Complications of the rigid spine

Complications of The Rigid Spine

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Keywords

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Abstract

This article provides a short overview of the most common causes and imaging findings of a rigid spine including longstanding spondylarthritis, Diffuse Idiopathic Skeletal Hyperostosis, and less common Ossification of the Posterior Longitudinal Ligament.

Furthermore, it focuses on the pathogenesis and imaging findings of acute complications of the rigid spine due to fractures. These fractures may occur even after minor trauma and are difficult to detect on initial radiographs, resulting in a delayed diagnosis. They are often unstable with a high risk of severe neurological complications, resulting in a high morbidity and mortality both in the initial phase and in the months following the fracture.

As the negative predictive value of CR is low, every patient with a rigid spine with newly appearing pain should be referred for subsequent cross-sectional imaging.

I. Introduction

Extensive and generalized spinal fusion manifests clinically as a rigid spine with altered posture.

Chronic complications of the rigid spine include osteopenia due to disuse, muscle atrophy and cauda equina syndrome.

This article emphasizes the pathogenesis and imaging findings of acute complications of the rigid spine e.g., fractures that may occur after minor trauma in patients with generalized spinal fusion.

II. Causes and imaging features of vertebral fusion

Fusion between different vertebral segments may either be limited to 2 adjacent vertebra and isolated (*focal fusion*) or involve multiple segments (*generalized fusion*). Isolated fusion results most often from a congenital segmentation defect, although involvement of multiple segments is rarer e.g. due to Klippel-Feil syndrome.

Generalized fusion involving fusion between multiple levels is often due to an acquired disease such as longstanding spondylarthritis (SpA), Diffuse Idiopathic Skeletal Hyperostosis (DISH) and Ossification of the Posterior Longitudinal Ligament (OPPL).

Postoperative fusion may either involve 2 adjacent or multiple segments.

Focal fusion

Focal or isolated fusion is either due to a congenital segmentation defect or acquired due to postoperative fusion.

Congenital fusion occurs most frequently between two adjacent vertebra at the cervical and less frequently at the thoracic and lumbar spine.

On imaging, a characteristic “wasp-waist sign” can be seen on CT, in which the anteroposterior diameter is smaller than the diameter of the superior and inferior limits of the vertebra adjacent to the affected level (▸ **Fig. 1**)[1]. There is a concave delineation of the anterior and posterior wall of the affected vertebra. Often the fusion is incomplete with a discal remnant which may be calcified. There may be an associated fusion of the posterior elements.

Isolated fusion may clinically present with a limited range of motion and can predispose to accelerated disk degeneration on adjacent non-fused levels (▸ **Fig. 1**).

The same mechanism is responsible for accelerated disk degeneration adjacent to postoperative fusion.

Generalized fusion

Generalized and extensive fusion is caused by acquired diseases including longstanding spondylarthritis, Diffuse Idiopathic Skeletal Hyperostosis, and less commonly Ossification of the Posterior Longitudinal Ligament.

Longstanding spondylarthritis

Spondylarthritis (SpA) consists of a spectrum of clinically and genetically interrelated rheumatic diseases, including ankylosing spondylitis, psoriatic arthritis, Reiter syndrome and arthritis-associated inflammatory bowel disease[2].

The typical disease onset ranging from childhood to middle age (>20–40 years)[3 4].

Ankylosing spondylitis -as the best-known prototype of SpA- has a predilection for the axial skeleton.

Typically, the inflammatory process starts at the vertebral enthesis at the anterior vertebral corners where Sharpey's fibers attach the outer portion of the annulus fibrosus to the vertebral body. Early active inflammation is seen as bone marrow edema on Magnetic Resonance Imaging (MRI), whereas conventional radiography (CR) is negative at this stage. Healed inactive lesions show fatty metaplasia on MRI. Later in the disease, erosion, reactive bone sclerosis and finally ossification may occur in longstanding and untreated disease on conventional radiographs. This ossification will extend from Sharpey's fibers along the deep layers of the longitudinal ligaments, forming initially thin, vertical outgrowths along the contour of the disc, known as syndesmophytes. These syndesmophytes ossify one vertebral body to the adjacent vertebral body in a succinct fashion. The disc spaces are generally preserved. Disc calcification or ossification may be present. With disease progression, the syndesmophytes thicken and involve the anterior longitudinal ligament and paravertebral soft tissues. This may lead to extensive spinal fusion with so-called bamboo spine formation[5](▸ **Fig. 2**).

Vertebral outgrowths in psoriatic arthritis may be morphologically distinct and present as focal paravertebral ossification (parasyndesmophytes)[2].

Similarly, the process of inflammation may also involve other target joints such as the apophyseal joints resulting in consecutive erosions, reactive bone formation with subchondral sclerosis and finally ankylosis of the facet joints. Narrowing and osseous fusion of these joints can be apparent on CR at this stage. Apophyseal joint ankylosis can be particularly extensive at the cervical spine[5].

Enthesitis of the posterior ligamentous attachments of the spine leads to subligamentous erosion and ossification. On frontal radiographs ossification of the supraspinous and interspinous ligaments can be seen as a central radiodense line, known as the "dagger sign".

Ossification of the apophyseal joint capsules forming two vertical radiodense lines lateral to this central line is apparent as the “trolley- or tram-track sign” on the frontal view[5].

(▸ Fig. 3).

The ongoing process of ankylosis results in progressive stiffness, flattening of the lumbar lordosis and increased thoracic kyphosis, together with a limitation in spinal flexion[6].

DISH

Diffuse Idiopathic Skeletal Hyperostosis (DISH)[2,7], previously known as Forrestier disease[8], is a common incidental finding on imaging. Compared to SpA, it is seen in middle-aged and elderly patients (>50 years)[4]. It may involve either the axial and appendicular skeleton[4].

The diagnosis of end-stage DISH of spinal involvement relies on the radiographic criteria of Resnick and Niwayama, showing flowing osteophytes over at least four contiguous vertebrae of the thoracic spine with relative preservation of the intervertebral disc space **(▸ Fig. 4a**) and the absence of apophyseal and costovertebral joint ankylosis and sacroiliac joints erosion, sclerosis, or bony fusion[7]. Ustinger suggested a threshold of flowing osteophytes over three contiguous vertebrae and the presence of pelvic enthesophytes to the criteria[9], although modification of these criteria is not generally accepted[4].

Due to pulsation of the adjacent thoracic aorta, preventing development of bony proliferation on the left side, there is typically predominant involvement on the right side, which is best illustrated on axial CT images **(▸ Fig. 4b**). As the anterior longitudinal ligament is loosely at the level of disc space and more firmly attached at the anterior vertebral body, this may allow expansion of the bony outgrowth at the disc space whereas expansion at the anterior wall of the vertebral body is limited.

As a consequence, vertebral outgrowths have a typical horizontal orientation and there is a subjacent L-T- and Y radiolucency at the disc space **(▸ Fig. 4a**)[7,4].

Compared to SpA, vertebral outgrowths are thicker[4].

In addition to involvement of the thoracic spine, irregular and pointed bony excrescences at the superior and inferior vertebral margins in the cervical and lumbar regions may be present[7].

OPPL

Ossification of the Posterior Longitudinal Ligament (OPLL) typically affects the cervical spine and is more common in Asian people than in Europe[2]. Its cause is not completely clear. It may coexist with DISH[10]. OPPL predominates at the midcervical region and less commonly the thoracic and lumbar spine[11].

CR shows a linear ossification of variable thickness posterior to the vertebral bodies and intervertebral discs, with a radiolucent line between the vertebral body and the ossified posterior longitudinal ligament. The disc space is usually spared[12] (▸ **Fig. 5**) and the disease may result in narrowing of the spinal canal.

III. Pathogenesis of acute complications of the rigid spine

There are several factors that contribute to increased fracture risk in the ankylosed spine, including increased stiffness, spinal deformity, and thoracic kyphosis, muscle weakness and osteopenia.

Increased stiffness

Due to its high number of articulations and spongious trabecular structure, the normal spine is highly flexible and is able to absorb forces without significant damage[13].

Fusion of the spine results in loss of flexibility, in which the fused spine behaves biomechanically like a long bone, acting as a rigid lever, incapable of appropriately dissipating the energy of a traumatic event with transient deformity[13].

Spinal deformity

Impaired mobility due to a rigid and kyphotic spine is another factor that increases the fracture risk. In addition, patients with longstanding spondylarthritis or DISH may present with joint arthritis or degenerative disease of the peripheral joints respectively, which may further compromise patient's mobility [14 13]. Spinal kyphosis also results in changes to the body's center of gravity, shifting it anteriorly, and impairing horizontal gaze, which may result in an increased tendency to fall [13 15 16].

Muscle weakness and osteoporosis

Decreased mobility of the rigid spine results in muscle weakness (▸ **Fig. 6)** and disuse osteopenia (▸ **Fig. 2)**, reducing the mechanical strength of the bone. Muscle weakness is associated with both increased fracture risk and low bone mineral density (BMD)[17]. Spondylarthritis is known to be associated with osteoporosis with reduced trabecular bone within the vertebrae, further compromising the flexibility on loading[13 18].

The 4 main predisposing factors for increased fracture risk factures in the rigid spine are summarized in ▸ **Fig. 7**[19]

Due to the above mentioned biomechanical factors, fractures in the rigid spine may occur after an even minor trauma often due to hyperextension of the spine[20]. In up to 35% of cases, there is no clear history of trauma[21 14]. As previously stated, the age of occurrence is usually older in patients with DISH than in SpA.

The diagnostic delay is often more pronounced in patients with DSIH than in SpA, because of the lower awareness of acute complications in DISH [21].

IV. Location

The morphology of the fractures may be either nonspecific or occur through the disk space (often seen in cases of incomplete ossification) or transvertebral (occurring in longstanding disease in which the disk is ossified)[22] (▸ **Fig. 8**).

These fractures almost invariably involve the 3 columns of Denis[23] and are highly unstable which may result in a high risk of severe neurological complications[24 25 14 19].

Fractures occurring in SpA patients involve predominantly the cervical spine (▸ **Fig. 9**) followed by the lower thoracic spine and lumbar spine [21 22] (▸ **Fig. 8**).

In patients with DISH, fractures occur more commonly at the thoracic and lumbar spine (▸ **Fig. 8**) [19].
Fractures may also occur at the transition either of the cervical lordosis and thoracic kyphosis or thoracic kyphosis and lumbar lordosis.

V. Imaging

Conventional Radiography (CR)

CR is not sensitive for detection and up to 50 % of fractures may be missed, due to several factors. These factors include osteoporosis with difficult delineation of the fracture line, absence of displacement in the initial stage, complex fracture course with superimposition of adjacent structures and the difficult positioning of patients with a rigid spine, especially in patients affected by SpA[26] .

Therefore, the diagnosis is often delayed in up to 30% of cases[19 27].

Fractures complicating the rigid spine have a high morbidity and mortality both in the initial phase as in the months following the fracture[19].

As the negative predictive value of CR is low, every patient with a rigid spine with newly appearing pain should be referred for subsequent cross-sectional imaging [28].

Computed Tomography (CT)

CT is the imaging method of choice for fracture detection and its extent[29]. Due to its capability for multiplanar reformatting, CT allows to detect small curvy fractures lines with extension in the posterior elements. One should particularly look for transdiscal fracture lines or transvertebral fractures close to the disk space[26] (▸ **Figs. 8-9)**.

Another advantage of CT is its possibility to visualize the whole spine in a very short acquisition time.

However, CT is not well suited for evaluation of associated spinal cord injury or extra-axial hematoma.

Therefore, any patient with a rigid spine, presenting with clinical-neurological sign following an even minor trauma should undergo MRI without any delay.

Later on, persistent motion at initial occult fractures can cause pseudoarthrosis. In this scenario, CT may reveal irregular osteolysis at the endplates surrounded by reactive sclerosis often with a vacuum phenomenon in the intervertebral space, that may be subtle[30].

Magnetic Resonance Imaging (MRI)

MRI is the method of choice for detection of intra-axial lesions in the spinal cord as well as extra-axial hematoma, even before the onset of clinical signs of compression of the spinal cord (**Figs. 8, 10)**. Bone marrow edema may pinpoint to the presence of fractures in the vertebral bodies adjacent to the disc space with extension in the posterior elements (▸ **Figs. 8,**

9). Transdiscal fractures less commonly exhibit bone marrow edema[31]. Due to a mechanism of hyperextension, these transdiscal or transvertebral fractures are often open anteriorly with the presence of a fluid collection along with the course of the fracture (▸ **Figs. 8, 10)**. These fluid collections often indicate a recent fracture, whereas a hypointense signal on T2 may indicate the presence of an old fracture with pseudarthrosis [19 20 31]. Involvement of the posterior elements is a helpful imaging clue in the differential diagnosis with spondylodiscitis in which the posterior elements are usually spared with the exception of tuberculous spondylitis[19].

Fractures can often occur at multiple levels in the vertebral column in these patients (▸ **Fig. 11)**. It is therefore important to image the whole spine to identify these other fractures.

MRI also enables evaluation of ligamentous injuries (▸ **Figs. 8-10)**, as well paraspinal and soft-tissue hematomas[29] and multifocal fractures, which are commonly associated with spinal cord injury[31] .

Catastrophic spinal cord injuries include hemorrhagic cord contusion, intra-medullary hematoma, extradural hematoma and complete cord transection.

Posttraumatic cord contusion may even occur in the absence of overt fracture[6].

A drawback of MRI is that patients with a marked ankylosis and thoracic kyphosis are often difficult to position in the MR machine.

The degree of bone marrow edema due to fractures varies with fracture mechanism and tends to be maximal in compression fractures and limited in distraction injuries. Fractures in fused spine, which are usually related to extension distraction mechanism, may be difficult to visualise easily even on STIR images on MRI examination due to the limited or absent bone marrow edema (▸ **Fig. 11)**.

MRI is also suited to document cauda equina syndrome due to longstanding ankylosing spondylitis. Although its pathogenesis has not been completely elucidated, it has been

suggested to result from dural stiffening and formation of dural ectasias, causing downstream nerve root damage (▸ **Fig. 12**)[32].

VI. Conclusion

Fractures occurring in the rigid spine are often initially unrecognized and have a delayed diagnosis.

Because they are often unstable, they are associated with a high rate of neurological complications and have a high morbidity and mortality.

A high index of suspicion is needed in any patient with a rigid spine and new onset of pain or neurological symptoms.

In these scenario's, cross-sectional imaging by CT for detection of the fracture and its extent.

MRI is imperative for every case where neurological complications are suspected.

Conflict of interest

None declared

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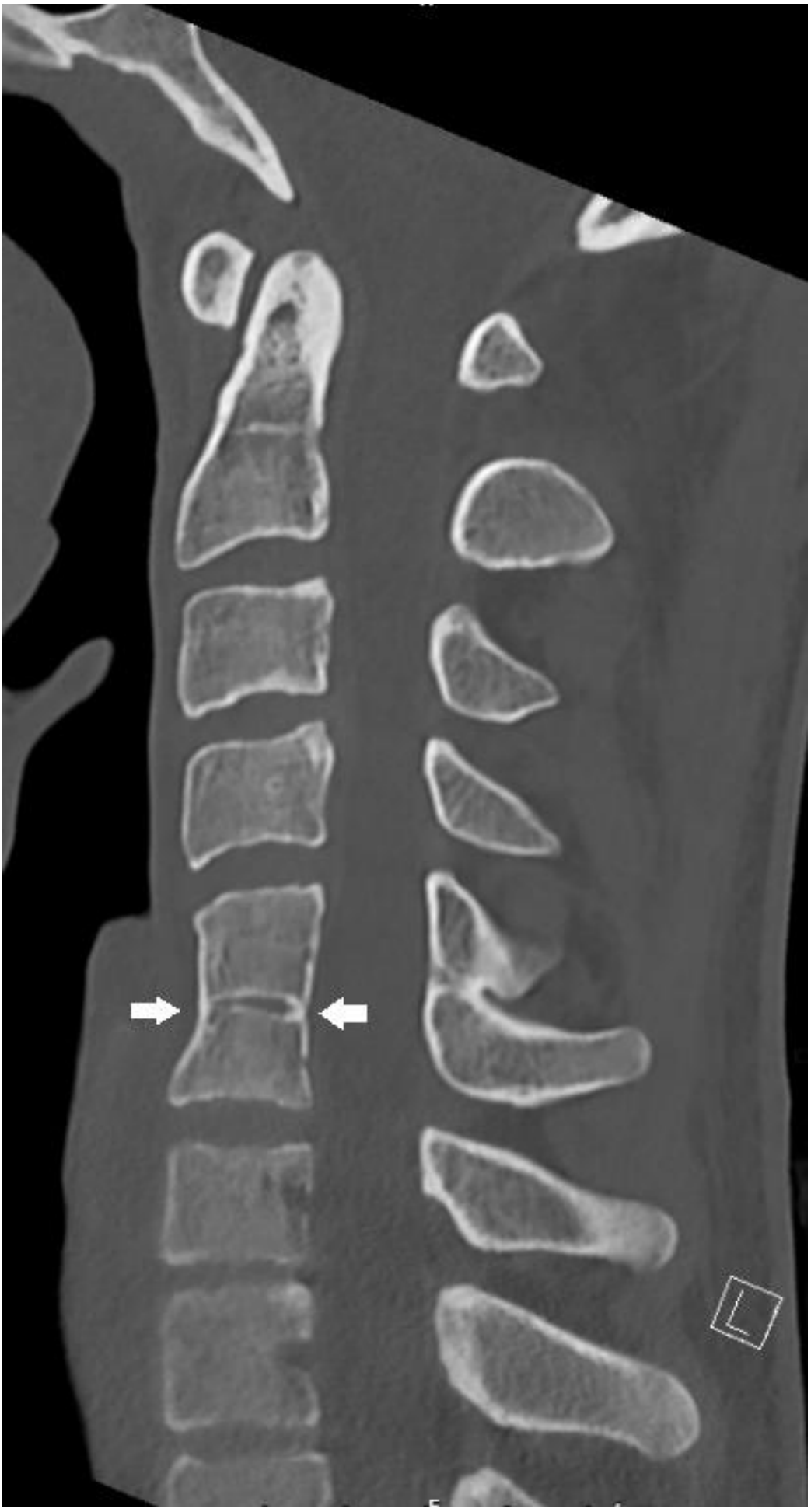
Figure legends



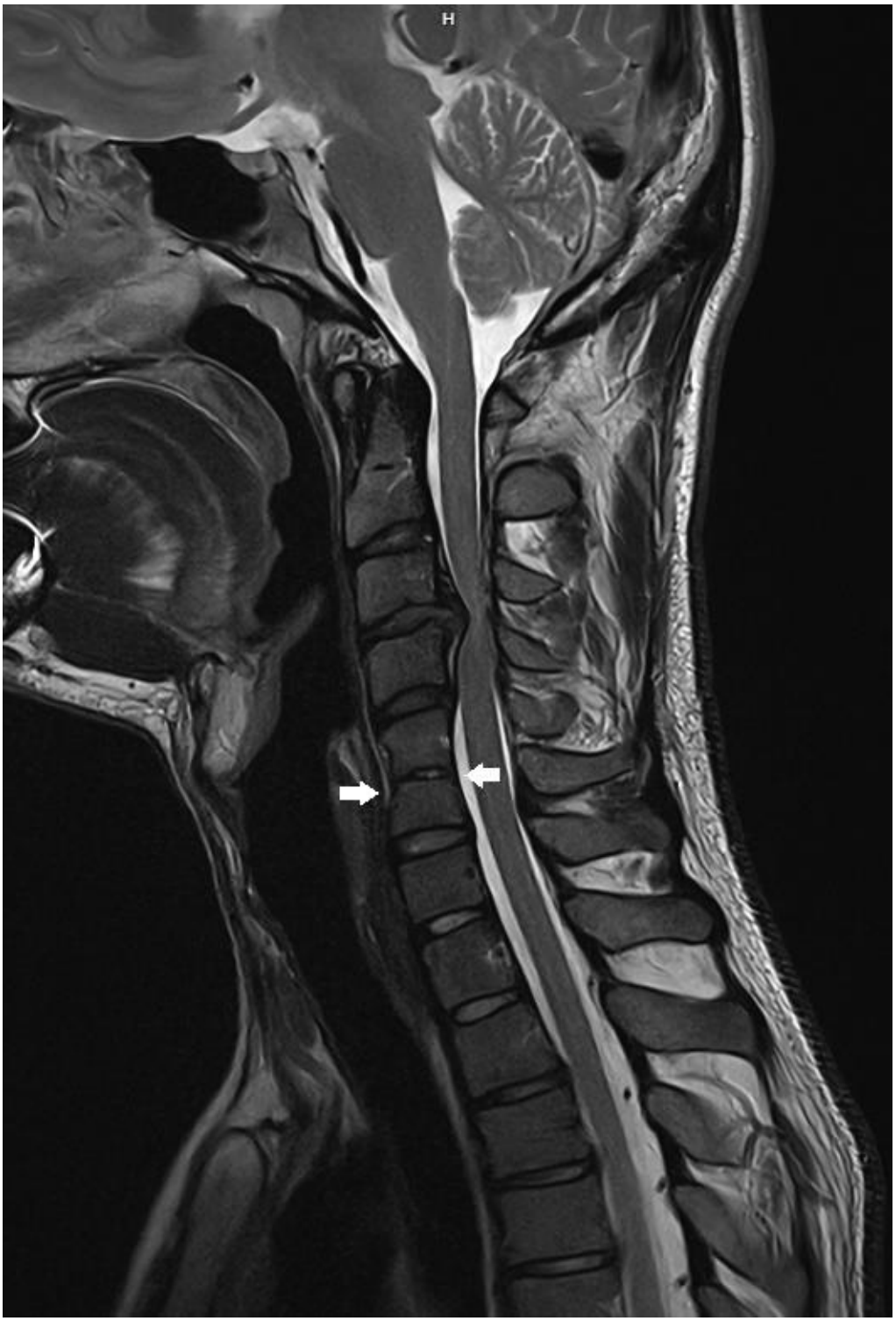


Fig. 1 Congenital fusion of C5-C6 in a 22-year-old male. **a.** Sagittal reformatted CT **b.** Sagittal T2-Weighted image. Note partial fusion of disc space anteriorly with disc remnant and anterior and posterior concavity of the vertebral bodies C5 and C6, resulting in a wasp-waist deformity (white arrows). There is premature disc degeneration with disc extrusion C3-C4, compressing the spinal cord and disc protrusion C4-C5.

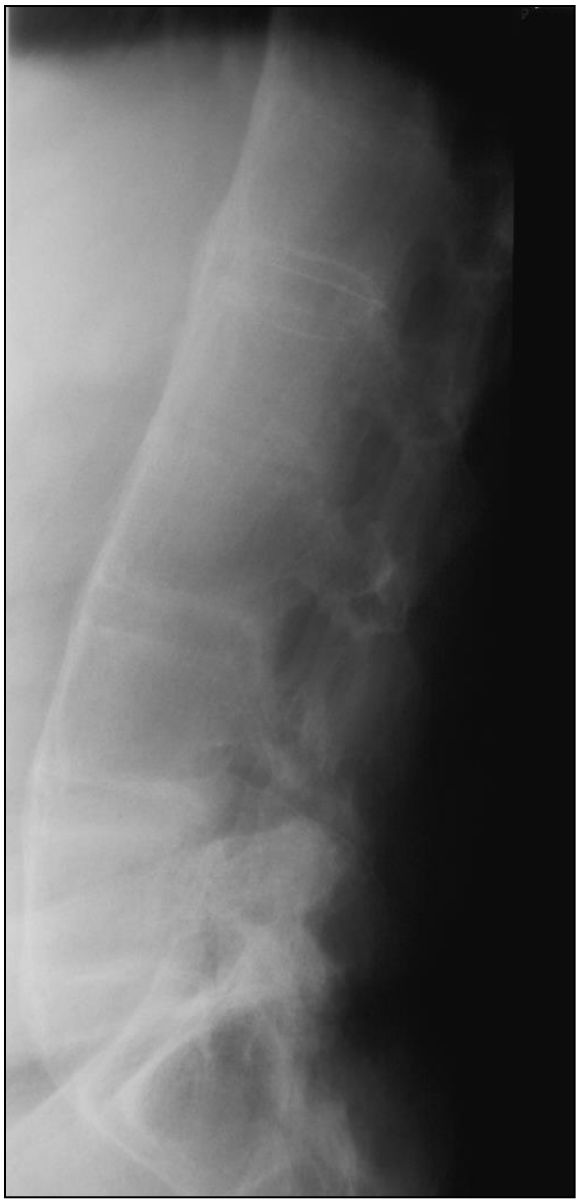


Fig. 2 Bamboo spine in a patient with longstanding SpA. Lateral radiograph showing thin syndesmophyte formation and fusion of the facet joint. This is also osteopenia secondarily to disuse as a consequence of decreased mobility.

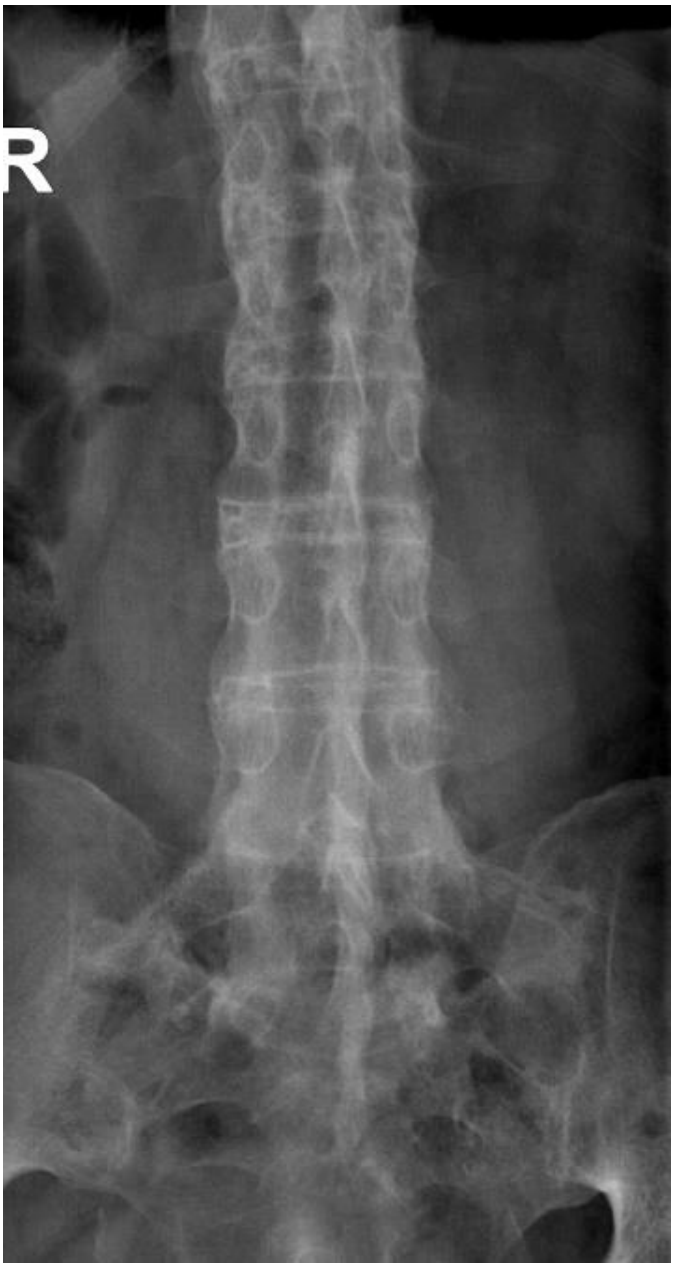
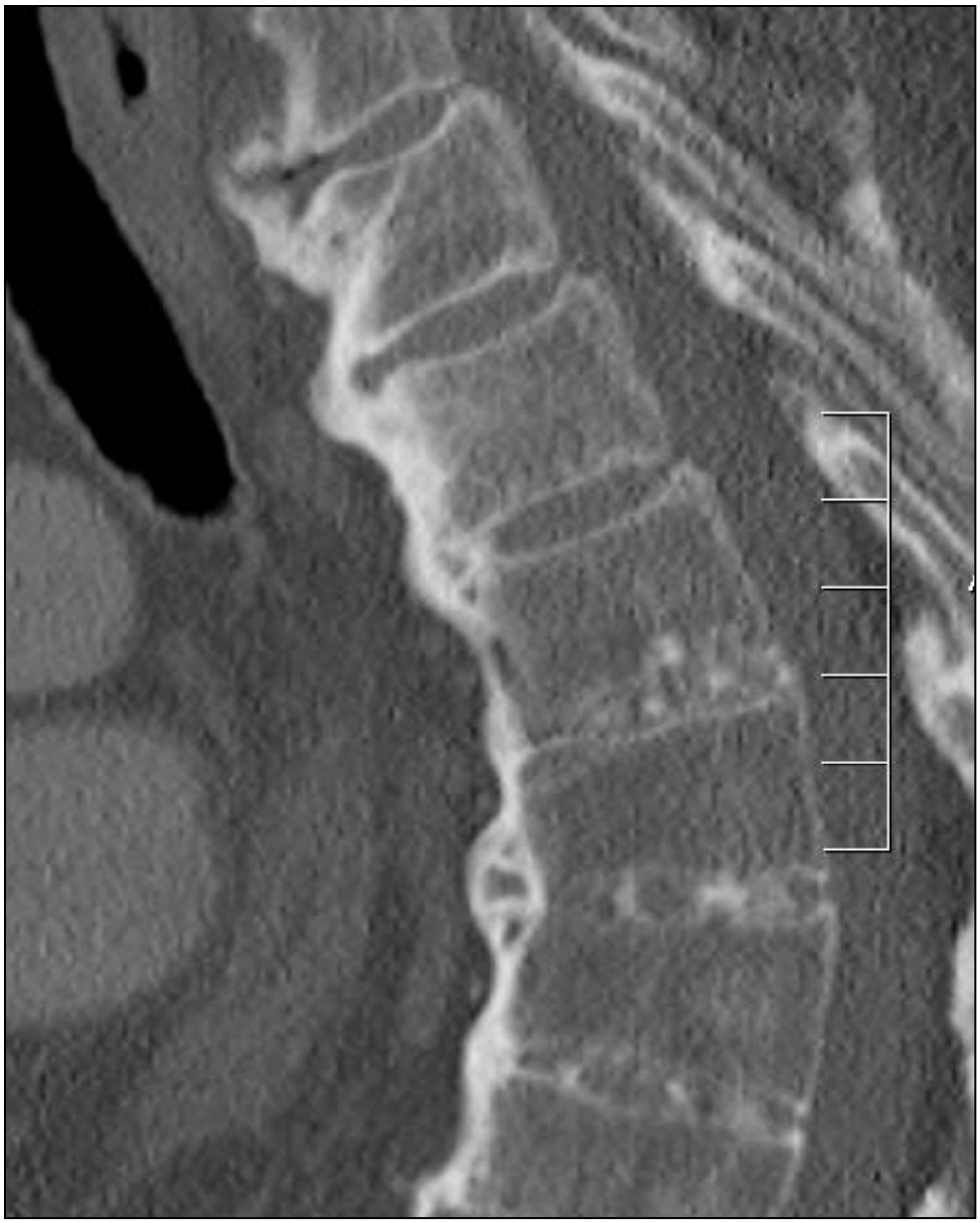


Fig. 3 "Trolley-track" on an frontal view of the lumbar spine sign due to fusion of the spinous process at the midline ("dagger sign") and ankylosis of the facet joints laterally in a 68-year-old male patient with longstanding SpA. Note also ankylosis of the sacro-iliac joints.



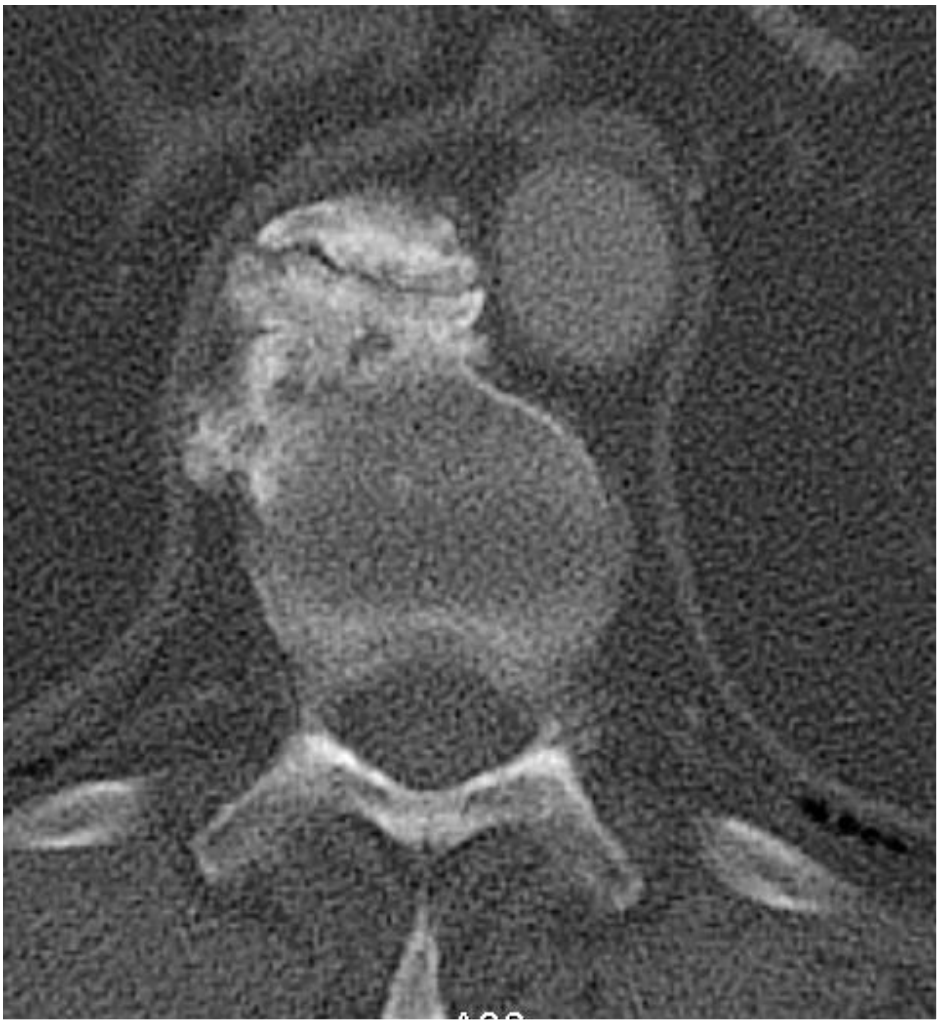


Fig. 4 DISH. **a.** Sagittal reformatted CT shows flowing osteophytes over at least four contiguous vertebrae at the thoracic spine with relative preservation of the intervertebral disc space. Note the typical horizontal orientation of the bony excrescenses and L-T-, or Y radiolucency at the disc space (white arrows) **b.** Axial CT shows predominant involvement on the right side because pulsation of the adjacent thoracic aorta prevents development of bony proliferation on the left side.

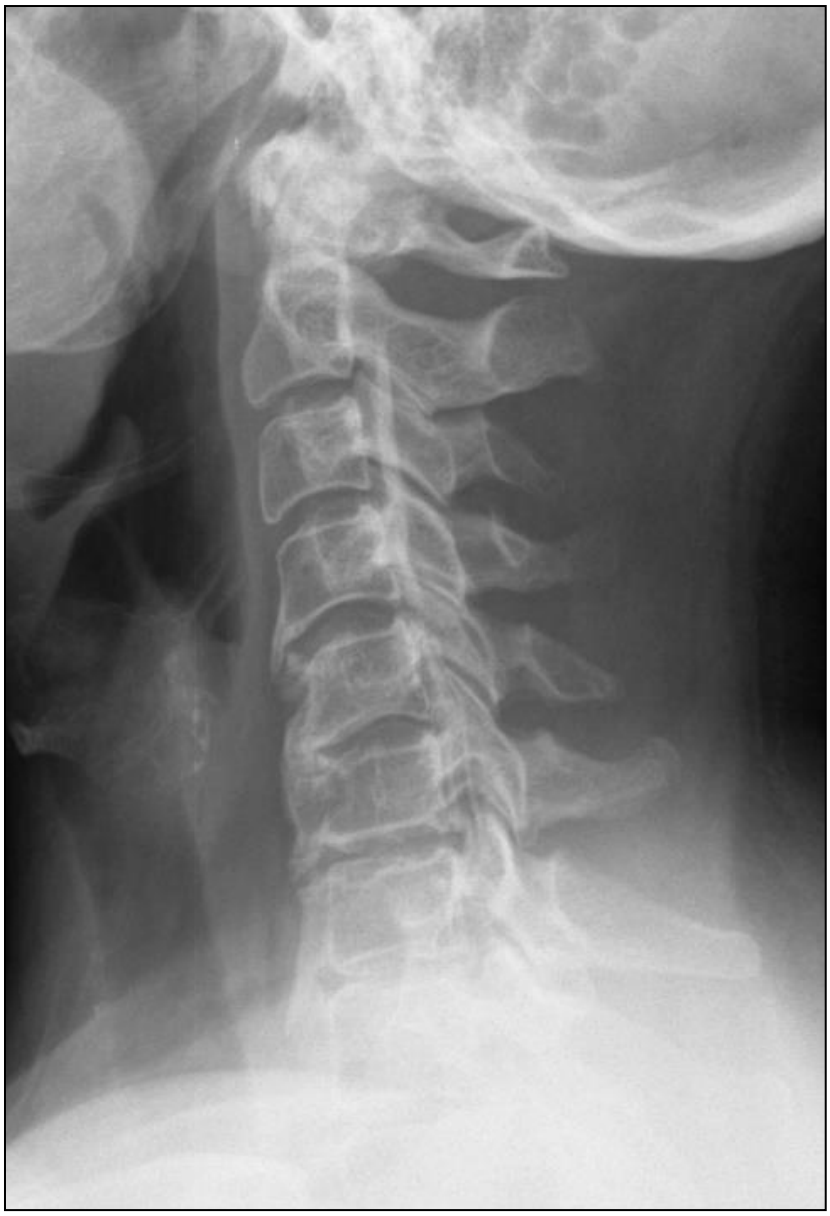


Fig. 5 OPLL. Lateral radiograph shows linear ossification posterior to the vertebral bodies and intervertebral discs (black arrows), with radiolucency (white arrows) between the vertebral body and the ossified posterior longitudinal ligament. The disc space is spared.

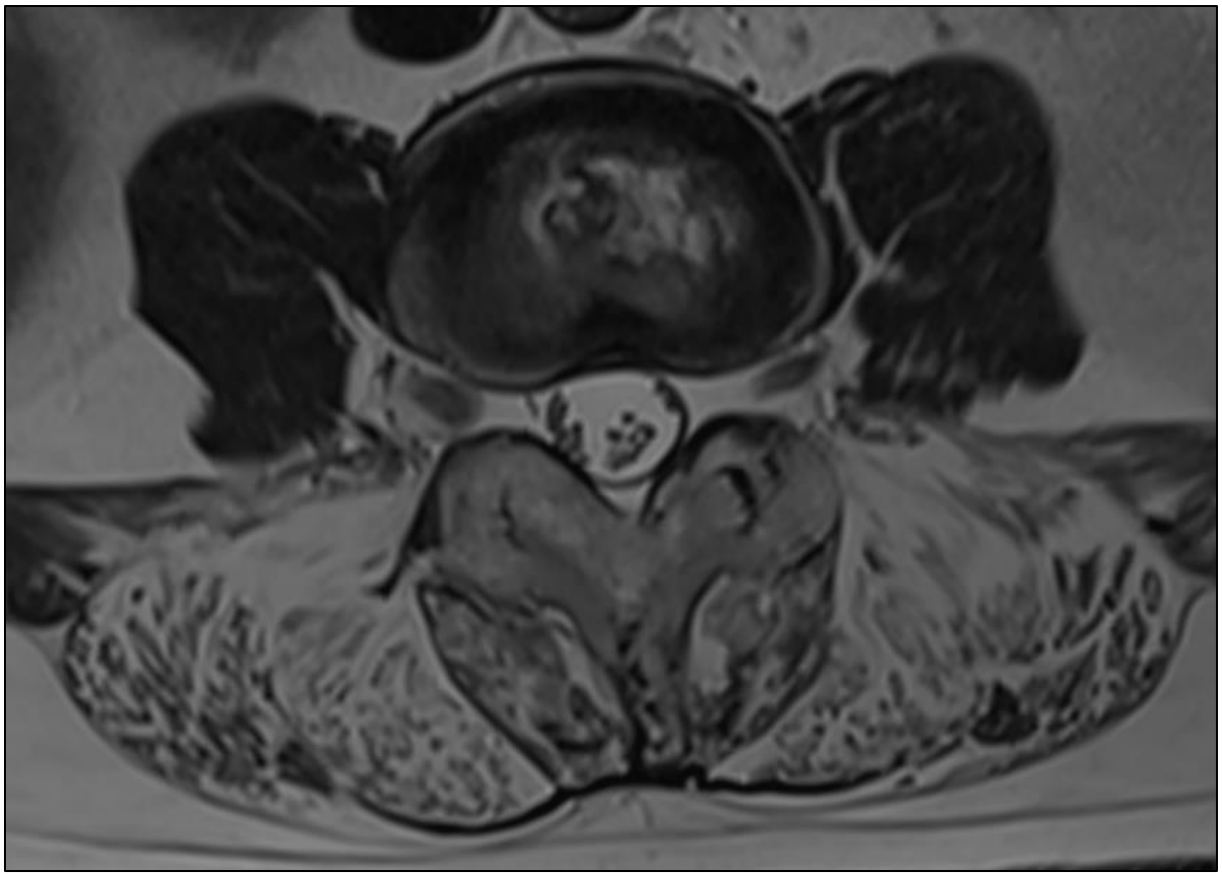


Fig. 6 Atrophy and fatty infiltration of the paravertebral muscles in a patient with longstanding SpA.

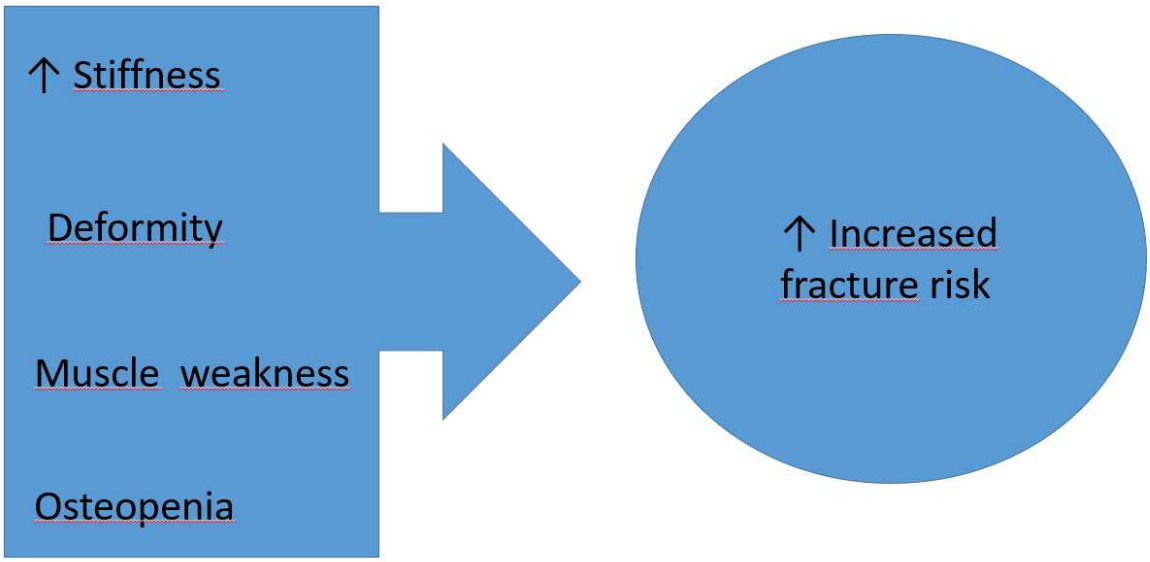
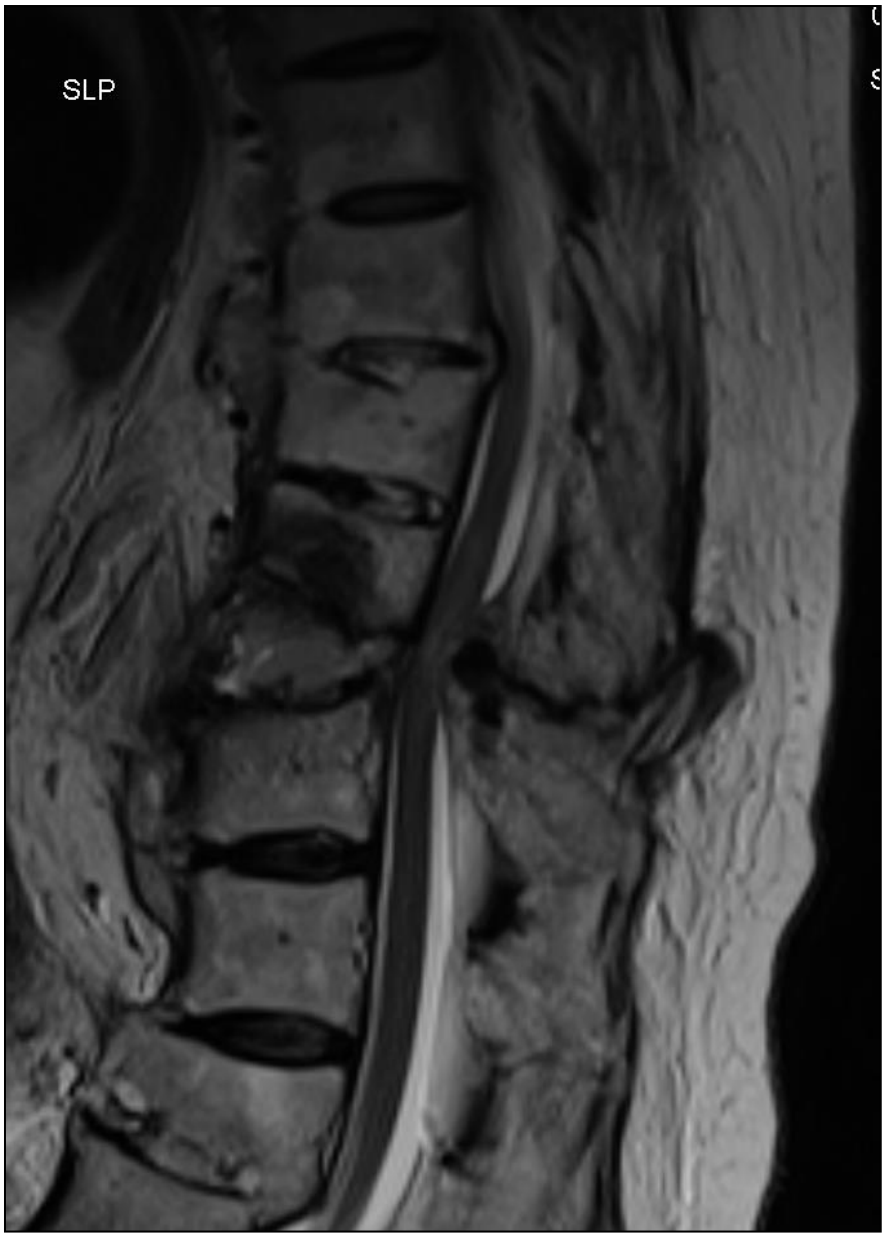
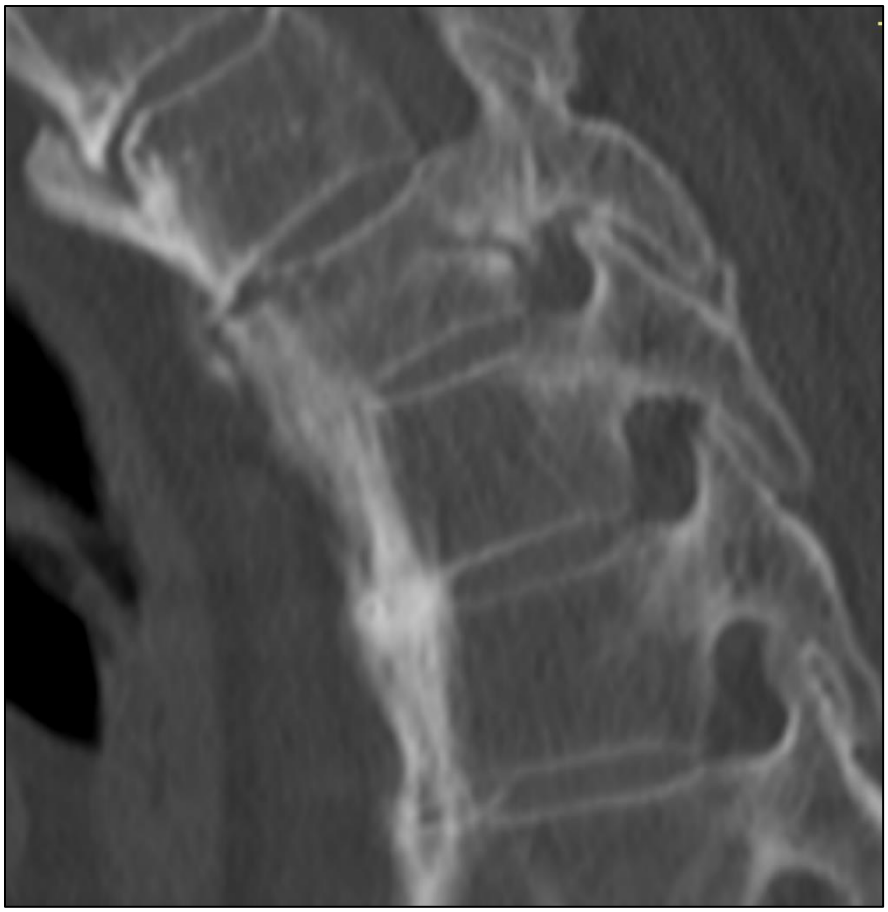
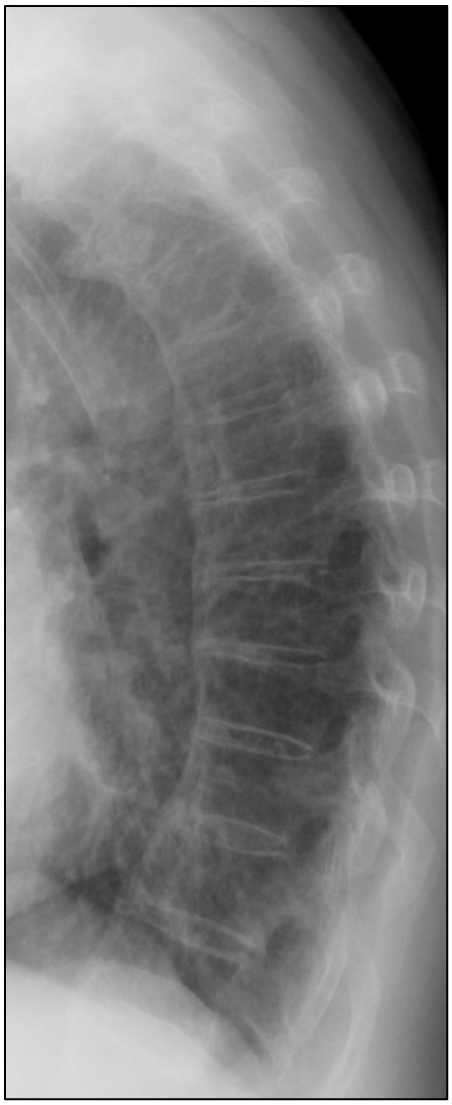


Fig. 7 Summary of the main predisposing factors for increased fracture risk factures in the rigid spine.


SLP





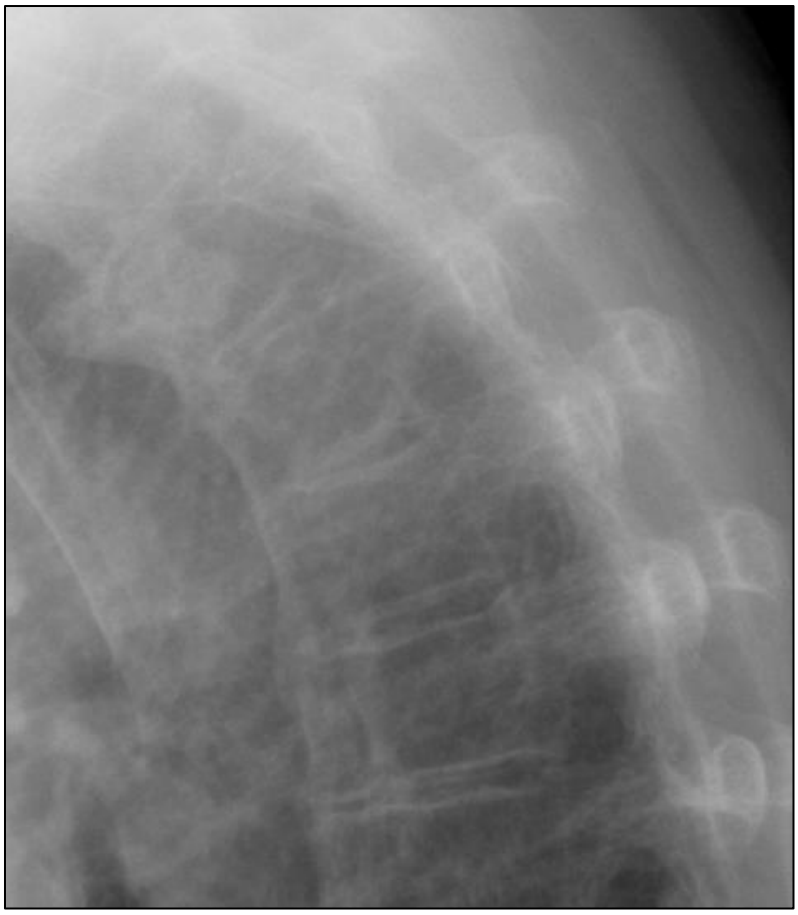
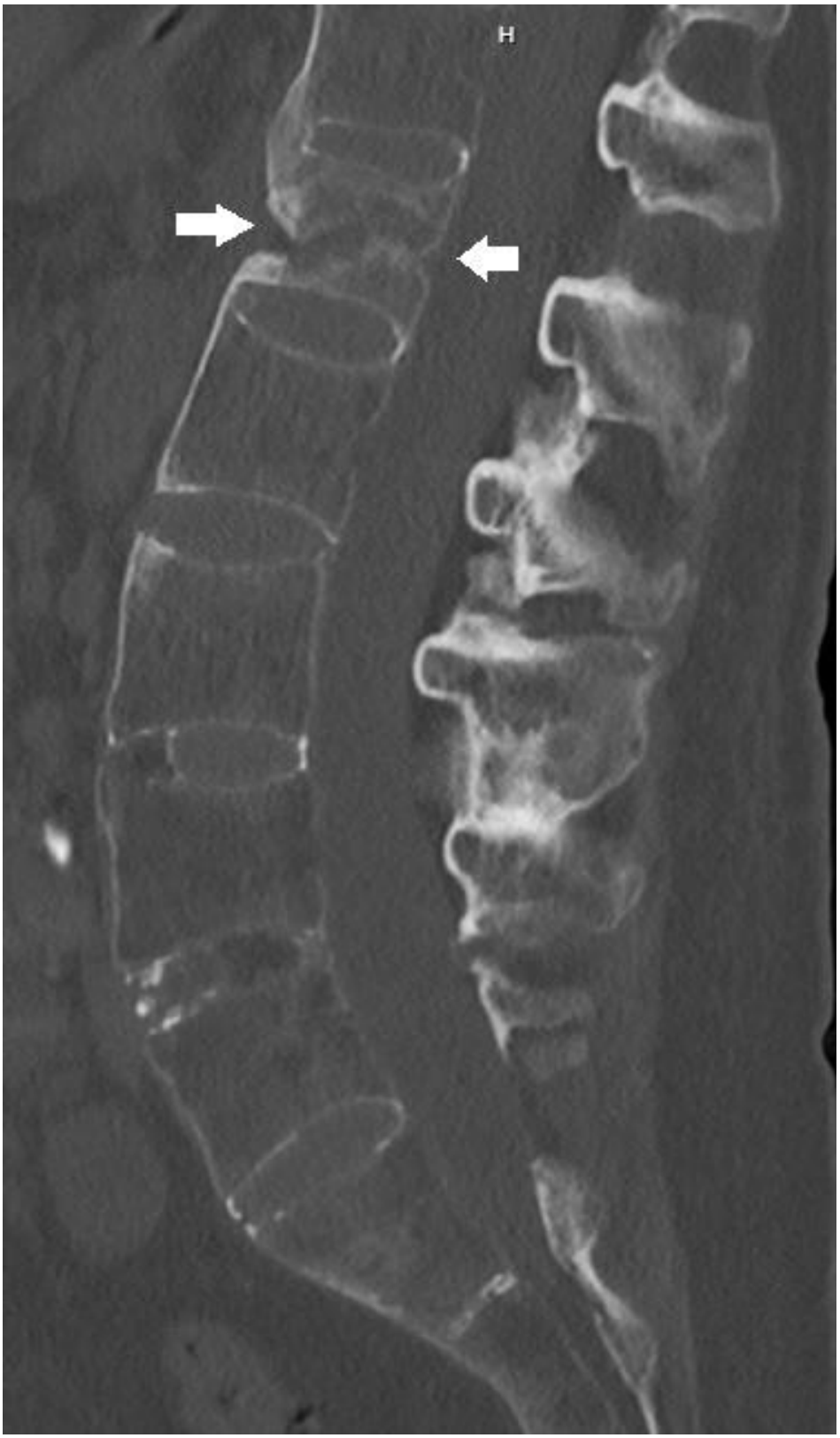


Fig. 8 Fractures in patients with DISH. **a.** Sagittal reformatted CT showing a transdiscal fracture which is open anteriorly (black arrow). Compared to other levels, there is discontinuity of the ossified anterior longitudinal ligament. **b.** Sagittal T2-Weighted image of the same patient as in **a** confirms the transdiscal fracture extending into the posterior elements with disruption of the anterior (white arrow), posterior longitudinal, interspinous and supraspinous ligaments. There is a large fluid cleft in the disc space and a posterior epidural hematoma compressing the spinal cord with central myelomalacia.
c. Sagittal reformatted CT in another patient showing a transvertebral fracture parallel to the endplate (white arrows). **d-e** Lateral radiograph of the thoracic spine (**d**) and spot view (**e**). This fracture was -even in retrospect- not visible on CR (same patient as in **c**).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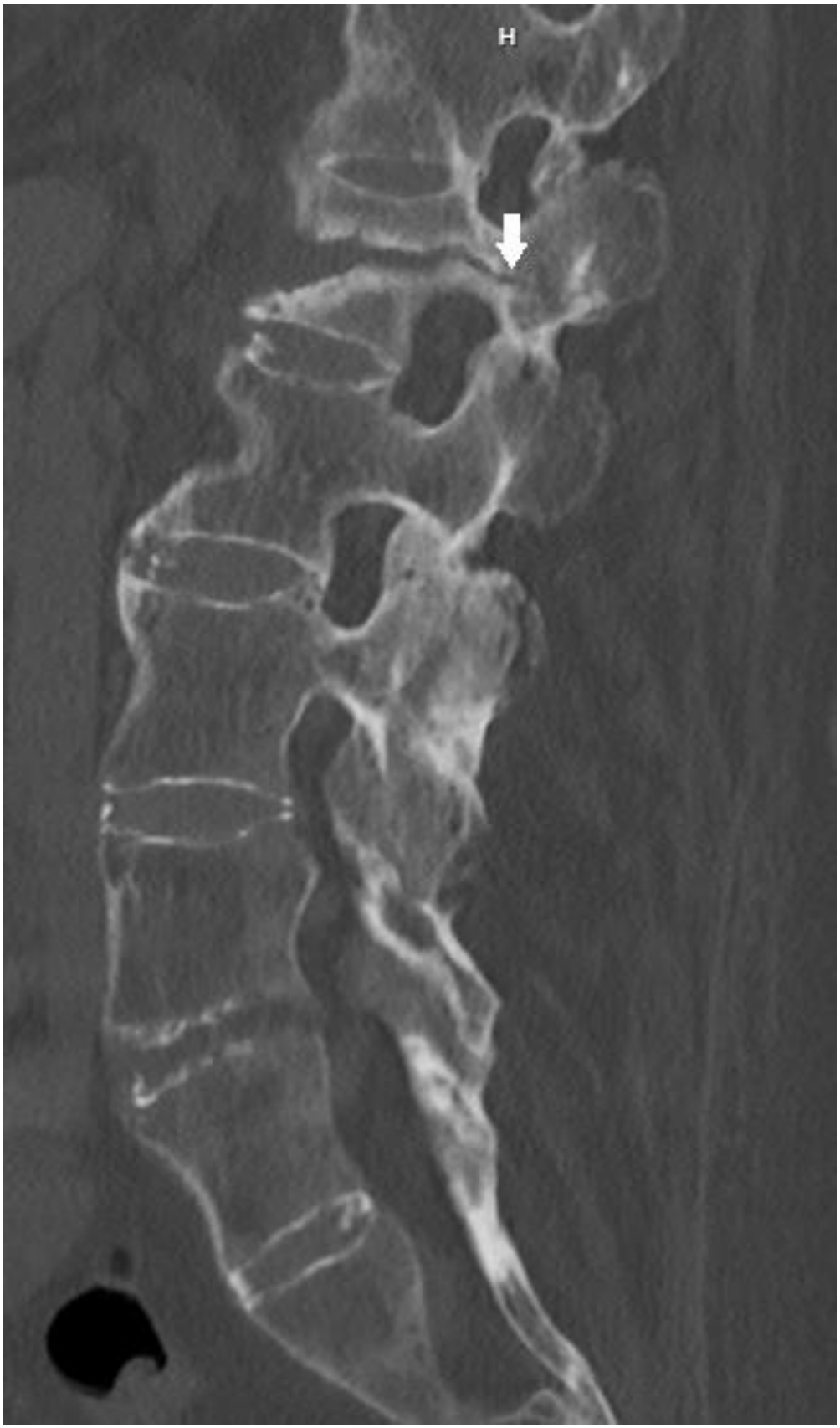
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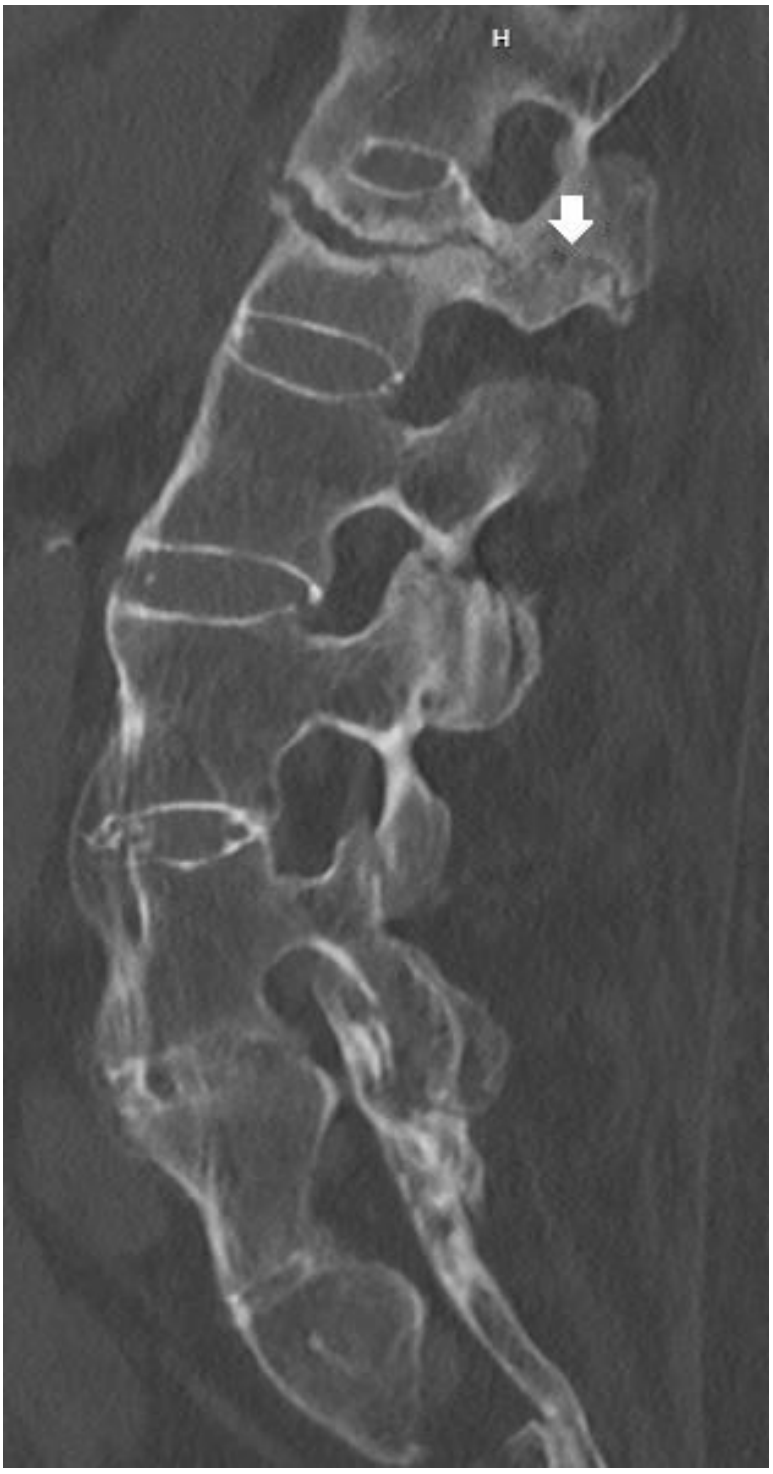
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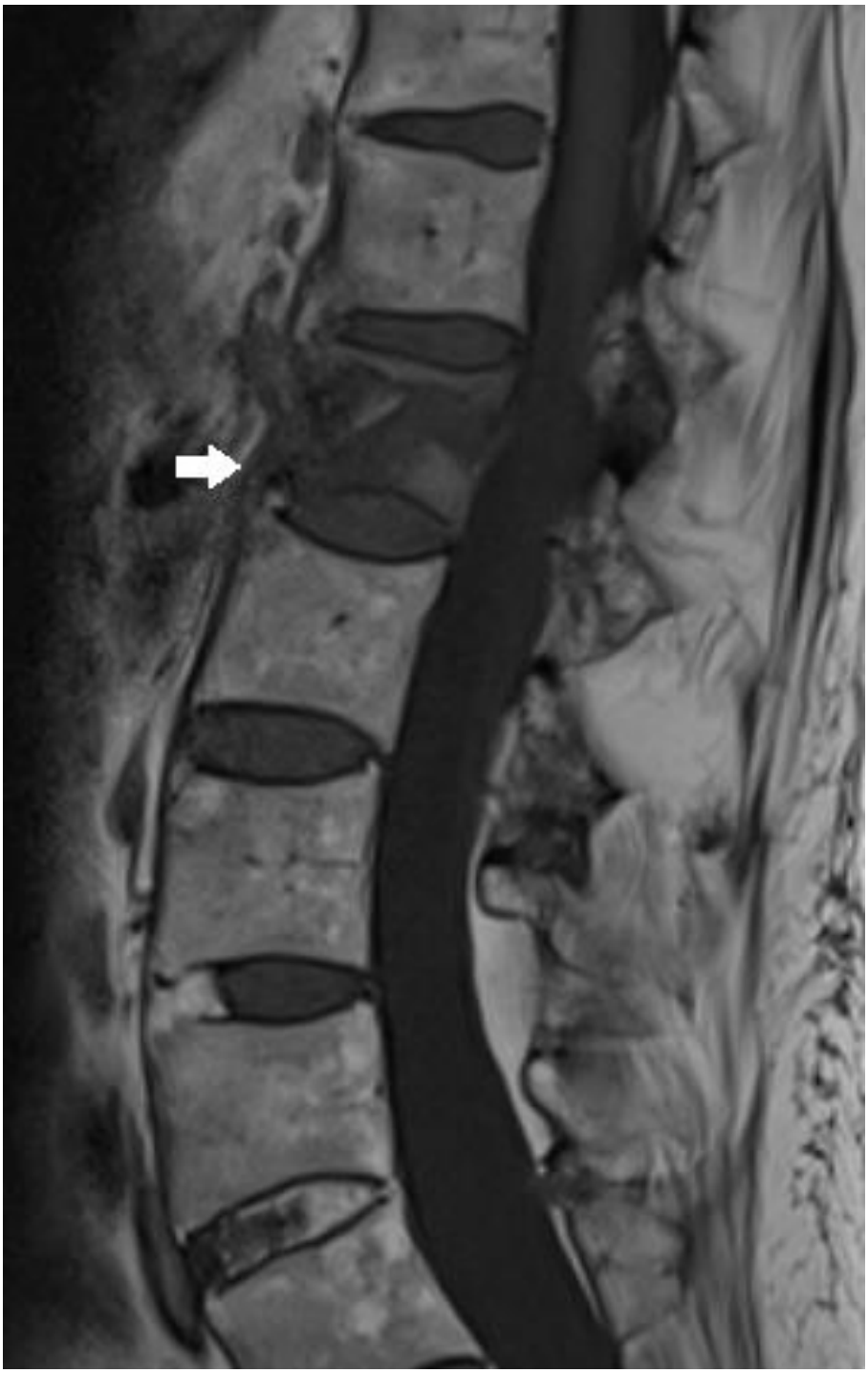
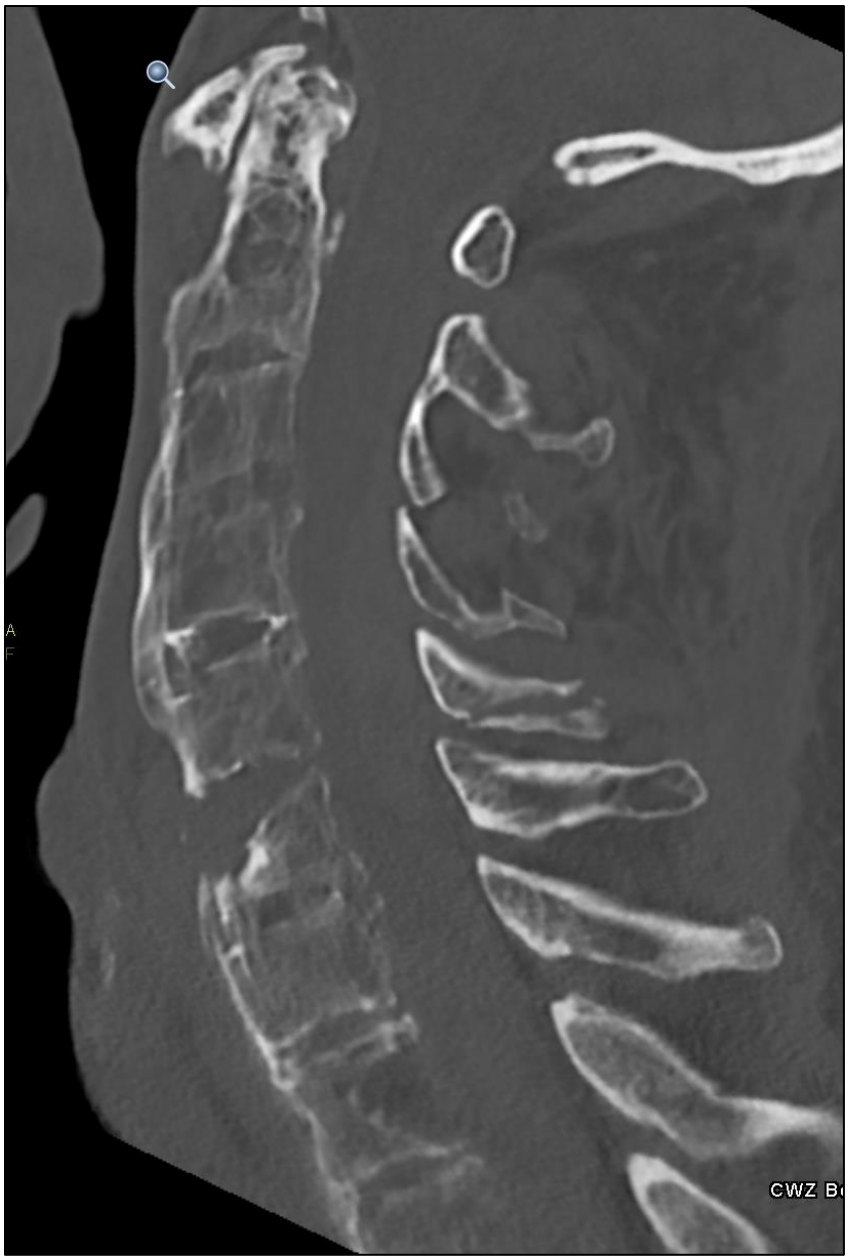


Fig. 9 Fractures in a 62-old female patient with ankylosing spondylitis. **a.** Mid**s**agittal reformatted CT showing a transvertebral fracture L1 (white arrow). **b.** Right parasagittal reformatted CT extension into the right pedicle (white arrow). **c.** Left parasagittal reformatted CT extension into the left pedicle (white arrow). **d.** Sagittal T1-weighted image bone marrow edema in the vertebral body L1 and disruption of the anterior longitudinal ligament (white arrow).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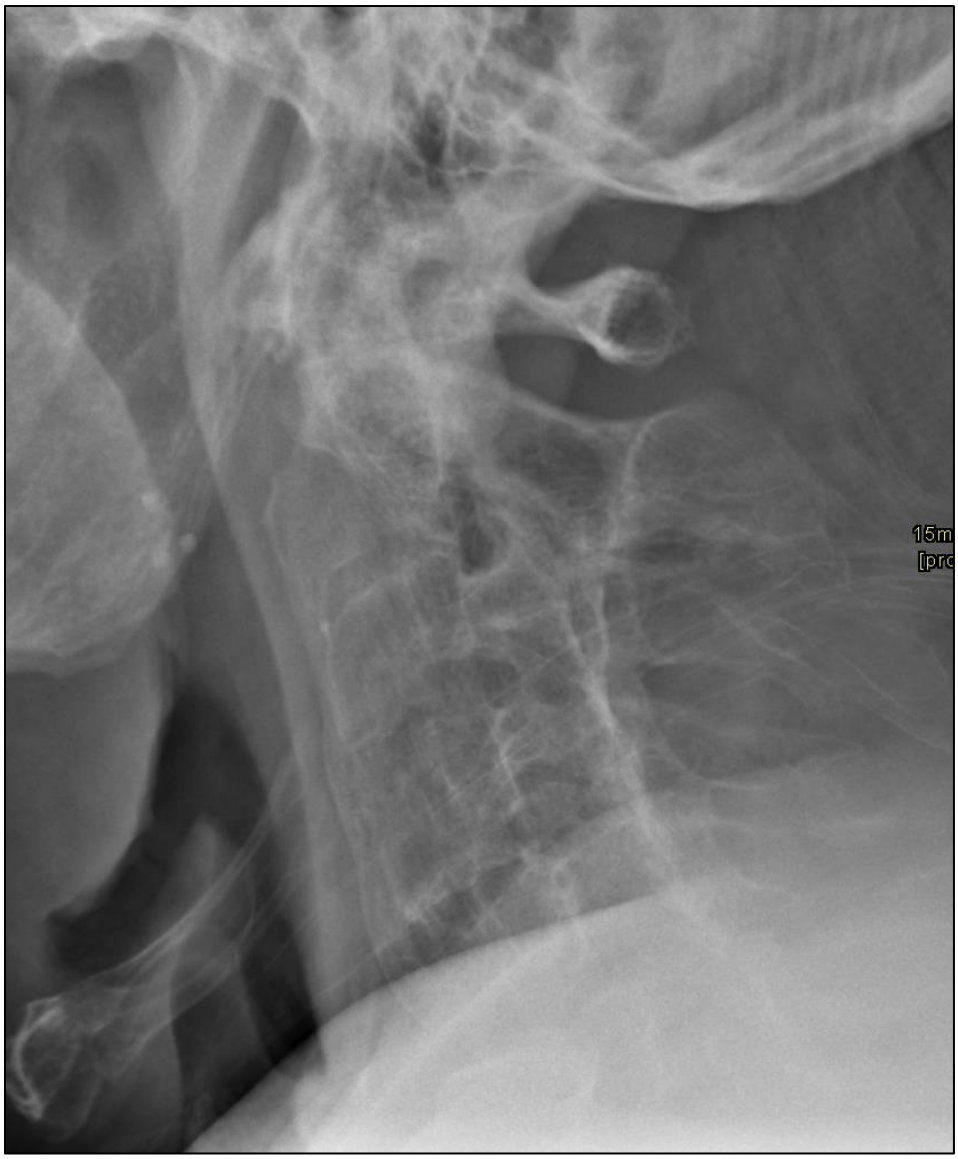
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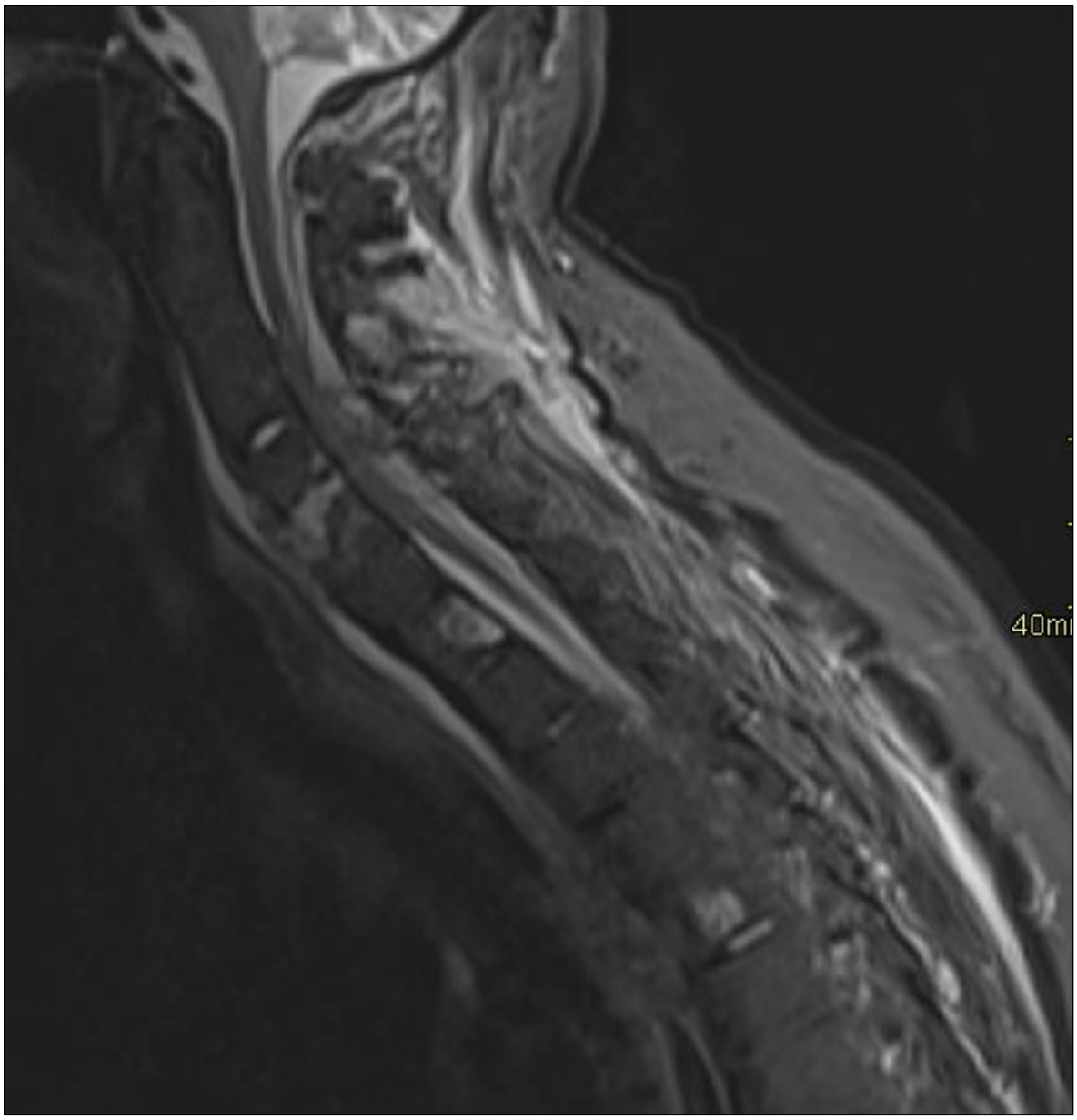
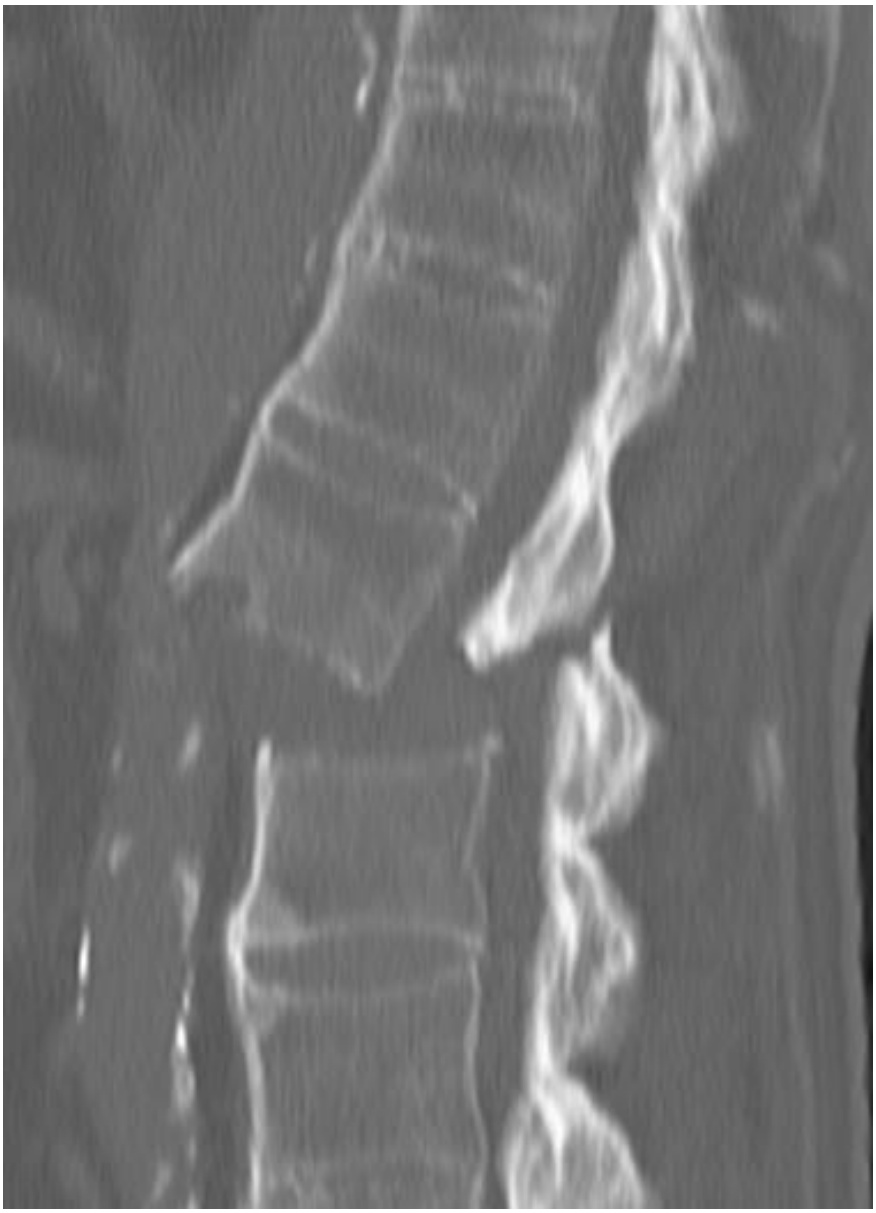
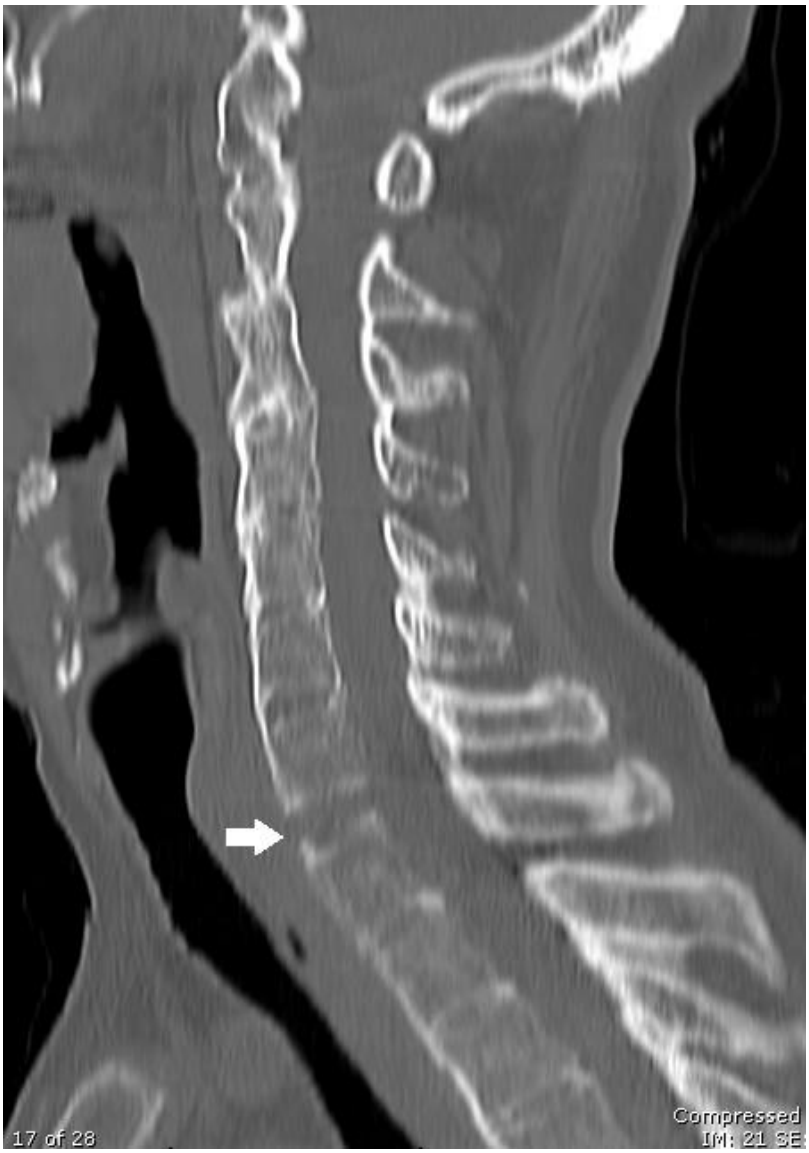


Fig. 10 Fracture of the cervical spine in a patient with DISH. **a**. Sagittal reformatted CT showing a transdiscal fracture C5-C6 partially involving the vertebral body C6 (white arrow) extending into the posterior elements (spinous process, black arrow) in keeping with involvement of the 3 columns of Denis. **b.** Lateral radiograph with improper positioning due to spinal rigidity failed to detect the fracture. **c.** Sagittal fatsuppressed T2-weighted image confirms partially transdiscal fracture (white arrow) extending into the posterior elements with disruption of the anterior longitudinal, interspinous, and supraspinous ligaments. The fracture is open anteriorly. There is a large fluid cleft in the disc space C5-C6 with adjacent bone marrow edema in the vertebral body C6. Note a posterior epidural hematoma compressing the spinal cord. Incidental haemangiomas are seen at the vertebral bodies Th1 and Th5.

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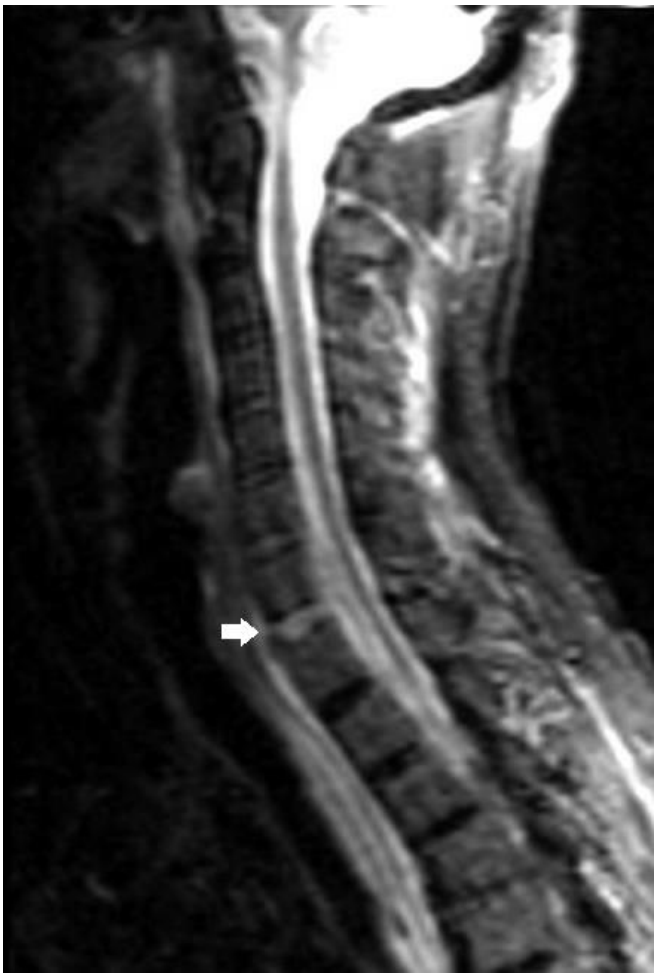

Fig. 11 Occurrence of multiple fractures at 2 different locations in the same patient. **a**. Sagittal reformatted CT of the thoracolumbar spine shows a transdiscal fracture extending into the posterior elements in keeping with involvement of the 3 columns of Denis. **b.** Sagittal reformatted CT of the cervical spine showing a transdiscal fracture between C7 and Th1 causing an enlarged disc space at this level (white arrow). **c.** On sagittal STIR of the cervical spine there is lack of bone marrow edema adjacent to the fracture (white arrow) because of the extension distraction mechanism of the trauma. The images are blurred due to motion artefact.

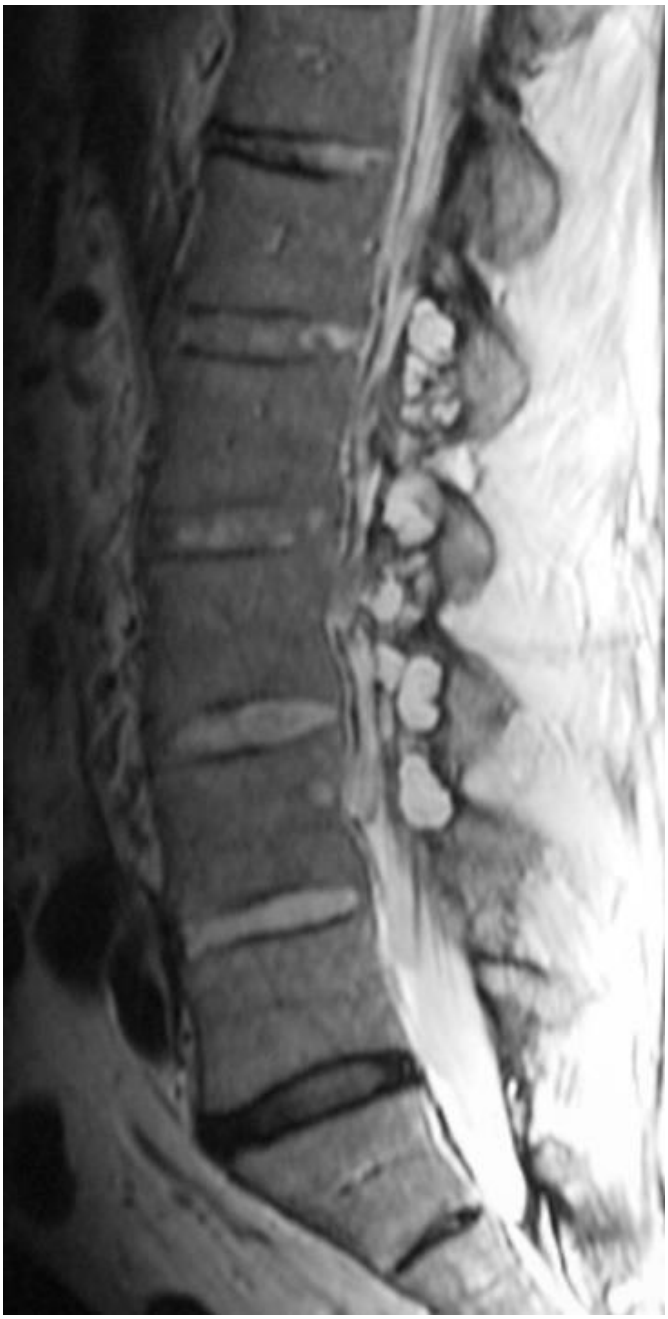

Fig. 12 Cauda equina syndrome complicating longstanding ankylosing spondylitis. Sagittal T2-WI showing adherent nerve roots and thecal diverticula causing lamina erosion.