Injuries to the Rigid Spine: What the Spine Surgeon Wants to Know

Nandish G. Shah, MD
Abhishek Keraliya, MD
Diego B. Nunez, MD, MPH
Andrew Schoenfeld, MD
Mitchel B. Harris, MD
Christopher M. Bono, MD
Bharti Khurana, MD

Abbreviations: ALL = anterior longitudinal ligament, AS = ankylosing spondylitis, DISH = diffuse idiopathic skeletal hyperostosis, DS = degenerative spondylosis, OPLL = ossification of the posterior longitudinal ligament, STIR = short τ inversion-recovery

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From the Department of Radiology, Brigham and Women's Hospital, 75 Francis St, Boston, MA 02115. Presented as an education exhibit at the 2017 RSNA Annual Meeting. Received April 1, 2018; revision requested May 15 and received June 12; accepted June 15. For this journal-based SA-CME activity, the authors A.S. and C.M.B. have provided disclosures (see end of article); all other authors, the editor, and the reviewers have disclosed no relevant relationships. **Address correspondence to** B.K. (e-mail: *bkhurana@bwh.harvard.edu*).

SA-CME LEARNING OBJECTIVES

After completing this journal-based SA-CME activity, participants will be able to:

- Describe the imaging features and altered biomechanics of various rigid spine entities.
- Characterize the injury patterns of a rigid spine at CT and MRI and the findings that may prompt surgical intervention.
- Recognize the causes of delay in diagnosing a rigid spine injury, its impact on outcomes, and how it can be prevented.

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The biomechanical stability of the spine is altered in patients with a rigid spine, rendering it vulnerable to fracture even from relatively minor impact. The rigid spine entities are ankylosing spondylitis (AS), diffuse idiopathic skeletal hyperostosis, degenerative spondylosis, and a surgically fused spine. The most common mechanism of injury resulting in fracture is hyperextension, which often leads to unstable injury in patients with a rigid spine per the recent AOSpine classification system. Due to the increased risk of spinal fractures in this population, performing a spine CT is the first step when a patient with a rigid spine presents with new back pain or suspected spinal trauma. In addition, there should be a low threshold for performing a non–contrast-enhanced spine MRI in patients with a rigid spine, especially those with AS who may have an occult fracture, epidural hematoma, or spinal cord injury. Unfortunately, owing to insufficient imaging and an unfamiliarity with fracture patterns in the setting of a rigid spine, fracture diagnosis is often delayed, leading to significant morbidity and even death. The radiologist's role is to recognize the imaging features of a rigid spine, identify any fractures at CT and MRI, and fully characterize the extent of injury. Reasons for surgical intervention include neurologic deficit or concern for deterioration, an unstable fracture, or the presence of an epidural hematoma. By understanding the imaging features of various rigid spine conditions and vigilantly examining images for occult fractures, the radiologist can avoid a missed or delayed diagnosis of an injured rigid spine.

Introduction

Rigid spine refers to a group of conditions that result in spontaneous or postsurgical ossification and fusion of the spinal segments. This pathologic condition leads to altered biomechanics and an increased susceptibility to fracture. Injuries of the rigid spine can be substantial even after seemingly innocuous events such as a low-energy fall from standing or sitting. The rigid spine entities are ankylosing spondylitis (AS), diffuse idiopathic skeletal hyperostosis (DISH), degenerative spondylosis (DS), and a surgically fused spine (Table 1). The manifestations of these underlying disorders and their imaging findings remain poorly understood, often leading to a delay in diagnosis on the part of the initial providers such as primary care or emergency

TEACHING POINTS

- The altered biomechanics and long lever arm of a rigid spine make the spine prone to fracture, even as a result of minor impact, such as a fall from standing or sitting.
- Due to the increased risk of spinal fractures, performing spine CT is the first step when patients with AS present with new back pain or suspected spinal trauma. Performing CT of the entire spine is recommended for patients with AS after an injury is detected owing to the increased risk of multiple noncontiguous fractures. In addition, there should be a low threshold to perform nonenhanced spine MRI in patients with a rigid spine, especially those with AS who may have an occult fracture, epidural hematoma, or spinal cord injury.
- For all rigid spine entities, the risk of fracture increases with a longer fused segment. Rigid spine fractures happen both within the ankylosed segment and the junctional zone between the fused and nonfused spine.
- Delayed diagnosis occurs because patients with a rigid spine present with minor trauma in the setting of acute on chronic pain, leading to insufficient imaging for diagnosis. Reasons for delayed diagnosis leading to secondary deterioration include a wrong initial assessment, missed fractures at imaging, and unrecognized epidural hematomas.
- The radiologist's role is to recognize the imaging features of a rigid spine and closely scrutinize the spine for subtle fractures at CT and MRI. One must fully characterize the extent of injury to help guide management. Reasons for surgical intervention include neurologic deficit or concern for deterioration, an unstable fracture, or the presence of an epidural hematoma.

physicians, as well as radiologists. For radiologists, a delay in diagnosis of fracture results from inadequate imaging, lack of awareness of the fracture patterns, and poor perception of the clinical significance of these injuries. Unfortunately, for patients with a rigid spine, a delay in diagnosis can impact the timing of surgical intervention, which can also lead to neurologic deterioration with significant morbidity and even death. The purpose of this article is to educate radiologists about the biomechanical alterations in the rigid spine, the fracture patterns associated with each entity, and the relevant features of injury that may require spine surgeons to intervene. By raising awareness, we hope that radiologists will avoid delays in diagnosing an injured rigid spine.

Biomechanical Alterations in the Rigid Spine

Stability is defined as the ability of the spine to prevent neurologic injury or pain during physiologic range of motion or axial loading (1). In a healthy spine, the normal lordosis and kyphosis of the spine allow for the balanced weight distribution of the head and upper body. The spinal components (vertebral body, intervertebral disk, and spinal ligaments) work together to provide biomechanical stability.

A rigid spine alters the biomechanics by affecting different spine-stabilizing components. In AS, there are three main ways this occurs. First, osseous fusion of the vertebral bodies eliminates the resistance and shock absorption provided by the intervertebral disks. More load bearing shifts to the vertebral bodies, which are generally already compromised from osteoporosis. A 25% decrease in mineralization leads to a 50% decrease in resistance (1). Second, the ankylosis transforms the spine into a singular rigid construct, creating a long lever arm that is more susceptible to fracture. Third, a kyphotic deformity of the spine is common in AS, which shifts the center of gravity forward, creating more shear stress forces (2). Unfortunately, end-stage kyphosis also leads to a fixed downward gaze affecting spatial recognition, making patients even more prone to falls (3).

In DISH, ossification of the ALL with osteophyte bridging to the anterior vertebral bodies results in the placement of the load on the anterior aspect of the vertebral body with minimal contribution from the disk (4). As the fused segment of the spine poorly dissipates axial loads, the proximal and distal nonfused segments are placed under increased stress, predisposing them to injury (5).

The motion segment (intervertebral disk, facet joints, and associated ligaments) is affected in advanced DS (6). Specifically, there is loss of the disk space and facet joint osteoarthritic hypertrophy. The loss of the disk space shifts load-bearing disproportionately to the facets, which are ill-equipped to handle the weight. Per Wolff law, the lack of stress on the anterior aspect of the vertebral bodies leads to focal bone loss, predisposing them to compression fractures (1). In a normal spine, axial loading is predominantly borne by the intervertebral disks, with the facet joints sharing up to one-third. In advanced DS, the altered biomechanics shift the axial load to the facet joints, which can be up to 70% in the lumbar spine. This significantly increases the risk for fracture even from low-impact trauma (6).

A surgically fused spine is a rigid segment that becomes unable to dissipate axial loads. Thus, the stress is transferred to the proximal and distal adjacent nonfused segments. The increased stress and hypermobility of the nonfused spine segments compared with those of the fused segments places the junction between the two at high risk for fracture (7).

In summary, regardless of the underlying cause, the altered biomechanics and long lever arm of a rigid spine make the spine prone to fracture, even as a result of minor impact, such as a fall from standing or sitting.

Table 1: Key Features of Rigid Spine Entities

Entity	Epidemiology	Imaging Features
AS	Diagnosed in men in their 3rd decade of life; 1% prevalence; male predominant	Symmetric arthropathy of the sacroiliac joints occurs first Ossification occurs and progresses upward to involve the vertebral bodies, facet joints, intervertebral disks, and spinal ligaments Costovertebral joint ankylosis
DISH	Affects patients in their 6th or 7th decade of life Up to 25% prevalence, which is increasing owing to conditions such as obesity and diabetes Male predominant	Bridging ossification of the anterior longitudinal ligament (ALL) spanning four or more vertebral bodies Spares the sacroiliac joints Normal intervertebral disks Commonly involves T7-T11 and thoracolumbar junction Spinal stenosis from ossification of the posterior longitudinal ligament (OPLL)
DS	Affects elderly patients, with incidence increasing with age	Advanced osteoarthritis of the spine Facet joint osteoarthropathy Narrowing of the intervertebral disk space Commonly involves C3-C5 and L4-S1 Posterior osteophytes can cause spinal stenosis
Surgically fused spine	Can affect patients of any age, but older patients are more at risk for fracture	Osseous fusion, with or without spinal hardware Fusion is of the intervertebral disks or transverse processes Degenerative disk disease is at the junction of the fused and nonfused spine

AOSpine Classification for Injuries of the Rigid Spine

Spinal instability results from the inability of the vertebrae to maintain a normal range of displacement under physiologic loads. This puts a patient at risk for neurologic deficit, deformity, and pain (8). Traumatic instability refers to the acute effects of trauma on the vertebrae that make it unstable and prone to spinal cord injury (9).

Several classification criteria have been used to characterize spine stability. A classification system must provide accurate injury characterization in a clinically relevant manner and show intra- and interobserver reliability (10). The Denis system, which uses a 3-column approach, and the Thoracolumbar Injury Classification System (TLICS) each had drawbacks that affected their clinical utility (11). The authors of the recent AOSpine classification system learned from these prior classification systems how to best direct clinical management. This system encompasses both cervical and thoracolumbar injuries (11).

The AOSpine classification system incorporates fracture morphology and neurologic injury and has two modifiers, with M2 describing rigid spine entities, especially AS (10). It is important to recognize this M2 modifier as it can alter surgical management. The fracture morphology is categorized into three injury types: *(a)* compression; *(b)* lengthening or distraction, which is a loss of the physiologic tension band; and *(c)* translation (10). Type A injuries focus mostly on the vertebral body, with the highest category, A4, representing a complete burst fracture. Type B injuries involve fractures that go beyond the vertebral body and those that also cause ligamentous disruption. The highest category is B3, or hyperextension. Type C injuries are the most severe and involve displacement or dislocation of the spine that often makes it unstable (11).

Studies to validate this classification system have shown that almost all type C injuries warrant surgical intervention (11). In the thoracolumbar spine, type B3, or hyperextension injuries, also warrant surgery (11). In the cervical spine, while a hyperextension injury alone may not warrant surgery, it is warranted when there is a rigid spine M2 modifier (11). Neurologic injury at clinical presentation is also an important factor when determining the benefit of surgical intervention (10).

As the AOSpine classification system enters clinical practice, understanding its nomenclature will be essential for radiologists. Knowing there is a modifier for a rigid spine like that seen in AS and mentioning this in the radiology report will assist the spine surgeon in management.

Ankylosing Spondylitis

AS is an immune-mediated spondylosis and the most common seronegative spondyloarthropathy (5). The prevalence is 14 per 100 000 patients per year and may be as high as 1.4% (12). There is a 2:1 male predominance, and the average age of onset is 28 years (5). Eighty percent of patients see symptoms before age 30 (12). Women are often diagnosed later in life than men, have milder

Table 2: Classic Radiographic Signs of AS

Radiographic Sign	Description
Sacroiliitis	Symmetric and bilateral subchondral erosions and sclerosis followed by ankylosis at the inferior aspect of the sacroiliac joints
Shiny comers	Reactive sclerosis at anterior vertebral body erosions, also known as Romanus lesions
Vertebral body squaring	Loss of the normal concavity of the anterior vertebral body; progression of Romanus lesions
Bamboo spine	Syndesmophytes, or marginal osteophytes, cause ankylosis of the vertebral bodies, creating a bamboo-like appearance
Dagger spine	Ossification of the supraspinous and interspinous ligaments creates a long thin ossified line, which looks like a dagger

symptoms, and have more cervical spine involvement (13). There is a strong association with the human leukocyte antigen (HLA)-B27 antigen, a genetic factor, among white patients and up to 20% have a positive family history (12).

The pathophysiology of AS involves a T-cell–mediated attack on the attachment sites of ligaments, tendons, and joint capsules. The inflammation causes cartilage destruction and bone erosion, which lead to reactive sclerosis and eventually new bone formation (5). New bone formation in AS manifests as syndesmophytes (ossifications inside a ligament) that occur at the margins of the annulus fibrosus, creating bony bridging of the vertebral bodies. These ankyloses of AS have also been described as marginal osteophytes in that they do not extend beyond the margin of the vertebral bodies.

A hallmark of AS is sacroiliitis, which is seen on anteroposterior (AP) radiographs. This and other pathophysiologic changes lead to classic imaging signs depicted on radiographs as detailed in Table 2 (14). The facet joints are also affected in AS by capsular enthesitis, which causes ossification in a bony shell–enclosing pattern (15).

AS is predominantly a clinical diagnosis with sacroiliitis manifesting as morning stiffness with lower back, sacrum, and/or buttock pain that improves with activity (5). Uveitis is the most common extra-articular symptom and is seen in up to 20% of patients with AS (5). The Modified New York criteria for AS does include radiographic evidence of sacroiliitis as a criterion, which is graded from 0 (normal) to 4 (ankylosis) (12). As the clinical diagnosis of early AS remains challenging, radiologists must be vigilant in looking for imaging signs of disease, especially in patients in whom disease may have gone undiagnosed.

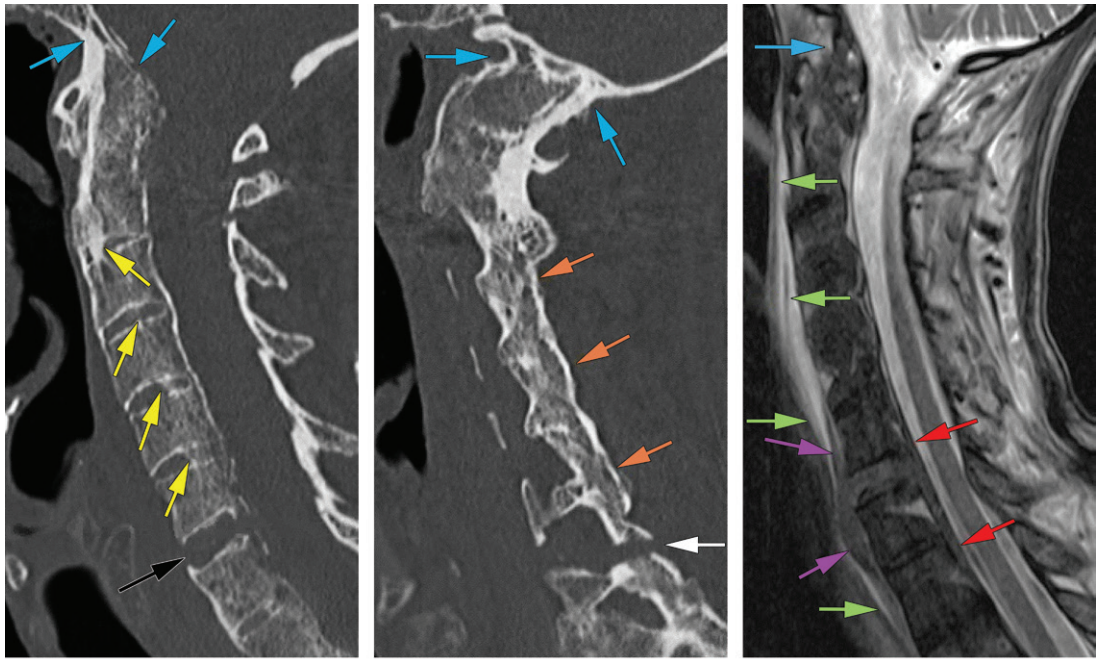
AS also predisposes patients to spine fracture by causing osteoporosis, which manifests in up to 62% of patients with AS (5). Osteoporosis occurs owing to an increase in bone resorption and inadequate bone formation in areas of chronic inflammation (2). In addition, an immobile fused spine inhibits the neurologic signals for new bone formation in the vertebral bodies (16). The severity of osteoporosis correlates with the degree of ankylosis (5). Importantly, osteoporosis increases the risk of vertebral compression fractures and can create problems during surgical fusion (12).

Additional aspects that differentiate AS from other rigid spine entities include kyphotic deformity, ankylosis of the craniocervical junction, and ankylosis of costovertebral joints, which also increase the fracture risk. Ankylosis of costovertebral joints, a hallmark finding of AS, results in chest wall rigidity, which makes patients highly susceptible to lung infections and deterioration (12).

Overall, patients with AS have a sevenfold increase in the incidence of spinal fracture compared with that of the general population (5). The risk of fracture increases with the length of ankylosed spine and with disease duration (16). One meta-analysis found the average age at the time of injury to be 63 years, while in another study over 50% of the injuries occurred after 70 years of age (12,17). Importantly, severe fractures can occur even with minimal trauma such as a fall from standing or sitting (18). Other causes include patient transfers and intubation (5).

The most common mechanism of injury is hyperextension, occurring in over 70% of cases, followed by flexion injuries seen 20% of the time (19,20). In fact, hyperextension is the most common cause of injury in the rigid spine. Cervical spine fractures are more common than thoracic or lumbar spine fractures, with most occurring from C5 to C7 and at the cervicothoracic junction, C7-T1 (20,21) (Fig 1).

As the rigid spine acts as a long lever arm, like a long bone in the extremities, fractures are often transverse. In AS, the fracture line more commonly passes through the disk space rather than the vertebral body. This is likely because the calcified disk is the weakest link along the ankylosed vertebral bodies (16). Nonetheless, the fractures



a. b. c.

Figure 1. Hyperextension fracture in an 86-year-old man with AS who fell in his bathroom and presented with neck pain. **(a)** Sagittal spine reformatted CT image shows a hyperextension fracture (black arrow) at the C6-7 disk. Atlanto-occipital ankylosis (blue arrows) is also depicted, also known as craniocervical ankylosis, which can occur in patients with AS. Another characteristic feature of AS is the bridging syndesmophytes (yellow arrows), which cause ankylosis of the vertebral bodies. **(b)** Sagittal spine reformatted CT image shows the fracture (white arrow) extending through the posterior elements in the left C6-C7 ankylosed facet joint. There is diffuse ankylosis of the facet joints (orange arrows) and atlanto-occipital ankylosis (blue arrows). **(c)** Sagittal T2-weighted MR image shows extensive prevertebral soft-tissue edema (green arrows) and disruption of the ALL, with a hematoma anterior to C6 and C7 (purple arrows). Note the disruption of the posterior longitudinal ligament, with a ventral epidural hematoma extending from C6 to C7 (red arrows), and the atlanto-occipital ankylosis (blue arrow).

often extend to involve the vertebral bodies, especially in those with long-standing disease. Importantly, multiple noncontiguous fractures occur in up to 20% of the cases (5,12) (Fig 2). The majority are a combination of cervical and thoracic fractures followed by thoracic and lumbar fractures (12).

The prevalence of spinal cord injury in patients with AS is more than 11 times greater than that in the general population (22,23). In one study, it was seen in over 20% of cases (17). In another, AS fractures were complicated by neurologic deficit 60% of the time (5). The rate of neurologic injury is similar for both cervical and thoracic fractures (5,23). Up to 23% of spine fracture cases reported concurrent epidural hematomas, which can extend for a long segment and predispose patients to neurologic injury (12) (Fig 3).

While radiographs can help diagnose AS through the depiction of bridging osteophytes, they are poor at depicting fractures (12,24). One reason is that the lower cervical spine and cervicothoracic junction are hard to visualize on spinal radiographs. Also, subtle hyperextension and transverse spinal fractures can be missed at radiography, and the degree of injury can be underestimated (16). Due to the increased risk of spinal fractures, performing spine CT is the first step when patients with AS present with new back pain or suspected spinal trauma. Performing CT of the entire spine is recommended for patients with AS after an injury is detected owing to the increased risk of multiple noncontiguous fractures. In addition, there should be a low threshold to perform nonenhanced spine MRI in patients with a rigid spine, especially those with AS who may have an occult fracture, epidural hematoma, or spinal cord injury (Fig 4).

MRI can help in the diagnosis of unstable fractures by assessing the posterior ligamentous complex (PCL) and spinal cord compression (5). As injury in the setting of a rigid spine can be substantial, any patient with a rigid spine with acute back pain or trauma must be treated with a high suspicion for fracture until proven otherwise.

Diffuse Idiopathic Skeletal Hyperostosis

DISH is a noninflammatory condition where there is widespread ossification and formation of large osteophytes adjacent to the spinal segments,

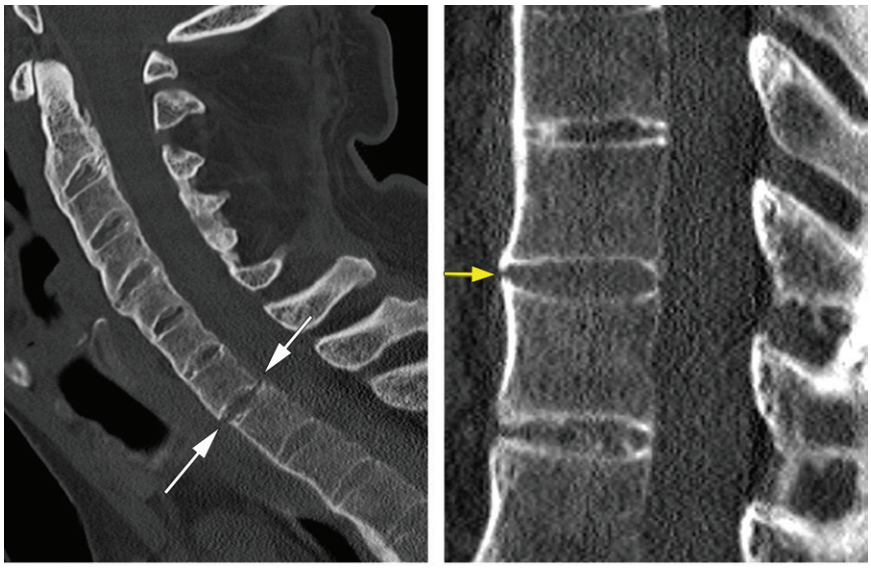


Figure 2. Noncontiguous fractures in a 48-year-old man with AS who fell from standing and presented with back pain. **(a)** Sagittal CT image shows ossified anterior and posterior ligaments with facet joint ankylosis, findings consistent with AS. There is a mild anterior translation (arrows) of C7 on T1, with discontinuity of anterior fusion. Of note, the patient was newly diagnosed with AS on the basis of these imaging findings. A whole-spine CT was performed owing to the risk of multiple noncontiguous fractures. **(b)** Sagittal CT image shows an additional nondisplaced T11-T12 fracture (arrow). Note that the gap at the anterior aspect of T12-L1 is an area that is not ankylosed and is similar to that seen at prior imaging.

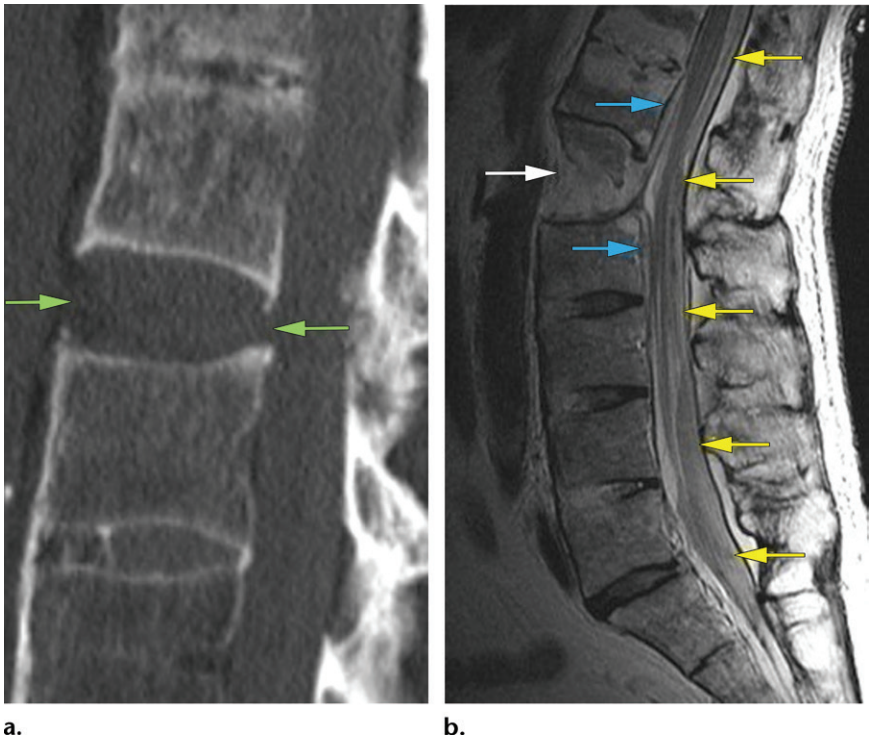
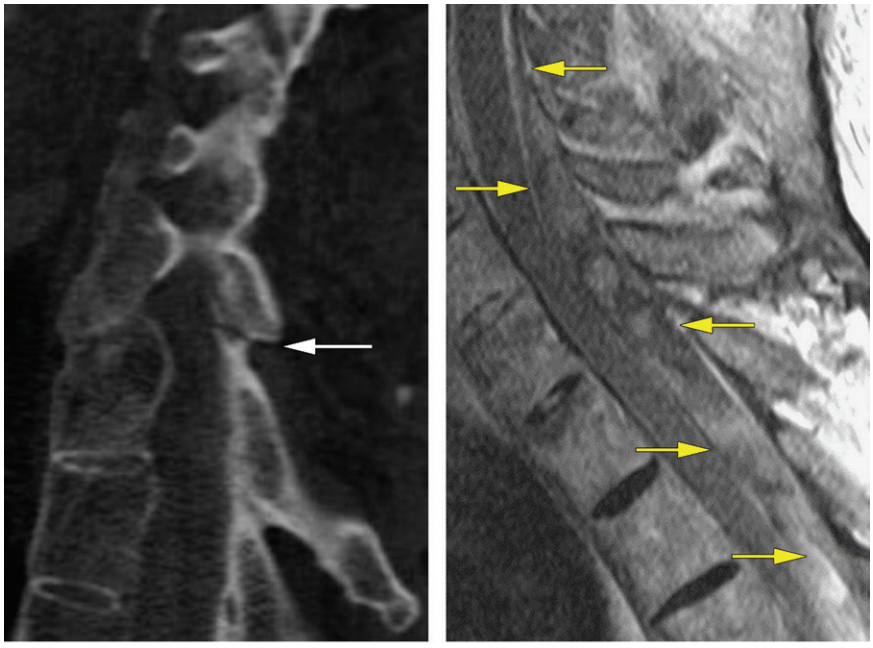


Figure 3. Hyperextension injury in a 50-year-old man with AS who slipped on ice and landed on his back. **(a)** Sagittal CT image shows distraction of the L1–2 disk space, with disruption of anterior and posterior fused ossified ligaments (arrows), a finding consistent with hyperextension injury. **(b)** Sagittal T2-weighted MR image shows the disruption of the ALL, disk, and posterior longitudinal ligament, with mixed intradiscal fluid and blood (white arrow), epidural hematoma (blue arrows), and extensive subdural and subarachnoid hematoma (yellow arrows).

sometimes associated with osseous fusion. This condition occurs in patients in their 6th or 7th decade of life, and there is a male predominance. The prevalence in the United States is 25% in men and 15% in women over 50 years of age (25). This is likely to increase as there is an association with obesity and diabetes and as the life expectancy of this population is increasing (5).

The pathogenesis of DISH remains unknown, but there is an association with metabolic abnormalities. DISH involves ossification of the ALL. This leads to bridging ossification of multiple vertebral bodies with the ALL forming the imaging characteristic of flowing osteophytes. The most common area affected is the lower thoracic spine, specifically T7 to T11, and the thoracolumbar junction (26). In the thoracic spine, the bridging ossification is seen in the anterior and right lateral side of the vertebral bodies; the left lateral side is spared owing to pulsations from the descending aorta (4). DISH may also involve other areas of the spine, namely the upper lumbar and lower cervical spine (5). Ossification of the ligamentum flavum in the lumbar spine and OPLL in the cervicothoracic spine can lead to spinal stenosis (12).



a. b.

Figure 4. Dorsal epidural hematoma in an 89-year-old man with AS who presented with neck pain after a fall. **(a)** Sagittal reformatted CT image shows a nondisplaced C7-T1 right facet fracture (arrow). Because of the patient's known AS diagnosis, neck pain, and trauma, MRI was performed. **(b)** Sagittal T2-weighted MR image shows an extensive C3-T3 dorsal epidural hematoma (arrows) adjacent to the thecal sac.

DISH is often diagnosed at radiography or CT (Table 1). Classically, the flowing osteophytes along the anterior spine involve at least four contiguous vertebral bodies, but more recent studies suggest three is sufficient to meet the criteria for the condition if there is peripheral enthesopathy (4). The osteophytes in DISH are described as nonmarginal in that they protrude beyond the borders of the vertebral bodies. Many patients with DISH have only one segment of bridging vertebral body ossification. Also, unlike those in patients with AS and DS, the intervertebral disks are preserved. Lastly, there is no facet joint ankylosis, bone changes from sacroiliitis, or costovertebral joint ankylosis, which are findings specific to AS (27).

Most commonly, DISH is asymptomatic. Pain and stiffness of the spine are the common presentations when patients are symptomatic. In advanced disease, patients may develop a kyphotic deformity like that encountered in AS. DISH in the cervical spine can also make intubation difficult. In the lumbar spine, patients can develop lumbar radiculopathy (12).

Unlike in AS, studies do not show vertebral osteoporosis in association with DISH (26). Therefore, risk for spinal fractures is primarily related to the altered biomechanics of a rigid spine. A hallmark of the rigid spine is fracture in the setting of low-impact trauma such as a fall from standing. For all rigid spine entities, the risk of fracture increases with a longer fused segment. Rigid spine fractures happen both within the ankylosed segment and the junctional zone between the fused and nonfused spine (Fig 5). This is from the increased mechanical stress on the nonfused spine adjacent to the rigid spine.

Unlike in AS, DISH fractures more often occur through the vertebral bodies, especially in older patients who have had DISH for a longer time (25). This is because the degree of anterior ossification is highest at the intervertebral disk space and lowest at the midvertebral body (28). The midvertebral body becomes a weak point at higher risk for fracture (5). Fractures at the ends of the fused spine can extend from the vertebral-body endplates through the adjacent intervertebral disk (4). The lower thoracic spine, specifically the thoracolumbar junction from T11 through L2, and the lower cervical spine from C5 through C7, like that seen in AS, are common sites of fracture (4,19,29). Hypertension injury is the most common mechanism, but it is followed closely by spine-translation injuries, which are classified as type C under the AOSpine classification system and inherently unstable spine injuries (19).

The rate of neurologic injury with DISH fractures is high, with one study reporting some degree of compromise in 40% of patients at the time of admission (19). This high rate is relatively proportional to the rate of unstable spinal fractures in patients with DISH (5). Due to a high prevalence of OPLL in patients with DISH, which causes spinal stenosis, the cervical spine is most vulnerable to neurologic injury (25,30). Studies show a significant correlation between the likelihood of neurologic injury and the length of the ankylosed segment rather than the trauma severity (29). In addition, the longer the ankylosed segment, the more severe the resultant spinal cord injury (31).

CT is the imaging modality of choice when assessing for fracture in a patient with DISH. Radiography should not be used to diagnose injuries. If a fracture is detected, subsequent CT imaging of the entire spine is strongly recommended as patients can have multiple noncontiguous fractures (Fig 6). For nondisplaced fractures, it is important to determine the horizontal extent of the fracture line (29). DISH fractures that do not extend to the posterior elements—facets, lamina, and spinous process—may be managed conservatively if a patient does not have neurologic deficit. This includes placing a brace and obtaining standing plain radiographs (32).

The use of MRI in DISH is an ongoing area of research without consensus. Unlike those in AS, fractures in DISH often do not extend to the posterior elements because there is no facet joint ankylosis or ossification of the spinous process ligaments. One study showed that patients without fracture of the posterior elements or spinous ligaments at CT did not show additional injury at MRI to suggest an unstable fracture pattern (29) (Fig 7). Of course, in the setting of an unstable spine injury at CT or neurologic deficit, spine MRI can provide additional information to guide surgical management (29).

Degenerative Spondylosis

DS refers to age-related osteoarthritis of the spine (12). The loss of the spine's motion segment (intervertebral disk space and bilateral facet joints) alters the biomechanics and creates a rigid spine (6). It is more common in men and associated with obesity. Also, the prevalence for advanced DS increases with age. One group of authors researched facet joint osteoarthritis in the cervical spine and found a prevalence of 57% in adults aged 65 years and older, while another group researched the lumbar spine and found a prevalence of 89% in the same age group (6).

The pathophysiology of DS is like the osteoarthritic changes in the extremities, namely loss of joint space, subchondral sclerosis, and eventually osteophyte formation. Changes are seen predominantly in the cervical and lumbar spine. Facet joint osteoarthritis is strongly associated with disk degeneration and often occurs in tandem (6). In advanced DS, a complete loss of the disk and facet joint space along with bridging osteophytes can restrict motion. The midcervical spine (C3-C5) and the lower lumbar spine (L4-S1) are the most affected.

An important aspect of advanced DS, one that it shares with DISH, is its propensity for causing spinal stenosis, which occurs from osteophytes at the posterior intervertebral disk space and hypertrophic changes around the facet joints (33).

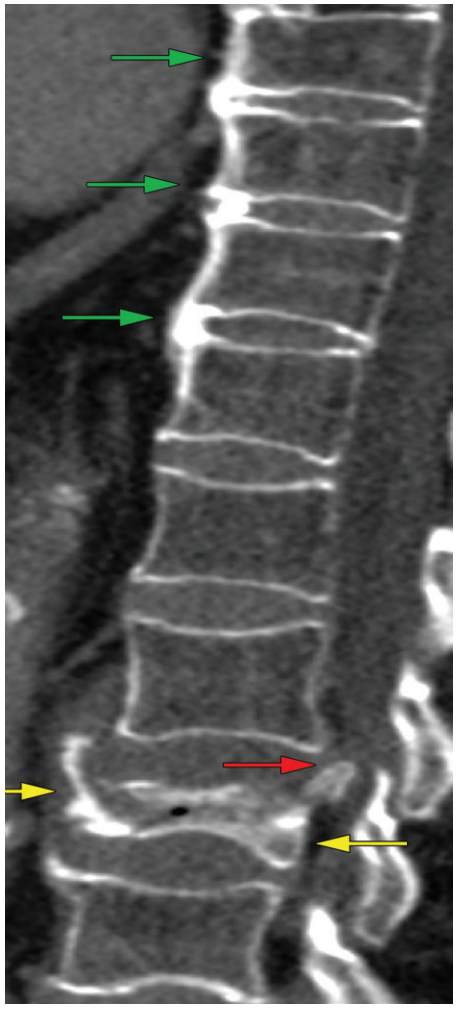
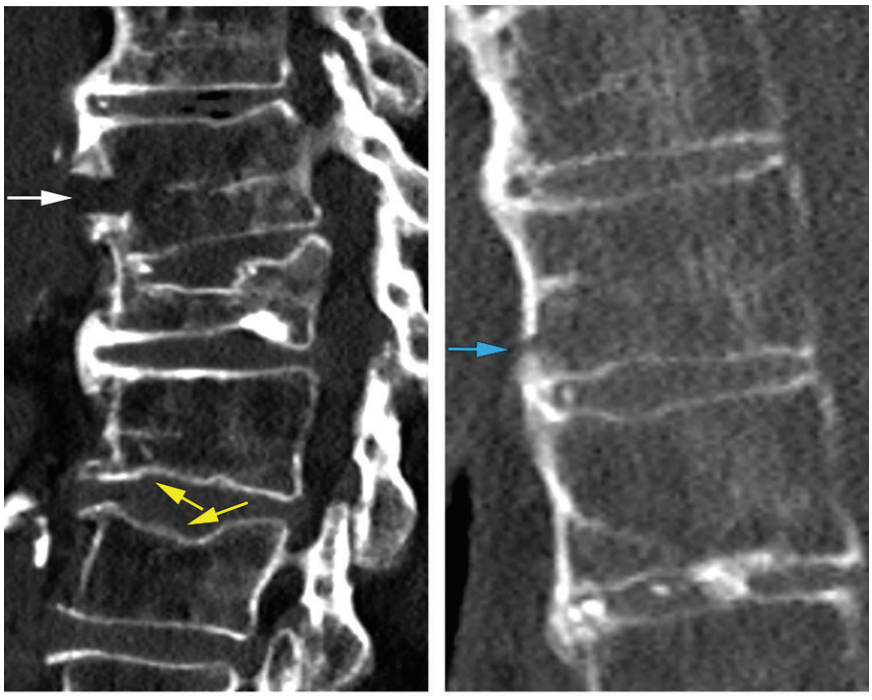


Figure 5. Rigid spine fracture in an 89-year-old man with DISH who fell back onto his bed and developed back pain. Sagittal CT image shows fused anterior flowing osteophytes (green arrows) in the lower thoracic spine, but the fracture was lower. An L3 burst compression fracture (yellow arrows) with retropulsion causing severe canal stenosis (red arrow) is depicted. Rigid spine fractures can occur just above or below the fused segment.

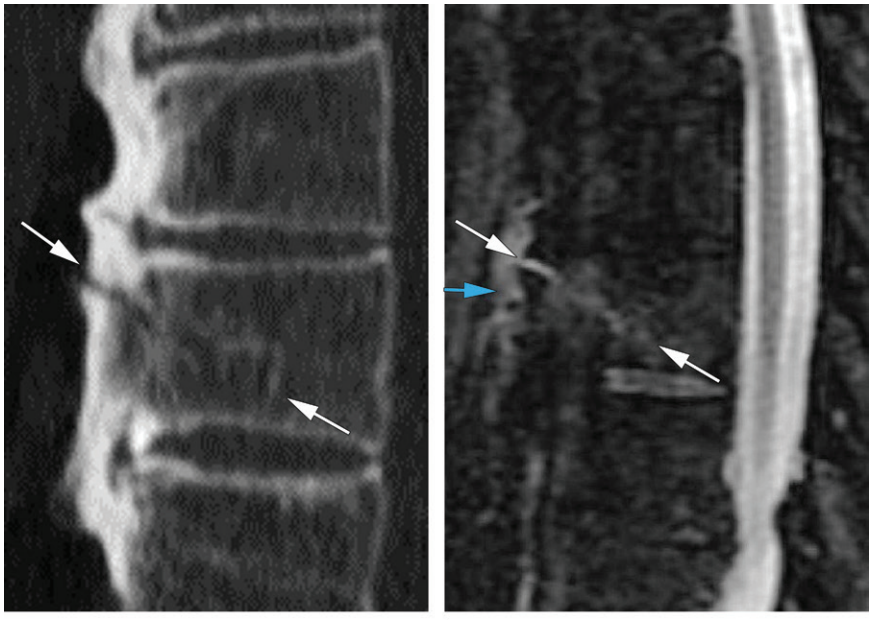
DISH and DS differ in that the intervertebral disks are preserved in DISH. Like AS, DS does affect the facet joint, but it does not cause spinal ligament ossification or bridging syndesmophytes. Also, ankylosis of the costovertebral joints is a feature of AS, not of DS (12). Transverse fractures, often through the vertebral bodies, are seen in advanced DS and often extend to the posterior elements, the facets. Clinically, patients present with neck or low back pain that can radiate to the proximal upper and lower limbs, respectively. Unlike that of AS, the pain is relieved with rest (5).

Performing CT is imperative for fracture assessment. Degenerative changes obscure fracture lines and the degree of injury on a radiograph. Even at CT, subtle fracture lines can be challenging to find in the setting of joint-space narrowing



a. b.

Figure 6. Noncontiguous fractures in a 91-year-old woman with DISH who fell from her walker and presented with acute on chronic back pain. **(a)** Sagittal CT image shows a transverse distraction fracture (white arrow) of the L1 vertebra. Mild L3 and L4 compression injuries (yellow arrows), subacute to chronic in appearance, and a severe L2 compression deformity were unchanged from a prior imaging study. CT of the whole spine was performed after these fractures were diagnosed. **(b)** Sagittal CT image through the midthoracic spine shows an additional anterior transverse fracture (arrow) of T8.



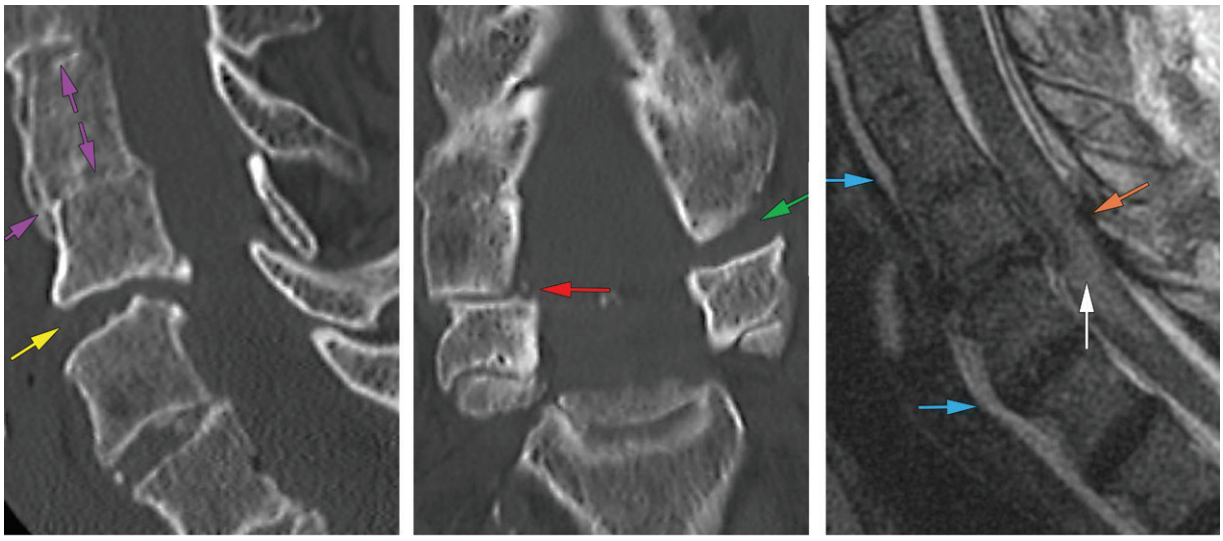
a. b.

Figure 7. ALL injury in a 55-year-old man with DISH who slipped in the bathtub and presented with back pain. No neurologic deficit was diagnosed. **(a)** Sagittal CT image shows a transverse midvertebral body fracture (arrows) at T8 that does not extend through the posterior vertebral cortex or posterior elements. MRI was performed to assess for ligamentous injury. **(b)** Sagittal short τ inversion-recovery (STIR) MR image shows the fracture (white arrows) and edema around the ALL (blue arrow), but there is no injury to the posterior longitudinal ligament or spinal cord. The injury was conservatively managed.

and osteophytes. For patients with advanced DS affecting the facet joints, careful attention must be given to the posterior elements, as a fracture line extending there suggests an unstable injury (Fig 8). Therefore, the spine should be assessed by using spine CT protocols, not bone windows at CT of the neck, chest, abdomen, or pelvis (34). This is true for all rigid spine entities. Because of underlying spinal stenosis, patients with these entities are at increased risk of neurologic injury from low-impact trauma (6). MRI must be performed if patients are diagnosed with acute neurologic deficits (Fig 9).

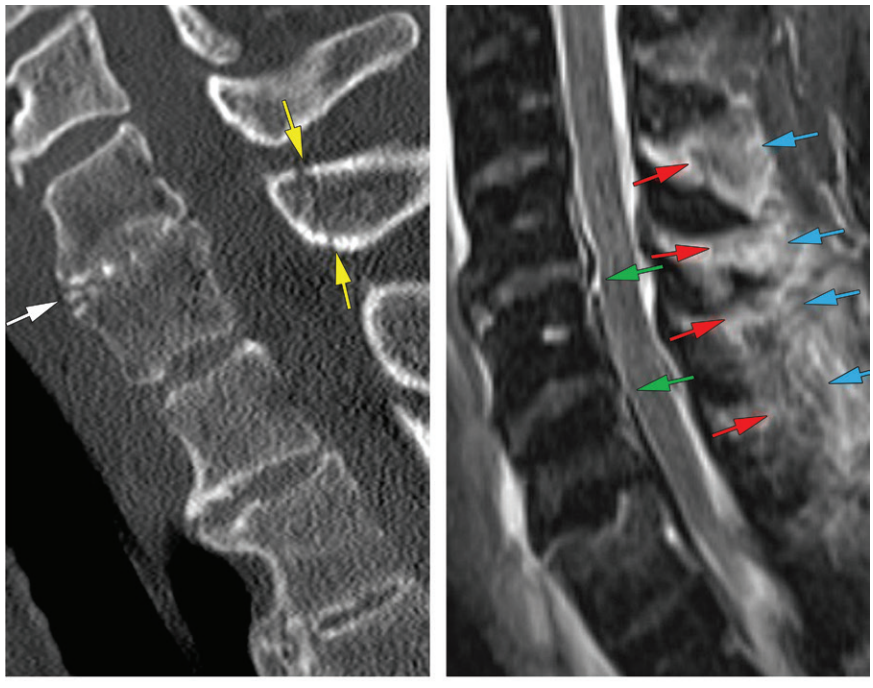
Surgically Fused Spine

Surgical fusion of the spine is a common procedure with many indications, such as an acute fracture causing neurologic deficit and spinal stenosis, with spondylolisthesis from age-related degeneration. Unlike surgical fixation, spinal fusion, with or without spinal hardware, involves using bone graft material to fuse adjacent vertebral structures.



a. b. c.

Figure 8. Unstable injury in an 85-year-old woman with DS who presented after a fall from standing. **(a)** Sagittal CT image shows degenerative osseous bridging of the cervical spine (purple arrows) and a C5-C6 hyperextension injury (yellow arrow) next to the fused segment. **(b)** Coronal CT image shows a fracture-translation injury of the right C5 facet (red arrow) and widening of the contralateral C5-C6 facet joint (green arrow). **(c)** Sagittal T2-weighted MR image shows severe canal narrowing (orange arrow), cord edema (white arrow), rupture of the ALL, and prevertebral soft-tissue edema (blue arrows).

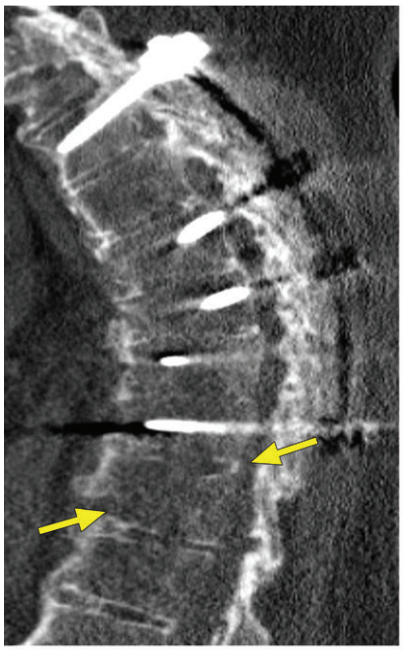


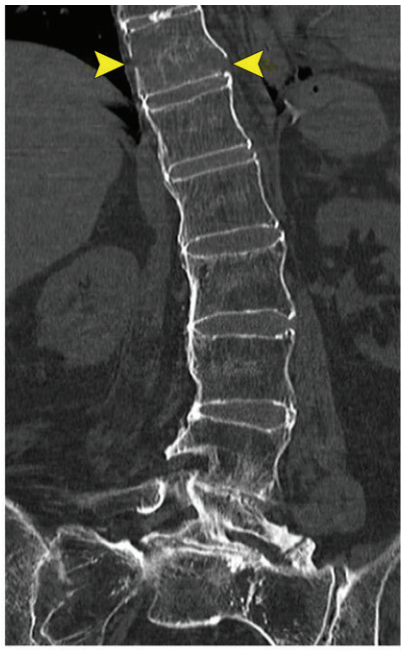
a. b.

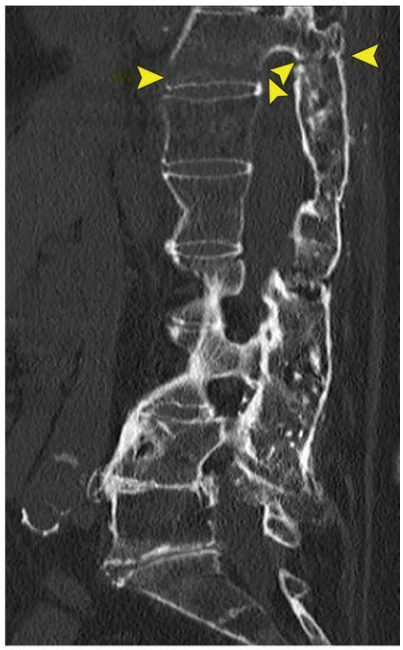
Figure 9. Neurologic injury from low-impact trauma in a 60-year-old woman with DS who fell from standing and developed left leg weakness and neck pain. **(a)** Sagittal CT image of the cervical spine shows degenerative C7-T1 fusion (white arrow) and a nondisplaced C7 spinous process fracture (yellow arrows). **(b)** Sagittal STIR MR image shows disruption of the posterior longitudinal ligament (green arrows) at C4-C5 and C5-C6, multilevel edema of the interspinous ligaments (red arrows), and disruption of the supraspinous ligament (blue arrows).

It can be placed in the intervertebral disk space or between transverse processes (35). However, the surgery is not without risk. Specifically, a surgically fused spine becomes a rigid spine and can be a risk factor for spinal fracture (7).

Well known in the orthopedic literature is an entity called adjacent segment disease (ASD), which refers to the nonfused spine segments proximal and distal to the fused spine (36). The abnormal processes that occur in these nonfused







10. **11a.** **11b.**

Figures 10, 11. **(10)** Hyperextension fracture in a 65-year-old man with T5-T10 fusion who fell backward from a walker. Sagittal CT image shows a T11 hyperextension fracture (arrows) within the mobile segment adjacent to the fused spine. Fractures often occur at the junction of the mobile and fused spine. **(11)** Transverse fracture in a 65-year-old man with thoracic and lumbar osseous surgical fusion who fell backward in his kitchen and landed on his shoulder. He presented with left collarbone pain and left low back pain. Coronal (**11a**) and sagittal (**11b**) CT images show a transverse fracture (arrowheads) extending through the vertebral body and fused facets.

segments include disk degeneration, osteophyte growth, facet joint hypertrophy, spinal stenosis, spondylolysis, and loss of bone mineral density (7). The development of ASD predisposes the nonfused segments to spinal fracture, especially the junction between the fixed and mobile spine (37). Of note, studies show that the longer the surgically fused segment, the higher the risk of ASD and eventual fracture (7). Interestingly, a fracture of the first adjacent vertebrae typically occurs within 8 months of surgery, while more distant fractures occur later, up to 2 years after fusion surgery (38). Compression fractures of the adjacent vertebrae are often seen (39).

Fusion surgery is also performed for and is often more challenging in degenerative scoliosis and sagittal imbalance, a condition in which the normal kyphosis and lordosis of the spine is altered. Sagittal imbalance is seen in AS where the kyphosis can be so severe it causes chin-to-chest syndrome (40). Unfortunately, correcting severe sagittal malalignment, while necessary at times for patient function, can predispose patients with AS to translation injuries of the spine (41).

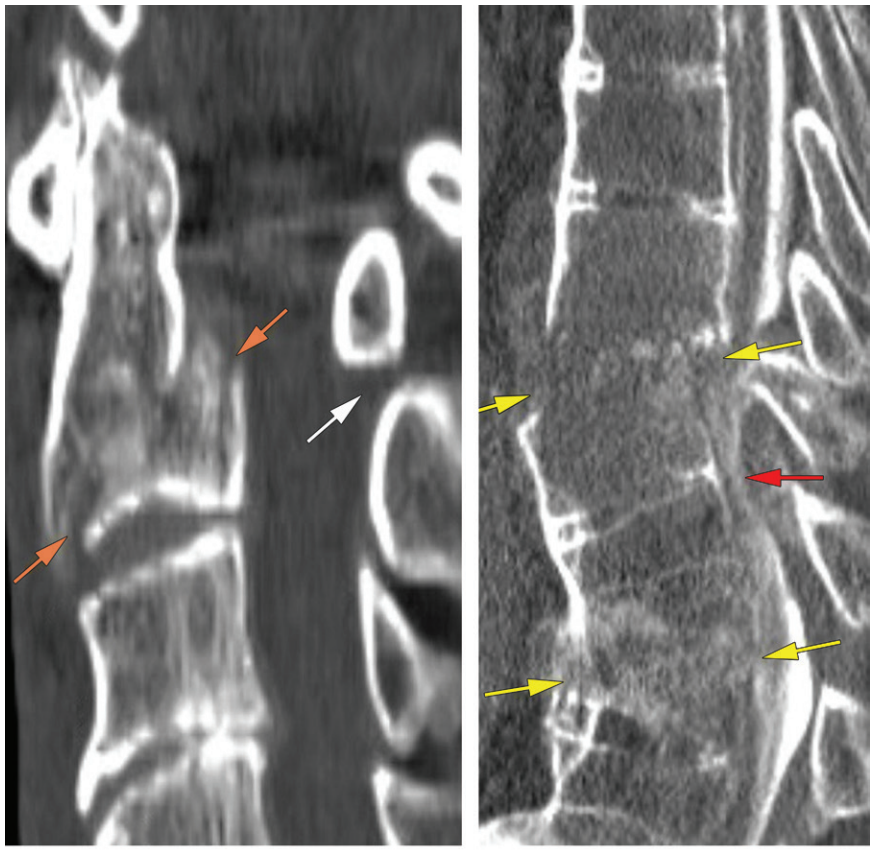
Patients with a surgically fused spine can present with acute back pain with or without trauma. Due to a risk of spine fractures in the proximal and distal nonfused spine, one must look closely in at least three vertebral bodies above and below the fused segment at CT. Fracture types include compression fracture of the adjacent vertebrae and transverse fractures within the fused spine and at the junction between the nonfused and fused spine (37,41) (Figs 10, 11). MRI is indicated if there is neurologic deficit such as neuropathy or myelopathy. It is important to assess both the spinal cord and nerve roots for impingement to guide any planned surgery.

Delayed Diagnosis and Outcomes

Diagnosing fractures in a patient with a rigid spine can be challenging owing to the fracture location in the transition zones, spinal deformity, and marked bony dystrophic changes. These challenges can lead to a delay in diagnosis. One study found a delay in diagnosis in 20% of rigid-spine cases (16). In other studies, delay in diagnosis has ranged from 10% to 50% for patients with AS or DISH (18).

In AS, diagnostic delay can also result because patients do not recognize the signs of acute injury or primary providers fail to obtain adequate imaging. Delayed recognition by the patient occurs because many patients with AS live with chronic pain and take pain medications, making acute fracture pain difficult to distinguish (12). Often, many patients with AS do not present until they experience abrupt neurologic deterioration (Fig 12).

For the initial providers, such as those in the emergency department and primary care clinic, the lack of high-impact trauma may lower suspicion for spinal fractures. Delayed diagnosis



a. b.

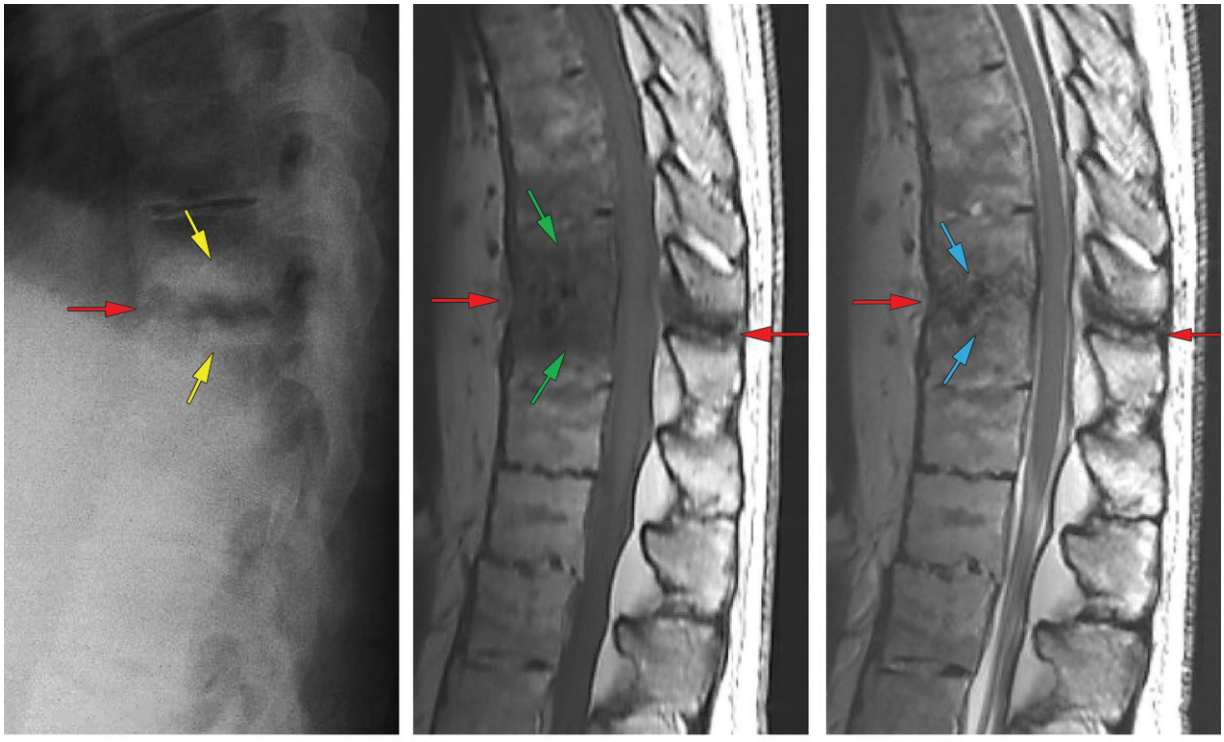
Figure 12. Neurologic deterioration in an 81-year-old man with AS who presented with headache and neck pain after falling. **(a)** Sagittal CT image of the cervical spine obtained on April 4 shows a comminuted fracture (orange arrows) of C2 at the base of the dens, with an 8-mm anterior translation of C1 on C2 (white arrow). No additional imaging was performed, and the patient was released in a C-spine hard collar. On April 18, he presented with confusion and new-onset weakness in the left lower extremity. The patient's weakness quickly progressed to bilateral lower extremity weakness the following day. **(b)** Sagittal CT myelogram shows a T9-T10 hyperextension-translation injury (yellow arrows) with severe canal narrowing and cord compression, findings suggestive of an extensive epidural hematoma (red arrow). Of note, MRI could not be performed owing to the presence of a pacemaker.

occurs because patients with a rigid spine present with minor trauma in the setting of acute on chronic pain, leading to insufficient imaging for diagnosis. Reasons for delayed diagnosis leading to secondary deterioration include a wrong initial assessment, missed fractures at imaging, and unrecognized epidural hematomas (19).

In patients with DISH, delayed diagnosis often transpires because the patients are frequently asymptomatic and present only in the setting of acute pain (19). Radiologists must be cognizant of the imaging features of different rigid spine entities and challenges associated with each entity to avoid a missed or delayed diagnosis of an injured rigid spine.

An especially challenging diagnosis in AS is an Andersson lesion because it can be mistaken for infection instead of a fracture and managed inappropriately. It refers to a pseudarthrosis from nonunion of a traumatic or more commonly an insufficiency fracture and can happen at the transdiscal or transvertebral location (42). Osseous union is limited by constant movement of the mobile fracture segment between two long autofused segments (43). They are primarily located at the junctional zones, especially the thoracolumbar junction, and are often solitary (44). There is widespread destruction of the vertebral body and disks and frequently localized kyphosis. Patients may present with progressively worsening pain or acute pain after trauma, or they may be asymptomatic and diagnosed years later.

At MRI, the fracture line has a hypointense signal, with the surrounding bone having low signal intensity on T1-weighted images and high signal intensity on T2-weighted images (Fig 13). Unfortunately, the radiographic and MRI appearances can overlap with those from infectious causes such as osteomyelitis or tuberculosis. However, one finding characteristic of an Andersson lesion is the



a. b. c.

Figure 13. Classic Andersson lesion in a 48-year-old man with AS. **(a)** Lateral radiograph shows the transdiscal fracture at T10-T11 (red arrow), with destructive changes of the surrounding vertebral bodies, and reactive sclerosis (yellow arrows), a finding that represents pseudarthrosis from nonunion. **(b)** Sagittal T1-weighted MR image shows the hypointense transdiscal fracture (red arrows) extending to the posterior elements. The reactive inflammatory changes to the bone (green arrows) surrounding the fracture line are T1 hypointense. **(c)** Sagittal T2-weighted MR image shows the hypointense transdiscal fracture extending to the posterior elements (red arrows). The reactive inflammatory changes (blue arrows) to the bone surrounding the fracture line are mildly T2 hyperintense.

presence of a fracture line extending to the posterior elements such as the facets (44). Also, there may be minimal soft-tissue swelling, unlike that which is depicted in cases of infection (12).

The clinical history, fracture location, and underlying history of AS can further help differentiate AS from an Andersson lesion. This distinction is important as Andersson lesions can be unstable when they manifest with acute pain and may require surgery rather than treatment with unnecessary antibiotics or antituberculosis medication (Fig 14) (12,42).

The likelihood of secondary decline in neurologic function when there is a delay in diagnosis is over 80% (16). In 35% of the patients with delayed diagnosis, the condition is incorrectly diagnosed at initial assessment as a compression fracture or degenerative disk disease (29). Fractures of the posterior elements are substantially associated with neurologic deterioration (29). Another source of diagnostic delay can be attributed to referrals from another hospital (Fig 15). In one study, 71.4% of patients with AS and 42.5% of patients with DISH were patients referred from other hospitals (30.6% in the control group), resulting in a longer period between fracture diagnosis and definitive treatment (45).

The radiologist's role is to recognize the imaging features of a rigid spine and closely scrutinize the spine for subtle fractures at CT and MRI. One must fully characterize the extent of injury to help guide management. Reasons for surgical intervention include neurologic deficit or concern for deterioration, an unstable fracture, or the presence of an epidural hematoma (Fig 16). Studies show that owing to the high chance of neurologic deterioration in patients with rigid spine who present with fracture, surgical stabilization is recommended in most cases. Importantly, the risks and benefits of surgery should be considered on an individual basis.

Morbidity and mortality from spinal trauma are higher in patients with a rigid spine than in the general population (46). Increased age, degree of neurologic deficit, and comorbid conditions are predictive of increased mortality (18) (Fig 17). Morbidity and mortality are greater among patients treated conservatively than among those treated surgically (18). In fact, surgical intervention leads to a 50% improvement in neurologic function, with up to 35% of cases showing complete recovery (5).

Research shows that age is the primary factor in predicting mortality. In one study, there was an 84% mortality for patients aged 80 years and older

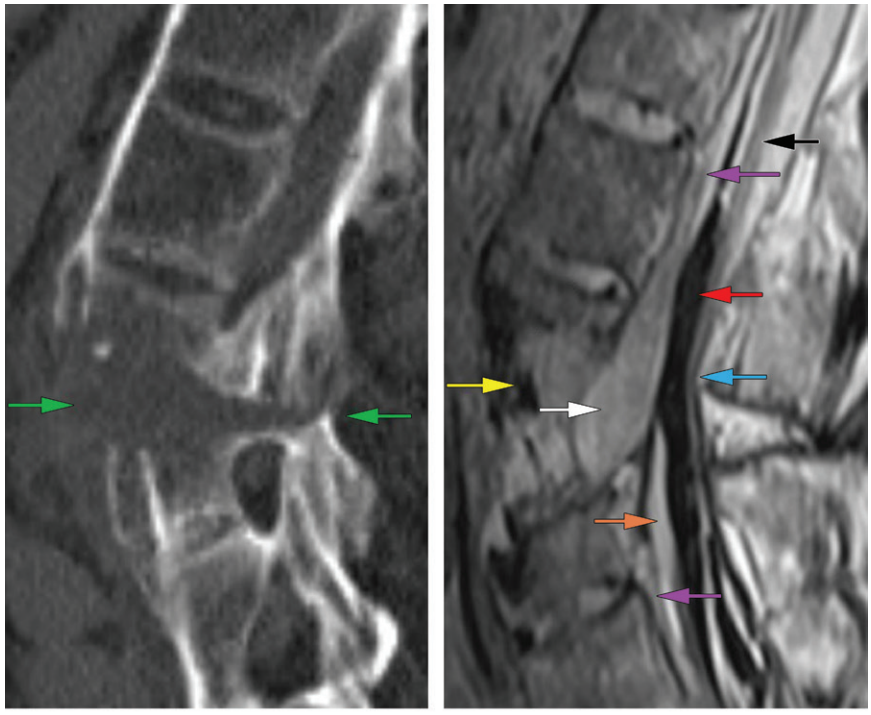


Figure 14. Acute injury to an underlying Andersson lesion in a 65-year-old man with AS who had acute on chronic back pain for 4 days with no inciting trauma. He developed urinary retention and went to the emergency department where lumbar spine radiographs were obtained, which were interpreted as negative. CT was not performed until the next day. **(a)** Sagittal CT image shows an L3-L4 hyperextension injury (arrows) extending through the posterior column. MRI was not performed until 2 days later, when the patient experienced symptoms of ongoing pain. **(b)** Sagittal T2-weighted MR image shows multiple abnormalities. The normal structures include the dural sac (purple arrows) and the cauda equina (black arrow). There is epidural and intrathecal blood. The epidural hematoma (white arrow) has intermediate signal intensity, while the subdural hematoma (orange arrow) has a higher signal intensity. The subarachnoid hemorrhage has markedly low signal intensity (red arrow). The epidural and intrathecal hematoma causes effacement of the dural sac (blue arrow), which is a finding concerning for cauda equina syndrome. Note the destruction of the L3-4 disk space (yellow arrow), which is a finding that likely represents an underlying Andersson lesion. The acute component of back pain and new urinary retention were concerning symptoms for an acute process, likely a hyperextension injury and mixed epidural and intrathecal hematoma causing cauda equina syndrome.

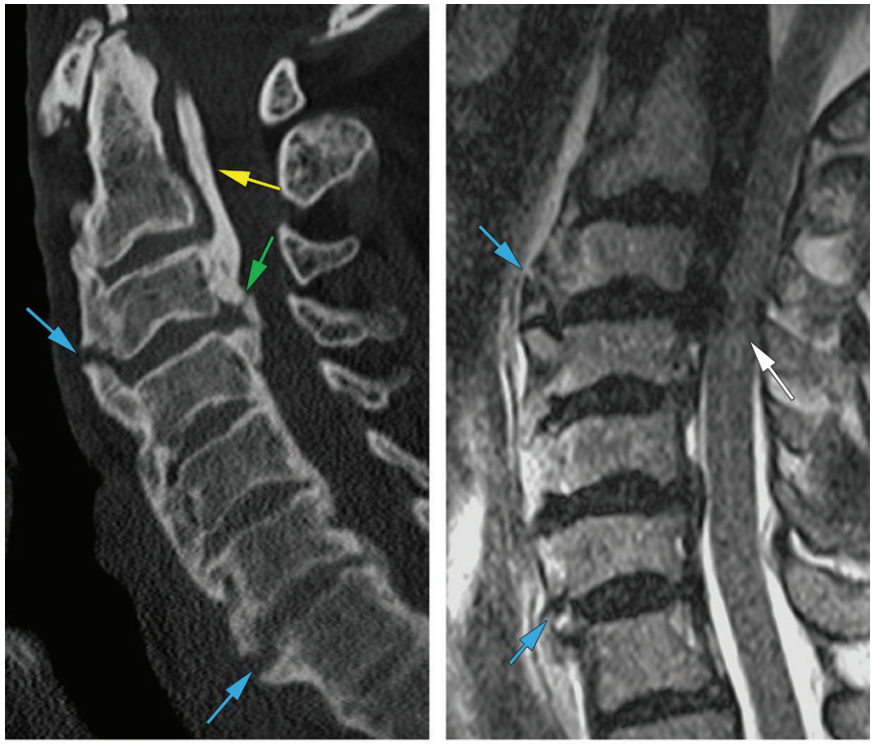
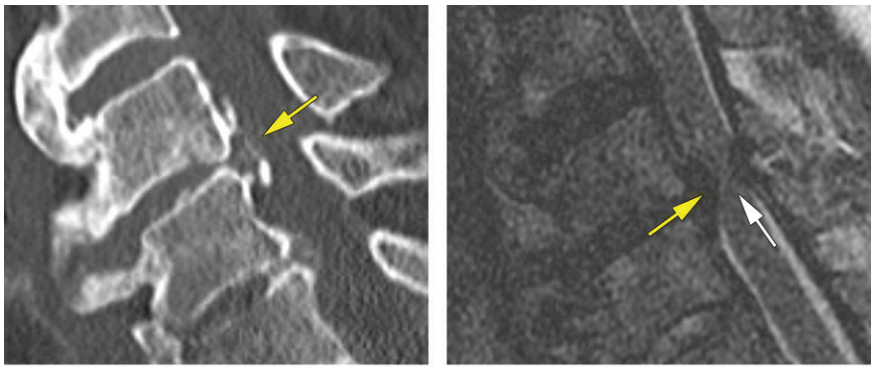
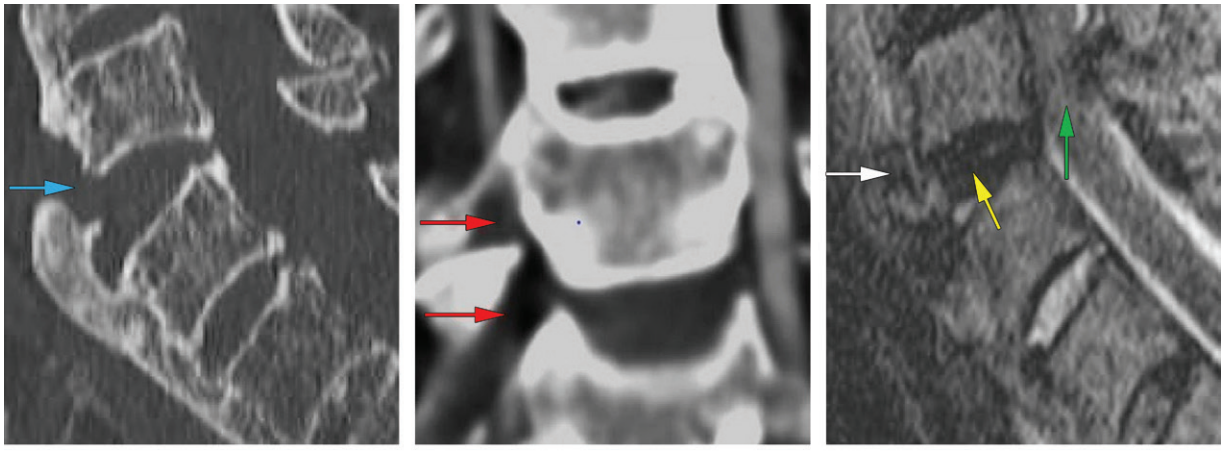


Figure 15. Diagnostic delay in a 69-year-old man with DISH who fell onto his face while walking and immediately noted weakness in his arms and legs with normal sensation. C-spine CT images obtained at an outside hospital (not shown) were interpreted as negative, so the patient was cleared from wearing a neck collar. Because he remained quadriplegic, he was transferred to our institution. Repeat C-spine CT was performed and also indicated no fracture. **(a)** Sagittal CT image shows areas of discontinuity in the anterior osteophytes (blue arrows) and posterior C3-C4 osteophyte (green arrow), as well as ossification of the posterior longitudinal ligament (yellow arrow). MRI was performed owing to the patient's neurologic symptoms. **(b)** Sagittal T2-weighted MR image shows acute fractures (blue arrows) of the anterior osteophytes at C3-C4 and C6-C7 with surrounding marrow edema, as well as severe central canal stenosis at C3-C4 with cord contusion (white arrow). These imaging findings support the clinical diagnosis of central cord syndrome. The discontinuity at the posterior C3-C4 osteophyte is not well depicted at MRI but was also diagnosed as an acute fracture.



a. b.

Figure 16. Central cord syndrome in a 77-year-old man with DISH who fell down stairs and presented with sensory deficit and bilateral arm and leg weakness. **(a)** Sagittal CT image of the cervical spine shows an area of discontinuity of the C4-C5 posterior osteophytes (arrow) but was interpreted as negative for fracture. MRI was performed owing to neurologic deficits. **(b)** Sagittal T2-weighted MR image shows a large C4-5 disk extrusion (yellow arrow), causing cord compression and edema (white arrow). These imaging findings support the clinical diagnosis of central cord syndrome.



a. b. c.

Figure 17. Acute vertebral artery dissection in a 75-year-old man with DISH and a recent history of falls who presented with right upper extremity weakness. **(a)** Sagittal CT image shows DISH, with asymmetric anterior disk space widening at C4–5 and discontinuity of the anterior fused mass, a finding representing a hyperextension injury (arrow). **(b)** Coronal CT angiogram shows narrowing of the right vertebral artery (arrows), a finding that represents acute dissection. **(c)** Sagittal T2-weighted MR image shows anterior disruption (white arrow), disk rupture (yellow arrow), and severe canal stenosis with some spinal cord edema (green arrow).

and 38% for patients in their 7th decade of life among patients with AS and DISH. These findings were statistically different from mortality rates in those patients aged 69 years and younger (16). This is likely from the associated comorbidities, which increase in number and severity with age (16).

The most frequent cause of death, both post-trauma and at follow-up, for patients with AS and DISH is pneumonia and respiratory failure (19). This is especially worrisome in patients with AS, in whom costovertebral ankylosis can cause chest wall rigidity and restrictive lung disease (12). Patients with AS are more likely to have lethal complications of spinal fracture such as aortic dissection and tracheal rupture, but these can also be seen in patients with DISH (16) (Fig 18). In addition, epidural hematomas, which have a very high risk of neurologic decline, are more common in patients with AS and contribute to increased mortality (5,12,32) (Fig 19).

Some recent research suggests that patients with AS are at a greater risk of mortality up to 2 years after injury compared with that of patients with DISH (46). Possible reasons include a higher likelihood of delayed diagnosis, younger age when ankylosis occurs, increased frequency of epidural hematomas after injury, and a greater incidence of spinal fractures owing to osteoporosis related to AS (46) (Fig 20). CT and MRI have important roles in diagnosing traumatic injuries in patients with a rigid spine. While performing CT is essential and performing MRI is usually necessary in patients with AS and in patients with rigid spine with neurologic deficit, additional

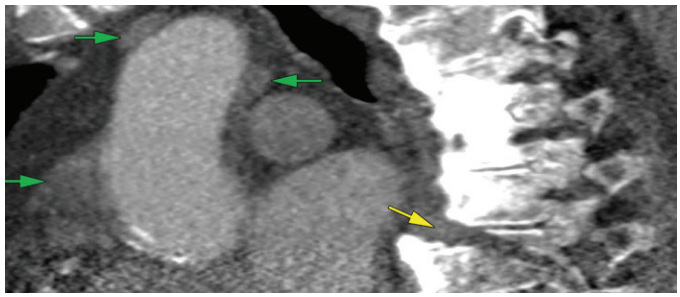


Figure 18. Complications following a motor vehicle collision in an 83-year-old man with DISH throughout the thoracic spine. Sagittal CT angiogram shows a hyperextension-translation injury (yellow arrow) at T7-T8 and a mediastinal hematoma (green arrows).

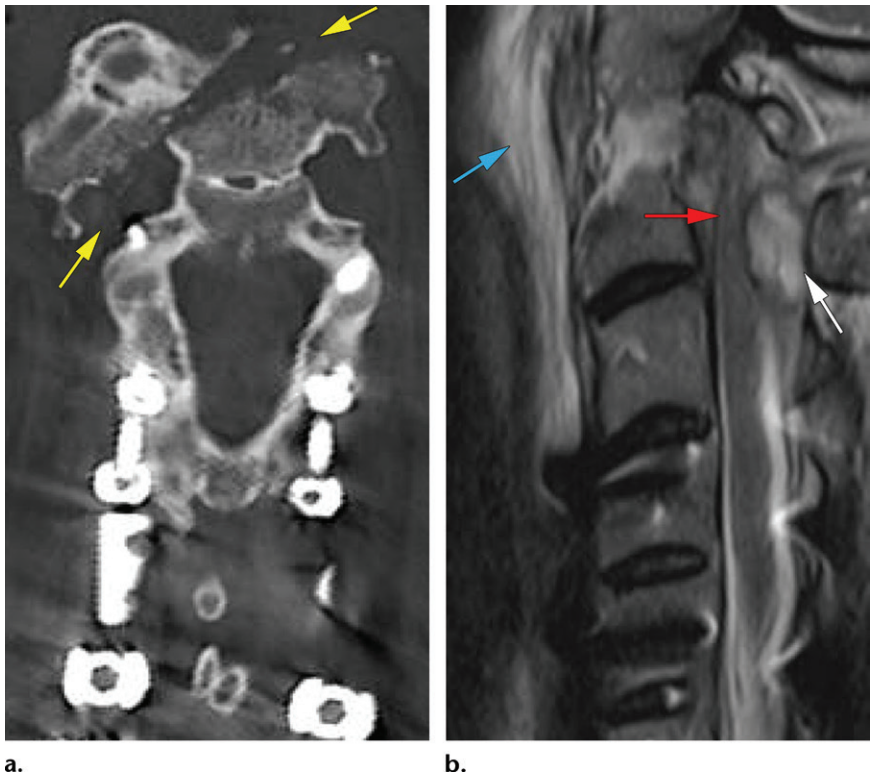


Figure 19. Epidural hematoma in a 53-year-old man with AS and prior cervicothoracic fusion who was found down. **(a)** Coronal CT image shows a severe fracture-translation injury of the craniocervical junction (arrows), a region prone to ankylosis in AS. **(b)** Sagittal T2-weighted MR image shows a large epidural hematoma (white arrow), spinal cord compression and edema (red arrow), and prevertebral edema (blue arrow).

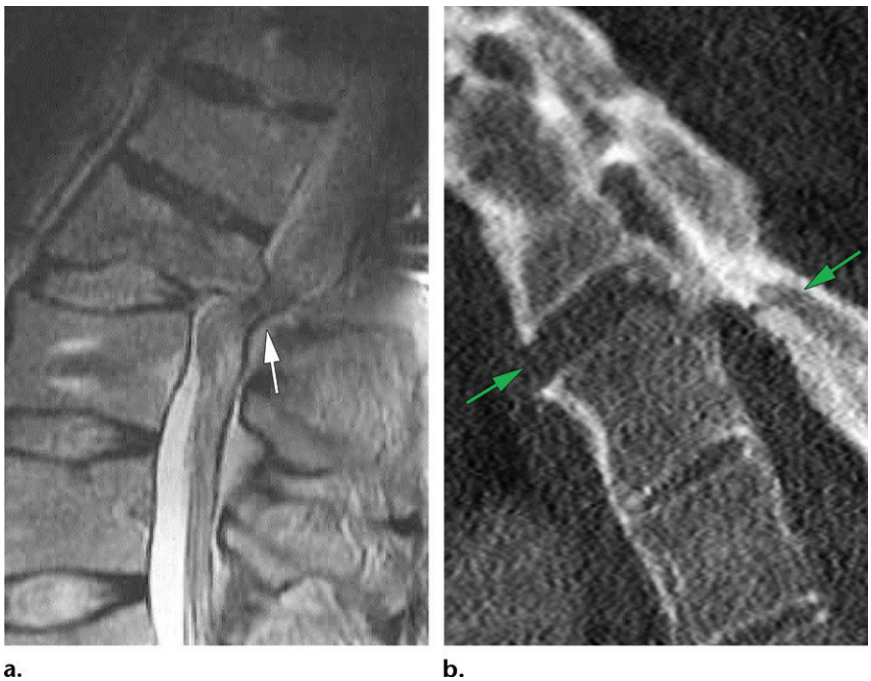


Figure 20. Trauma in two young patients with AS. **(a)** Sagittal T2-weighted MR image shows an L1 burst fracture (arrow) with canal narrowing and cauda equina syndrome in a 28-year-old man with AS who developed lower extremity weakness a day after undergoing surgery to address a kyphotic deformity. **(b)** Sagittal CT image shows a T2-T3 hyperextension-distraction injury with widening of the disk space and a transverse fracture through the ankylosed posterior elements (arrows) in a 30-year-old man with AS and cardiomyopathy who fell down stairs.

research needs to be performed to strengthen the imaging guidelines and indications for patients with a rigid spine. By doing so, the radiologist will prevent a delayed diagnosis and guide appropriate surgical management.

Conclusion

A radiologist must first identify the fractures in patients with a rigid spine and then fully characterize the extent of injury, paying attention to the relevant imaging features that may indicate surgical intervention, such as in cases with an unstable fracture or epidural hematoma. By recognizing the fracture and these relevant features in patients with rigid spine presenting with back pain or after trauma, radiologists can avoid a missed and/or delayed diagnosis of an injured rigid spine.

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References

1. Izzo R, Guarnieri G, Guglielmi G, Muto M. Biomechanics of the spine. I. Spinal stability. Eur J Radiol 2013;82(1):118–126.
2. Werner BC, Feuchtbaum E, Shen FH, Samartzis D. Ankylosing spondylitis of the cervical spine. In: Shen FH, Samartzis D, Fessler RG, eds. Textbook of the cervical spine. Maryland Heights, Mo: Elsevier Saunders, 2015.
3. Vergara ME, O'Shea FD, Inman RD, Gage WH. Postural control is altered in patients with ankylosing spondylitis. Clin Biomech (Bristol, Avon) 2012;27(4):334–340.
4. Taljanovic MS, Hunter TB, Wisneski RJ, et al. Imaging characteristics of diffuse idiopathic skeletal hyperostosis with an emphasis on acute spinal fractures: review. AJR Am J Roentgenol 2009;193(3 suppl):S10–S19; quiz S20–S24.
5. Finn M, Kumar R. Fractures in the ankylosed spine. In: Browner BD, Jupiter JB, Krettek C, Anderson PA, eds. Skeletal trauma: basic science, management, and reconstruction. 5th ed. Philadelphia, Pa: Elsevier Health Sciences, 2015.
6. Gellhorn AC, Katz JN, Suri P. Osteoarthritis of the spine: the facet joints. Nat Rev Rheumatol 2013;9(4):216–224.
7. Park P, Garton HJ, Gala VC, Hoff JT, McGillicuddy JE. Adjacent segment disease after lumbar or lumbosacral fusion: review of the literature. Spine 2004;29(17):1938–1944.
8. Panjabi MM. Clinical spinal instability and low back pain. J Electromyogr Kinesiol 2003;13(4):371–379.
9. Izzo R, Guarnieri G, Guglielmi G, Muto M. Biomechanics of the spine. II. Spinal instability. Eur J Radiol 2013;82(1):127–138.
10. Vaccaro AR, Koerner JD, Radcliff KE, et al. AOSpine subaxial cervical spine injury classification system. Eur Spine J 2016;25(7):2173–2184.
11. Kepler CK, Vaccaro AR, Schroeder GD, et al. The thoracolumbar AOSpine injury score. Global Spine J 2016;6(4):329–334.
12. Rustagi T, Drazin D, Oner C, et al. Fractures in spinal ankylosing disorders: a narrative review of disease and injury types, treatment techniques, and outcomes. J Orthop Trauma 2017;31(suppl 4):S57–S74.
13. El Maghraoui A, Bensabbah R, Bahiri R, Bezza A, Guedira N, Hajjaj-Hassouni N. Cervical spine involvement in ankylosing spondylitis. Clin Rheumatol 2003;22(2):94–98.
14. Chaudhary SB, Hullinger H, Vives MJ. Management of acute spinal fractures in ankylosing spondylitis. ISRN Rheumatol 2011;2011:150484.
15. Anghelescu A, Onose LV, Popescu C, et al. Evolution of traumatic spinal cord injury in patients with ankylosing spondylitis, in a Romanian rehabilitation clinic. Spinal Cord Ser Cases 2016;2(1):16001.
16. Caron T, Bransford R, Nguyen Q, Agel J, Chapman J, Bellabarba C. Spine fractures in patients with ankylosing spinal disorders. Spine 2010;35(11):e458–E464.
17. Lukasiewicz AM, Bohl DD, Varthi AG, et al. Spinal fracture in patients with ankylosing spondylitis: cohort definition, distribution of injuries, and hospital outcomes. Spine 2016;41(3):191–196.
18. Hartmann S, Tschugg A, Wipplinger C, Thomé C. Analysis of the literature on cervical spine fractures in ankylosing spinal disorders. Global Spine J 2017;7(5):469–481.
19. Westerveld LA, Verlaan JJ, Oner FC. Spinal fractures in patients with ankylosing spinal disorders: a systematic review of the literature on treatment, neurological status and complications. Eur Spine J 2009;18(2):145–156.
20. Gonzalez-Beicos A, Nuñez DB Jr, Fung AW, Sanchez M, Gahbauer H. Trauma to the ankylotic spine: imaging spectrum of vertebral and soft tissue injuries. Emerg Radiol 2007;14(6):371–378.
21. Altun I, Yuksel KZ. Ankylosing spondylitis: patterns of spinal injury and treatment outcomes. Asian Spine J 2016;10(4):655–662.
22. Alaranta H, Luoto S, Konttinen YT. Traumatic spinal cord injury as a complication to ankylosing spondylitis: an extended report. Clin Exp Rheumatol 2002;20(1):66–68.
23. Jacobs WB, Fehlings MG. Ankylosing spondylitis and spinal cord injury: origin, incidence, management, and avoidance. Neurosurg Focus 2008;24(1):E12.
24. Rao SK, Wasyliw C, Nunez DB Jr. Spectrum of imaging findings in hyperextension injuries of the neck. RadioGraphics 2005;25(5):1239–1254.
25. Balling H, Weckbach A. Hyperextension injuries of the thoracolumbar spine in diffuse idiopathic skeletal hyperostosis. Spine 2015;40(2):E61–E67.
26. Bransford RJ, Koller H, Caron T, et al. Cervical spine trauma in diffuse idiopathic skeletal hyperostosis: injury characteristics and outcome with surgical treatment. Spine 2012;37(23):1923–1932.
27. Olivieri I, D'Angelo S, Palazzi C, Padula A, Mader R, Khan MA. Diffuse idiopathic skeletal hyperostosis: differentiation from ankylosing spondylitis. Curr Rheumatol Rep 2009;11(5):321–328.
28. Verlaan JJ, Westerveld LA, van Keulen JW, et al. Quantitative analysis of the anterolateral ossification mass in diffuse idiopathic skeletal hyperostosis of the thoracic spine. Eur Spine J 2011;20(9):1474–1479.
29. Okada E, Tsuji T, Shimizu K, et al. CT-based morphological analysis of spinal fractures in patients with diffuse idiopathic skeletal hyperostosis. J Orthop Sci 2017;22(1):3–9.
30. Kim B-S, Moon M-S, Yoon MG, et al. Prevalence of diffuse idiopathic skeletal hyperostosis diagnosed by whole spine computed tomography: a preliminary study. Clin Orthop Surg 2018;10(1):41–46.
31. Hendrix RW, Melany M, Miller F, Rogers LF. Fracture of the spine in patients with ankylosis due to diffuse skeletal hyperostosis: clinical and imaging findings. AJR Am J Roentgenol 1994;162(4):899–904.
32. Whang PG, Goldberg G, Lawrence JP, et al. The management of spinal injuries in patients with ankylosing spondylitis or diffuse idiopathic skeletal hyperostosis: a comparison of treatment methods and clinical outcomes. J Spinal Disord Tech 2009;22(2):77–85.
33. Goode AP, Carey TS, Jordan JM. Low back pain and lumbar spine osteoarthritis: how are they related? Curr Rheumatol Rep 2013;15(2):305.
34. Carter B, Griffith B, Mossa-Basha F, et al. Reformatted images of the thoracic and lumbar spine following CT of

chest, abdomen, and pelvis in the setting of blunt trauma: are they necessary? Emerg Radiol 2015;22(4):373–378.
35. Coding Education Team. Spinal fusion and fixation. Department of Health, Government of Western Australia. http://ww2.health.wa.gov.au/~/media/Files/Corporate/general%20documents/Clinical%20coding/Guides%20and%20summaries/summ-spinal.pdf. Published 2016. Accessed March 4, 2018.
36. Carrier CS, Bono CM, Lebl DR. Evidence-based analysis of adjacent segment degeneration and disease after ACDF: a systematic review. Spine J 2013;13(10):1370–1378.
37. Kobayashi H, Otani K, Watanabe K, et al. Vertebral fracture at the caudal end of a surgical fusion for thoracic vertebral fracture in a patient with diffuse idiopathic skeletal hyperostosis (DISH). Fukushima J Med Sci 2017;63(2):112–115.
38. Toyone T, Ozawa T, Kamikawa K, et al. Subsequent vertebral fractures following spinal fusion surgery for degenerative lumbar disease: a mean ten-year follow-up. Spine 2010;35(21):1915–1918.
39. Lawrence BD, Hilibrand AS, Brodt ED, Dettori JR, Brodke DS. Predicting the risk of adjacent segment pathology in the cervical spine: a systematic review. Spine 2012;37(22 suppl):S52–S64.
40. Kaiser M, Angevine PD, Mandigo CE , Reid PC, and Anderson RCE. Sagittal imbalance. The Spine Hospital at The Neurological Institute of New York. http://columbiaspine.org/condition/sagittal-imbalance/. Published 2018. Accessed March 7, 2018.
41. Watanabe K, Lenke LG, Bridwell KH, Kim YJ, Koester L, Hensley M. Proximal junctional vertebral fracture in adults after spinal deformity surgery using pedicle screw constructs: analysis of morphological features. Spine 2010;35(2):138–145.
42. Bron JL, de Vries MK, Snieders MN, van der Horst-Bruinsma IE, van Royen BJ. Discovertebral (Andersson) lesions of the spine in ankylosing spondylitis revisited. Clin Rheumatol 2009;28(8):883–892.
43. Park Y-S, Kim J-H, Ryu J-A, Kim T-H. The Andersson lesion in ankylosing spondylitis: distinguishing between the inflammatory and traumatic subtypes. J Bone Joint Surg Br 2011;93(7):961–966.
44. Hong SH, Choi J-Y, Lee JW, Kim NR, Choi J-A, Kang HS. MR imaging assessment of the spine: infection or an imitation? RadioGraphics 2009;29(2):599–612.
45. Westerveld LA, van Bemmel JC, Dhert WJA, Oner FC, Verlaan JJ. Clinical outcome after traumatic spinal fractures in patients with ankylosing spinal disorders compared with control patients. Spine J 2014;14(5):729–740.
46. Schoenfeld AJ, Harris MB, McGuire KJ, Warholic N, Wood KB, Bono CM. Mortality in elderly patients with hyperostotic disease of the cervical spine after fracture: an age- and sex-matched study. Spine J 2011;11(4):257–264.