

Structural Lesions Detected by Magnetic Resonance Imaging in the Spine of Patients with Spondyloarthritis – Definitions, Assessment System, and Reference Image Set

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ABSTRACT. Objective. There is no reliable and sensitive magnetic resonance imaging (MRI) assessment system for structural lesions in patients with spondyloarthritis (SpA). We sought to develop and illustrate a detailed anatomy-based set of MRI definitions and an assessment system for structural lesions in the spine of patients with SpA.

Methods. MRI definitions of different structural (“chronic”) lesions at various anatomical locations in the spine, and an accompanying assessment system, were agreed by consensus within the Canada-Denmark MRI working group. Subsequently, a reference image set of representative examples of the individual pathologies, as well as borderline cases and important artefacts, were collected.

Results. The defined lesions were (a) Bone erosions, subdivided into corner and non-corner vertebral body erosions and facet joint erosions; (b) Focal fat infiltration at vertebral corners; (c) Bone spurs, subdivided into corner and non-corner vertebral body spurs; and (d) Ankylosis, subdivided into corner and non-corner vertebral body ankylosis and facet joint ankylosis. All definitions were based on their appearance on sagittal T1-weighted MR images. Vertebral body structural lesions are assessed at each vertebral endplate at all 23 spinal levels from C2/3 to L5/S1, whereas facet joint lesions are to be assessed by segmental level (cervical, thoracic, and lumbar).

Conclusion. An anatomy-based set of definitions and an assessment system for structural lesions in the spine of patients with SpA were developed and illustrated. The system is designed to study the spatial pattern of the lesions and their relation to spine inflammation and clinical and radiographic outcomes. *J Rheumatol* 2009;36 Suppl 84:18-34; doi:10.3899/jrheum.090617

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The great majority of magnetic resonance imaging (MRI) studies of the spine of patients with spondyloarthritis (SpA), including ankylosing spondylitis (AS), have focused on inflammatory changes. While MRI scor-

ing systems for inflammatory activity are available for use in clinical trials¹⁻³, so far only one MRI scoring system for structural lesions, the AS-spi-MRI-c (Ankylosing Spondylitis Spine MRI Chronicity score system), has been proposed and tested in longitudinal studies¹. This system identifies lesions only in the discovertebral units as a whole, without any information on the localization of the changes, and without any possibility for specific documentation of the different types of lesions. Further, it does not identify any changes in the posterior elements of the vertebrae, such as the facet and costotransverse joints. Moreover, the method is validated only as sum scores, not on the level of the individual vertebra.

For exploration of the disease process in SpA, e.g., the spatial and temporal pattern of structural changes in the spine, such as bone erosions, fat infiltration, and syndesmophytes, the currently available scoring system is not sufficient. A more detailed anatomy-based assessment system, separating not only the different types of changes and their occurrence at different anatomical areas, such as vertebral body versus facet joint, but also in different locations within the vertebral body itself, could provide important additional information. Such a system would also be valuable in detailed studies of the relationship between inflammation and the development of different types of structural change, including radiographic evidence of syndesmophyte formation.

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It is very important to be able to detect the presence of changes in structure in the spine. First, it is important to monitor structural lesions in clinical trials and practice. Second, MRI signs of structural change may have prognostic value for longterm disability, pain, and other key patient-related outcomes. Currently, the standard method for assessment of structural spine lesions in SpA is conventional radiography, most often evaluated according to the modified Stoke AS Spinal Score (mSASSS)^{4,5}. This method was rated the preferred radiographic method by the OMERACT consensus conference in 2004, based on its superior reproducibility and sensitivity to change, compared with the competing options – the original SASSS and the Bath AS Radiology Index (BASRI)⁶. However, the method is not very sensitive to change, as it allows reliable detection of change only after at least 2 years^{5,6}. Considering the ability of MRI to visualize the diverse pathologies comprising the structural changes in the SpA spine, a comprehensive MRI system would be expected to record lesions that remain undetected by radiography, and to have a higher sensitivity to change.

In summer 2007, a Canadian-Danish collaboration of researchers from a mixed rheumatological and radiological background (the Canada-Denmark MRI working group) was formed to develop and validate a detailed MRI assessment system for inflammatory and structural changes in the spine of patients with SpA. This report describes and illustrates the group's proposed definitions and assessment system of structural lesions.

METHODS

At a 3 day meeting in Edmonton, Canada, in September 2007, preliminary definitions of different structural lesions (bone erosion, squaring, focal fat infiltration, bone spurs, and ankylosis) at various anatomical locations in the spine of patients with SpA were agreed by consensus between the participants in the Canada-Denmark MRI working group. After subsequent review and testing of the definitions on SpA spine image sets, and discussion at video teleconferences, the definitions were slightly modified. Moreover, squaring was left out, because it was considered very difficult to make a relevant and robust definition, and because the feature was not considered of clinical importance. Thereafter, a series of representative examples of the individual pathologies, as well as borderline cases, were collected. These were discussed, revised, and finally agreed upon by consensus at video teleconferences and a 2 day meeting in Edmonton in May 2008. The selected examples thereafter constituted a "reference image set." Key examples from this reference image set are presented in this article (Figures 1–10), while a more comprehensive collection of reference images can be found online at www.arthritisdoctor.ca.

Two critical factors influenced the group's decisions concerning definitions, which images to assess, and an assessment system: (i) The definitions were developed bearing in mind the key requirement for correlation of MRI data with other measures and outcomes, most particularly radiographic correlation; and (ii) Future screening of SpA patients with MRI will always, and sometimes only, include images in the sagittal plane. It is intended that the definitions would, as far as possible, allow:

1. Separation of precursors of anterior vertebral body syndesmophyte (visible on lateral radiographs) from vertebral body syndesmophyte at other locations (less consistently seen on radiographs).
2. Separation of discovertebral involvement from costovertebral involvement.
3. Separation of vertebral body changes clearly due to discovertebral disease from vertebral body changes that could be manifestations of processes emanating from the posterior elements.

All illustrations in Figures 1–10 are designed according to the following format:

Left panel: Sagittal short-tau inversion recovery (STIR) MR image of the spine.

Center panel: Matching sagittal T1-weighted (T1w) MR image of the spine.

Right panel: Diagram of T1w image depicting anatomy and significant pathological lesions. All T1w MR images were acquired with repetition time (TR) 400–500 ms and echo time (TE) 13–18 ms. All STIR images were acquired with TR 4000–4500 ms, inversion time (TI) 140–145 ms, and TE 50–55 ms.

RESULTS

Below are key points of the MRI definitions of structural lesions, which anatomical areas to assess for them, and the proposed assessment system, with references to illustrations. Table 1 provides a detailed list of the definitions and the assessment system.

Definitions

Signal alteration. All definitions of structural lesions relate to their appearance on sagittal T1w MR images.

1. The term "increased signal in bone marrow" refers to a signal intensity higher than the "normal bone marrow signal." The bone marrow signal in the center of the vertebra, if normal, constitutes the reference for designation of normal signal or, alternatively, in the center of the closest available normal vertebra.

Anatomical location of MR image. The images of the thoracic and lumbar spine on a sagittal MRI scan are divided into "central" and "lateral" slices, defined as follows:

1. Central sagittal slices: The sagittal slices that include the spinal canal. The pedicle may be partially seen but is

Table 1. Definitions of structural lesions in the spine of patients with spondyloarthritis.

DEFINITIONS	
A. Bone erosion	Full-thickness loss of dark appearance of cortical bone <i>and</i> loss of normal bright appearance of adjacent bone marrow on T1w images
A1. Corner bone erosion (COBE)	Bone erosion involving the vertebral corner, in at least one central sagittal slice
Location	Anterior COBE (aCOBE): COBE at the anterior corner Posterior COBE (pCOBE): COBE at the posterior corner
Size	Large: Involvement of more than 25% of the anteroposterior AP diameter of original height of the vertebra, in any central sagittal slice Not large: Does not fulfil definition of large
A2. Non-corner bone erosion (NOBE)	Bone erosion adjacent to the vertebral endplate on any slice, but involving neither the anterior nor the posterior vertebral corner of any central sagittal slice
Location	Central: Involvement of any central sagittal slice Lateral: Involvement of any lateral sagittal slice Note: The same NOBE can be both central and lateral, if present in both central and lateral sagittal slices
Size	Large (central NOBE only): Involvement of more than 25% of AP diameter of the original endplate <i>and</i> more than 25% of original height of the vertebra Not large: Does not fulfil definition of large
Type	Type A: The diameter of neck (at cortical break) is less than the maximal diameter of the loss of high signal in the bone marrow Type B: The diameter of neck (at cortical break) is at least equal to the maximal diameter of the loss of high signal in the bone marrow
A3. Facet joint bone erosion (FABE)	Bone erosion adjacent to the facet joint
B. Focal fat infiltration	Focal increased signal in bone marrow on T1w images. Only fat infiltrations involving the vertebral corners on any central sagittal slice (corner fat infiltration) are assessed
Location	Anterior corner fat infiltration (aFAT): FAT at the anterior corner Posterior corner fat infiltration (pFAT): FAT at the posterior corner
C. Bone Spur	Bright signal on T1w images extending from the vertebral endplate towards the adjacent vertebra
C1. Corner spur (COS)	Bone erosion involving the vertebral corner, in at least one central sagittal slice
Location	Anterior corner spur (aCOS): COS at the anterior corner Posterior corner spur (pCOS): COS at the posterior corner
C2. Non-corner spur (NOS)	Bone spur involving the endplate on any slice, but neither the anterior nor the posterior vertebral corner on any central sagittal slice
Location	Central: Involvement of any central sagittal slice Lateral: Involvement of any lateral sagittal slice. Note: the same NOS can be both central and lateral, if present in both central and lateral sagittal slices
D. Ankylosis	Bright signal on T1w images extending from a vertebra and being continuous with the adjacent vertebra
D1. Corner ankylosis (CANK)	Ankylosis involving the vertebral corner, in at least one central sagittal slice
Location	Anterior corner ankylosis (aCANK): CANK at the anterior corner Posterior corner ankylosis (pCANK): CANK at the posterior corner
D2. Non-corner ankylosis (NANK)	Ankylosis involving the vertebral endplate on any slice, but neither the anterior nor the posterior vertebral corner on any central sagittal slice
Location	Central: Involvement of any central sagittal slice Lateral: Involvement of any lateral sagittal slice Note: The same NANK can be both central and lateral, if present in both central and lateral sagittal slices
D3. Facet joint ankylosis (FANK)	Ankylosis at the facet joint
E. Additional definitions	Normal bone marrow signal: The bone marrow signal in the center of the vertebra, if normal. If not, the signal in the centre of the closest available normal vertebra

Table 1. Continued

Increased signal in the bone marrow	A signal intensity higher than the normal bone marrow signal
Central sagittal slices (thoracic and lumbar spine only)	The sagittal slices that include the spinal canal. The pedicle may be partially seen but is not continuous between the vertebral body and posterior elements
Lateral sagittal slices (thoracic and lumbar spine only)	The sagittal slices that are located lateral to the spinal canal. These slices do not include the spinal canal, and the pedicle must be continuous between vertebral body and posterior elements unless the slice is lateral to the pedicle
ASSESSMENT SYSTEM	
	Vertebral body lesions (bone erosions, fat infiltration, bone spurs, and ankylosis) are assessed at each vertebral endplate at all 23 spinal levels from C2/3 to L5/S1 Facet joint lesions (erosion and ankylosis) are to be assessed by spinal segment — cervical, thoracic, and lumbar
A. Bone erosion	For each of the 46 vertebral endplates from C2/3 to L5/S1: <ul style="list-style-type: none"> • Vertebral body bone erosion in any slice: 0: no; 1: yes • Anterior corner bone erosion (aCOBE): 0: no; 1: yes, not large; 2: yes, large • Posterior corner bone erosion (pCOBE): 0: no; 1: yes, not large; 2: yes, large • Non-corner bone erosion (NOBE): 0: no; 1: yes • Central NOBE: 0: no; 1: yes, not large 2: yes, large. If yes, type of NOBE: Type A or Type B • Lateral NOBE: 0: no; 1: yes. If yes, type of NOBE: Type A or Type B For each of the 3 spinal segments (cervical, thoracic, and lumbar), the following should be noted: <ul style="list-style-type: none"> • Vertebral body bone erosion in any facet joint (FABE): 0: no; 1: yes
B. Fat infiltration	For each of the 46 vertebral endplates from C2/3 to L5/S1: <ul style="list-style-type: none"> • Corner fat infiltration in any central sagittal slice: 0: no; 1: yes • Anterior corner fat infiltration (aFAT): 0: no; 1: yes • Posterior corner fat infiltration (pFAT): 0: no; 1: yes
C. Bone spur	For each of the 46 vertebral endplates from C2/3 to L5/S1: <ul style="list-style-type: none"> • Vertebral body bone spur in any slice: 0: no; 1: yes • Anterior corner spur (aCOS): 0: no; 1: yes • Posterior corner spur (pCOS): 0: no; 1: yes • Non-corner spur (NOS): 0: no; 1: yes • Central NOS: 0: no; 1: yes • Lateral NOS: 0: no; 1: yes
D. Ankylosis	For each of the 46 vertebral endplates from C2/3 to L5/S1: <ul style="list-style-type: none"> • Vertebral body ankylosis in any slice: 0: no; 1: yes • Anterior corner ankylosis (aCANK): 0: no; 1: yes • Posterior corner ankylosis (pCANK): 0: no; 1: yes • Non-corner ankylosis (NANK): 0: no; 1: yes • Central NANK: 0: no; 1: yes • Lateral NANK: 0: no; 1: yes For each of the 3 spinal segments (cervical, thoracic, and lumbar): <ul style="list-style-type: none"> • Ankylosis in any facet joint (FANK): 0: no; 1: yes

not continuous between the vertebral body and posterior elements.

2. Lateral sagittal slices: The sagittal slices that are located lateral to the spinal canal. These slices do not include the spinal canal, and either the pedicle must be continuous between vertebral body and posterior elements or the slice is lateral to the pedicle.

In the cervical spine all slices through the vertebral body are “central,” because the pedicle is localized posterolaterally to the vertebral body.

Anatomical location of lesion. The structural lesions are divided into:

1. Vertebral body lesions of 4 types: (1) bone erosion, (2) focal fat infiltration in bone marrow, (3) bone spur, and (4) ankylosis. All are subdivided into vertebral corner lesions and non-corner lesions except for fat infiltration, which is assessed only at the corners.

2. Vertebral lesions not involving the vertebral body, of which there are 2 types: (1) facet joint bone erosion and (2) facet joint ankylosis. Fat infiltration and bone spurs are not assessed. Erosions and ankylosis are not assessed in posterior elements other than the facet joints.

Detailed definitions of structural lesions.

A. Bone erosion: Bone erosion is defined as full-thickness loss of the dark appearance of cortical bone and loss of normal bright appearance of adjacent bone marrow on T1w MR images. These are further subdivided by location, size, and morphology:

A1. Corner bone erosion (COBE) is defined as a bone erosion involving the vertebral corner, in at least one central sagittal slice, with an anterior COBE (aCOBE) being a COBE at the anterior corner (Figures 1, 2, and 4) and a posterior COBE (pCOBE) being a COBE at the posterior corner (Figures 3 and 4). aCOBE and pCOBE may be further categorized as “large” if a lesion extends to more than 25% of the anteroposterior (AP) diameter of the original endplate and/or of the original height of the vertebra, in any sagittal slice (Figures 1 and 7).

A2. Non-corner bone erosion (NOBE) is defined as a bone erosion adjacent to the vertebral endplate on any slice and not involving the vertebral corner on any central sagittal slice. A NOBE may be further categorized as “central” if it involves any central sagittal slice (Figures 1, 4, and 9) or as “lateral” if it involves any lateral sagittal slice (Figure 8). Note that the same NOBE can be both central and lateral if present in both central and lateral sagittal slices. A central NOBE may also be further categorized as “large,” if it involves more than 25% of AP diameter of the original endplate and more than 25% of the original height of the vertebra (Figure 1). Finally, a NOBE is further categorized as “Type A” or “Type B,” depending on whether the diameter of neck (at the cortical break) is less (Type A) or at least equal to (Type B) the maximal diameter of the loss of the high signal in the bone marrow (Figure 1).

A3. Facet joint bone erosion (FABE) is defined as a bone erosion adjacent to the facet joint (Figure 10).

B. Focal fat infiltration. Focal fat infiltration is defined as focal increased signal in bone marrow on T1w images. Only fat infiltration involving the vertebral corners on any central sagittal slice (corner fat infiltration) is assessed. By location these lesions are subdivided into anterior corner fat infiltration (aFAT), which is FAT at the anterior corner (Figures 5 and 9), and posterior corner fat infiltration (pFAT), which is FAT at the posterior corner (Figures 5 and 9).

C. Bone spur. Bone spur is defined as bright signal on T1w images extending from the vertebral endplate towards the adjacent vertebra. These are subdivided by location.

C1. Corner spur (COS) is a bone spur involving the vertebral corner, in at least one central sagittal slice, and can be either an anterior COS (aCOS; Figures 6 and 7) or a posterior COS (pCOS; Figure 7).

C2. Non-corner spur (NOS) is a bone spur involving the endplate on any slice, but neither the anterior nor the posterior vertebral corner on any central sagittal slice. A NOS may be further categorized as “central” if it involves any central sagittal slice (Figures 6 and 9) or as “lateral” if it involves any lateral sagittal slice (Figures 7 and 8). Note that the same NOS can be both central and lateral if present in both central and lateral sagittal slices.

D. Ankylosis. Ankylosis is defined as bright signal on T1w images extending from a vertebra and being continuous with the adjacent vertebra. Ankylosis is subdivided based on location.

D1. Corner ankylosis (CANK) is ankylosis involving the vertebral corner, in at least one central sagittal slice, with an anterior CANK (aCANK) being at the anterior corner (Figures 2, 5, 6, and 7) and a posterior CANK (pCANK) being at the posterior corner (Figure 7).

D2. Non-corner ankylosis (NANK) is ankylosis involving the endplate on any slice, but neither the anterior nor the posterior vertebral corner on any central sagittal slice. A NANK may be further categorized as “central” if it involves any central sagittal slice (Figure 9), or as “lateral” if it involves any lateral sagittal slice (Figures 3 and 8). Note that the same NANK can be both central and lateral, if present in both central and lateral sagittal slices.

D3. Facet joint ankylosis (FANK) is ankylosis of a facet joint. (Figure 8).

Assessment system

Vertebral body lesions (bone erosions, fat infiltration, bone spurs, and ankylosis) are assessed at each vertebral endplate at all 23 spinal levels from C2/3 to L5/S1. Facet joint lesions (erosions and ankylosis) are assessed by spinal segment – cervical, thoracic, and lumbar.

A. Bone erosions. For each of the 46 vertebral endplates from C2/3 to L5/S1, the following should be assessed: Vertebral body bone erosion in any slice, Anterior corner bone erosion (aCOBE) (including size), Posterior corner bone erosion (pCOBE) (including size), Non-corner bone erosion (NOBE), Central NOBE (including size and type), and Lateral NOBE (including type).

For each of the 3 spinal segments (cervical, thoracic, and lumbar), the following should be assessed: Bone erosion in any facet joint (FABE).

B. Fat infiltration. For each of the 46 vertebral endplates from C2/3 to L5/S1, the following should be assessed: Vertebral body corner fat infiltration in any central sagittal slice, Anterior corner fat infiltration (aFAT), and Posterior corner fat infiltration (pFAT).

C. Bone spur. For each of the 46 vertebral endplates from C2/3 to L5/S1, the following should be assessed: Vertebral body bone spur in any slice, Anterior corner spur (aCOS), Posterior corner spur (pCOS), Non-corner spur (NOS), Central NOS, and Lateral NOS.

D. Ankylosis. For each of the 23 discovertebral units C2/3 to L5/S1, the following should be assessed: Vertebral body ankylosis in any slice, Anterior corner ankylosis (aCANK), Posterior corner ankylosis (pCANK), Non-corner ankylosis (NANK), Central NANK, and Lateral NANK.

For each of the 3 spinal segments (cervical, thoracic, and lumbar), the following should be assessed: Ankylosis in any facet joint (FANK).

DISCUSSION

Our article presents an anatomy-based set of definitions and an assessment system for structural lesions in the spine of patients with SpA proposed by the Canada-Denmark MRI working group. In contrast to the previously described assessment system, the AS-spi-MRI-c system, the present (CanDen) system is designed to study the temporal and spatial pattern of bone erosion, fat infiltration, and new bone formation, as well as their relation to inflammatory lesions.

Systematic evaluation of structural changes such as bone erosions and new bone formation in the spine by MRI has been limited to the AS-spi-MRI-c scoring system, which scores sclerosis, squaring of vertebrae, syndesmophytes, and ankylosis according to each discovertebral unit. Unfortunately, reliability has been shown to be poor, and in a comparative study this MRI system was not superior to radiography for detection of new bone formation^{1,7,8}. However, no specific definitions for syndesmophytes and ankylosis seen on MRI were proposed, and it was not clear whether the poor reliability was due to unreliable detection of all or only some lesions since data were reported for the score as a whole only. The present CanDen system is therefore a novel and unexplored approach that would be expected to provide important new knowledge. Future studies are obviously needed to document this.

It should be emphasized that interpretation of MR images is frequently challenging, even for experienced readers. Subtle areas of signal alteration will be seen somewhere in most scans, and quite frequently the reader will not be confident that the change constitutes a true lesion (e.g., Figures 3 and 4). The interpretation of such borderline lesions may influence the overall assessment of the patient. In this reference image set we provide examples of such borderline lesions. We suggest that lesions more apparent than these should be considered pathological and should be scored. Findings less obvious than

these should not be scored as pathological. Whether the borderline lesion (at the threshold for detection) is scored or not will depend on several factors, including overall image quality, artefact in the immediate vicinity, observation of the same borderline lesion on multiple images, and reader experience. The reader of MR images should be aware of different causes of artefacts, and should become familiar with their appearances and how they may cause misinterpretation. Spine MRI scans are generally done with large fields of view resulting in variation in the strength of the signal reaching the receiver coils (coil artefact) as the thoracic kyphosis and lumbar lordosis cause variability in the distance of vertebrae from the antennae. Signal from blood flowing in the great vessels (aorta and inferior vena cava) can cause phase-encoding artefacts, and artefacts due to patient movement and breathing, incomplete fat suppression, or partial voluming effects are also commonly encountered.

The cervical spine is generally the most difficult spinal segment to assess for several reasons. In particular, the anatomical structures are much smaller, resulting in proportionally less spatial resolution than in the rest of the spine. The shape of the cervical posterior elements is very different from the thoracic and lumbar spine. In the cervical spine, the facet joints are situated posterolaterally to the intervertebral disc, which comprises less than half the overall diameter of the vertebra. Consequently, any "lateral slice" in the cervical spine is lateral to the vertebral body and therefore the distinction between central and lateral slices in the cervical spine is not meaningful. Consequently, the term lateral slice is only relevant in the thoracic and lumbar segments.

Fat infiltration is a feature uniquely detected by MRI, and areas of fat infiltration in the corners of vertebral bodies are frequent in SpA. However, the exact significance of such findings is not known, as no data are available on its sensitivity and specificity for SpA, or on the spatial or temporal relation with inflammation or with development of erosions or syndesmophytes. However, it is likely that fat infiltration represents a reparative phenomenon. In the CanDen system, we chose to restrict our assessment of fat infiltration to the anterior and posterior corners of the vertebral bodies, because these areas are easy to delimit, fat infiltrative lesions at these sites are quite distinct, and these areas provide optimal possibilities for exploration of the relationship to radiographic syndesmophytes, which are normally visualized at exactly these sites on the routinely acquired lateral radiographs.

New bone formation, such as bone spurs (syndesmophytes) and ankylosis, is generally accepted as an important longterm consequence of SpA/AS. A recent study demonstrated that syndesmophyte formation is related to previous MRI corner inflammatory lesions⁹. Further studies are needed to confirm and further explore this

relationship. Syndesmophytes are the main feature identified by the current standard for assessment of structural damage in the spine using lateral spine radiographs in the modified SASSS method⁴⁻⁶. However, this method identifies only anterior syndesmophytes, and only in the cervical and lumbar spine. The present MRI system assesses syndesmophytes throughout the disc space as well as the anterior and posterior aspects of adjacent vertebrae, and in all segments of the spine. MRI is not ideal for visualization of cortical bone, which appears as a black signal void indistinguishable from ligamentous structures. However, the tomographic perspective and the assessment at many different sites may be expected to provide a higher sensitivity than conventional radiographs for detection and monitoring of new bone formation in the spine, especially the thoracic spine. Further studies are needed to explore the validity and relative sensitivity to change of the proposed assessment system. The difficult differentiation between the cortical bone of a bone spur and adjacent ligaments is the reason we required the bright signal reflecting fat infiltration and/or visualizing cartilage metaplasia inside the spur in order to allow the process to be scored as a bone spur.

Erosions are also not always easy to identify. The main difficulty is to verify with certainty that a lesion has a clear break of the bone cortex. The dark appearance of an erosion is frequently not easily discernible from the appearance of the cortical bone at the vertebral corner. Focal bone sclerosis may also resemble an erosion because it appears as a dark area in contrast with the usual bright bone marrow. Sensitivity and specificity of erosions for SpA need to be assessed, as well as their relationship with inflammation and new bone formation.

It should be noted that this article does not claim that the described features are pathognomonic for SpA; rather, it provides a standardized approach to defining pathological features observed in the spine of patients

with SpA, whereas other studies, mainly longitudinal studies of patients with undifferentiated inflammatory back pain, are needed to clarify the diagnostic and prognostic value of spine MRI.

It could be debated whether the term “chronic lesions” or “structural lesions” or “structural damage lesions” are the most appropriate notation for erosions, fat infiltration, and spurs/ankylosis. They have all previously been used for such lesions (e.g., “chronic” lesions^{7,8}, “structural damage” lesions¹⁰). Chronic implies a protracted and sustained process, while only one set of images is usually available and does not permit conclusions as to how long the lesion may have been present or how long it will persist. “Structural damage” lesion may be more appropriate as the T1w MR images display the changes directly and provide information as to the extent of damage that may have occurred in the relevant anatomical area. Further, damage is what we want to monitor and predict. On the other hand, “structural damage” lesion does not adequately reflect the possibility that the lesion instead reflects repair. A recent Assessment of SpondyloArthritis International Society (ASAS) article used the term structural damage lesion for such changes in the sacroiliac joints of SpA patients¹⁰. For the reasons described above we feel that either term can be used, but we have chosen to use “structural lesion” in this article because we cannot be certain about the chronicity of the lesion and whether it reflects damage or a repair response.

In conclusion, an anatomy-based set of definitions and an assessment system for structural lesions in the spine of patients with SpA have been developed and illustrated. The system is designed to study the spatial patterns of spine lesions and their relation to the development of structural change. Further studies are urgently needed to elucidate the validity of the system and its usefulness for study of the disease course in SpA and as markers of disease progression.

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Figure 1. Corner bone erosion (COBE) and non-corner bone erosion (NOBE). An aCOBE is present at the L1 inferior endplate on the T1 image. The STIR image shows increased signal indicating active inflammation at this corner [anterior corner inflammatory lesion (aCIL)]. There is a large central Type A NOBE at the L2 inferior endplate [on the STIR image a non-corner inflammatory lesion (NIL) with a dimorphic appearance is seen]. A conspicuous area of marrow fat signal loss at the L3 inferior endplate is easily seen on the T1 image. However, at this level, the large central Type A NOBE is in fact at the threshold of detection because most of the cortex is intact on the T1w image. However, there is just enough

cortical destruction for this to be positive. A rim of active inflammation is identified on STIR (NIL, with dimorphic appearance). A large central Type B NOBE at the L5 superior endplate is also considered to be at the threshold of detection but for a quite different reason: although there is clear evidence of cortical destruction, the lesion is relatively subtle on the T1 image as bone marrow signal is only slightly reduced, whereas associated active inflammation on the STIR image is easily seen. Note that the apparent presence of a cortex on the STIR image is in part related to intact hyaline cartilage of the endplate rather than intact cortical bone. At the L5 inferior endplate, there is a large central Type B NOBE.

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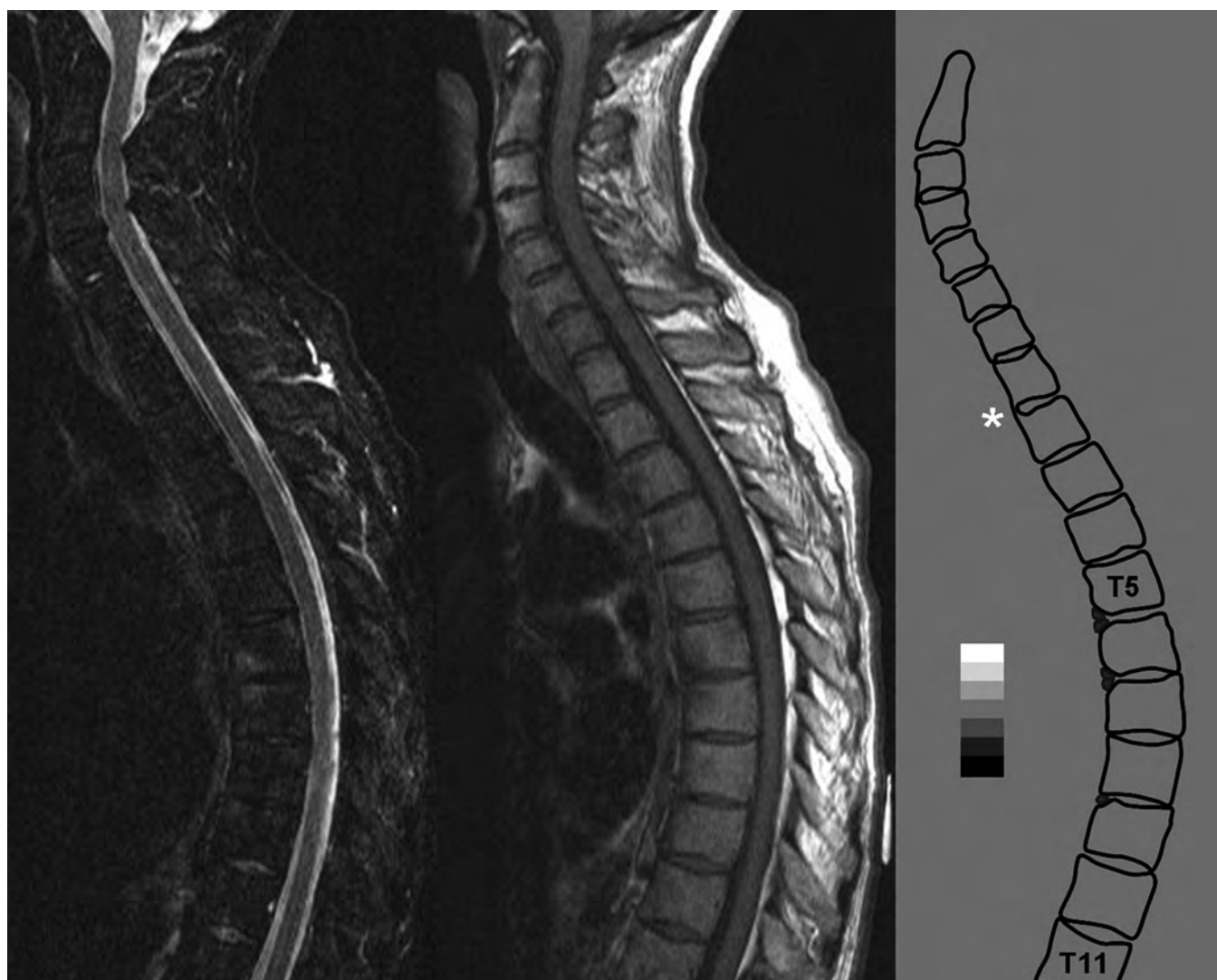


Figure 2. Corner bone erosion (COBE) and corner ankylosis (CANK). Scanning at large fields of view presents particular challenges. The smaller structures and suboptimal spatial resolution are sometimes compounded by limited image quality. In this MRI of the cervical and thoracic spine, subtle anterior corner bone erosions (aCOBE) are present at the threshold for detection at

T5/6, T6/7, and T9 superior. Possible lesions at other levels are below the detection threshold except for ankylosis anteriorly (aCANK) at T1/2. Note how the anterior corners at T6/7 are bright on the STIR sequence (i.e., they are “active”), while the erosions at T5/6 are normal on STIR.



Figure 3. Corner bone erosion (COBE) and non-corner ankylosis (NANK). A posterior corner bone erosion (pCOBE) is present at the L5 superior endplate. However, some chronic lesions are very difficult to ascertain and classify. In this case, 3 areas of abnormality are seen posteriorly at the corners of the T11 inferior, T12 superior, and L3 superior endplates. The cortices appear to be intact or at least not clearly eroded. The bone marrow signal is reduced, likely due to fibrosis and/or sclerosis, and these may be the MRI equivalent of the well known radiographic sign, “the shiny corner.” Unfortunately, it is

rarely possible to be certain that this truly represents sclerosis, and bridging compact bone cannot be distinguished from the normal fibrous tissue of the annulus fibrosus or longitudinal ligament. Some corner fat can be seen, but subtle bony prominence at some corners is below the threshold for a corner spur, except at L4 superiorly where a tiny spur is present at the posterior corner. Posterior ankylosis at T10/11 is present on a lateral slice at this level (lateral NANK) and is easier to see because of the bright fat signal in the bridging spur.



Figure 4. Corner (COBE) and non-corner bone erosion (NOBE). Small erosions are difficult to discern in many instances. In this case non-corner bone erosions (NOBE) are seen on either side of the L3/4 intervertebral disc. However, subtle deformities of the anterior corners of these 2 endplates are consistent with tiny corner bone erosions (aCOBE) at the threshold for detection. The difficulty for the reader is 2-fold: (1) Does the subtle defor-

mity of the corner represent anatomical variation or erosion of the original corner with sclerosis at the edge of the erosion? (2) If erosion is present, is it continuous with the NOBEs? A more obvious erosion is present posteriorly at the L4 inferior endplate (pCOBE). Slight irregularities of other vertebral corners do not constitute detectable lesions.



Figure 5. Focal fat infiltration at vertebral corners (aFAT, pFAT) and vertebral corner ankylosis (CANK). Multiple foci of fatty infiltration of bone marrow are present at many vertebral body corners, anteriorly and posteriorly. The aFAT lesion at the L1 inferior endplate and the pFAT lesion at the L3 inferior endplate are at the threshold of detection. But faint increased signal at the anterior

corners of the L2 and L3 superior endplates are below threshold for detection. At T11/12 there is subtle anterior ankylosis (aCANK) with bright signal in bridging bony spurs at the threshold for detection. Normal irregularity of vertebral endplates with normal bone marrow does not meet the definition of a NOBE at any level.



Figure 6. Vertebral corner spur (COS), non-corner spur (NOS), and vertebral corner ankylosis (CANK). Multiple bony spurs are present with varying configuration. At L1/2 there is anterior corner ankylosis (aCANK) at the threshold of detection. At L2 inferior, there is anterior bony irregularity that is below the threshold for detection of a spur. At L3 superior there is an anterior corner spur (aCOS) that is large and easily seen. At L3 inferior there

is anterior bony irregularity that is below the threshold for detection of a spur. At L4 superior, there is an anterior corner spur (aCOS) at the threshold of detection. At L4 inferior there is a large central non-corner spur (NOS). At L5 superior there is a large central non-corner spur (NOS), which is discontinuous with an anterior corner spur (aCOS) at the threshold of detection.

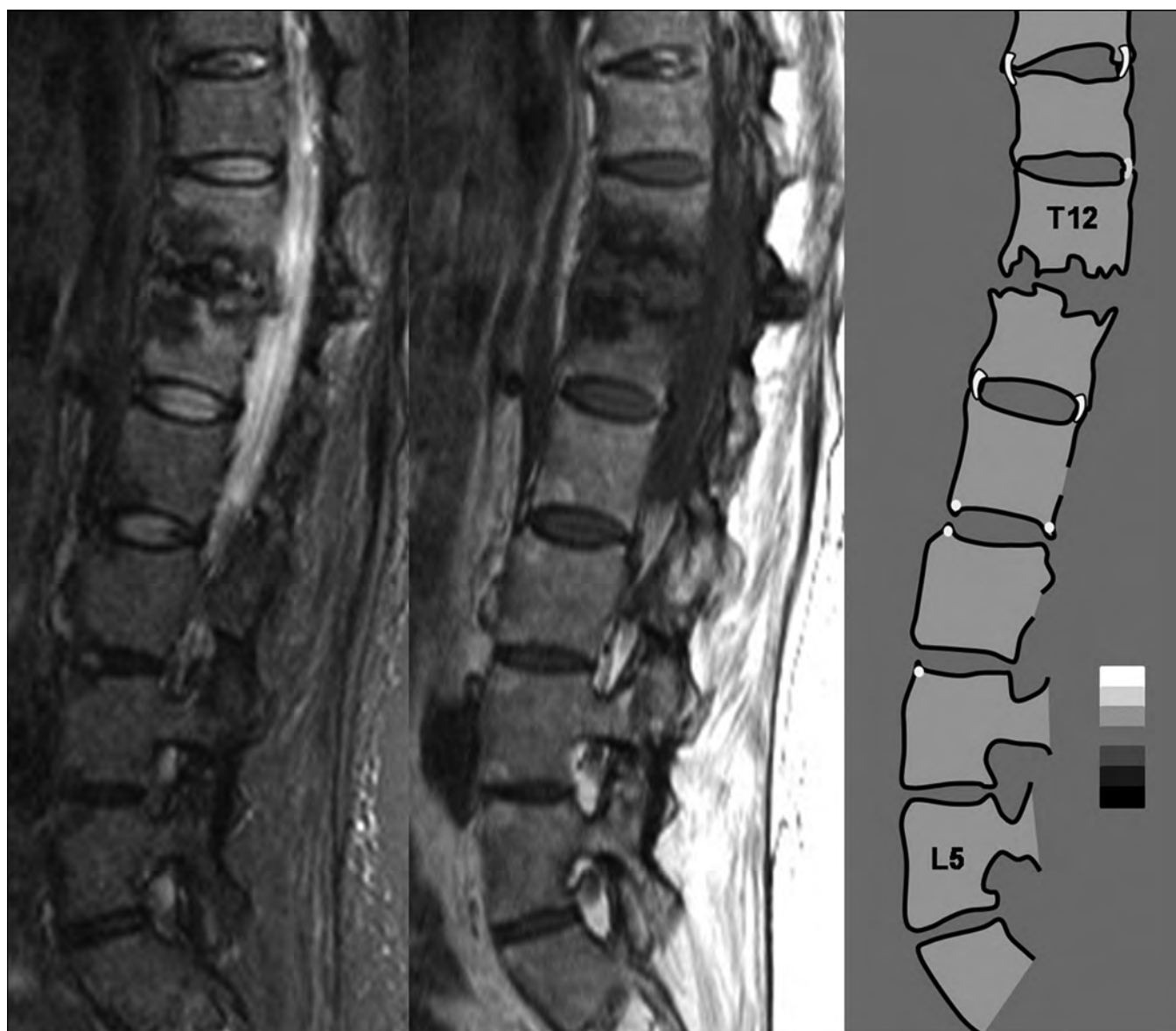


Figure 7. Corner bone erosions (COBE), ankylosis (CANK), and spurs (COS and NOS). There is extensive bone erosion at T12/L1 that involves the entirety of the adjacent endplates including all 4 vertebral corners. These lesions therefore all qualify as corner bone erosions (COBE) and constitute 2 aCOBE and 2 pCOBE. There is ankylosis anteriorly and posteriorly at T10/11 and L1/2 (aCANK and pCANK). At T11/12 there is ankylosis posteriorly (pCANK). Although ankylosis may be present anteriorly as well, this is below the threshold for detection

on this image. Down to L3, the MR image is a “central slice.” However, because of mild scoliosis, L4 and below is a “lateral slice.” Both anterior and posterior corner spurs are present at L2 inferior (aCOS and pCOS), and there is an anterior corner spur at L3 superior. A tiny bony spur is present anteriorly at the L4 level superior on this lateral slice (since the pedicle is included) at the L4 level. This lesion is therefore a “lateral NOS” at the threshold of detection.



Figure 8. Lateral non-corner spur (NOS) and ankylosis (NANK) and facet joint ankylosis (FANK). A lateral slice of the thoracic spine demonstrates extensive ankylosis at all levels. Ankylosis is most pronounced in the facet joints (FANK) and in costovertebral joints posterolaterally in the vertebral body (lateral NANK). Subtle spurs and/or ankylosis can also be seen at some levels anteriorly

classified as non-corner spur (lateral NOS) and non-corner ankylosis (lateral NANK), respectively. Despite complete ankylosis of the spine (confirmed radiographically), the patient has active spondylodiscitis at T11/12 with a non-corner bone erosion (lateral NOBE) in the T11 inferior endplate and a “massive inflammatory lesion” (MIL).



Figure 9. Central non-corner ankylosis (NANK), spur (NOS), and erosion (NOBE), and focal fat infiltration (aFAT and pFAT). Ankylosis in the thoracic spine is visualized at only one level. Complete ankylosis has developed across the T7/8 intervertebral disc. This is a central non-corner ankylosis (NANK). Erosion of the vertebral endplates is also present at other levels without involvement of the vertebral body corners (non-corner bone erosion; NOBE). Some of these are at the threshold

of detection, and a tiny irregularity at the superior endplate of T10 is below the threshold. A non-corner spur (NOS) is seen extending superiorly from the T11 superior endplate incorporating continuity of bone marrow signal with the vertebral body. There is also extensive fatty infiltration at multiple levels extending to the anterior and posterior corners (aFAT and pFAT), prominent at T9, T10, T11 and T12.

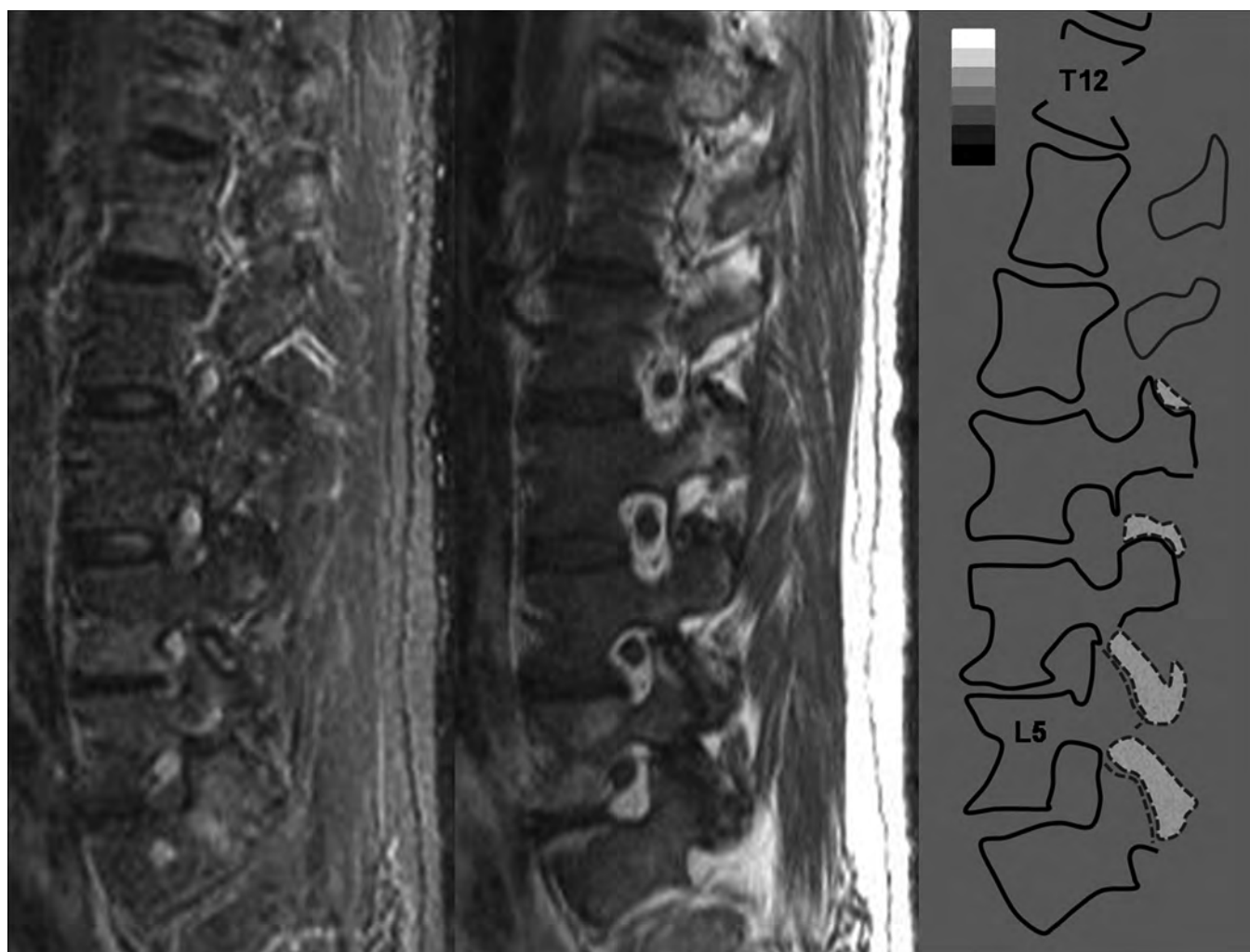


Figure 10. Facet bone erosion (FABE). The facet processes of the lumbosacral spine are markedly irregular in contour and signal intensity related to extensive erosion of the articular surfaces and some fatty infiltration of bone marrow.