

# Magnetic Resonance Imaging for Spondyloarthritis — Avoiding the Minefield

Diagnostic evaluation and management of inflammatory arthritis based primarily in the axial skeleton is arguably more problematic than in most other forms of inflammatory arthritis. Physical findings are typically confined to later stages of disease and are insensitive to change. Laboratory investigations are essentially limited to acute phase reactants and HLA-B27, which alone have limited diagnostic value. Plain radiographic abnormalities occur late, are insensitive to change,

and depict structural abnormalities only<sup>1</sup>. Consequently, diagnostic evaluation and longterm management is still largely dependent on historical inquiry and subjective patient self-report. The advent of magnetic resonance imaging (MRI) has proven to be a milestone in the evaluation of spondyloarthritis (SpA) through its ability to depict objective features of active inflammation, thereby facilitating earlier diagnosis and ongoing management, and permitting quantitative assessment



**Figure 1.** A. Sagittal STIR image of spine in a 51-year-old man with SpA (TR 3060 ms, TE 61 ms, TI 140 ms). Small white arrows: multiple small Romanus lesions in the lower thoracic spine seen only on STIR sequence. Large white arrow: inflammation in T10/11 facet joint. \*extensive vertebral inflammation at L1/2 and L5/S1. B. Sagittal T1SE image of spine (TR 420 ms, TE 13 ms). Arrows: bright signal due to repopulation of marrow with fat after healing of a Romanus lesion at L2/3.

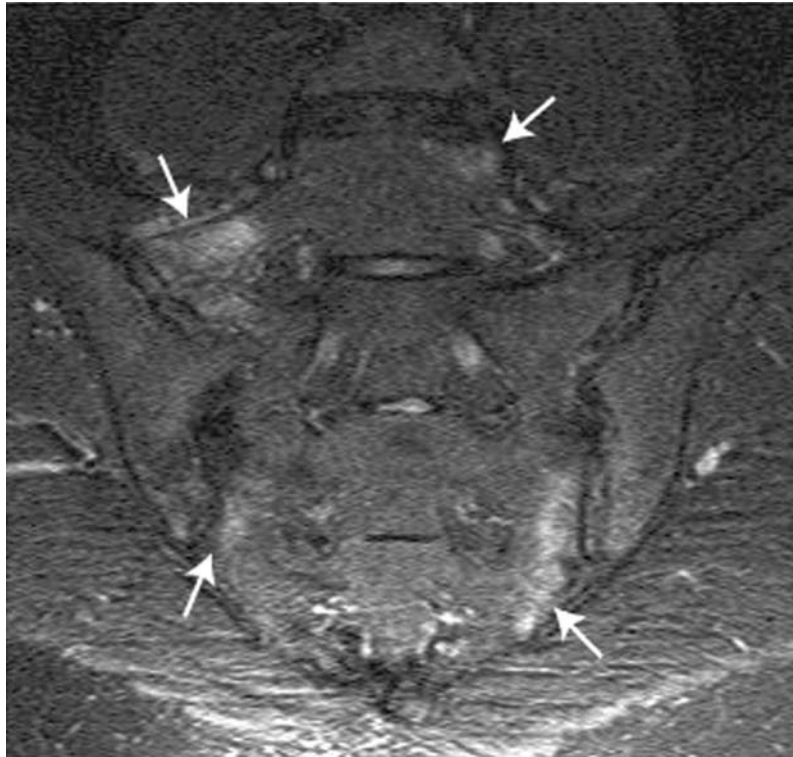
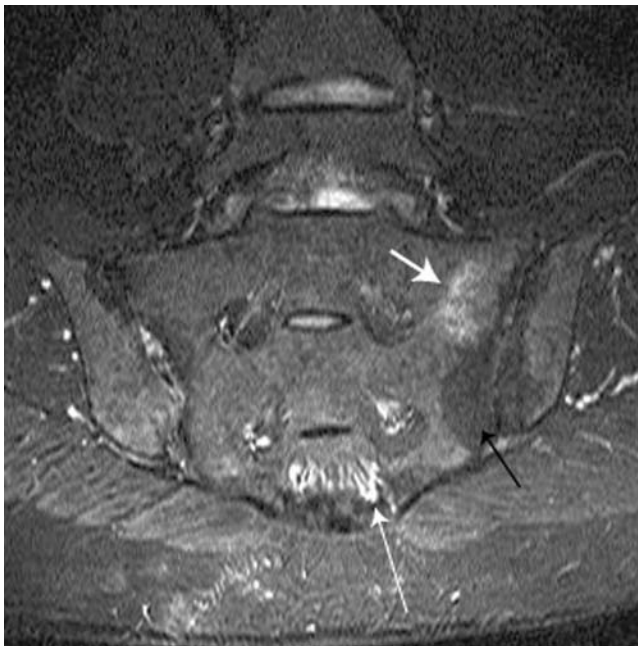
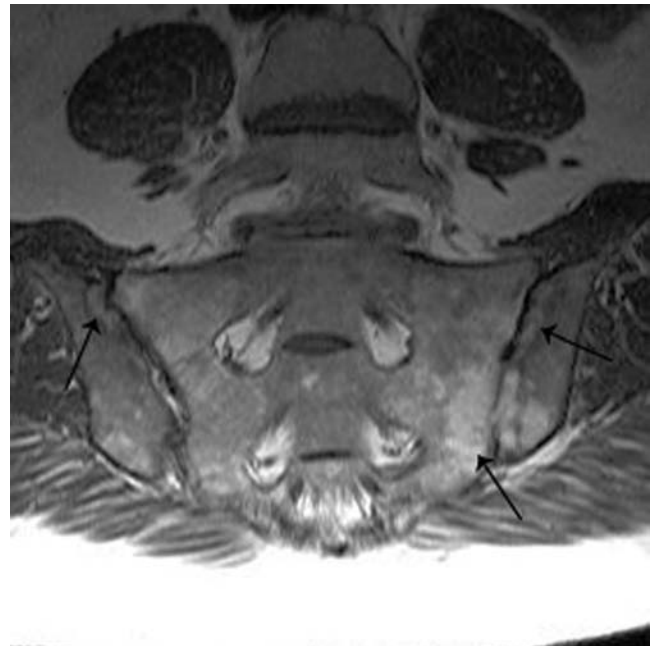


Figure 2. Coronal STIR image of SI joints in a 42-year-old man with SpA (TR 3700 ms, TE 38 ms, TI 140 ms). Arrows: multiple sites of active inflammation in both SI joints, left lumbosacral pseudarthrosis (transitional L5 vertebra), and at L5 inferior endplate.



**A**



**B**

Figure 3. A. Coronal STIR image of SI joints in a 35-year-old man with SpA (TR 3700 ms, TE 38 ms, TI 140 ms). Short white arrow: marrow inflammation (not seen on T1SE image). Long white arrow: presacral veins. Black arrow: "healed inflammation as reflected by fatty bone marrow infiltration." B. Coronal T1SE image (TR 414 ms, TE 13 ms). Black arrows: multiple foci of postinflammatory change with ankylosis in lower half of left SI joint.



Figure 4. Same patient as Figure 2; sagittal STIR image of spine (TR 3060 ms, TE 61 ms, TI 140 ms). Large white arrow: focal discovertebral inflammation in the center of the T12 inferior endplate. Small white arrows: Romanus lesions. Black arrows: bright signal is projected over the bones due to artefact from blood flowing in great vessels, creating appearance very similar to the Romanus lesions.

of extent and severity of spinal inflammation. There is clearly increasing awareness of the clinical utility of MRI, as reported in a recent survey of Canadian rheumatologists, although most are unfamiliar with imaging techniques, the spectrum of abnormalities observed, and pitfalls in assessment.

The availability of numerous MR sequences and approaches to imaging has the potential to confuse and deter clinicians in the interpretation of MR images. We recommend that rheumatologists focus on 2 MR sequences that provide most of the imaging details required for the assessment of SpA, require the least imaging time, and incur the least cost. In T1-weighted images (T1WI), normal bone marrow fat is bright, while bone is dark, and these contrasting features con-

tribute to the enhanced anatomical resolution provided by T1 compared to other MR sequences. Loss of the fat signal within bone marrow is a nonspecific finding and may be observed following fat replacement by most pathological processes such as sclerotic bone, fibrosis, tumor, cyst, and inflammation. However, the presence of acute inflammation in the marrow may be obscured by the signal from marrow fat. The short tau inversion recovery (STIR) sequence is a T2-weighted sequence that suppresses the fat signal so that the marrow now appears dark, and any fluid within the marrow due to active inflammation is relatively bright (Figure 1). The signal detected by the STIR sequence is a nonspecific indicator of increased free water content (both intra- and extracellular), so that the differential diagnosis for abnormal signal on this sequence includes any pathology associated with fluid accumulation in bone marrow (e.g., tumor, type I Modic change related to disc degeneration, infection, and trauma). Images of the spine are typically obtained in the sagittal orientation, and STIR images can be readily discerned from T1WI by the presence of high signal emanating from the cerebral spinal fluid in the spinal canal. Images of the sacroiliac (SI) joints are typically obtained in a tilted coronal orientation along the long axis of the sacral bone. Consecutive slices in this plane allow visualization of the cartilaginous portion of the SI joint, which is convex-shaped with the apex facing antero-inferiorly.

One of the earliest features of sacroiliitis is the presence of subchondral bone marrow edema in the postero-inferior region of the synovial portion of the joint, as seen on STIR images (Figure 2). This reflects the subchondral marrow inflammation reported on histopathological examination of early sacroiliitis<sup>2</sup>. The lesion develops into an erosion as defined by loss of the marrow fat signal on T1WI together with loss of subarticular bone. Healing of erosion/inflammation or progression to ankylosis is typically associated with repopulation of the marrow space by fat (Figure 3). The spectrum of abnormalities is easiest to interpret when T1WI and corresponding STIR images are viewed concurrently. Characteristic abnormalities observed in the spine may also be seen on STIR images, which depict increased marrow signal at the anterior and posterior corners of the vertebrae, adjacent to the insertion of the annulus fibrosis, corresponding to the Romanus lesion observed histopathologically (Figure 1). A lesion that had been underestimated prior to the advent of MRI is the occurrence of spondylodiscitis characterized as inflammatory marrow signal within vertebrae adjacent to the central portion of the vertebral endplate, which occurs in up to 15% of patients (Figure 4)<sup>3</sup>. Acute lesions may also occur in the posterior elements of the spine including the facet joints, pedicles, and ligamentary insertions at spinous and transverse processes (Figure 1). We have also documented a high frequency of acute changes in the costovertebral joints that may represent the major portion of the inflammation occurring in the thoracic spine (Figure 5)<sup>4</sup>. Syndesmophytes tend to be less well visualized on MRI than by radiography, and a systemat-



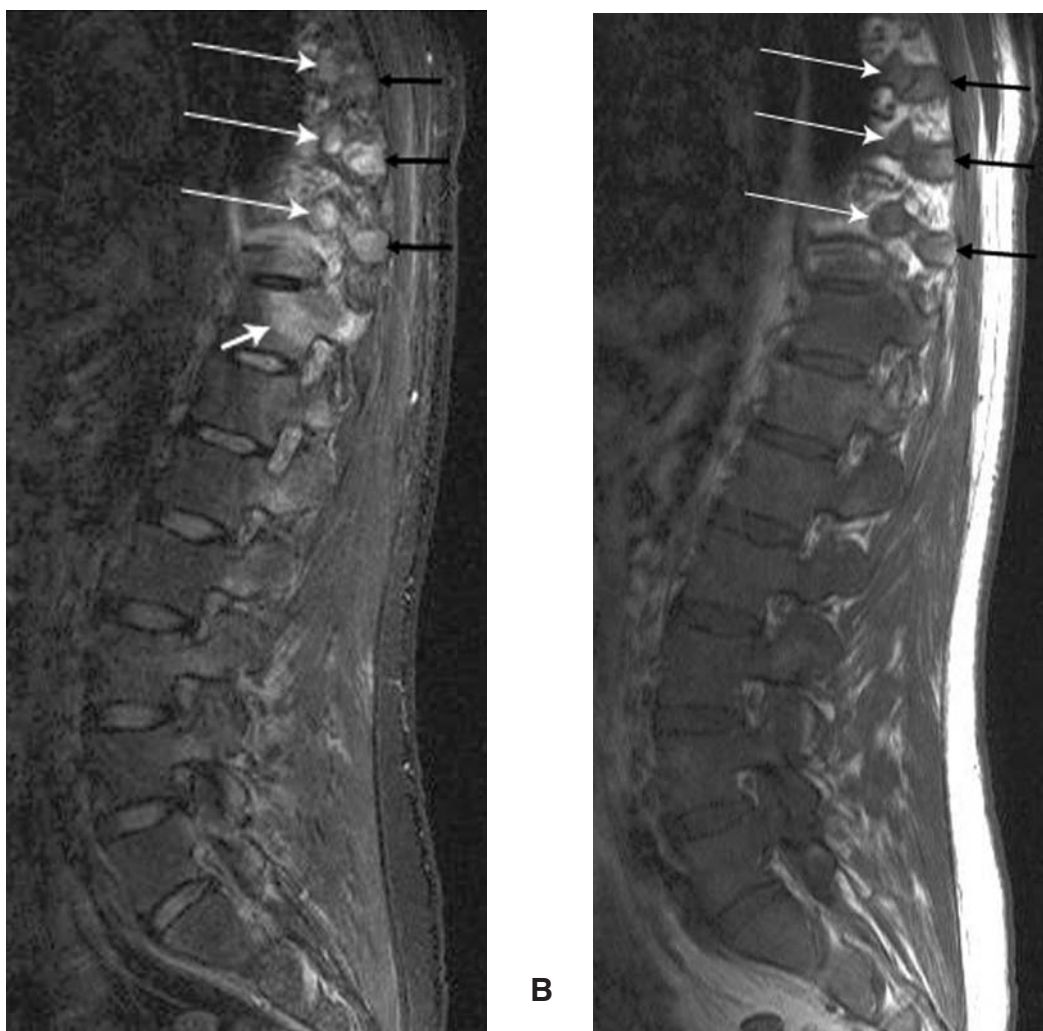


Figure 5. A. Sagittal STIR image of spine in a 29-year-old man (TR 3060 ms, TE 61 ms, TI 140 ms). Extensive inflammation in costovertebral and costotransverse joints. Long white arrows: ribs. Black arrows: transverse processes. Short white arrow: vertebral side of costovertebral joint. B. Sagittal T1 SE image of spine (TR 420 ms, TE 13 ms). Long white arrows: ribs. Black arrows: transverse processes.

ic comparison showed no advantages of MRI over radiographs for the assessment of chronic lesions<sup>5</sup>.

Pitfalls to the use of MRI can be broadly subdivided into those that are primarily technical and those that relate to interpretation. MRI is subject to numerous artefacts that are invariably present on these types of scans. Motion artefact is a particular problem and this includes any patient movement (including breathing, swallowing) and physiological motion (e.g., arterial, venous, or cerebrospinal fluid pulsation artefact). These commonly simulate low-grade inflammation and so are particularly relevant to patients with SpA (Figure 6). The spine is typically assessed in 2 halves to reduce imaging time, an upper cervico-thoracic and a lower thoraco-lumbar. This results in a relatively large field of view so that the cervical vertebrae appear small, limiting anatomical resolution and detectability of lesions. Detection of abnormalities also depends on viewing conditions, which are much superior when the image is displayed on a computer monitor (in con-

trast to hard copy film), allowing the observer to manipulate the display properties. The setting of the thresholds and grey-scale (windowing) for viewing markedly affects the visualization of subtle lesions, and is particularly important in the assessment of STIR images, where signal to noise ratios are usually limited. On suboptimal quality scans, it may be quite difficult to reliably determine what constitutes abnormal increased signal intensity referable to background. This is particularly germane to the development of scoring systems for axial inflammation, where explicit definition of what constitutes abnormal inflammatory signal in the SI joint and spine, such as in the SpA Research Consortium of Canada (SPARCC) MRI index<sup>6,7</sup>, is essential for observer consistency.

Pitfalls in interpretation are often related to a lack of systematic assessment, limitations in anatomical resolution, failure to apply consistent definitions of abnormalities, and fixed postural abnormalities (e.g., scoliosis) in the patient. Examination of the lateral and posterior segments of the spine



**Figure 6.** Sagittal STIR image of spine in a 64-year-old man (TR 3000 ms, TE 54 ms, TI 140 ms). Cerebrospinal fluid signal in upper thoracic spine (upper black arrow) does not appear nearly as bright as in the lower thoracic spine (lower black arrow). This artefact is unavoidable and is usually caused by the greater distance of the upper thoracic spine from the table top (and thereby the receiver coil) due to normal kyphotic curvature in the thoracic spine. White arrows: “noise” created by flowing blood in great vessels projects bright signal vertically across the image and over the T4 and T5 vertebral bodies. On this image, it is impossible to exclude inflammation in these vertebrae.

that may harbor inflammation in the costovertebral joints, facet joints, pedicles, and entheses may be difficult due to the complexity of the anatomy. Training is essential if readers of MRI are to avoid frequent misinterpretation of scans, and even radiologists with considerable experience in MRI are frequently unaware of the nuances of interpretation in this field. A systematic approach to detection of lesions in SpA is essential, and a diagnostic imaging protocol has been posted on our website [www.arthritisdoctors.org]. We have recently shown that when the spine is assessed systematically, a major portion of the inflammation in the thoracic spine is primarily observed laterally, such as in the costovertebral joints<sup>4</sup>. Some MRI reports have primarily focused on descriptions of individual lesions in a single dimension and described lesions primarily in vertebral bodies<sup>8</sup>. This may be sufficient in framing

a scoring system but is clearly insufficient for diagnostic evaluation. It is important to emphasize that interpretation of abnormalities on MRI (e.g., erosions) may be rather different from radiography, and few reports clearly define such abnormalities. Where this has been done, an erosion has been defined as loss of marrow fat signal together with loss of the overlying subchondral bone on a T1WI<sup>9</sup>. However, because bone is dark, loss of subchondral bone may not be readily apparent and sometimes may even be more evident on STIR images. Of particular relevance to the interpretation of spinal abnormalities, degenerative discal lesions are common in the age group with SpA and may include loss of the discovertebral endplate with adjacent bone marrow edema that may be indistinguishable from the spondylodiscitis of SpA. Unfortunately, there have been no reports that systematically analyzed the sensitivity and specificity of lesions in the spine.

Inflammation in bone may also be detected using T2-weighted spin-echo sequences with fat suppression or with T1WI with fat suppression following gadolinium contrast enhancement (T1FS-postGd). Contrast-enhanced sequences do offer specific advantages that may be evident in early inflammatory lesions; for example, inflammatory changes in soft tissues and at sites of enthesitis may be seen first or be easier to identify with T1FS-postGd<sup>2</sup>. However, comparative studies have yet to be conducted, and the method of fat suppression required is problematic as it is frequently subject to significant artefact when scanning with a large field of view in the spine (Figure 7). Consequently, most approaches to the quantitative assessment of acute MRI lesions are primarily focused on the evaluation of STIR images and can generally be subdivided into 2 related methods (ASSpMRI, Berlin score) that score individual lesions in a single dimension (anterior-posterior extent for the spine)<sup>7</sup> and one method (SPARCC MRI index) that scores lesions in 3 dimensions<sup>6</sup>. Inflammatory lesions within bone are often highly asymmetrical and therefore are more precisely quantified with a method that systematically assesses lesions in several dimensions. With the former methods, evaluation of the vertebral body of all 23 vertebral segments is mandatory, necessitating the scoring of regions subject to artefact and/or limited anatomical resolution. The median number of affected vertebral segments is typically 5–6 in a cohort of severely affected patients that would be regarded as eligible for trials of anti-TNF therapies<sup>10</sup>. In patients with many levels affected, the less conspicuous lesions are often subtle, and therefore the scoring of these lesions is less responsive to therapy. It is therefore not surprising that a comparison of the SPARCC scoring method for all 23 DVU versus only the 6 most severely affected discovertebral unit shows similar reliability but better responsiveness for the latter approach<sup>10</sup>.

One can conclude that MRI is now established as an essential component of the clinician’s toolkit, although in need of further educational initiatives. For the clinical researcher, much greater scrutiny is required to address issues



Figure 7. A. Sagittal T1SE image of spine in a 40-year-old man (TR 618 ms, TE 13 ms). Focal fatty infiltration in the anterior border of C2 is a postinflammatory phenomenon (black arrow). C2/3 disc is normal. Note erosion/defect in T6 inferior endplate (\*). B. Sagittal T1FS-postGd (TR 618 ms, TE 13 ms) image of spine. Upper thoracic spine is clearly visible (lower white arrow), and contrast enhancement is present in the erosion/defect in the T6 inferior endplate (\*), consistent with active inflammation. An important artefact is present in the cervical spine. The signal from the spinal cord, cerebrospinal fluid, paraspinal muscle, and intervertebral disc are all artificially decreased at the C2/3 level (upper white arrow). This is due to erroneous “water signal suppression” at these levels, where the fat suppression has also failed to occur. Note that only the C2/3 disc appears black. This results in the false impression of a contrast-enhancing Romanus lesion anteriorly in the C2 inferior endplate (black arrow) due to the unsuppressed fat signal. Not only is this a problem, but in addition if active inflammation was present it could not be detected, as the signal from any true contrast enhancement would be eliminated by the effect of the “water signal suppression.”

of standardization of methodology and pitfalls in assessment. However, there is also a need to recognize and incorporate the unique opportunities afforded by this imaging modality in the evaluation of lesions in patients with SpA.

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## REFERENCES

1. Wanders AJB, Landewé RBM, Spoorenberg A, et al. What is the most appropriate radiologic scoring method for ankylosing spondylitis? *Arthritis Rheum* 2004;50:2622-32.
2. Muche B, Bollow M, Francois RJ, Sieper J, Hamm B, Braun J. Anatomic structures involved in early- and late-stage sacroiliitis in spondylarthritis: a detailed analysis by contrast-enhanced magnetic resonance imaging. *Arthritis Rheum* 2003;48:1374-84.
3. Braun J, Sieper J, Bollow M. Imaging of sacroiliitis. *Clin Rheumatol* 2000;19:51-7.
4. Rennie WJ, Dhillon SS, Conner-Spady B, Maksymowych WP, Lambert RGW. Standard MRI assessment of spinal inflammatory lesions in AS may omit a significant component of inflammation in the thoracic spine. *Ann Rheum Dis* 2006;65 Suppl 2:534.
5. Braun J, Baraliakos X, Golder W, et al. Analysing chronic spinal changes in ankylosing spondylitis: a systematic comparison of conventional x-rays with magnetic resonance imaging using established and new scoring systems. *Ann Rheum Dis* 2004;63:1046-55.

6. Maksymowych WP, Inman RD, Salonen D, et al. Spondyloarthritis Research Consortium of Canada magnetic resonance imaging index for assessment of spinal inflammation in ankylosing spondylitis. *Arthritis Rheum* 2005;53:502-9.
7. Maksymowych WP, Inman RD, Salonen D, et al. Spondyloarthritis Research Consortium of Canada magnetic resonance imaging index for assessment of sacroiliac joint inflammation in ankylosing spondylitis. *Arthritis Rheum* 2005;53:703-9.
8. Braun J, Baraliakos X, Golder W, et al. Magnetic resonance imaging examinations of the spine in patients with ankylosing spondylitis, before and after successful therapy with infliximab. Evaluation of a new scoring system. *Arthritis Rheum* 2003;48:1126-36.
9. Bird P, Conaghan P, Ejbjerg B, et al. The development of the EULAR-OMERACT rheumatoid arthritis MRI reference image atlas. *Ann Rheum Dis* 2005;64 Suppl 1:i8-10.
10. Maksymowych WP, Dhillon SS, Park R, Salonen D, Inman RD, Lambert RGW. Validation of the Spondyloarthritis Research Consortium of Canada (SPARCC) MRI Spinal Inflammation Index: Is it necessary to score the entire spine? *Arthritis Rheum* (in press).