial-SpA, which may be useful where inflammatory changes are absent on fat-suppression MRI and where radiography is normal.

INTRODUCTION
The hallmark of the seronegative spondyloarthropathies (SpA) and of ankylosing spondylitis (AS), in particular, is the presence of axial-skeleton disease that may lead to sacroiliac joint or spinal fusion. A number of additional radiographic features including vertebral ‘shiny corners’, also known as Romanus lesions, have been described.1 The major limitation of plain radiography for the evaluation of SpA/AS is that it is insensitive, especially in early disease, as diagnostic radiographic changes may take up to 10 years to appear.2 3

The diagnostic and prognostic utility of MRI in SpA is currently an area of intense investigation.4–6 However, what is clear is that patients with symptomatic axial-SpA may have no MRI evidence of acute inflammatory changes—namely, peri-enthesal osteitis.5 7 8 The basis for this apparent normality on MRI fat-suppressed scans in the face of inflammatory back pain is unclear but may relate to the comparatively low resolution of spinal MRI for showing subtle bone and soft tissue inflammation or possible resolution of inflammatory lesions between disease flares.

We describe the fatty Romanus lesion (FRL). The name corresponds to an MRI description based on that fact that in the natural history of SpA inflammatory vertebral corners may undergo a fatty transformation that is best appreciated with high signal on T1-weighted images in the location corresponding to the area in which radiographic Romanus lesions have been described. We believe that the FRL may be a useful imaging biomarker for the diagnosis of SpA in the absence of MR-determined inflammatory lesions.

METHODS
Detailed methods have previously been described.5 In summary, patients were recruited from Calderdale Royal Hospital, UK and were identified as having had an MRI of the whole spine, for back pain, in the past 5 years. A control group with no back pain was also recruited.

The ‘gold standard’ diagnosis was that given by the treating doctor based on history, examination, non-radiological/radiological investigations, histology, when available, and clinical outcome data. Patient case records were evaluated with the benefit of up to 5 years’ outcome data to confirm the treating doctor’s diagnosis. SpA, spinal malignancy and degenerative arthritis (DA) of the spine were the most common diagnoses. In the SpA cohort all patients met the European Spondyloarthritis Study Group criteria for SpA8a (20 with AS, 17 with undifferentiated spondyloarthritis (uSpA), 14 with inflammatory bowel disease (IBD)-SpA, 9 with psoriatic arthritis (PsA), 4 with reactive arthritis (ReA)), had clinically active disease, 68% were HLA-B27 positive and the mean disease duration was 8.5 years.

MRI scoring
All 185 patients had standardised sagittal T1 SE and STIR sequences of the whole spine.5 Scoring was by consensus, using the Leeds Scoring System2 5 modified to score FRLs in the spine. FRLs were defined as well-demarcated triangular lesions, high signal on T1-weighted images in the location corresponding to the area in which radiographic Romanus lesions have been described. Present on at least one sagittal slice on the corner of any vertebrae from lower C2 to upper S1 (figure 1).

FRLs were recorded as present/absent. Other lesions, on STIR sequences, typical of SpA, malignancy and DA were recorded and their diagnostic utility has been reported.5 Fatty changes that extended diffusely along the vertebral end plate were not scored as FRLs but as endplate changes.
Before fat-suppression MRI these latter endplate changes with a distinctive pattern were commonly referred to as Modic type II endplate changes. Modic type II end plates differ from FRLs as the latter are confined to the vertebral corners and do not extend across the end plate. The overall imaging diagnosis and frequency and pattern of lesions on MRI were compared with the ‘gold standard’ diagnosis to assess diagnostic utility.

Intraobserver reliability for FRLs was substantial in the cervical (agreement = 100%, κ = 1.00), thoracic (95.7%, 0.76) and lumbar spine (95.2%, 0.811).

RESULTS
One hundred and eighty-five participants were recruited (mean age 52.5 years (SD 17.3), range 19–91). Sixty-four (35%) had a ‘gold standard’ diagnosis of SpA, 45 (24%) DA, 45 (24%) spinal malignancy, 11 (6%) were normal and 20 (11%) were diagnosed with other conditions including TB abscess and osteoporotic fractures.

Fatty Romanus lesions
Twenty-nine patients had MRI FRLs: 31% (20/64) of patients with SpA (8/20 (40%) with AS, 6/14 (43%) with IBD-SpA, 2/9 (22%) with PsA-SpA, 4/17 (24%) with uSpA), 45 (24%) spinal malignancy, 11 (6%) were normal and 20 (11%) were diagnosed with other conditions including TB abscess and osteoporotic fractures.

The majority of FRL in SpA were present in the thoracic spine (60%) (figure 2) with 35% being present in the lumbar spine and only 5% in the cervical spine. In contrast only 15% in DA were in the thoracic spine and 73% were present in the lumbar spine. There were significantly more thoracic FRLs in SpA than DA (Mann-Whitney U test, Z = −2.46, p = 0.014).

The presence of FRLs had a higher sensitivity (33%), specificity (93%) and significantly higher (p<0.05) likelihood ratio (4.7) for SpA than for DA, or spinal malignancy (table 1). This
increased even further to a LR = 12.6 (p<0.05) and specificity of 98%, if more than five FRL lesions were present in any given case (LR >10 is a large and often conclusive increase in the likelihood of disease) (table 1).

FRLs and spinal enthesitis/osteitis

We determined the relationship between FRLs and bone oedema as depicted on T2 fat-suppressed sequences which in SpA histologically represents an osteitis. None of the FRLs had associated bone oedema on fat-suppressed imaging, suggesting that they might represent a disease phase that follows osteitis/enthesitis.

Of the 20 patients with SpA with FRLs, five did not have diagnostic changes on fat-suppression MRI. Ten per cent (2/20) of these had no inflammatory lesions suggestive of active spinal disease anywhere on fat-suppressed MRI and 15% (3/20) had only one or two non-diagnostic low-grade lesions on fat-suppressed MRI. This suggests that FRLs may be of diagnostic utility where MRI imaging is otherwise normal or nondiagnostic.

DISCUSSION

This study describes a spinal lesion causing MRI evidence of vertebral ‘shiny corners’ by virtue of an increased fat content as depicted by T1-weighted imaging. Although these lesions are similar to radiographic Romanus lesions in that they occupy the same territory, FRLs are fatty rather than sclerotic and appear bright rather than dark on T1-weighted MRI. Therefore, we named these lesions FRLs and found that they were highly specific to SpA and had a significantly higher likelihood ratio for axial-SpA, especially when multiple (likelihood ratio = 12.56, p<0.05), than for other causes of back pain. Surprisingly, the presence of a normal spinal fat-suppressed MRI in the face of clinically active AS is well described. The MRI FRLs might, therefore, have an extremely useful role in the diagnosis of patients with SpA with no active inflammation at the time of scanning and who also have normal spinal radiographs.

Kim et al have recently reported an ‘MRI corner sign’ in a cohort of 52 patients with AS according to modified New York criteria and found it useful diagnostically, with sensitivity and specificity of 44% and 96%, respectively. However, no reliability data were reported, the study was limited to patients with AS established radiographically in the target group rather than a broad spectrum of patients with axial-SpA, only the lumbar spine was scanned and fat-suppressed sequences were not obtained. Moreover, a young control group (mean age 34 years) was used. Such a young control group would only have minimal degenerative changes that might be mistaken for inflammatory SpA lesions, and hence the report by Kim et al of diagnostic specificity of the ‘MRI corner sign’ is likely to be artificially high. These findings are substantiated as Kim et al also report that the ‘MRI corner sign’ increased with age. Also, as no fat-suppression sequences were performed, as is recommended in the assessment of AS, Kim et al therefore included acute inflammatory and chronic lesions in their ‘MRI corner sign’, which could account for their higher reported sensitivity.

Acute inflammatory MRI lesions in the spine in SpA have been reported to be most commonly found in the thoracic spine. It is noteworthy that we found that FRLs were present in an identical territory, which supports the theory that these lesions may be post-inflammatory. In keeping with our findings, Kim et al found that these lesions were most abundant in the thoracolumbar regions.

One common pathophysiological theory for AS is that the initial vertebral corner inflammation is followed by fatty replacement and, subsequently, by sclerotic bone formation. It might be, therefore, that FRLs are lesions detectable in the post-inflammatory phase of SpA, which would agree with the finding that the disease duration of our patients with FRL was 8.3 years—that is, before the sclerotic bone formation phase, which is evident on radiography and often takes up to 10 years. However, given that the study was performed on a relatively large cohort of 185 patients (64 with SpA) it is somewhat surprising that only 20 patients with SpA had FRLs present. Longitudinal studies are needed to fully determine the significance of these lesions with respect to prior inflammatory lesions and spinal fusion in SpA.

One may feel it is slightly surprising to have a proposed postinflammatory lesion, the FRL, found relatively frequently in DA, as in this study. However, inflammatory Romanus lesions have previously been reported to be common in DA, and hence explains the frequency of the FRL in DA, but in more classically degenerative locations.

In conclusion, this study defines the FRLs as a diagnostic MRI feature specific to axial-SpA. Although many patients with SpA will be diagnosed on STIR MRI sequences, the FRLs may be diagnostically useful in patients with SpA, especially in those with no acute inflammatory findings on fat-suppression MRI and/or no diagnostic radiographic change.

Competing interests None.

Ethics approval This study was conducted with the approval of the Halifax and Huddersfield NHS trust research and ethics committee.

Provenance and peer review Not commissioned; externally peer reviewed.

Table 1 Diagnostic value of FRLs in SpA

<table>
<thead>
<tr>
<th></th>
<th>Sensitivity (95% CI)</th>
<th>Specificity (95% CI)</th>
<th>PPV (95% CI)</th>
<th>NPV (95% CI)</th>
<th>Positive likelihood ratio (95% CI)</th>
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<tr>
<td>FRLs&gt;0</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SpA</td>
<td>0.33 (0.21 to 0.48)</td>
<td>0.93 (0.86 to 0.97)</td>
<td>0.69 (0.48 to 0.85)</td>
<td>0.74 (0.66 to 0.81)</td>
<td>4.71 (2.19 to 10.14)</td>
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<tr>
<td>DA</td>
<td>0.13 (0.05 to 0.28)</td>
<td>0.83 (0.76 to 0.89)</td>
<td>0.19 (0.07 to 0.40)</td>
<td>0.75 (0.67 to 0.82)</td>
<td>0.76 (0.30 to 1.87)</td>
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<td>Malignancy</td>
<td>0.02 (0.00 to 0.14)</td>
<td>0.80 (0.71 to 0.86)</td>
<td>0.04 (0.00 to 0.22)</td>
<td>0.70 (0.61 to 0.77)</td>
<td>0.11 (0.02 to 0.80)</td>
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<tr>
<td>FRLs&gt;5</td>
<td></td>
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<tr>
<td>SpA</td>
<td>0.22 (0.12 to 0.36)</td>
<td>0.98 (0.93 to 1.00)</td>
<td>0.86 (0.56 to 0.97)</td>
<td>0.73 (0.65 to 0.79)</td>
<td>12.56 (2.91 to 54.14)</td>
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<tr>
<td>DA</td>
<td>0.03 (0.00 to 0.15)</td>
<td>0.90 (0.83 to 0.94)</td>
<td>0.07 (0.04 to 0.36)</td>
<td>0.75 (0.67 to 0.81)</td>
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<tr>
<td>Malignancy</td>
<td>0.00 (0.00 to 0.10)</td>
<td>0.89 (0.81 to 0.93)</td>
<td>0.00 (0.00 to 0.27)</td>
<td>0.71 (0.63 to 0.78)</td>
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REFERENCES


