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REVIEW ARTICLE

Review of the updated definitions and concepts of spinal lesions in axial spondyloarthritis

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Abstract

Spinal imaging may support the diagnosis of axial spondyloarthritis when typical findings are recognized in an appropriate clinical context and it can also indicate disease activity. In May 2022, the definitions for inflammatory and structural spinal lesions in axial spondyloarthritis were updated and validated by the Assessment of SpondyloArthritis international Society (ASAS) magnetic resonance imaging (MRI) working group. The aims of this paper are to demonstrate and describe imaging findings of the spine in patients with axial SpA, including the latest updated definitions by the ASAS, and to show complications in patients with long-standing disease.

Keywords Axial spondyloarthritis · Ankylosing spondylitis · Spinal lesions

Abbreviations

AS	Ankylosing spondylitis
ASAS	Assessment of SpondyloArthritis international Society
CES	Cauda equina syndrome
CT	Computed tomography
T2FS	Fat-suppressed T2-weighted
MRI	Magnetic resonance imaging
SIJ	Sacroiliac joints
STIR	Short tau inversion recovery
SpA	Spondyloarthritis

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Introduction

The spondyloarthritis (SpA) family is a group of chronic inflammatory diseases which share some common features, such as enthesitis and strong association with HLA-B27 gene. SpA is classified as axial or peripheral according to the predominance of clinical manifestations. Ankylosing spondylitis (AS) is the prototype of axial disease affecting predominantly the spine, sacroiliac joints (SIJ), and thoracic cage. Peripheral SpA, such as psoriatic arthritis, reactive arthritis, undifferentiated spondyloarthropathy, and inflammatory bowel-disease-associated arthritis, mainly affects the lower limbs [1–3].

AS is the major subtype of SpA. The disease typically affects people under 45 years old, with men being nearly twice as likely to be affected as women, with an estimated prevalence between 0.1 and 1.4%. Clinical features are back pain, spinal stiffness, and loss of spinal mobility, which are explained by spinal inflammation and structural damage [2, 4]. Chronic spinal inflammation contributes to bone loss and development of enthesophytes, syndesmophytes, and ankylosis. This can lead to a combination of spinal rigidity and reduced bone density, which increases the susceptibility to vertebral fracture [4, 5].

The most involved anatomical structures in the inflammatory process of SpA are the entheses, which are the sites where tendons, ligaments, and joint capsules attach to bone [6]. Fibrocartilaginous entheses are typically affected in SpA and are characterized by having a transitional tissue, which

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is the fibrocartilage close to the bone insertion [6]. The most affected fibrocartilage enthesis in the vertebral spine is the insertion of the peripheral fibers of the annulus fibrosus in the corner of the vertebral body. Other entheses and joints commonly involved are the anterior and posterior longitudinal ligaments, interspinous ligaments, supraspinous ligament, *ligamenta flava*, capsuloligamentous attachments of the facet joints, facet joints, and costovertebral and costotransverse joints [6–8].

The inflammation of the enthesis leads to vasodilation and migration of inflammatory cells, which determines bone erosion adjacent to the enthesis insertion sites. The inflammatory reaction induces osteoblast differentiation and enables new bone apposition. Therefore, in the healing process of erosion, enthesophytes or syndesmophytes are formed. Inflammatory lesions are characterized by bone edema, which can be best assessed by magnetic resonance imaging (MRI). Radiography and conventional computed tomography do not depict active lesions, and better demonstrate structural lesions, such as sclerosis, erosions, syndesmophytes, and ankylosis [9, 10].

The aims of this paper are to demonstrate and describe imaging findings of the spine in patients with axial SpA, including the latest updated definitions by the Assessment of SpondyloArthritis international Society (ASAS), and to show complications in patients with long-standing disease [11].

Classification of spinal lesions and imaging methods

Both SIJ and spine imaging are important for diagnosing and monitoring SpA, but only SIJ imaging is included in the ASAS criteria for the classification of axial spondyloarthritis. One main reason for this is that spinal changes, detectable by MRI or radiography, typically occur later in the disease course. SIJ involvement is seen in over 95% of patients with early spondyloarthritis, while fewer than 5% present with inflammation limited to the spine. Furthermore, characteristic SpA lesions on MRI are frequently present in degenerative spinal disease as well, increasing the risk of misdiagnosis, especially in older individuals [11–14].

Although the ASAS does not value spinal findings in the classification criteria, literature data demonstrate that some AS patients with clinically active disease may only have spinal inflammation and no evidence of active sacroiliitis [13, 15–17]. Marzo-Ortega et al. evaluated MRI scans of the SIJ and lumbar spine in patients with early inflammatory back pain. Their study demonstrated that inflammation can occur simultaneously in both the SIJ and lumbar spine in one-third of cases, with some patients showing spinal inflammation without sacroiliitis. This suggests that limiting imaging to a single region may result in missed diagnoses [18].

McEwen et al. concluded that ankylosing spondylitis/enteropathic spondylitis presents with early, progressive, symmetrical sacroiliac involvement and extends progressively from the lumbar into the dorsal and finally the cervical spine. In contrast, psoriatic spondylitis/reactive spondylitis demonstrated frequent asymmetrical and occasionally unilateral sacroiliitis progressing in random fashion throughout the spine [19].

Vertebral alterations are classified as active and structural lesions. Active lesions are characterized by bone marrow edema or osteitis (which are equivalent terms according to the ASAS) in vertebral corners, aseptic spondylodiscitis, arthritis, and enthesitis. Structural lesions include fat lesions, bone erosions, bone sclerosis, syndesmophytes, and ankylosis [11].

MRI is the most sensitive imaging method for detecting active lesions. MRI sequences which are useful in the inflammatory phase are fluid-sensitive sequences such as fat-suppressed T2-weighted (T2FS) and short T1 inversion recovery (STIR). STIR has the advantages of being less sensitive to magnetic field inhomogeneities and high capacity to demonstrate edema; however, it has lower signal-to-noise ratio, which results in a slightly lower image quality. Alternatively, fat-suppressed gadolinium-enhanced T1-weighted can assess increased perfusion at the site of edema; nonetheless, the contrast agent does not usually provide more information in addition to T2FS or STIR [4, 8, 20]. Both the European Alliance of Associations for Rheumatology (EULAR) and ASAS do not recommend the routine use of gadolinium-based contrast media to assess disease activity in SpA [3, 21].

Structural lesions can be detected with conventional radiography, computed tomography (CT), and MRI. Conventional radiography has low sensitivity to detect early inflammatory findings, but may be useful to follow structural disease progression, particularly new bone formation. CT has better spatial resolution to assess structural lesions; however, the technique should not be used routinely because of a high exposure to radiation. In the spine, CT is useful in the diagnosis of complications of late disease such as spondylodiscitis or spinal fracture. Regarding MRI, T1-weighted sequences without fat suppression can demonstrate areas of bone erosion, bone marrow fatty degeneration, new bone formation, and ankylosis [11, 20, 21].

According to the ASAS update in spinal MRI, images in the sagittal orientation are used for all definitions of inflammatory and structural lesions. Thus, spine MRI field of view should be extended to include posterolateral structures. The central sagittal slices correspond to the spinal canal region; the pedicles may be partially seen. The lateral sagittal slices are located lateral to the spinal canal, either

including images in which the pedicles are continuous with the vertebral body and posterior elements, or images lateral to the pedicles. Although the ASAS definitions emphasize the use of sagittal images for defining SpA lesions, radiologists are not limited to this plane alone. Axial and coronal images, which are also acquired during MRI exams, can further enhance the categorization and interpretation of findings, depending on the clinical context and specifics of each case. For example, these additional planes can be particularly useful for a detailed assessment of the extreme lateral edges of the vertebral body and/or the costovertebral joint [11, 22, 23].

In the most recent ASAS update, active inflammatory lesions in the spine are categorized as those affecting vertebral bodies and those that do not. Vertebral body inflammatory lesions encompass vertebral corner inflammatory lesion (anterior/posterior spondylitis), vertebral endplate inflammatory lesion (aseptic spondylodiscitis), and thoracic lateral inflammatory lesion (arthritis of the costovertebral joints). Inflammatory lesions that do not involve vertebral bodies are facet joint inflammatory lesion (facet joint arthritis) and posterior element inflammatory lesion (including enthesitis of spinal ligaments and costotransverse joint arthritis) [11].

According to the ASAS, structural lesions are defined by fat lesions, erosions, sclerosis, syndesmophytes, and ankylosis. Those lesions can be accompanied by bone marrow edema [8, 11].

The following lesions must be visualized in at least two continuous sagittal slices on MRI to represent a valuable finding: vertebral corner inflammatory lesion, vertebral corner fat lesion, and vertebral endplate inflammatory lesion. The other lesions defined by the ASAS MRI working group can be identified in a single sagittal slice [11].

Definitions of various spinal lesions outlined by the ASAS MRI working group were also described by the Canada–Denmark MRI working group. The Canada–Denmark MRI working group detailed and illustrated definitions of both active inflammatory lesions and structural lesions in the spines of patients with SpA. Their definitions and some of their illustrations were incorporated into the ASAS update on spinal MRI published in 2022 [3, 11, 23–26].

Spinal imaging findings in SpA

Vertebral corner inflammatory lesion (anterior/posterior spondylitis)

Anterior/posterior spondylitis occurs at the site of attachment of the annulus fibrosus to the vertebral endplate and represents one of the earliest findings in patients with SpA [27]. The presence of anterior/posterior spondylitis in three or more sites is highly suggestive of axial SpA, especially in patients under the age of 50 [14].

On MRI, vertebral corner inflammatory lesions are small, sharply marginated, and triangular-shaped (Fig. 1). Degenerative corner lesions, on the other hand, are usually associated with disk degeneration and present a semicircular shape. The MRI corner sign is more commonly observed in the thoracolumbar junction, whereas for degenerative corner

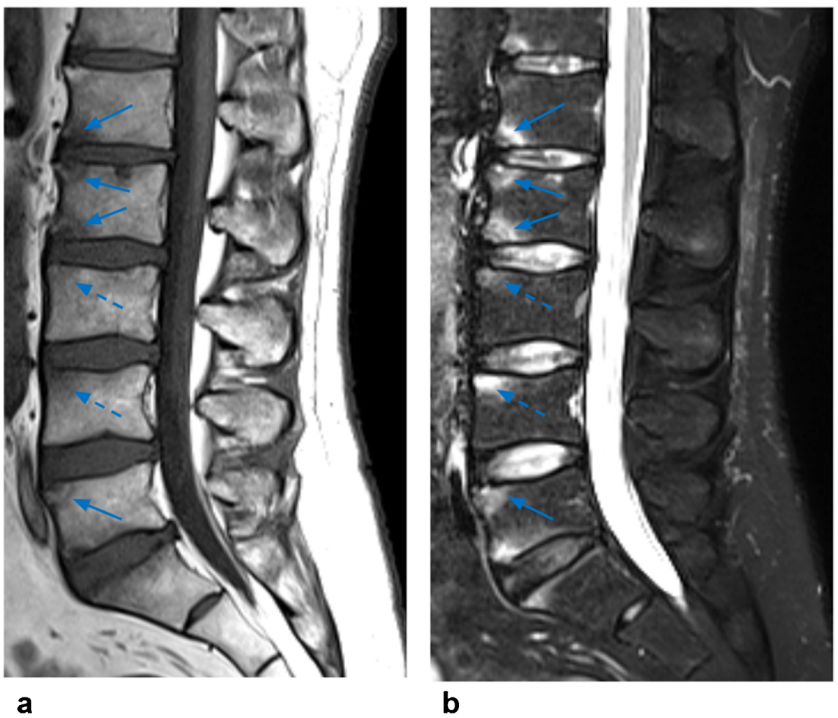

Fig. 1 A 45-year-old man with ankylosing spondylitis. **a**, **b** Sagittal T1-weighted MR image (**a**) and STIR MR image (**b**) show several monomorphic (dashed arrows) and dimorphic (arrows) inflammatory lesions located at the vertebral corners. Note that the lesions are small, sharply marginated and triangular-shaped, exhibiting low signal intensity on sagittal T1-weighted MR image (**a**) and high signal intensity on STIR MR image (**b**)

lesions, the sign is more frequently found at lower lumbar levels [28].

The updated ASAS definitions for axial SpA spinal lesions subdivide inflammatory activity at the vertebral corner into two categories: monomorphic and dimorphic. In the monomorphic category, bone edema extends to the vertebral corner. In the dimorphic category, edema does not cover the whole vertebral corner, as there may be bone erosion, sclerosis, or fatty degeneration at the corner itself [11].

It is worth noting that MRI vertebral corner inflammatory lesions are associated with radiographic progression in AS [29–31].

Vertebral corner fat lesions

Some of the earliest signs of structural lesions are bone marrow fatty lesions, which are best depicted by MRI T1-weighted images [32] (Fig. 2). Vertebral corner fat lesions are highly specific to SpA and have a significantly higher likelihood ratio for axial SpA, especially if more than five are present [33].

One common pathophysiological theory for AS states that the initial vertebral corner inflammation is followed by fatty replacement and, subsequently, by sclerotic bone formation. It is believed that it takes between 6 months and 2 years for an inflammatory lesion to become a fat lesion. Furthermore, literature has demonstrated that fat infiltration in vertebral corners increases the risk of subsequent radiographic syndesmophyte formation [29, 33–35]. It has been suggested that new syndesmophytes developed significantly more frequently in vertebral corners where inflammation had resolved than in those where inflammation persisted after anti-TNF treatment [14, 27, 28].

Bone erosion and Romanus lesion

Typical erosions are situated at the attachment of the annulus fibrosus to the vertebral endplate [20, 36]. The concept of erosions was updated by the ASAS and is now defined as full-thickness loss of the dark appearance of cortical bone and loss of the normal bright appearance of adjacent bone marrow on T1-weighted images (Fig. 3). Although they may occur in different locations in the spine for patients with SpA, only vertebral corner erosions are considered disease-specific [11].

The MRI corner erosions are radiographically equivalent to a Romanus lesion, which was first identified and described in studies based on conventional radiography; therefore, this term should not be transposed to MRI [28, 37] (Fig. 4).

Sclerosis/shiny corners

Frequently, in response to corner erosion, a focal area of bone sclerosis appears at the edges of the vertebral

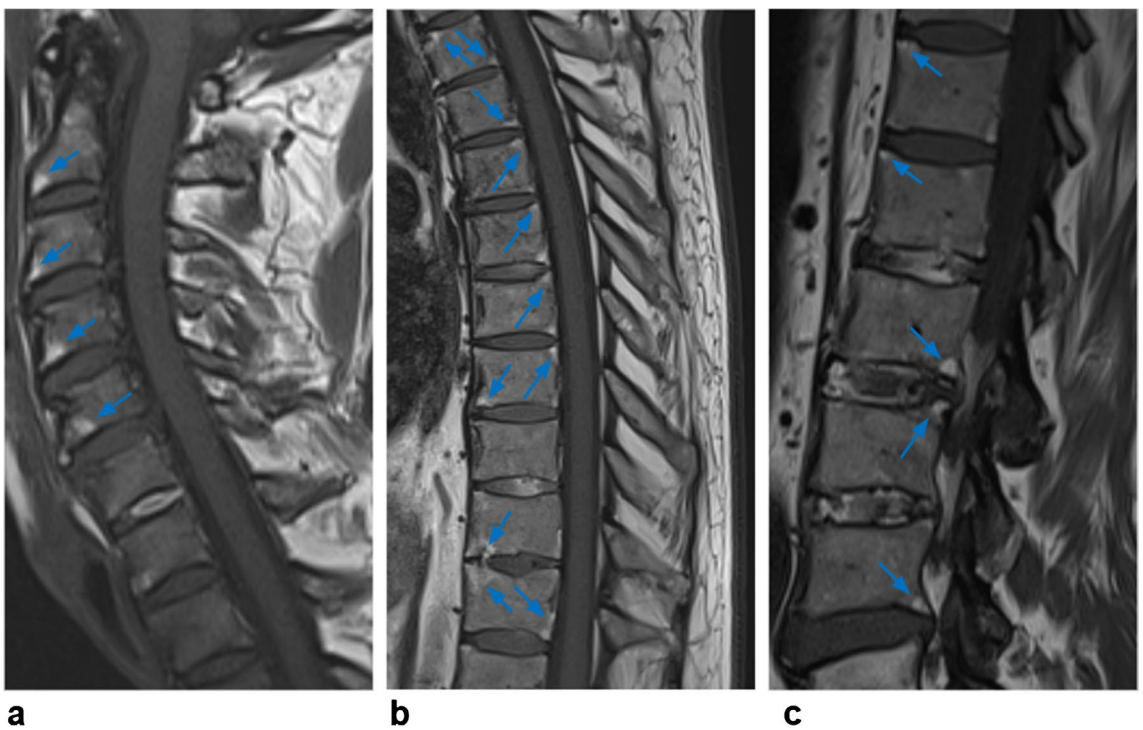

Fig. 2 A 60-year-old man with ankylosing spondylitis. **a**, **b**, **c** Sagittal T1-weighted MR images of the cervical (**a**), thoracic (**b**), and lumbar (**c**) spines reveal high signal intensity (arrows) in multiple vertebral corners, consistent with corner fat lesions

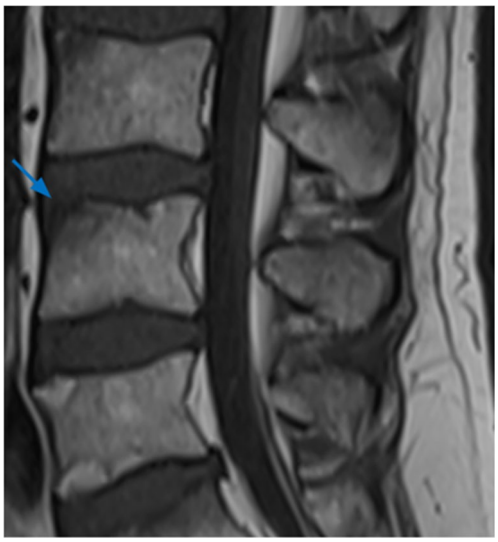

Fig. 3 A 45-year-old man with lumbar pain. Sagittal T1-weighted MR image shows full-thickness loss of the dark appearance of the cortical bone and low signal of the adjacent bone marrow (arrow), representing bone erosion

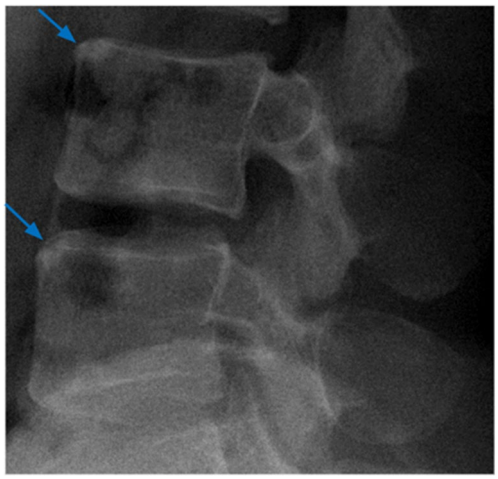

Fig. 4 A 41-year-old with psoriatic arthritis. Lateral radiograph of the lumbar region demonstrates Romanus lesions, appearing as bone erosions involving the anterior edges of the vertebral endplates with reactive sclerosis ("shiny corners")

endplates, known as shiny corners (Fig. 4). This is another descriptor whose use should be restricted to conventional radiography as it was first described by Romanus in that imaging modality [27, 28, 37].

On MRI, sclerosis is defined as low signal at a vertebral corner on all MRI sequences and is not specific for axial SpA [11].

Syndesmophytes

Non-bridging syndesmophytes are thin vertically oriented projections of bone which develop due to ossification within the outer fibers of the annulus fibrosus and do not reach another vertebra [8, 20] (Fig. 5 A and B).

It has been shown that new syndesmophytes can develop after vertebral corner inflammatory lesions or fat lesions, but it can also originate, although less commonly, in vertebrae that appear normal on prior MRI and conventional radiography [31]. However, this does not necessarily mean that there is no inflammation at histological examination as MRI may not be sensitive enough to detect a small amount of edema [38].

Classic marginal syndesmophytes are the predominant form in all types of SpA. However, patients with psoriatic arthritis and reactive arthritis tend to have a larger number of coarse nonmarginal syndesmophytes and fewer classic syndesmophytes than patients with AS [39, 40]. Some authors refer to this bone proliferation as parasyndesmophytes or paravertebral ossification, characterized by having a larger volume, not exactly following the course of the anterior longitudinal ligament (showing the so-called "paramarginal" localization), and frequently appearing in non-consecutive vertebrae (non-contiguous). This type of bone formation may result in less movement restriction in psoriatic arthritis compared to ankylosing spondylitis [19, 41–44].

The 2022 ASAS consensus on MRI spinal lesions suggests that bone spurs should not be scored as related to SpA (syndesmophytes) in the presence of disc degeneration, because distinguishing degenerative osteophytes from spondylitic syndesmophytes can be difficult [11, 39]. Degenerative bony spurring arises several millimeters away from the discovertebral junction, is typically triangular in shape, and has a horizontally oriented segment at the point of origin [20].

Vertebral corner ankylosis (bridging syndesmophytes)

Progressive growth of syndesmophytes will bridge the intervertebral disk causing ankylosis (Fig. 5C and D; Fig. 6) and extensive bone formation produces a smooth spinal contour: the bamboo spine (Fig. 7A) [20].

Squared vertebrae

Vertebrae may appear straight or squared due to new bone filling in the anterior vertebral body normal concavity, to which the anterior longitudinal ligament is attached, or even as a consequence of progression of erosions at the anterosuperior and anteroinferior vertebral margins [20, 36, 45, 46] (Fig. 7B).

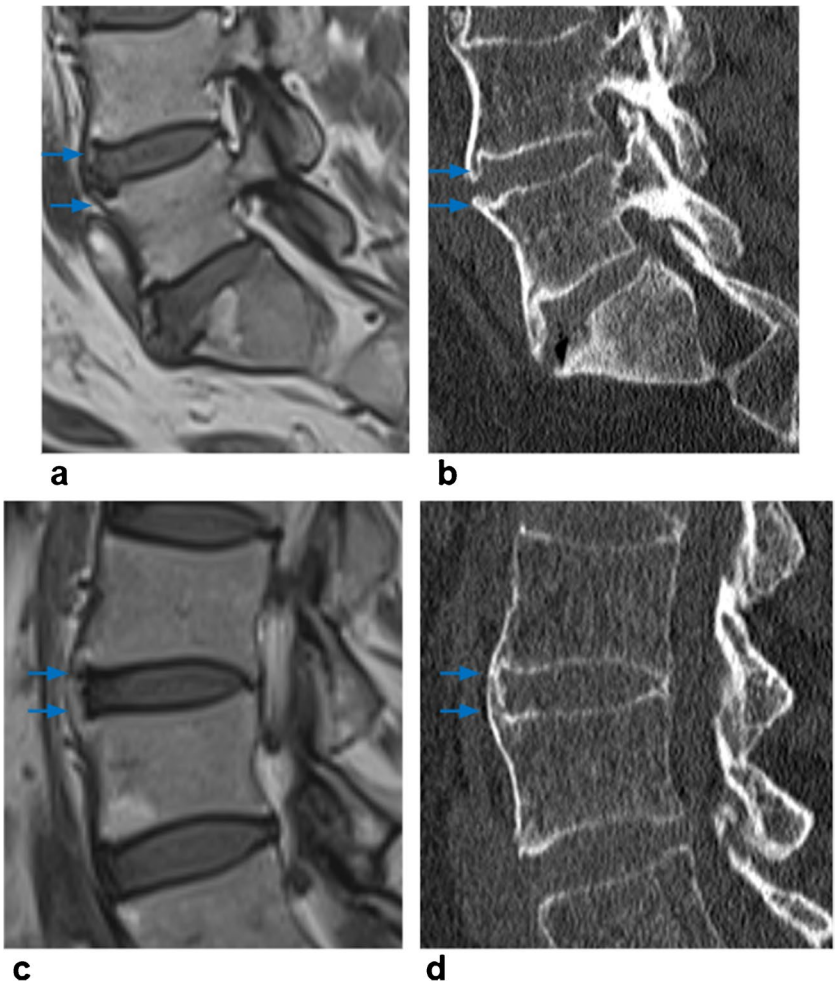

Fig. 5 A 40-year-old with ankylosing spondylitis. **a**, **b** Sagittal T1-weighted MR image (**a**) of the lumbar spine demonstrates thin vertically oriented projections of bone (arrows), consistent with syndesmophytes, also demonstrated on sagittal CT image (**b**). **c**, **d** Sagittal T1-weighted MR image (**c**) of the lumbar spine demonstrates bridging syndesmophytes (arrows), also demonstrated on sagittal CT image (**d**)

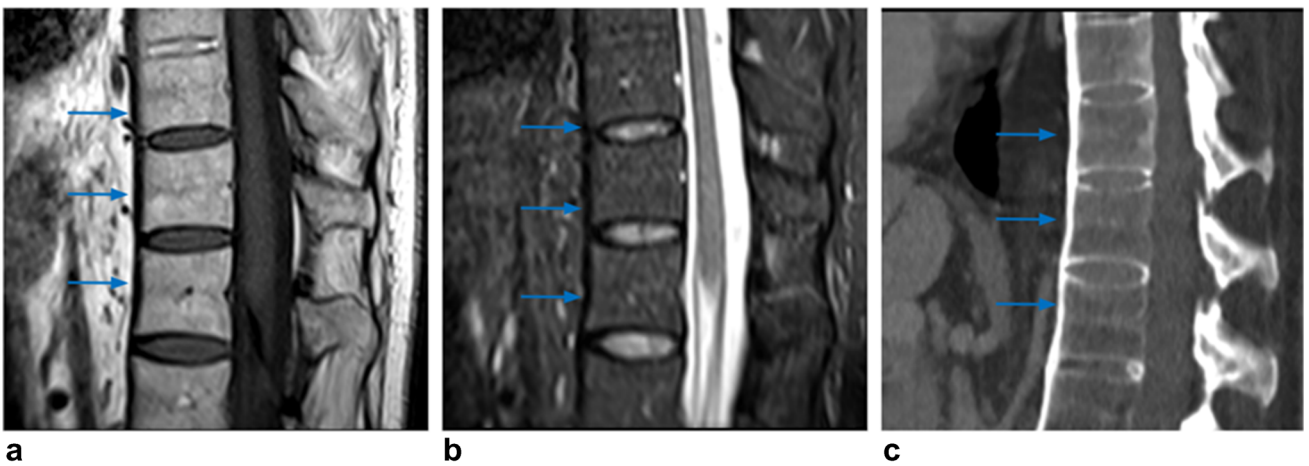

Fig. 6 A 57-year-old man with ankylosing spondylitis and spinal ankylosis. **a**, **b**, **c** Sagittal T1-weighted (**a**) and STIR (**b**) MR images of the thoracolumbar spine show bridging syndesmophytes (arrows), which are often difficult to distinguish from a slightly thickened anterior longitudinal ligament. In this case, the patient also had a CT scan, where the ankylosis is clearly visible on the sagittal CT image (**c**)

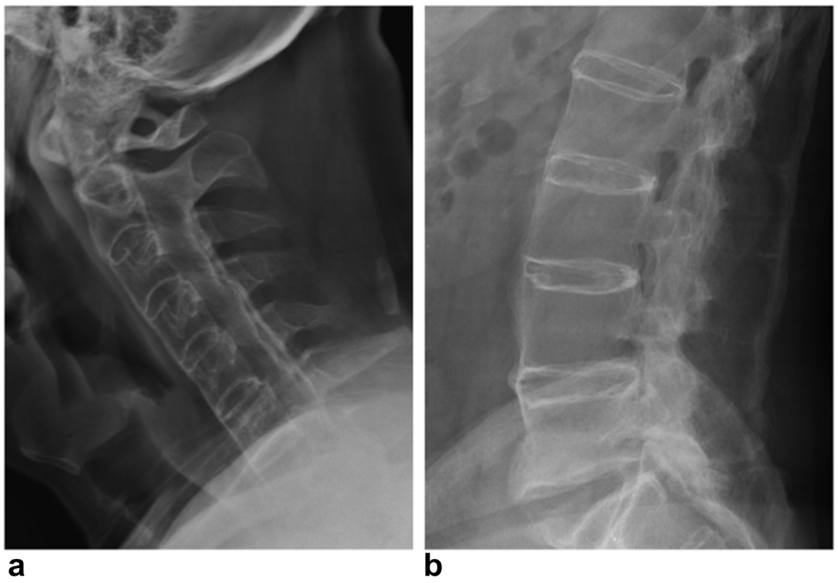

Fig. 7 A 52-year-old man with ankylosing spondylitis. **a**, **b** Lateral radiographs show bamboo spine appearance (**a**) and a squared vertebrae at L2, L3, and L4 (**b**)

Vertebral endplate inflammatory lesion (aseptic spondylodiscitis)

Inflammatory involvement of the intervertebral disks by SpA is known as aseptic spondylodiscitis or inflammatory Andersson lesion. They are noninfectious lesions that occur on vertebral endplates and do not extend to vertebral corners [11, 27].

On MRI these lesions occur in the bone marrow adjacent to the vertebral endplate and may affect one or both vertebral halves of a discovertebral unit. They appear hyperintense on T2FS or STIR images and hypointense on T1-weighted images, where they are often hemispherically shaped and may be accompanied by bone erosions of the vertebral endplate [8, 27, 47] (Fig. 8).

Vertebral endplate ankylosis (transdiscal ankylosis)

This lesion is defined as bony fusion across intervertebral disks, but not vertebral corners, with bridging of vertebral body cortical endplates. On MRI, new bone formations are isointense to normal bone marrow, with bright signal on T1-weighted images, crossing vertebral joint spaces through intervertebral disks, with loss of cortical endplates [8, 11, 47, 48] (Fig. 9).

Lallo et al. suggested that transdiscal ankylosis has potential as a specific and reliable sign of axial SpA, based on

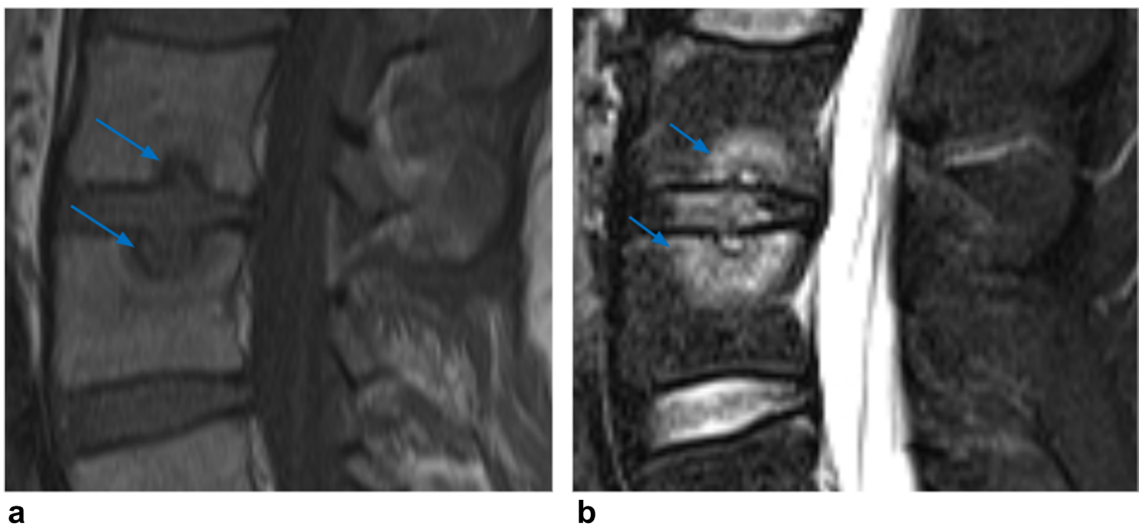

Fig. 8 A 41-year-old man with psoriatic arthritis. **a**, **b** Sagittal T1-weighted (**a**) and STIR (**b**) MR images of the lumbar spine demonstrate bone erosions (arrows) with adjacent bone marrow edema (short arrows) on vertebral endplates related to inflammatory lesions

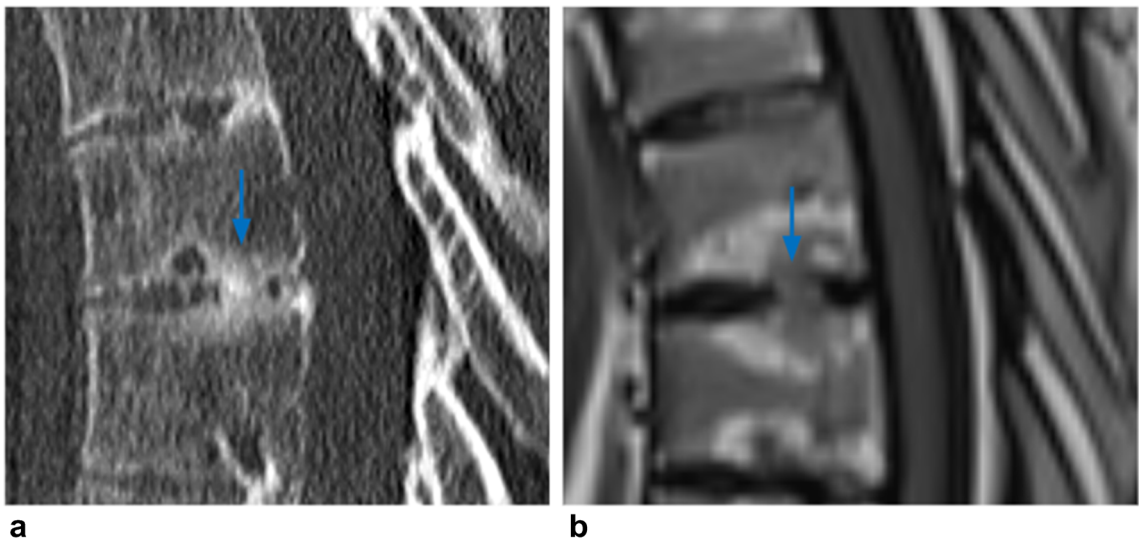

Fig. 9 A 41-year-old man with ankylosing spondylitis. **a**, **b** Sagittal CT (**a**) and T1-weighted MR (**b**) images of the thoracic spine demonstrate transdiscal ankylosis

a relatively small study population. Nonetheless, there are many other known causes of transdiscal ankylosis, including congenital vertebral fusion (due to a failure in the process of vertebral segmentation during fetal development), sequelae of infectious spondylodiscitis, surgical spinal arthrodesis, and post-traumatic causes [48, 49].

Intradiscal high signal intensity on T1-weighted images

It has been demonstrated that hyperintensity of the intervertebral disks in T1-weighted images is associated with disk calcification [50–54]. It is identified as high signal intensity on T1-weighted images within the intervertebral disk without loss of cortical endplate hypointense signal [48, 53].

Disk calcification in AS is a well-known finding. It occurs through endochondral ossification, starting in the cartilaginous endplate and gradually extending to the entire disk [36, 40, 41]. However, this finding is not restricted to SpA and can also be found in conditions like degenerative diseases and ochronosis [52, 54].

Intradiscal high signal intensity on T1-weighted images is not in the ASAS consensus.

Thoracic lateral inflammatory lesion (arthritis of the costovertebral joints)

To be evaluated on MRI, thoracic lateral inflammatory lesion requires additional sagittal slices that extend beyond the pedicles (a minimum of 16 sagittal slices, with 3–4 mm slice thickness) [22]. It is defined as periarticular bone marrow edema in the costovertebral joints, identified in at least one lateral sagittal slice [11] (Fig. 10A, B, and C).

Arthritis and enthesitis of the costovertebral complex may reduce spinal mobility [55].

Facet joint inflammatory lesion (facet joint arthritis)

Facet joints are directly involved in the inflammatory processes of SpA specifically in subchondral bone marrow and entheses of the articular processes [55, 56]. On MRI, it is defined as osteitis in one or both facets of a facet joint observed in at least one sagittal slice, which may be associated with surrounding soft tissue edema [8, 11] (Fig. 10D and E). Facet joint inflammation identified on MRI is associated with reduced spinal mobility and could affect patients in performing daily activities [55].

Patients with psoriatic arthritis are believed to experience less spinal mobility reduction compared to those with AS, as areas critical to spinal mobility, such as the facet joints, are less commonly affected in axial psoriatic arthritis [39].

Posterior element inflammatory lesion (including enthesitis of spinal ligaments and costotransverse joint arthritis)

Posterior element inflammatory lesion is characterized on MRI as bone marrow edema in at least one sagittal slice in a water-sensitive sequence in posterior elements at which there are ligamentous or muscular attachments, or at the costotransverse joint [11, 27].

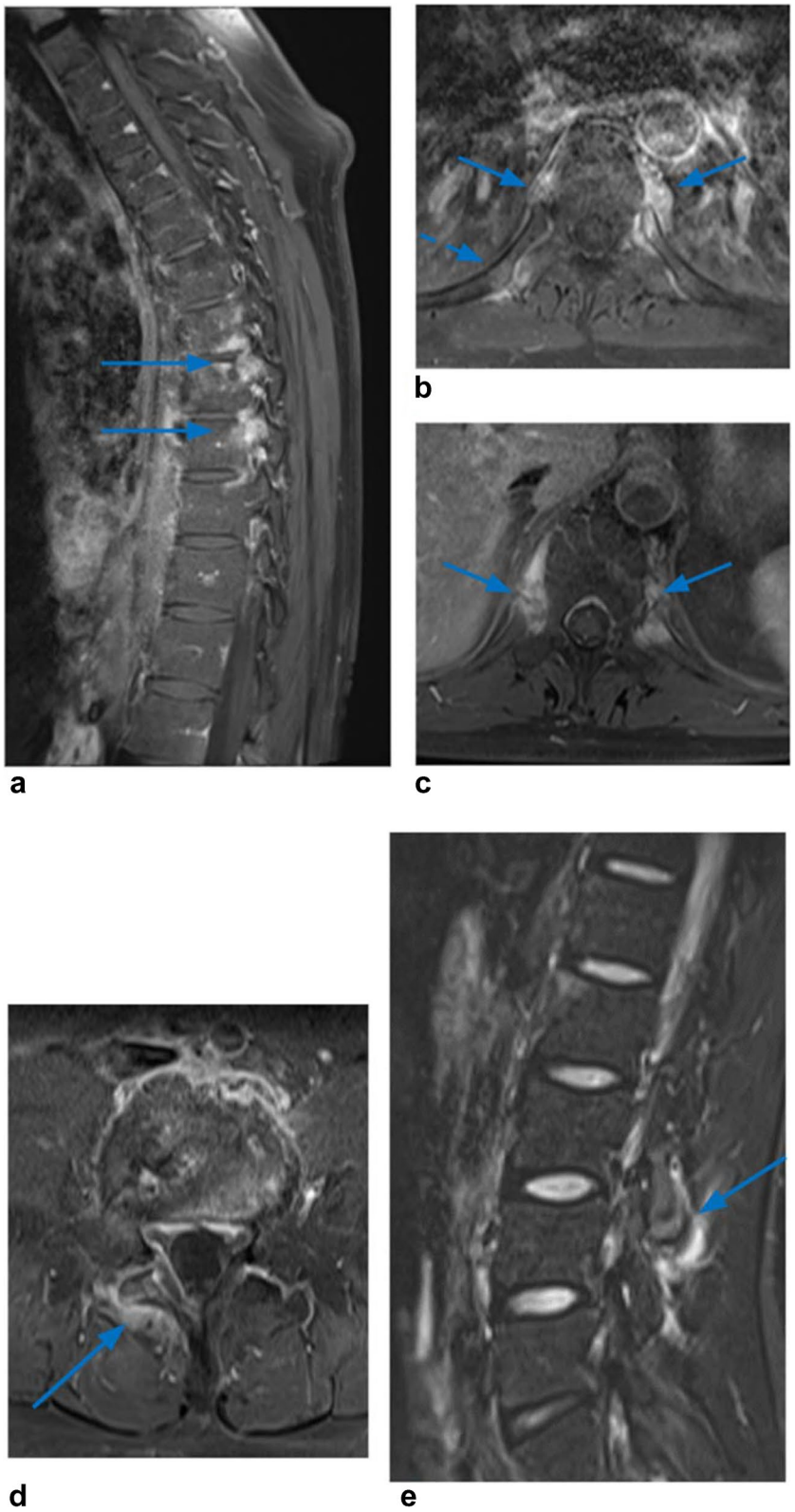

Fig. 10 A 31-year-old man with inflammatory bowel-disease-associated arthritis. **a**, **b**, **c** Sagittal (**a**) and axial (**b** and **c**) fat–suppressed contrast-enhanced T1-weighted image of the thoracic spine shows enhancement at the costovertebral joints (arrows) related to costovertebral arthritis. Additionally, enhancement is noted at the costotransverse joint (dashed arrow), suggestive of costotransverse arthritis. **d**, **e** Axial fat–suppressed contrast-enhanced T1-weighted MR image (**d**) and sagittal STIR MR image (**e**) of the lumbar spine reveal enhancement (**d**) and high signal intensity (**e**) in subchondral bone marrow and entheses of the articular processes with surrounding soft tissue edema (arrows)

Some of the entheses affected in posterior element inflammatory lesion are interspinous ligaments, supraspinous ligament, and *ligamenta flava* [20].

Braun et al. conducted a study comparing the prevalence of inflammatory abnormalities in the posterior arch on lumbar spine MRI between patients with axial spondyloarthritis

and those with low back pain. The analysis included the lumbar and thoracic lower spine using MRI, with images ranging from the ninth thoracic vertebra (T9) to the first sacral vertebra (S1). The study concluded that spinous process edema, costotransverse arthritis, transverse process edema, and pedicle edema above L3 are more prevalent in axial spondyloarthritis patients compared to those with low back pain [57].

Spinal ligaments ankylosis and synovial joints ankylosis (facet, costovertebral, and costotransverse)

On imaging, extensive ossification of ligaments results in the fusion of multiple segments of the interspinous processes and facet joints, leading to the appearance of both the dagger sign (Fig. 11) and the tram track sign. The dagger sign is characterized by a single central radiodense line resembling a dagger blade, indicating ossification of the interspinous ligaments on frontal X-rays. On the other hand, the tram track sign arises from ossification of both the interspinous ligaments and the facet joint capsules, presenting as multiple parallel vertical lines that resemble tram tracks on radiographs [36].

Cartilage degeneration is believed to be a hallmark of the facet joint pathology which may promote cartilage fusion and intra-articular ankylosis. Nonetheless, capsular ankylosis and development of extra-articular new bone formation have also been reported [58]. Facet joint fusion is associated with restricted lumbar motion [59] (Fig. 11).

Fusion of the costovertebral joints and ankylosis of the thoracic spine can contribute to restricted pulmonary function due to impairment of chest wall expansion. Although there is an association between reduced spinal and chest wall mobility and restrictive pulmonary function,

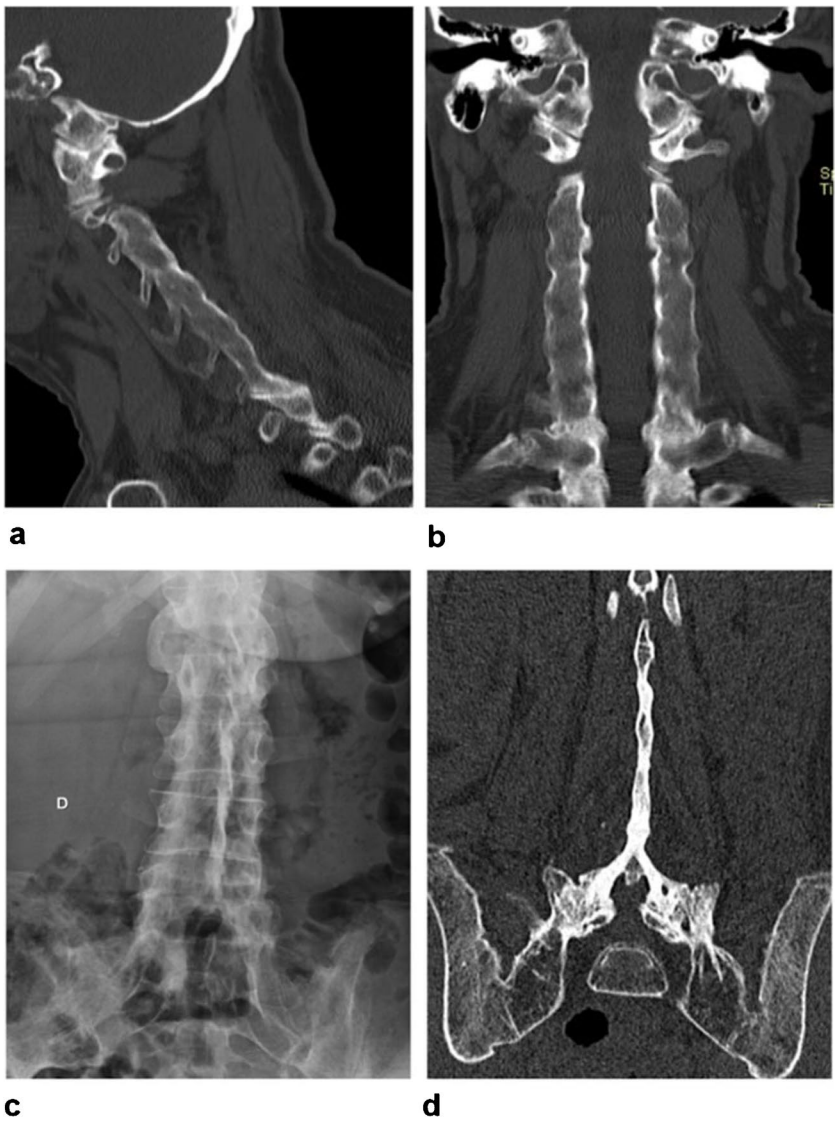

Fig. 11 A 57-year-old man with ankylosing spondylitis. **a**, **b** Sagittal (**a**) and coronal (**b**) CT images of the cervical spine show diffuse involvement of facet joints with complete ankylosis. **c**, **d** Frontal radiograph (**c**) and coronal CT image (**d**) of the lumbar spine show ossification of supraspinous and interspinous ligaments (dagger sign)

pulmonary manifestations in these patients are rare and typically asymptomatic [60, 61].

Spinal complications of long-standing SpA

Cauda equina syndrome in ankylosing spondylitis

Cauda equina syndrome (CES) is a rare complication of long-standing AS, whose etiology is not completely understood. A suggested theory is that the inflammatory process starting at the spinal ligaments leads to secondary involvement of the meninges and nerve roots, which in turn causes arachnoiditis and dural diverticula formation [62, 63]. MRI is the preferred imaging method for the evaluation of CES and may show bone erosions in the posterior elements and pedicles, tethered *conus medullaris*, peripheral clumping of nerve roots, dural ectasia, diverticula, and calcifications [36, 62–64].

Patients' symptoms and signs are related to progressive loss of function of the lower spinal nerve roots, such as pain and sensory and/or motor deficits of the pelvis and lower limbs, sphincter, and sexual dysfunction [62, 63].

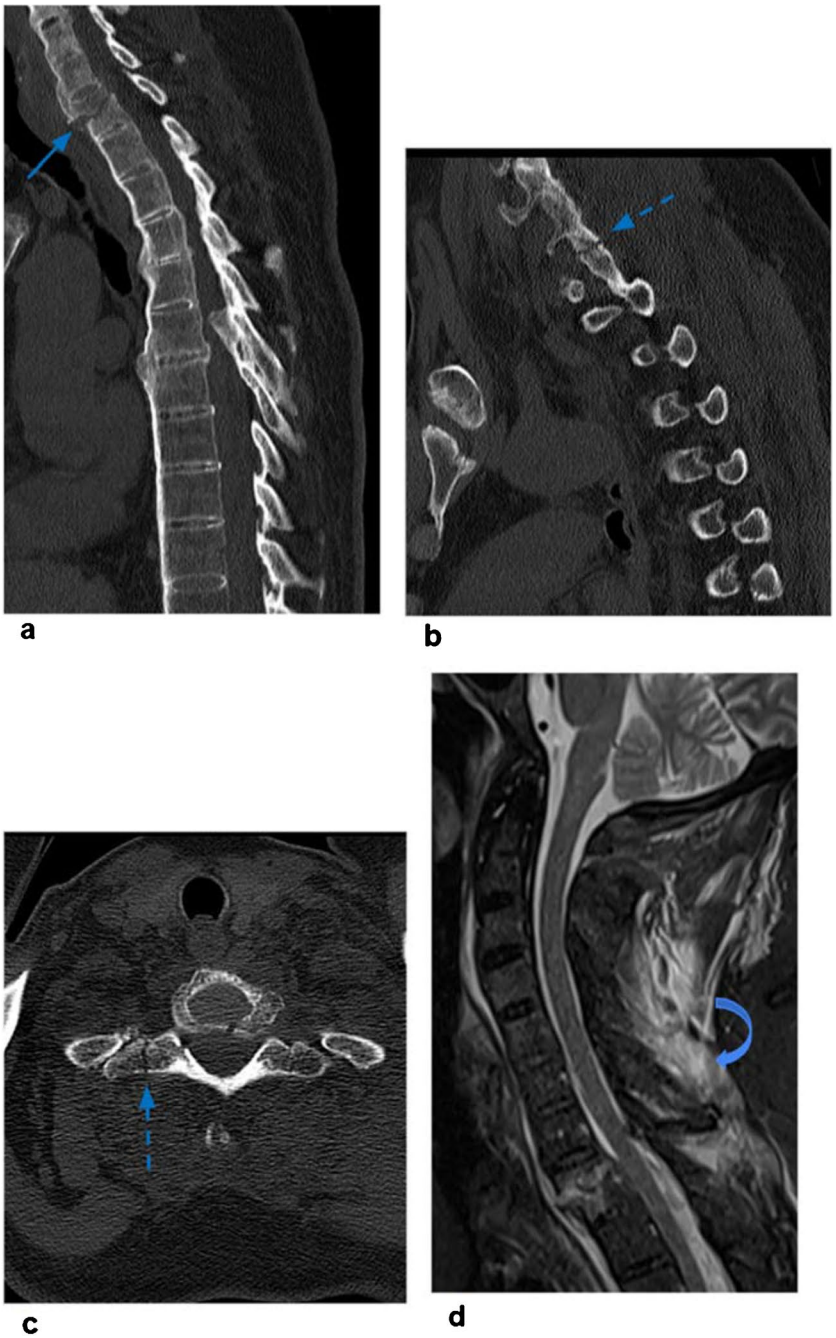

Fig. 12 A 56-year-old man with ankylosing spondylitis. **a**, **b**, and **c** Sagittal (**a** and **b**) and axial (**c**) CT images of the thoracic spine show a transvertebral fracture of T1 (arrow) with extension to the right transverse process (dashed arrows), resulting in an unstable fracture. **d** Sagittal STIR MR image of the cervical spine shows the transvertebral fracture (arrow) and reveals edema in the interspinous ligaments and paravertebral soft tissues (curved arrow)

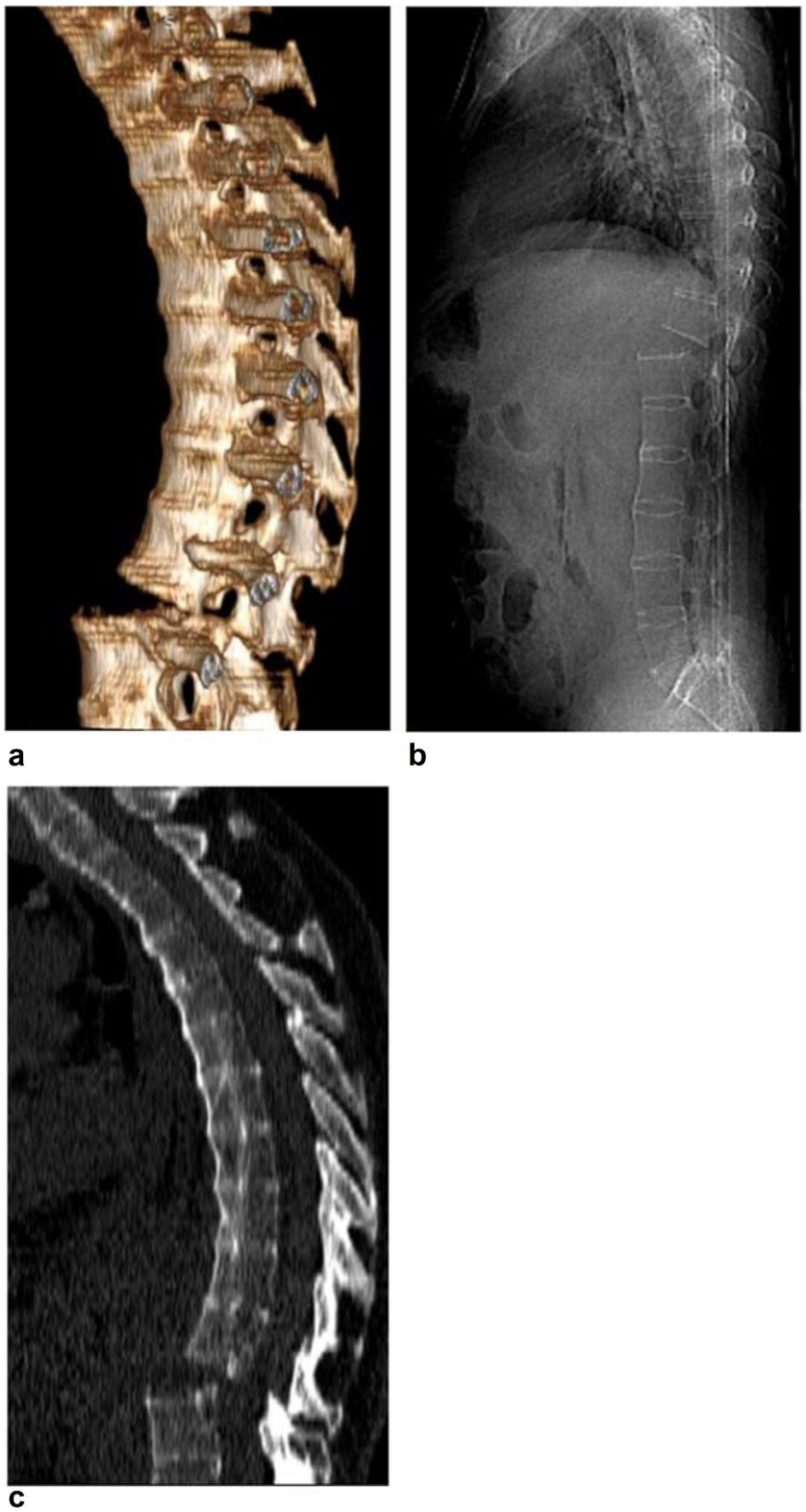

Fig. 13 A 68-year-old man with ankylosing spondylitis. **a**, **b**, and **c** Sagittal 3D bone reconstruction (**a**), CT scout (**b**), and sagittal CT images (**c**) (dual-detector CT scanner) of the thoracic spine demonstrate a transdiscal fracture at T11–T12. The patient choked while eating and sustained the fracture following the Heimlich maneuver

Spinal fractures associated with SpA

The most serious complication of AS is spinal fracture, which can occur even without reported trauma. Cooper et al. reported an increased odds ratio of 7.7% for vertebral fractures in AS compared with the general population [65]. The risk of vertebral fracture increases with the length of disease duration [65–67].

SpA patients are at increased risk of vertebral fractures due to decrease in bone mineralization; loss of spinal mobility, as an ankylosed spine has lower capacity to dissipate energy when subjected to trauma; and also because of kyphotic deformity, which shifts the center of gravity forward, creating more shear stress [68].

When spinal fracture is suspected, conventional radiography is the initial imaging method of choice. If conventional radiography is negative, CT should be performed [21]. MRI is recommended whenever spinal cord lesion is suspected, as it is more sensitive in assessing soft tissue lesions, such as ligamentous lesions and epidural hematomas [69, 70].

The three main patterns of vertebral fracture described in SpA are compression fractures, transversely oriented shear fractures after a direct trauma, and pseudoarthrosis (noninflammatory Andersson lesions) [71].

Vertebral osteoporosis is a well-recognized feature of advanced AS and may predispose patients to spontaneous compression fractures. Ralston et al. found that bridging syndesmophytes were rarely observed in vertebrae with compression fractures, inferring that they may protect against compression fractures by buttressing the edge of vertebral bodies [72].

The most common mechanism of acute traumatic fractures is hyperextension. Most cases of this type of fracture are observed in the lower cervical spine; however, multilevel fractures can also be found. Traumatic fractures often involve both the anterior and posterior vertebral elements resulting in an unstable fracture (Fig. 12), which can lead to spinal cord injury. A fracture of an ankylosed spine usually has a horizontal or oblique orientation and may pass through the vertebrae (transvertebral, Fig. 12), through the disk space (transdiscal, Fig. 13) or through both the disk and adjacent vertebral body or bodies [68, 70, 73–76].

The non-inflammatory Andersson lesion refers to pseudarthrosis from nonunion of a traumatic or more commonly an insufficiency fracture, which can occur with a transdiscal or transvertebral location [68]. An insufficiency fracture in the ankylosed spine may occur spontaneously or after minimal trauma. AS patients might not recognize a lesion and the fracture may be unnoticeable because of its relatively mild symptoms [27]. In the complete ankylosed spine, the persistent motion at the only moving segment, i.e., the fracture site, will impair healing and result in pseudoarthrosis. Stress fractures are primarily located at the junctional zones, especially the thoracolumbar. Such fractures have horizontal orientation with accompanying sclerosis and irregular osteolysis in its borders. The appearance of pseudoarthrosis mimics that of an infectious spondylitis and the differential diagnosis may be difficult [73, 75]. Imaging features which can help differentiate between these entities are minimal paraspinal swelling and extension of the fracture line into the posterior elements; both aspects are observed in pseudoarthrosis [68, 77].

The incidence of spinal cord injuries is 11 times more frequent in patients with AS than in the general population. This is related to the increased incidence of spinal fractures, epidural hematomas, and also related to the AS disease process, such as ossified spinal ligaments [68, 78].

Conclusions

The spinal imaging findings in axial SpA, including the latest ASAS consensus for MRI lesions of the spine, as well as complications of long-standing disease, were reviewed.

Although spinal MRI features are not included in the current ASAS classification criteria, its recognition may raise the diagnostic suspicion of SpA. Additionally, some patients with active disease may only have spinal inflammation and no evidence of sacroiliitis.

Declarations

Conflict of interest The authors declare no competing interests.

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