Degenerative Disorders of the Spine

65

Radiological Imaging

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Contents

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F. Barkhof et al. (eds.), *Clinical Neuroradiology*,
https://doi.org/10.1007/978-3-319-68536-6_38

Abstract

Spinal pain and low back pain (LBP) are ubiquitous in all Western countries and represents the second leading cause of disability worldwide. The estimated lifetime prevalence rates of LBP and neck pain in adults are 91% and 66.7%, respectively, but the incidence is disproportionally rising with respect to the population growth. Degenerative disc disease (DDD) is a generalized multifactorial process involving the disco-vertebral joints, having genetic, inflammatory, traumatic, and nutritional bases and leading to morphological and biomechanical spine modifications. It is by far the most frequent cause of acute and chronic spinal pain.

Acute back pain has most commonly a benign and self-limiting evolution being pathologically due to an underlying benign disease in about 95% of cases and most patients need neither imaging nor invasive approaches. Imaging in acute setting has no role in the absence of clinical suggestion of underlying systemic disease (red flags) as well as of a progressive or severe neurologic deficit. Chronic spinal pain is a major welfare and economic concern, being responsible of about 70–90% of the total national compensation for disease-related expenditure in USA. Imaging is crucial but represents only a step of the assessment of spinal pain and its findings must always be related to anamnesis data and physical evaluation in order to be correctly evaluated.

In many cases, the significance of imaging data and the source of pain are challenging to be determined due to the complex anatomy and function of the spine as well as to the occurrence of the similar changes in asymptomatic and diseased subjects.

Postoperative imaging finding is important to understand posttreatment persistent pain.

Keywords

Degenerative spine · Disc herniation · Degenerative disc disease · Disco genic pain · Internal disc disruption · Facet pain · Spinal canal stenosis · Postoperative spine

Abbreviations

AF	Annulus fibrosus
ALIF	Anterior lumbar interbody fusion
CSF	Cerebrospinal fluid
DDD	Degenerative disc disease
DSL	Degenerative spondylolisthesis
DWI	Diffusion weighted imaging
FBSS	Failed back surgery syndrome
FSE	Fast Spin Echo
IASP	International Association for the Study of Pain
IDD	Internal disc destruction
ISL	Isthmic spondylolisthesis
LBP	Low back pain
MDCT	Multi-detector computed tomography
MRI	Magnetic resonance imaging
MS	Motion segment
NP	Nucleus pulposus
NPV	Negative predictive value
PF	Posterior fusion
PLF	Posterior lateral fusion
PLIF	Posterior lumbar interbody fusion
PPV	Positive predictive value
SAC	Space available for the cord
SCS	Spinal canal stenosis
XLIF	Extreme lateral interbody fusion

Epidemiology

Spinal pain is diffuse worldwide and represents the second leading cause of disability, with an estimated 1% of the population disabled and 2–3% of people receiving medical care (Kang and Hanks 2008). In the Western countries, its lifetime reaches 80%.

Back pain, with peaks between 45 and 65 years with no gender prevalence, by far is caused by the degenerative disc disease (DDD).

Degenerative Disc Disease (DDD)

Pathology, Clinics, and Imaging

A normal intervertebral disc has both the tension-resisting properties of a ligament and the compression-resisting properties of a joint cartilage. It works as a shock absorber of axial loads, to preserve the integrity of vertebral endplates, and acts as a ligament to control the complex three-dimensional movements performed by every spinal MS (Izzo et al. 2013). According to nomenclature proposed by the Combined Task Forces formed by the North American Spine Society, American Society of Spine Radiology, and American Society of Neuroradiology, a normal disc respects the boundaries of the vertebral endplates and interspace, retains a normal volume and thickness, and shows a sharp distinction between nucleus pulposus and external annulus, irrespectively of the clinical setting (Fardon et al. 2014). However, the composition and morphology of the normal intervertebral disc change with aging and aging changes begin early in life.

Starting in childhood, disc mucoid matrix gradually loses proteoglycans and water while it is replaced by collagen. The dehydration process principally involves the NP transforming it from a semiliquid in a solid structure leading to a loss of distinction between NP and AF. The intervertebral disc affected by dehydration loses volume too. The reduced hydrostatic pressure, due to the dehydration of NP, makes the compressive loads to shift on the AF that folds inward and undergoes increased shearing stresses favoring the delamination and internal fissuring. The chemical composition and structure of the vertebral endplates resemble and run parallel to those of the rest of the disc. Vascular channels are gradually occluded close to the bone near to endplates with reduction of diffusion of metabolites to and from the disc. This impairing nutrition may contribute to biochemical changes inside the disc and perhaps represents the *primum movens* of disc degeneration. Fractures of the endplates and intra-vertebral disc herniation can also drastically reduce the internal disc pressure and accelerate annulus' degeneration and destruction. Over time, biochemical changes lead to structural and finally to biomechanical changes and can end in loss of disc function.

DDD is a generalized multifactorial process involving the disco-vertebral joints, having genetic, inflammatory, traumatic, and nutritional bases and leading to morphological and biomechanical spine modifications. The process can remain asymptomatic or manifests with acute/

chronic back or neck pain, radicular pain with or without radiculopathy, neurogenic claudication, more rarely with neurological deficits. On the basis of imaging, such as on pathology study, it is difficult to distinguish between normal aging, occurring with time in all subjects, and true degenerative changes having pathological significance, due to aging modifications that predispose to degenerative changes and merge with them. The distinction between simple aging and pathological degeneration from an abnormal acceleration of aging processes is often impossible.

DDD imaging features include

- Conventional radiographs only demonstrate variable degrees of disc interspace collapse during the delayed phases of disc dehydration and degeneration, intradiscal gas collections (vacuum), eventual calcifications, in most of cases flanked by endplate changes.
- CT has too limited contrast resolution for detecting early disc changes and then shows disc collapse, disc bulging, calcifications, vacuum, and endplate changes.
- MRI accurately reflects the disc biochemical and morphological changes from the beginning. On MRI, the measure of T2 relaxation times can offer an objective and very sensitive evaluation of ongoing disc aging and degeneration, but the 5-point qualitative grading by Pfirrmann offers a good and well-reproducible tool (Table 1).

An early sign of aging on MRI of young adults is the appearance of intranuclear cleft, a band-like deposition of collagen along the disc equator giving it a bipartite aspect (Fig. 1); its disappearance has to be considered an early sign of disc degeneration. Normal aging also includes signal loss contemporarily involving all the discs of a spinal segment, while an isolated black disc should rather be considered as abnormal. Disc degeneration begins early in life with a reported prevalence of MR signs among young people ranging from 21% in aged 13 to 42% in 18-years-old and a prevalence of Pfirrmann-grade 3 MRI changes and over in 47% of young subjects aged 20–22 years.

The degeneration first involves the most stressed discs in transitional motion segments, such as C5-C6, C6-C7, L4-L5, and L5-S1.

Two distinct processes involve the discovertebral joint (Resnick and Niwayama 1995):

– *Spondylosis deformans* involving the annulus fibrosus and its insertions on the apophyseal rings

Table 1 *Pfirrmann scale* (Pfirrmann et al. 2001)

Grade 1: Normal disc height, homogeneous bright signal of NP/inner annulus, and clear-cut distinction with outer annulus.
Grade 2: Inhomogeneous bright signal, clear distinction annulus/ NP disc. Eventual nuclear cleft (Fig. 1).
Grade 3: Inhomogeneous intermediate signal, unclear distinction annulus/NP, possible initial disc thinning.
Grade 4: Inhomogeneous dark signal throughout the disc, with normal to reduced disc height.
Grade 5: Collapsed black disc.

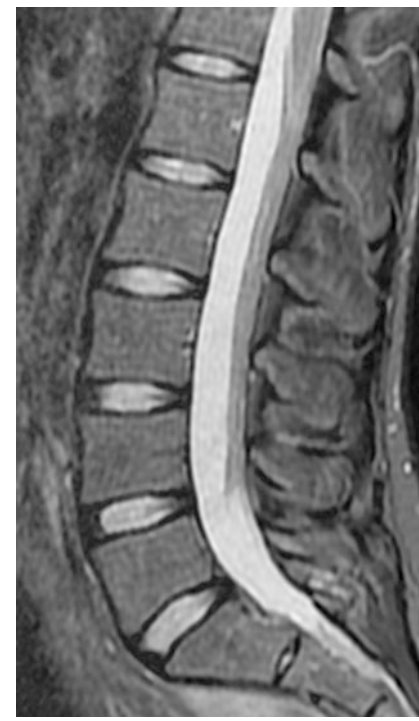


Fig. 1 MR Fast-STIR sagittal image. 34-year-old man spine, both the nucleus pulposus and inner annulus of all intervertebral discs show a linear and thin transversal hypointensity, referred to as nuclear cleft. It represents collagen deposits and this finding is common in young healthy discs in the second decade and is a normal sign of initial ageing, but not of degeneration. A disc with an intranuclear cleft corresponds to grade 2 of Pfirrmann scale

– *Intervertebral osteochondrosis* involving the nucleus pulposus and adjacent vertebral endplates

Spondylosis deformans is an enthesopathy which begins with the breakdown of annular anchorage into vertebral apophyseal rings and the anterior protrusion of disc. The stretching and the abnormal tension on the insertions of the Sharpey's fibers and anterior longitudinal ligament lead to reactive bone spurring. Anterolateral osteophytes are an adaptive answer to the increasing stresses concentrating during aging in the disc annulus, due to the ongoing fibrosis, through an expansion of the joint surface. In fact, they have been found in 100% of subjects over 40 years old. However, the posterior osteophytes are rare and are not considered a natural consequence of aging.

In the cervical spine, bony degenerative deformities also involve uncovertebral processes, causing the so-called "uncoarthrosis" that is an important cause of foraminal canal stenosis and nerve entrapment.

Findings of osteochondrosis are disc height loss, intradiscal gas collections (vacuum), endplate erosion and sclerosis, and intervertebral disc displacement (Figs. 2 and 3). Osteochondrosis is considered a true degenerative event and the degrees of disc signal and height loss on imaging can have clinical relevance.

A degenerated black and collapsed disc can be painful. The signal loss in MRI T2-weighted sagittal images can be a predictor of painful disc at provocative discography, with reported sensitivities ranging from 90% to 98% and specificities from 39% to 77% (Weishaupt et al. 2001). A moderate or severe disc height loss had sensitivity for painful

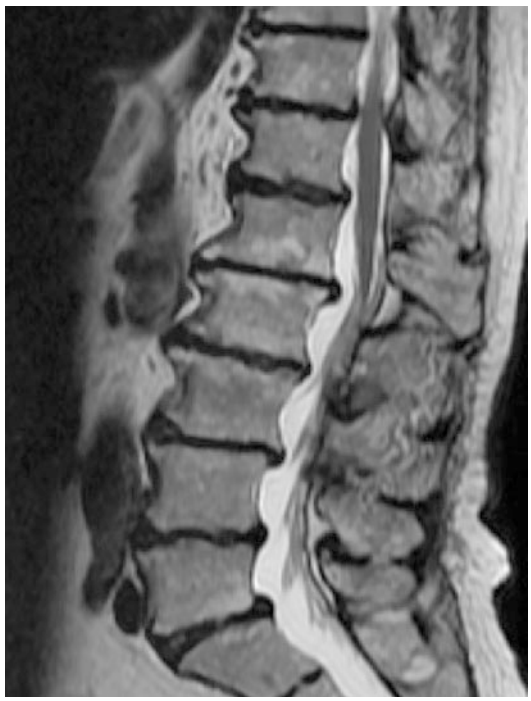


Fig. 2 MR FSE T2-weighted midsagittal image. 67-year-old man with chronic low back pain. Diffuse collapse and hypointensity of all lumbar discs with circumferential remodeling of vertebral bodies and deformans spondylosis. The dehydrated and degenerated discs show complete dedifferentiation between annulus and nucleus pulposus (Pfirrmann grade V)

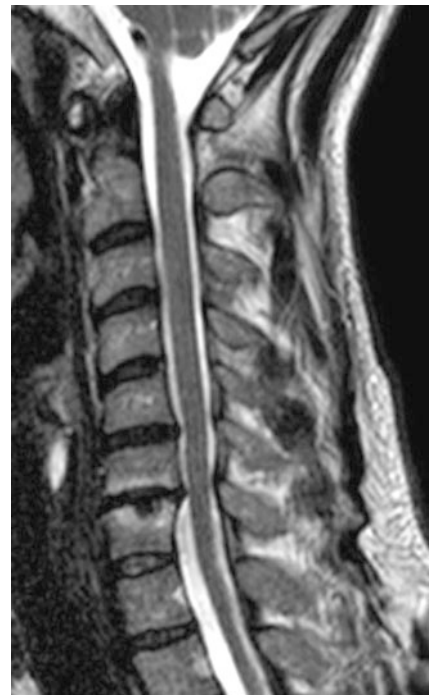


Fig. 3 MR midsagittalFSE-T2 w-image. 45-year-old man suffering from long-time cervical pain. Diffuse hypointensity of cervical discs. Partial collapse of C6-C7 disc and intervertebral disc herniation into the superior C6 endplate. Subchondral edema of both opposite C5 and C6 endplates (Modic type I changes). Both disc collapse and Schmorl's node are findings of intervertebral osteochondrosis and represent a true degeneration of interbody joint

disc of 87% and 73% with specificities between 69% and 81%, respectively. While a morphologically normal disc on MRI excludes the necessity of a discography, a severely collapsed black disc is a strong predictor of positive discography.

Internal Disc Disruption (IDD)

Pathology, Clinics, and Imaging

Only in a small percentage of back pain cases is due to a disc herniation. The most frequent cause of nonspecific back pain is the IDD responsible for discogenic pain. Discogenic axial pain is mechanical, exacerbated by weight loads and motion and relieved by recumbency and rest. It can either be axial and/or referred to sites distant from the spinal source. The composition and structure of a painful disrupted disc are different from that of a degenerated asymptomatic disc. IDD consists in annular fissures, disc collapse, and mechanical failure, with or without endplate fractures, with no significant modifications of disc contour or radicular compression. IDD accounts for 26–42% of cases of chronic low back pain and is described as a separate clinical entity from other forms of painful or asymptomatic disc pathology. Histological hallmarks of painful disc are the annulus radial tears, fissures running from the central part of the disc outwards for a variable extension either in a sagittal, oblique, or a horizontal plane (Fig. 4).

Inside and along the radial tear, a densely vascularized and neo-innervated reactive granulation tissue can develop coming from the external disc contour, as a tentative of repair after an injury. Through the radial fissures, pro-inflammatory metabolites can chemically sensitize the disc nociceptors of the external annulus, making painful even physiological loads. In addition, starting from the radial fissure, the inflammatory reaction may diffuse throughout the disc and lead to the degradation and proteolysis of the entire disc matrix, with final disc mechanical failure (disc resorption). There are three histopathological types of annular

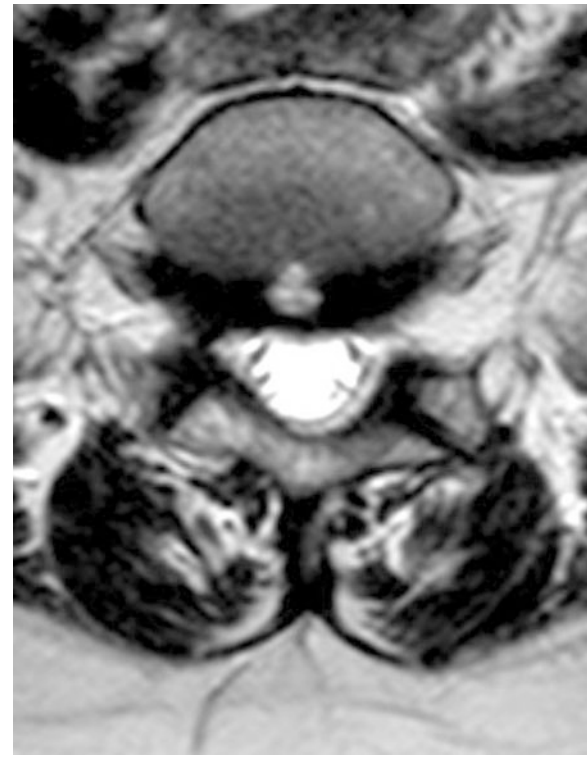


Fig. 4 MR FSE T2 axial image through L5-S1 disc showing an annular radial tear running inside the posterior annulus. The tear shows marked hypersignal completely surrounded by hypointense annular tissue, forming a high hyperintensity zone (HIZ). The hypersignal is due to inflammatory granulation tissue and fluid

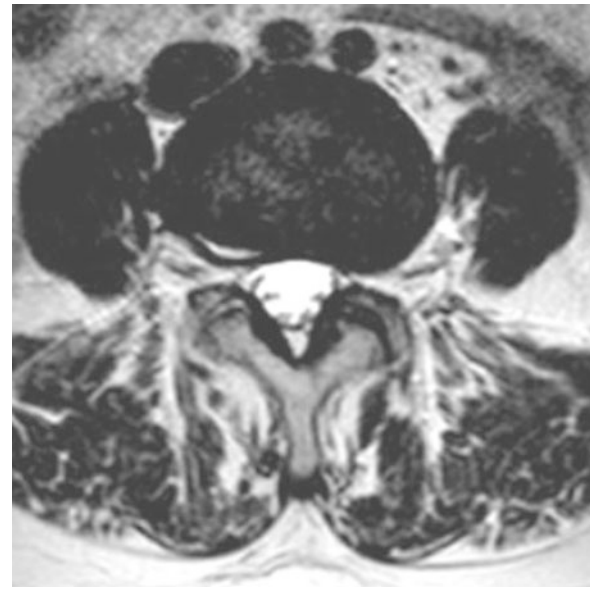


Fig. 5 MR T2-WI axial image showing HIZ inside posterolateral annulus corresponding to focal hyperintensity completely surrounded by hypointense annulus. Concentric tears consist of detachments between annulus lamellae expressing disc degeneration considered potentially painful. The patient complained of persistent low back pain

fissures: radial, concentric, and transverse ones. Concentric tears consist of delamination between adjacent lamellae, and they are considered of traumatic origin (Fig. 5). Transverse or peripheral tears consist of horizontal detachments of Sharpey's fibers traumatically induced near the insertions on the apophyseal rings.

While the clinical significance of transverse tears remains unclear, radial and concentric tears are considered potentially painful. On MR imaging, annular tears correspond to the hyperintensity zones (HIZ), bright spots on T2-weighted images, isointense or hyperintense to CSF owing to presence of mucoid fluid and reactive tissue, that are completely included in the hypointense posterior annulus, and enhance after contrast medium administration for being vascularized (Figs. 4, 5, and 6).

The clinical relevance of HIZs is debated. Most of studies, by correlating RM to provocative discography, reported high PPV of HIZs (86–87%) for painful disrupted disc (Aprill and Bogduk 1992). However, the high frequency of HIZs in asymptomatic people (25%) and the low sensitivity of MR imaging (26.7%), limit their clinical value. HIZs have to be distinguished from the low intensity zones (LIZs), annular tears appearing to be hypointense to surrounding annulus, inactive and silent for lacking inflammatory granulation tissue and fluid. There is no way to definitely diagnose clinically the IDD.

The diagnostic criteria proposed by the IASP are:

- Reproduction of typical patient's pain by discography
- Detection of an annular tear, by post-discography CT or by MR
- Modic lesions in vertebral endplates, by MR

Discography and CT: discography shows the IDD features with great accuracy. By discography, Peng distinguished two forms of discogenic pain, referred to as annular-disruption-induced LBP and endplate-disruption-induced LBP, both classified in progressive degrees of damage (Peng 2013). Grade I, II, and III radial fissures reach the inner, middle, and outer third of the annulus, respectively. Grade IV fissures are grade III types along with associated circumferential spread of contrast medium inside the outer annulus. Peng reported up to 70% of grade III fissures to be associated with pain and 70% of all painful discs show at least a grade III fissure. Discs with no fissures or having only grade I or II fissures are uncommonly symptomatic (Peng 2013).

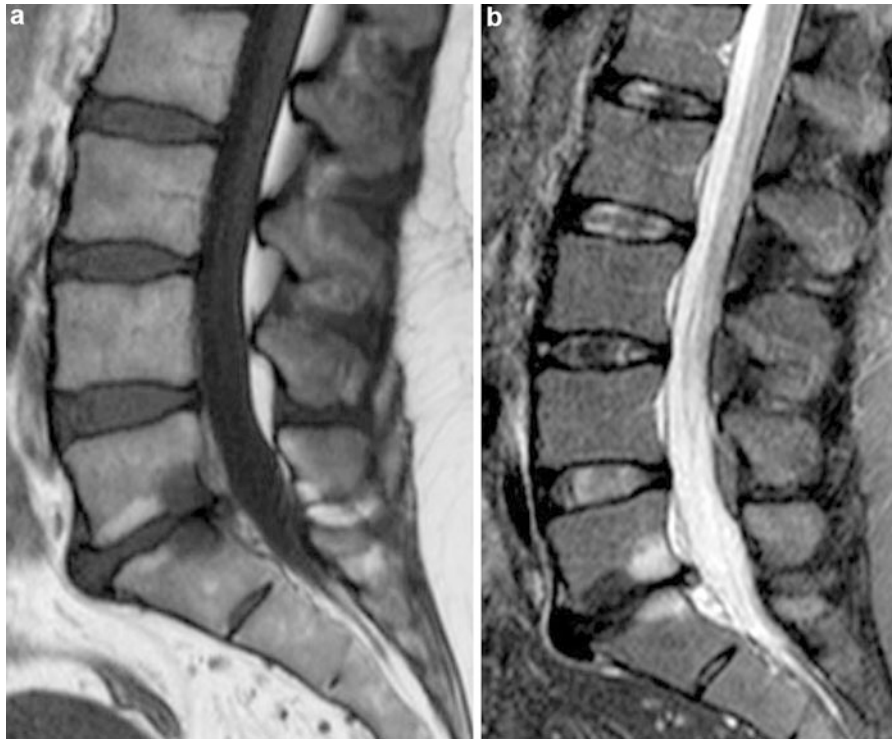


Fig. 6 (**a**–**b**) Sagittal FSE T1-WI (**a**) and T2-WI (**b**) images showing L5-S1 disc bulging associated to mixed type I and type II Modic changes involving posterior and anterior halves of both L5 and S1 endplates, respectively. Modic changes are an expression of intervertebral osteochondrosis and may coexist in the same interbody joint. Extended Modic I–II lesions can be painful and associated to segmental instability

Discography remains the only diagnostic test for low-back pain which consents the reproduction of pain suffered by patients, but significant controversy exists as to whether it really offers further diagnostic information in respect to MR imaging. In addition, morphological features on discography do not always correlate with pain response, and back pain may be elicited also in asymptomatic patients or when spinal pain is due to no spinal pathologies.

The negative predictive value of the absence of an HIZ on MRI for non-painful discography is 97%: A negative MRI eliminates for that the indication for discography. Surgical outcome of spinal fusion was better in patients with positive discography and an MR-documented abnormality than in those having positivity of discography alone. Discography alone has limited predictive value for surgery.

Endplate Changes

Pathology, Clinics, and Imaging

Disc degenerative and disruptive changes are often flanked or preceded by endplate changes. Endplate fatigue micro-fractures can even occur after repetitive loads even during the normal daily activities. The antigenic nuclear proteins are exposed to vessels in the vertebral spongiosa trough endplate micro-fractures and may elicit an autoimmune inflammatory response which diffuses throughout the disc and may cause its inflammatory degradation (Bogduk 2012). Furthermore, the endplates having similar density of innervations as the annulus may be a direct source of pain. Endplate-related LBP is thought to account for 16.7% of cases of chronic discogenic LBP (Peng 2013).

Modic described three features of endplates and subchondral bone degeneration detectable by MRI (Modic et al. 1998).

- Type-1 signal changes appear as hypointense on T1-WI, hyperintense on T2-WI in the sub-endplate bony areas, increasing after contrast enhancement, caused by ingrowth of reactive fibro-vascularized tissue and edema (Fig. 6).
- Type-2 lesions show hypersignal in T1-WI and T2-WI, reflecting fatty infiltration.
- Type-3 lesions show hypointensity on all sequences which reflects final sclerosis.

There is a natural trend of type-I lesions to convert over time in the other types. In fact, Modic changes represent different phases of a unique reactive process mounted by vertebral endplates in response to repetitive traumas or overloads and they can coexist in the same disco-vertebral junction. Occasionally, osteochondrosis can appear in a more aggressive, inflammatory, and painful form, with endplate erosions associated to extended edema and contrast enhancement of the vertebral bodies (erosive osteochondrosis), mimicking a spondylodiscitis, but lacking peridiscal soft tissue changes (Fig. 7).

By discography, endplate lesions were graded by Peng in five types (Table 2). Painful discs showed endplate lesions equal or more severe than grade III (local dispersion of contrast medium in the subchondral bone) (Peng 2013).

Modic changes become more frequent with aging and can involve both or just one endplate, entirely or only partially, mainly their anterior segments. While the Modic classification has proved to be reliable and well reproducible, the clinical relevance of changes has also been long debated. Modic type 1 lesions are reactive changes occurring with significantly greater frequency in patients suffering acute or chronic pain than in asymptomatic subjects. About 73% of patients with type I change and 11% with type II change have low back pain. Several studies found high specificity (87–98%), but a relatively low sensitivity (14–48%) of type I/II changes as a predictor of discogenic pain at provocative discography. However, most extended type 1 changes had a PPV of 100% for painful disc in a report by Weishaupt (Weishaupt et al. 2001). Modic type I changes are also associated to segmental instability with up to 70% of patients having segmental hypermobility. Successfully treated patients with surgical fusion show conversion of type I in type II lesions or complete disappearance thereof, whereas pseudo-arthrosis causes their persistence or reappearance. In several studies, the fusion surgery gave much better results in patients showing Modic type I changes than in subjects having isolated disc degeneration or type II changes' disc degeneration.

Intervertebral Disc Herniation

Pathology, Clinics, and Imaging

DDD and IDD can both progress to a disc herniation. A disc herniation is uncommon without other signs of spinal degeneration, even in cases of supposed traumatic genesis. A disc herniation is a focal displacement of disc material, cartilage, and apophyseal bone fragments, beyond the normal intervertebral disc space (osteophytes excluded) and can develop through a tear inside the annulus fibrosus or a focal endplate fracture (intervertebral herniation or Schmorl's node).

The nomenclature proposed by the Combined Task Force, distinguishes the disc herniation from bulging. A generalized and circumferential, symmetrical or asymmetrical, disc displacement spanning over 50% of disc circumference forms a bulging and can either be physiological at L5-S1 and at mid-cervical levels or an expression of DDD. The disc herniation, according to shape, can be a *protrusion*, either broad-based (involving 25–50% of disc circumference), or focal (<25% of circumference), or an *extrusion* having a base, in any plane, narrower than the height of the displaced disc material, which suggests the complete failure of annulus and the expression of disc substance into the epidural space, while a protrusion can correspond to a contained herniation with no violation of external annulus (Fardon et al. 2014).

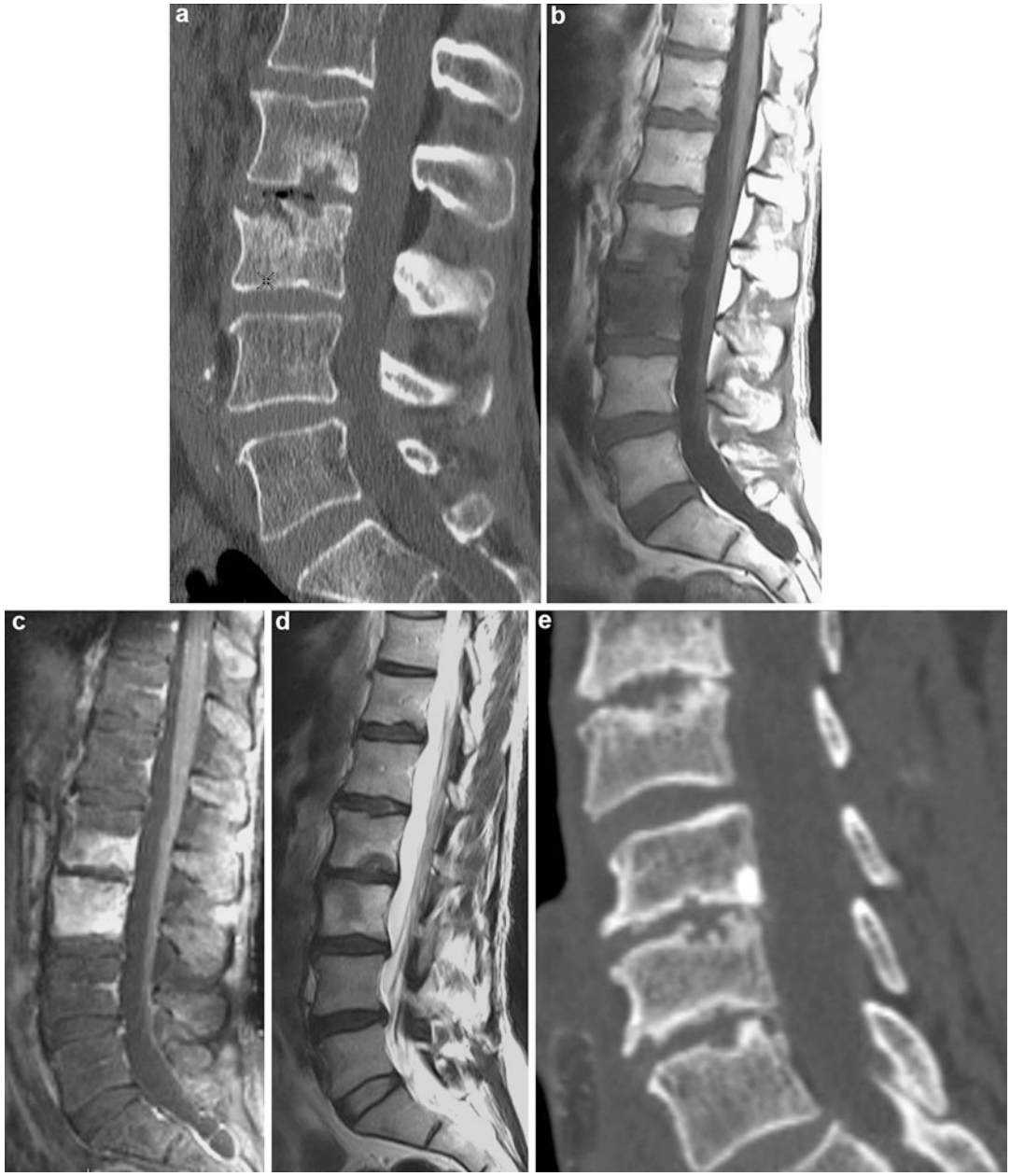


Fig. 7 CT reformatted midline image of lumbar spine showing erosive changes involving the L2-L3 endplates associated to disc collapse and extended subchondral sclerosis. The presence of gas inside the disc renders less probable a septic spondylodiscitis (**a**). The latter was definitively excluded by MRI. (**b**), (**c**), and (**d**) Sagittal T1, T1 fat-sat, and T2-WI demonstrate extensive bone marrow edema and contrast enhancement but no hypersignal and no enhancement of disc and complete absence of perivertebral inflammatory soft mass. (**e**) CT sagittal reformatted image in a different patient showing multilevel erosions of cervical endplates associated to disc spaces narrowing. In both patients, hematologic inflammatory markers and leucocytes were normal

A condition for disc extrusion to occur is probably the association of a nucleus pulposus degraded, no more intrinsically cohesive but converted in an expressible form, with a full thickness annular tear. Acute and isolated disc herniation is rare because experimental single compressive traumatic event usually provokes endplate fracture rather than an annular tearing.

Table 2 Endplate disruption grading scale by discography (Peng et al. 2009)

Grade 0: No disruption
Grade 1: Contrast medium flows into the cartilage endplate through a tear
Grade 2: Contrast medium flows into the bony endplate
Grade 3: Contrast medium flows into the subchondral cancellous bone with a local dispersion
Grade 4: Contrast medium disperses extensively in the cancellous bone

On CT, the herniated material maintains a density similar to parent disc, whatever the location and size are, even in case of free migrated fragments. Even tiny calcifications inside it are well demonstrated.

On MRI, the large extruded or migrated herniations often appear hyperintense to parent disc on T2 weighted images owing to fluid imbibition and inflammatory reaction they prime. The latter is responsible for surrounding and centripetal contrast enhancement (Fig. 8).

There are no studies comparing the diagnostic accuracy of actual MDCT with MR, but CT may not be inferior in the detection of disc herniation. High resolution reformatted sagittal views can improve the detection of larger herniations occupying the full area of spinal canal in the axial images. In both modalities, contrast medium

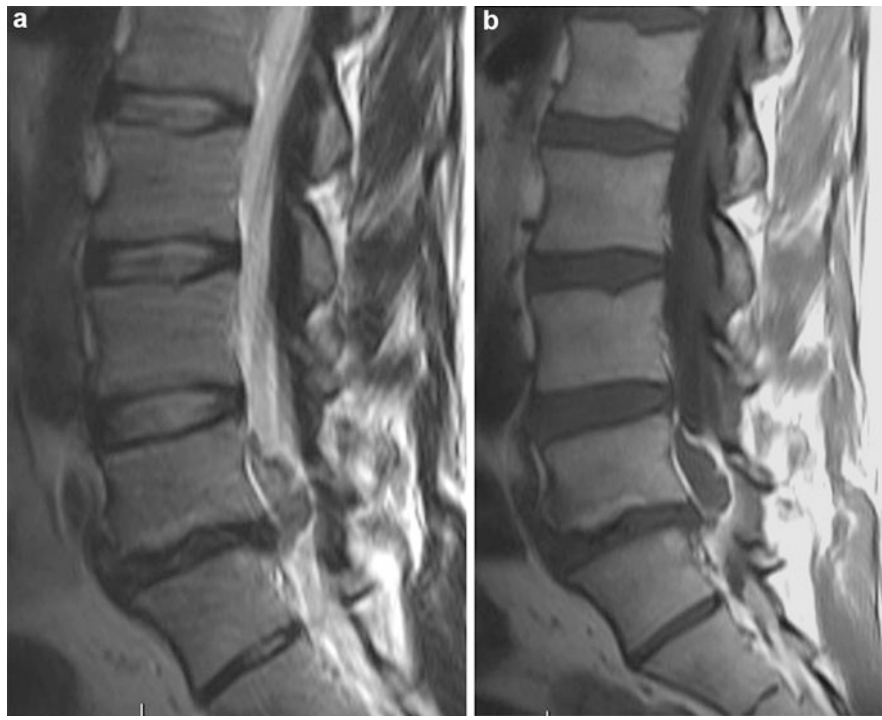


Fig. 8 Parasagittal FSE T1-WI (**a**), FSE T2-WI (**b**). Huge disc prolapse, migrating cranially from L5-S1 disc all along the L5 vertebral body. Large extruded and migrating disc herniations show hyperintense signal in relation to parent disc and are surrounded by a ring of contrast enhancement due to an inflammatory reaction which favors the spontaneous regression of the extruded material

administration may be necessary for excluding an epidural mass or, in postsurgical setting, for differentiating epidural fibrosis and residual/recurrent disc herniation. Contrast imaging may also reveal the real dimension of extrusions or sequestered fragments by distinguishing them from surrounding reactive tissue. Often the disc extrusion occupies only a limited part of a large soft tissue mass formed by reactive granulomatosis and congested and dilated epidural veins.

The precise spatial localization of the disc herniation and especially of migrate fragments in relation to anatomical landmarks used by surgeons is useful to prevent wrong-level surgery or incomplete disc removal and to guide the surgical approach.

- Axial plane: The location of a disc herniation can be central, paracentral, subarticular (located between the medial margin of the facet joint and that of pedicle), foraminal or lateral (between pedicle margins), far lateral (extraforaminal), and anterior.
- Sagittal plane: The location is referred to the pedicular, infrapedicular, discal, or suprapedicular levels.
- Spinal canal involvement through a disc herniation should be graded as mild, moderate, or severe, for degrees of occupation increasing by an additional one-third of the its cross-sectional area.
- A paracentral or subarticular herniation typically compresses the nerve root descending toward the subjacent foramen. The impingement of the nerve traversing the neural foramen at same level of disc requires a disc extrusion migrating cephalad into the neural foramen or lateral recess (Fig. 9).

A bulging disc or protrusion seldom encroaches the nerve root at a foraminal level and almost never do it at an extraforaminal site. An extraforaminal disc herniation, similar to a foraminal lesion, compresses the exiting nerve and should be searched for, whenever a patient complaints radicular pain without any evidence of pathology inside the spinal canal (Fardon et al. 2014). Large disc extrusions may penetrate the posterior longitudinal ligament and even the dura. The link of the disc herniation with the posterior longitudinal ligament (PLL) and with the dural sac is difficult to define. MR signs suggestive of extra-ligamentous disc herniation are

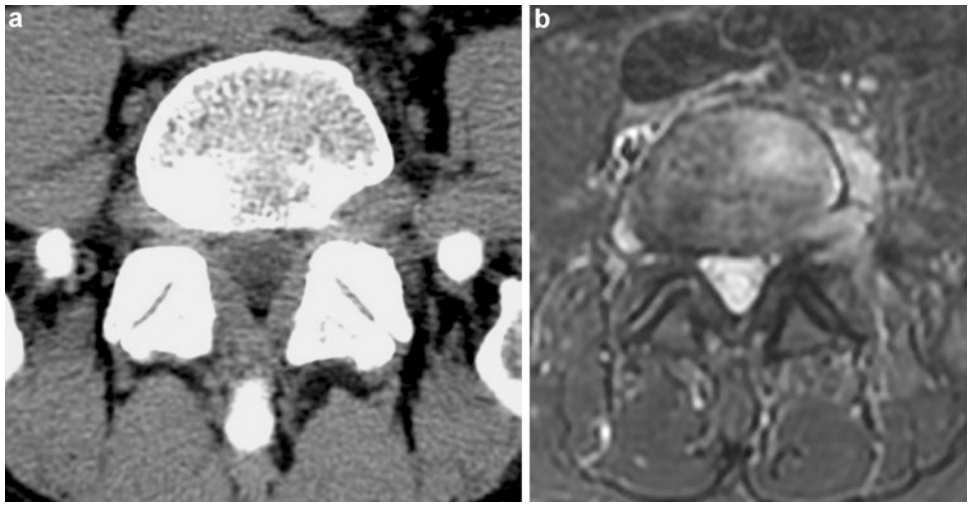


Fig. 9 (**a**) CT axial image though L5-S1 foramina showing a left intraforaminal hyperdense mass associated to disappearance of intraforaminal fat, compression of ganglion, and scalloping of left posterolateral vertebral body. Bone remodeling can occur also in the presence of inflammatory reaction related to a disc extrusion; (**b**) MR FSE postcontrast T1-WI image showing intraforaminal disc herniation inside inflammatory enhancing tissue

shown in the Table 3 (Oh et al. 2013). In combination, they had an accuracy of 76.1% at surgical report (Fig. 10).

Intradural herniations are rare and develop, owing to the passage of disc material into the subdural or subarachnoid space through a perforation of the annulus, the PLL, and the dura, favored by prior dense adhesion due to a disc herniation, surgery, trauma, or inflammation as well as spinal canal stenosis (Fig. 11).

Intradural lumbar disc herniation is more frequent at lumbar spine and at L4-L5 and represents less than 1% of all lumbar herniations, but at thoracic level, up to 7% of symptomatic herniations requiring surgery have this migration. Intradural disc herniations typically widen the ipsilateral subarachnoid space displacing away of neural elements, but when they are very large, this sign is not valuable. In the sagittal MR images, a discontinuity of the posterior longitudinal ligament may or may not be present along with a bilobular morphology of the extrusion, while a "hawk-beak" sign at axial T2-weighted imaging may be present. After administration of gadolinium, a typical peripheral rim enhancement does appear. The eventual presence of gas inside can be a clue to diagnosis, especially at CT.

Table 3 MR Signs Suggestive of Extra-Ligamentous Disc Herniation (Oh et al. 2013)

Spinal canal compromised for more than half its dimension
Internal signal difference in the disc herniation
Presence of an internal dark line corresponding to violated and interposed LLP
A budding appearance of the disc material
Disruption of the continuous low-signal-intensity line covering the herniation

The differential diagnosis of an intradural disc herniation includes nerve sheath tumor, inclusion cyst, meningioma, arachnoid cyst, and metastasis even though ring enhancement is not typical in these entities. Resection of an intradural disk herniation requires a transdural approach, and surgery is often difficult. A dural dehiscence is not always found at surgery of intradural herniations. Adhesions are typically present and are a clue to the presence of an eventual intradural disc, particularly if the intraoperative findings do not match the preoperative imaging findings. Over 90% of

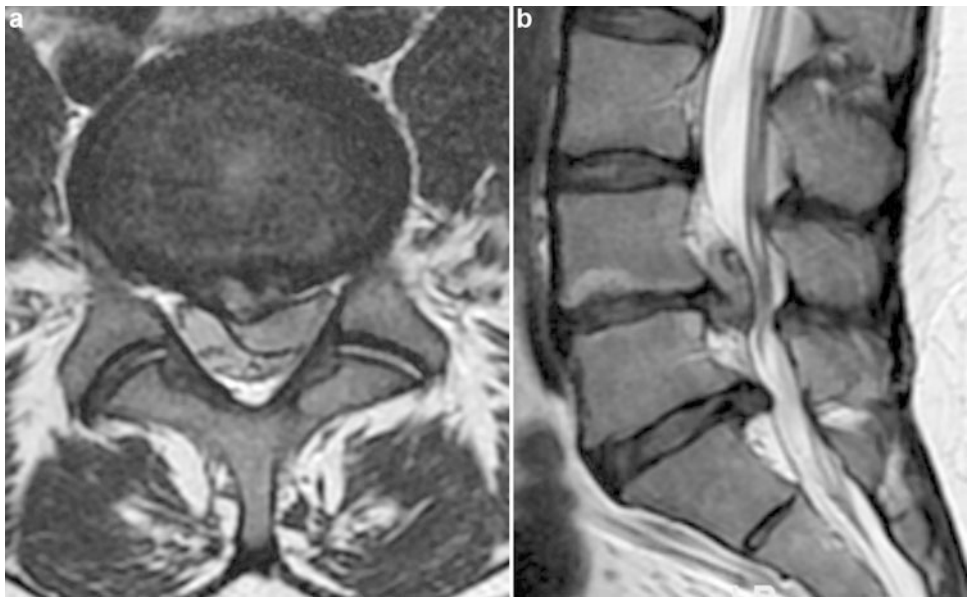


Fig. 10 Axial (**a**) and sagittal (**b**) FSE T2-WI. Large extruded disc herniation through an exploded posterior annulus and with signs suggestive of transligamentous extension, among which there is the black line of PLL traversing herniation material (Table 3). Notice the hypersignal of the extrusion in comparison with the parent disc

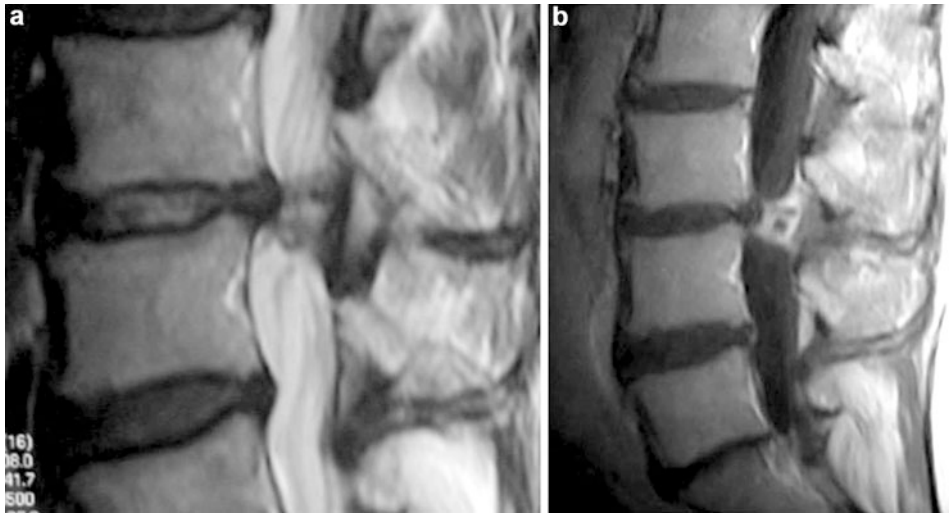


Fig. 11 (**a**) MR FSE T2-WI sagittal image showing a bilobate L3-L4 disc herniation, most of spinal canal are seemingly encircled by nerve roots; (**b**) MR FSE T1-WI sagittal postcontrast image shows an intradural enhancing mass including focal hypointensities corresponding to disc fragments

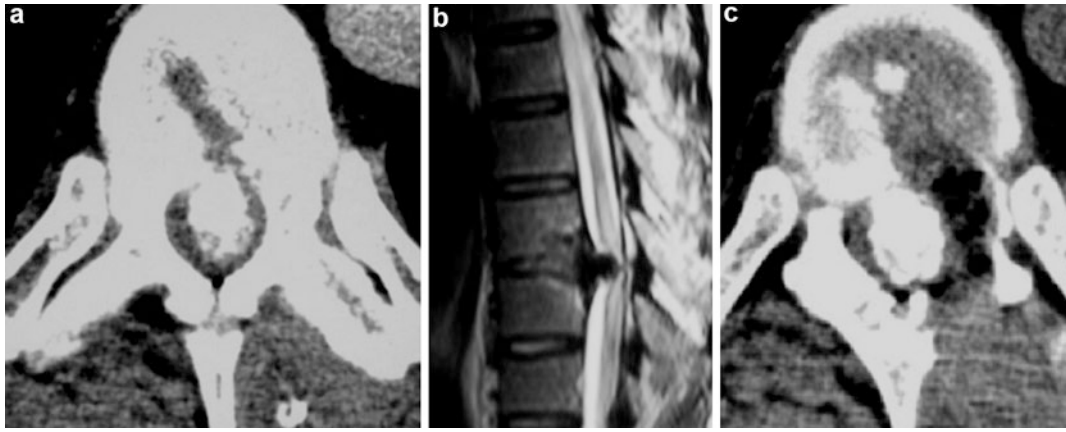


Fig. 12 48-year-old suffering from long-time dorsal axial pain with recent appearance of spastic gait and lower limb weakness. (**a**) axial CT scan through D7-D8 disc showing a giant densely calcified disc herniation occupying most of spinal canal. Notice the restricted base of contact with the parent disc, with acute angles and the dural sac encircling it without an *en bloc* displacement. (**b**) MR FSE T2-WI showing marked displacement and compression of an edematous spinal cord. (**c**) Postoperatory control CT demonstrating the persistence of the herniation. At surgery, the expected extradural herniation was not found neither a dural dehiscence was noted. The surgeon thought he was at a wrong level. The patient on awaking presented frank paraplegia. The displacement of dural sac during surgery had increased the compression of spinal cord by herniation

intradural herniation occurs in the lumbar spine, but symptomatic giant thoracic herniations also can occur. Giant herniations are those that occupy more than 40% of the spinal canal and, for being often densely calcified, tend to erode the dura (Fig. 12).

They prevail in women in the third to fifth decades and in 90% of time occur between T6 and T11. An extruded herniation can migrate and lose any contact with the parent disc forming a sequestered fragment (Fig. 13).

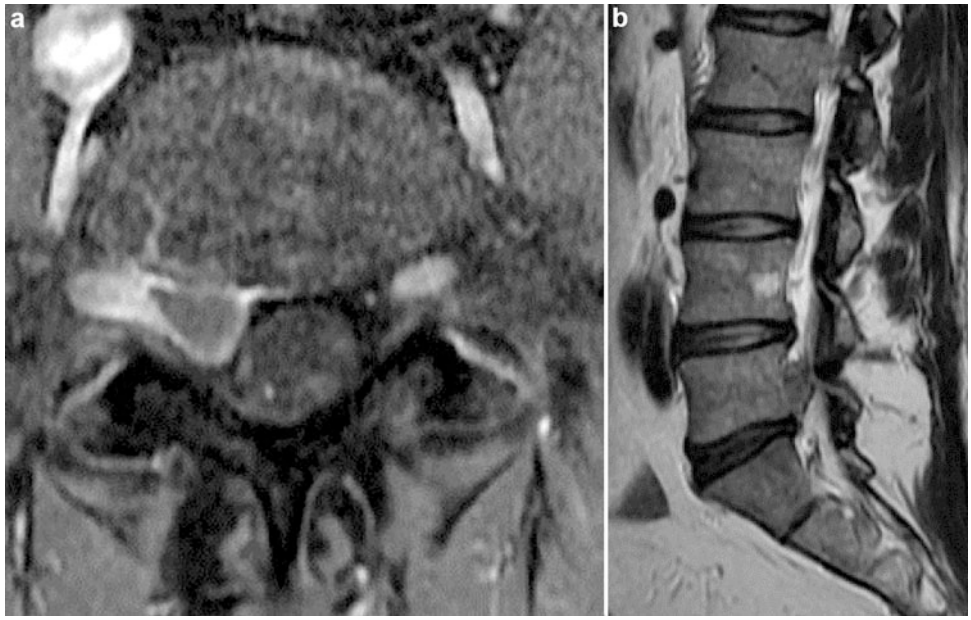


Fig. 13 (**a**) MR FSE T1-WI postcontrast axial image at L5-S1 showing a free fragment migrated cranially into the right lateral recess of L5 encircled by a thick halo of contrast enhancement. (**b**) MR parasagittal FSE T2-WI image demonstrating the migrated disc fragment with subarticular location and at infrapedicular level

Migration is usually confined to anterolateral epidural space by multiple anatomical barriers, including the dura, PLL, the sagittal midline septum, the lateral membrane, the epidural fat and venous plexus, and the nerve roots. Rarely a disc extrusion can migrate within the posterior epidural space where it may or may not be sequestered. Most of dorsally migrated disc herniations are located in the lumbar region, where the most common level is L3-L4. Isolated reports regard the cervical and thoracic spine. On imaging, posterior epidural disc herniations manifest as a nonspecific mass in the dorsal epidural space, hyperdense on CT and with a variable signal, more often hyperintense on T2-weighted and hypointense on T1-weighted MR images (Fig. 14). After gadolinium administration, it appears peripheral enhancement, like disc fragments elsewhere.

Dorsal disc herniations are in the differential diagnosis for a posterior epidural mass including a synovial cyst, abscess, hematoma, or even a neoplasm. Some thoracic herniations can have an anterior and lateral component (either disc material or granulation tissue), which can be a key for the diagnosis but is often missed by both radiologists and surgeons (Diehn et al. 2016). Synovial cysts are connected to degenerated facet joints and may contain gas and show peripheral enhancement, like dorsal disc fragments. Epidural abscesses also show ring enhancement, whereas neoplasms generally have solid enhancement. Surgery is performed in the vast majority of reported cases, in part because many patients show acute symptoms or cauda equina symptoms. Approximately, 7–10% are within the foramen or extraforaminal (Fig. 15).

Extraforaminal (far lateral, extreme lateral, retroperitoneal, and lateral) disc herniations more often have an intraforaminal component too, but about 20% are exclusively lateral to the foramen; L4-L5 is the most commonly involved level. The exact position in relation to neuroforamen is an important criterion for surgeons, who may decide a paraspinal approach rather than a classic laminectomy with facetectomy. However, far lateral herniations are often underdiagnosed because they are excluded by sagittal MR/CT imaging (usually performed within the range of the vertebral body width) that in addition is not optimal for

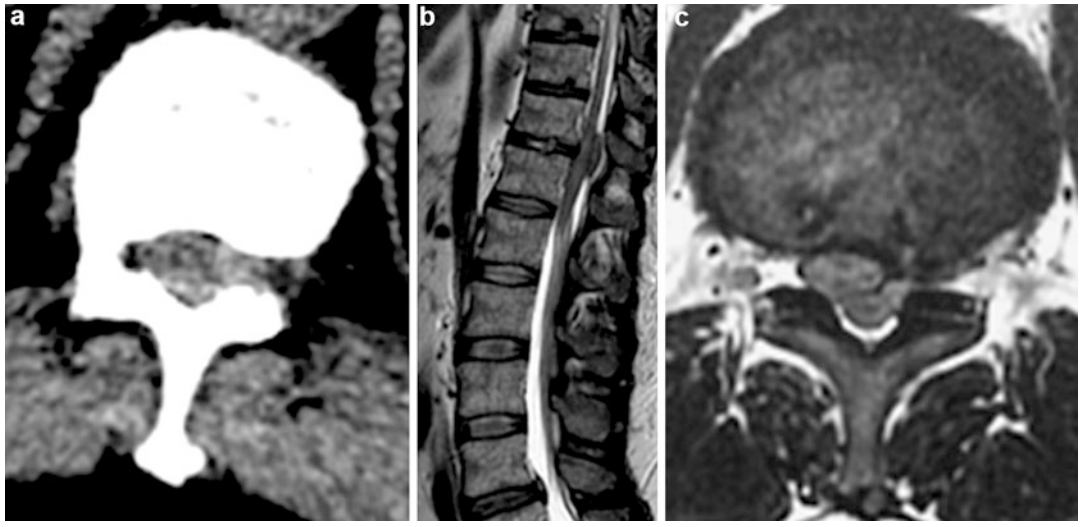


Fig. 14 40-year-old man after sneezing felt an electric discharge all along the spine and radiating into lower limbs followed by progressive paraparesis and cauda equine syndrome within 24 h. (**a**) CT done in emergency showing at D11-D12 level a posterolateral herniation. The dorsal epidural hyperdensity was initially overlooked. (**b**, **c**) RM sagittal and axial T2-WI demonstrated an epidural lateral and posterior mass compressing the thecal sac. The left epidural component in continuity with parent disc suggested a dorsally epidural migrated disc extrusion compressing the spinal cord showing edema

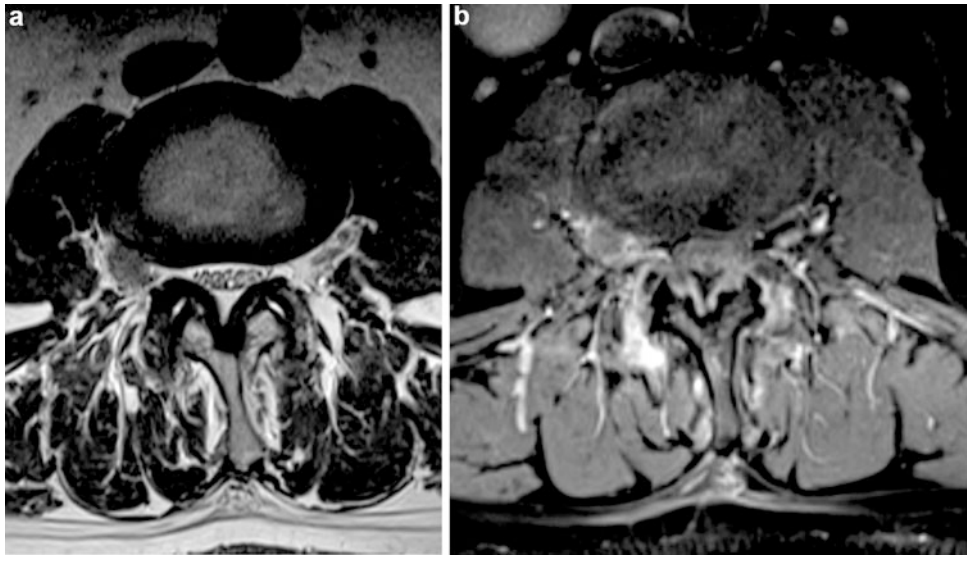


Fig. 15 Subject suffering acute radicular pain in the distribution of left L3 root. MR axial FSE T2-WI (**a**) and FSE T1-fat saturation (**b**) images. Extreme lateral purely extraforaminal L3-L4 left disc herniation

their visualization, while, on axial imaging, they remain often overlooked because they are uncommon and difficult to differentiate from an abnormal nerve root course.

Far lateral herniations may cause radicular axial pain or may be asymptomatic and incidentally discovered. Missed herniations in symptomatic patients may cause poor outcomes owing to

unsuccessful or wrong-level surgery. On MR imaging, a focal bumping of disc contour are clues for diagnosis, eventually with paraspinal fat displacement or obliteration, a change in diameter, and displacement of the exiting nerve root (Fig. 15). These four findings are not always contemporarily present, but even the presence of just one makes consider the diagnosis. Contrast imaging shows a rim-type enhancement like herniations in usual sites, with variable extension of inflammatory changes into the adjacent fat and muscle. In these cases, the differential diagnosis includes an abscess. However, more homogeneous enhancement has been described, making it hard to differentiate from metastasis, adenopathy, or a nerve tumor.

A disc cyst is a very rare pathological condition, consisting in a communication of intervertebral disc with epidural cyst that can behave like a disc herniation (Fig. 16). Because of its rarity, a disc cyst can be mistaken for other intraspinal cysts, mainly synovial or perineural cysts abutting the disc (Kono et al. 1999). The cyst content is typically hyperintense on T2-weighted MR images; the signal intensity on T1-imaging is usually hypointense, but hyperintense in case of internal hemorrhage. A blood-fluid level, if present, can be a clue to the diagnosis. Adjacent bone erosion may be present and is more evident on CT.

The natural trend of the disc herniations toward the spontaneous regression occur in patients undergoing conservative treatment. Modic reported a significant regression or a complete disappearance in one-third of individuals by 6 weeks, and in two-third by 6 months after clinical presentation (Modic et al. 2005). Bozzao reported regression in 68% of patients during an average follow up of 11 months (Bozzao et al. 1992). The regression involves to a greater degree the extrusions and sequestered fragments (Fig. 17) and especially massive herniations that occupy over than 50% of the spinal canal.

The cause of regression of the disc herniation can be dehydration, resorption trough an inflammatory response by macrophages, or a combination. The inflammatory mechanism prevails which appears as a thick halo of peripheral contrast enhancement in the epidural space, consisting of congest veins and reactive tissue. Disc bulges and contained herniations, confined within the annulus, are less likely to change over time and to respond to conservative treatments. The morphological changes of disc herniation do not always correlate to clinical changes.

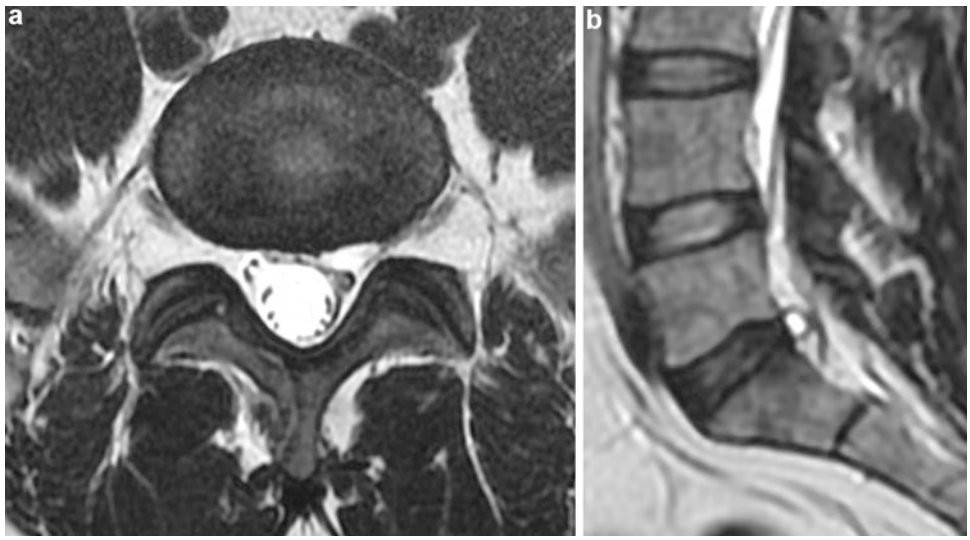


Fig. 16 Patient suffering of left L5 acute radiculopathy. (**a**, **b**) MR axial and sagittal FSE T2-WI showing a little cyst in strict contact with posterolateral left annulus with internal homogeneous fluid signal

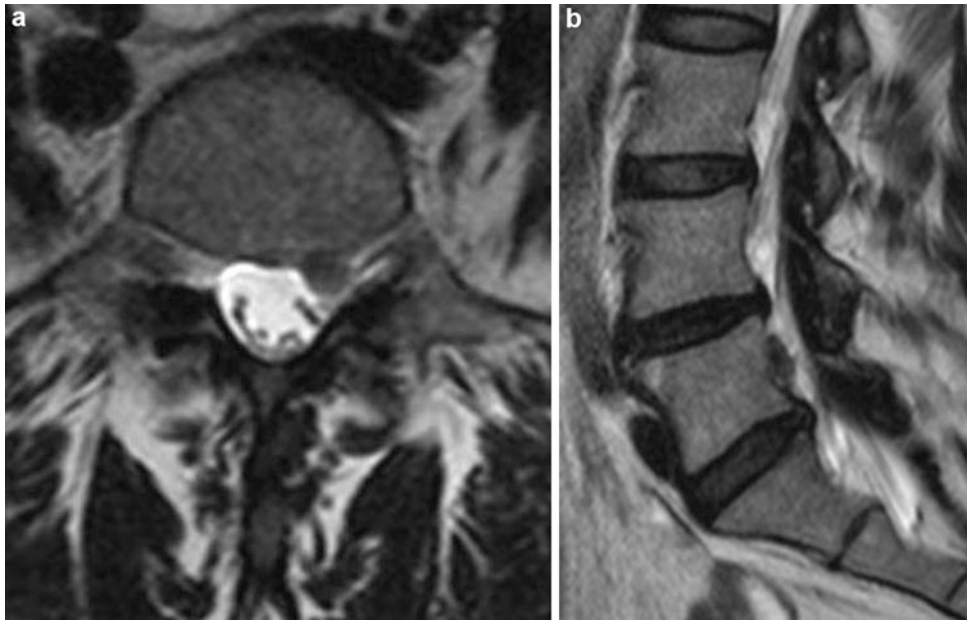


Fig. 17 MR axial (**a**) and parasagittal (**b**) FSE T2-WI images showing a cranially migrated free disc fragment from L5-S1 disc located in the ventral left epidural space causative of acute back pain and left L5 radiculopathy. Twelve months after a conservative therapy, the fragment is completely resorbed. Lower limb pain did regress, axial pain did not

According to Modic, the type, size, and location of a disc herniation at presentation and its changes in size and type over time do not correlate with outcome. As a consequence, early imaging in acute setting does not offer additional information over clinical assessment alone apt to modify the treatment (Modic et al. 2005). Even for massive lumbar disc herniations, the extent of the decrease in disc volume does not correlate with the degree of clinical improvement (Benson et al. 2010). The surgical option is essentially based on the clinical findings.

Intervertebral disc herniations occur owing to the focal disruption of the cartilaginous endplate of the vertebral body. The endplate or the subchondral bone can be weakened by the vascular channels perforating the endplate or by degenerative, metabolic, traumatic, neoplastic, and infectious processes. Repetitive traumas can provoke stress focal fractures in the endplates. In chronic form, intervertebral herniations are very common incidental spinal features with similar frequency in young and old individuals, and prevail at the thoracolumbar junction. Old stable lesions have signal intensities on T1- and T2-weighted MR images similar to that of the parent disc, with surrounding sclerosis. Acute lesions are associated to Modic-type I changes, often with a concentric morphology, enhancement after gadolinium administration, and are painful (Fig. 18).

Concentric rings of T2 hyperintensity surrounding the node had a negative predictive value of 72% for neoplasm, infection, and fracture. Signal and enhancement changes fade gradually as the Schmorl's node become inactive. Uncommonly, a peripheral Schmorl's node may focally deform or even detach a fragment from vertebral margin, forming a mixed hard disc-bony herniation or a retro-marginal dissecting herniation, respectively (Fig. 19). During youth, this mechanism may create a limbus vertebra.

Disc herniation is the most frequent cause of radicular pain. Radicular pain is that provoked by irritation of a spinal nerve or its root; it is shooting or lancinating in quality and descends in the lower limb along a narrow band well localized within a dermatome. The mechanical compression, alone, elicits sustained pain only when involving a dorsal

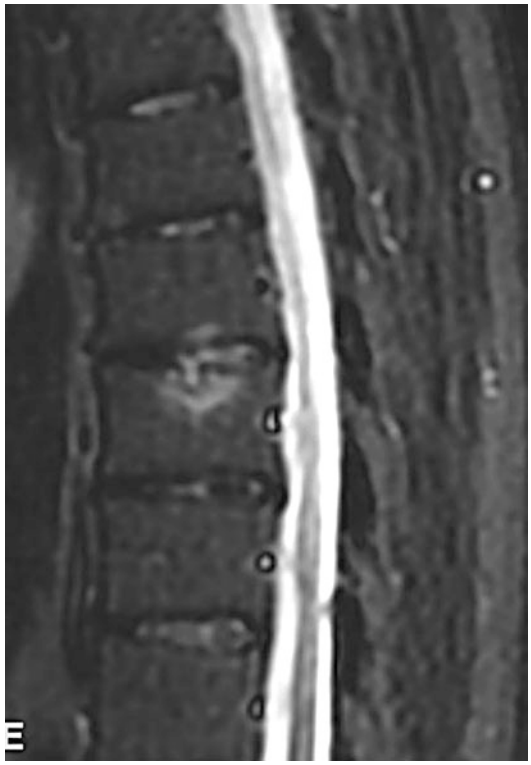


Fig. 18 MR sagittal STIR image showing a little subacute intervertebral herniation in a patient complaining of acute axial pain. The intervertebral node is encircled by a halo of edema

root ganglion. Otherwise, inflammatory mechanisms prevail and explicate the evidence that asymptomatic people may show compressive disc herniations, or pain can bereleaved even if patients shoe nerve roots compression on Imaging.

Like radicular pain, referred pain is also perceived in areas different from the real source, but it differs in quality and distribution, being aching, constant, poorly localized, and distributing within the deep tissues belonging to a dermatome. Referred pain would originate owing to the convergence of two different sensitive primary neurons on the same neuron inside the dorsal horns of spinal cord and in the thalami, generating a false mental localization of painful sensations. Failure to distinguish radicular pain from somatic referred pain may lead to misdiagnosis and thereby mismanagement. While imaging can often establish the causative lesion in case of radicular pain and radiculopathy, it is unable to reveal the real source of pain in the majority of cases of referred pain. To confusing the two types of pain carries the risk of false-positive interpretations and inappropriate surgery.

In a patient complaining radicular pain, the finding on imaging of an extruded or sequestered disc herniation with radicular compression may

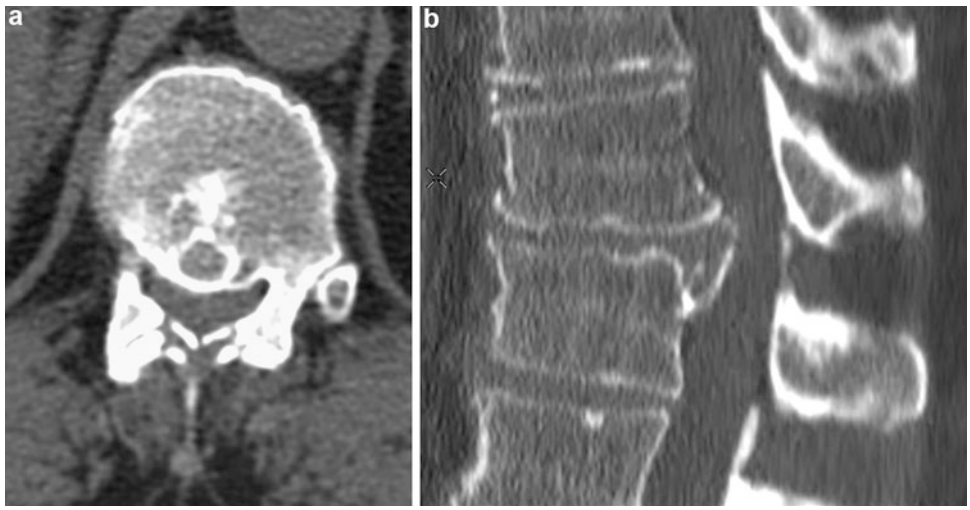


Fig. 19 Retromarginal intervertebral disc herniation. (**a**) axial CT image showing the presence of a Schmorl's node located inside the posterior margin of superior endplate and deforming it. (**b**) CT reformatted sagittal image confirms the focal bumping of posterior wall cortex by intervertebral disc herniation. In this case, the posterior cortex is not interrupted and the body edge is not detached

justify the clinical context, but with a prevalence of up to 28% in asymptomatic people, a disc herniation is not necessarily the cause of pain. Like for all other degenerative spine pathologies, any excessive confidence on imaging can lead to inappropriate treatments. Intradural disc herniation can commonly manifest with cauda equina syndrome, in the lumbar spine, and with Brown-Séquard syndrome or severe myelopathy, in the cervical and thoracic segments. As to symptomatic thoracic disc herniations, the most common manifestation is localized or axial pain. Myelopathy with motor impairment, hyperreflexia, and sensory impairment are also common. Bladder dysfunction is less common, while acute myelopathy is rare.

Facet Joints Degeneration

Pathology, Clinics, and Imaging

Facet joints control the direction and amplitude of mobile segment movement and share the axial loads with the disco-vertebral joints. Both disco-vertebral and facet joints degenerate with age, being parts of a unique three-joint complex where biomechanical changes affect over time all joints of the MS in a centrifugal way. While disc degeneration may exist without facets osteoarthritis, the inverse condition alone is uncommon.

Degenerative facet changes are another important cause of axial, radicular, or referred pain. Owing to the rich innervation of synovia and capsules, the facet joints can be a direct source of pain, but they often provoke nerve root compression in the spinal canal or in neural foramina. Degenerative changes of facet joints are typical of all synovial joints, consisting in hypertrophy, spurring, osteosclerosis, fibrillation thinning, and ulceration of cartilages with narrowing of the joint space, subchondral cysts, synovial cysts, joint effusion, capsular and ligament hypertrophy, and calcification.

On imaging, facet hypertrophy appears as a global enlargement of facets with preserved proportions of cortex and spongiosa, whereas the osteophytes are bony marginal mushroom-like productions where at the cortical margins. Often both changes coexist (Fig. 20).

Degenerative facet changes mainly develop in the lordotic segments, in obesity, scoliosis, orientation asymmetry (tropism). Weishaupt proposed a four-point scale for grading of facet changes by CT or MR (Table 4) (Weishaupt et al. 1999).

The complex geometry of facets does not consent an accurate evaluation by plain radiographs. CT and MR have moderate to good concordance, sufficient to avoid CT after an MR study. While CT overcomes MR in depicting facets sclerosis, MR directly analyze the cartilages and shows the joint effusions. Thanks to the routine use of fat-saturated T2-weighted images and post-gadolinium fat-saturated T1-weighted images, MRI can display inflammatory reaction within

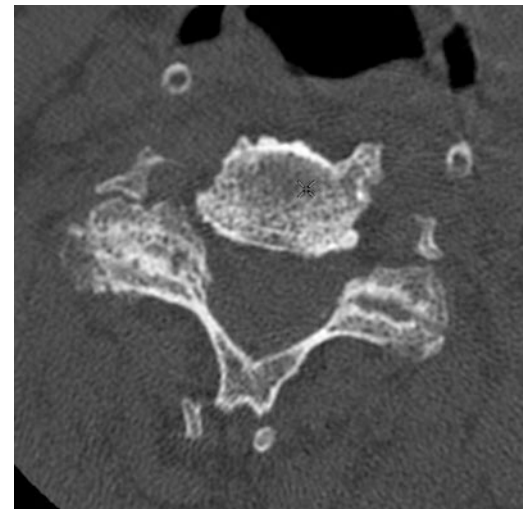


Fig. 20 Axial CT image in an old man showing marked degenerative and osteoarthritic changes involving facet joints

Table 4 Facet joints arthropathy grading scale by CT/RM (Weishaupt et al. 1999)

Grade 0: Normal space (2–4 mm width)
Grade 1: Initial narrowing (<2 mm) and/or small osteophytes, mild hypertrophy
Grade 2: Narrowing and/or moderate osteophytes/ hypertrophy, mild bone erosions
Grade 3: Narrowing and/or large osteophytes/severe bone erosions and/or subchondral cysts

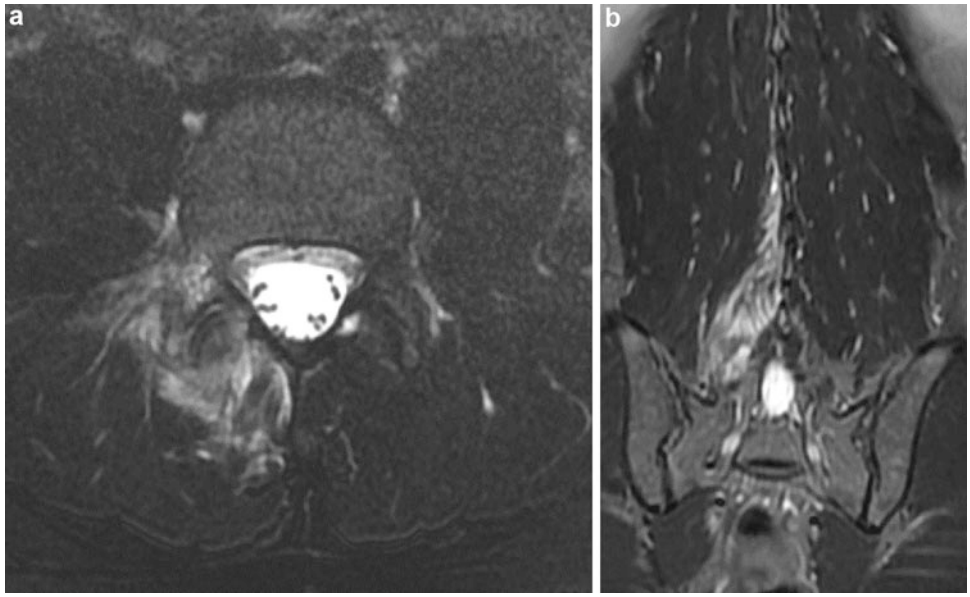


Fig. 21 Axial (**a**) and coronal (**b**) MR STIR images showing intra articular, bony, and periarticular inflammatory edema extending along paraspinal muscle fibers

and around the joints, as well as the marrow edema or fatty replacement in the articular processes (Fig. 21) (D'Aprile et al. 2007). However, the fluid-sensitive sequences are less sensitive in depicting the joints bony cortices and the amount of sclerosis present inside. Facet joint fluid seen on T2-weighted MRI sequences is a finding suggestive of spinal segmental instability. The acceptance of the interventional pain procedures and the development of motion preservation surgical technology have renewed the interest on the facet joints as a source of spinal pain.

Facet joints with no significant morphologic changes may be painful, while degenerative joints are most commonly asymptomatic. The low reliability of both clinical and imaging findings renders challenging choice as to which patient undergoes a diagnostic intra articular injection or medial branch block. The prevalence of the facet syndrome is variously reported. When complete relief of pain after diagnostic blocks was used as criterion, the prevalence was less than 10%. Degenerative changes of facet joints, capsules, and ligaments have an important pathological role in the genesis of spinal canal and foraminal stenosis.

Synovial cysts are out-pouching of synovia through defects of facet joint capsule due to traumas or degeneration (Fig. 22). Their prevalence was of about 10% in a population of patients undergoing MRI for back or leg pain, mostly with posterior, extraspinal location (7.3%). Synovial cysts differ from ganglion cysts which lack synovial lining and are not connected to the joint. Intraspinal cysts (2.7%) cause low back pain, radiculitis, and radiculopathy, sometimes neurogenic claudication. Symptoms can worse in case of inflammation surrounding the cyst.

Synovial cysts prevail in the lumbar spine and at L4-L5 level (60–70%) and are mainly favored by the increased motion of facets and by the instability, but they are also associated with rheumatoid arthritis and chondrocalcinosis. Cervical synovial cysts can also originate from the cruciate ligament of C1 (Fig. 22).

MR is the technique of choice, because it detects the cyst with great sensitivity as a rounded mass located in the posterior epidural space and centered on a facet joint. The internal signal of cysts varies from that of a fluid being equal to or slightly greater than that of CSF on T2-weighted

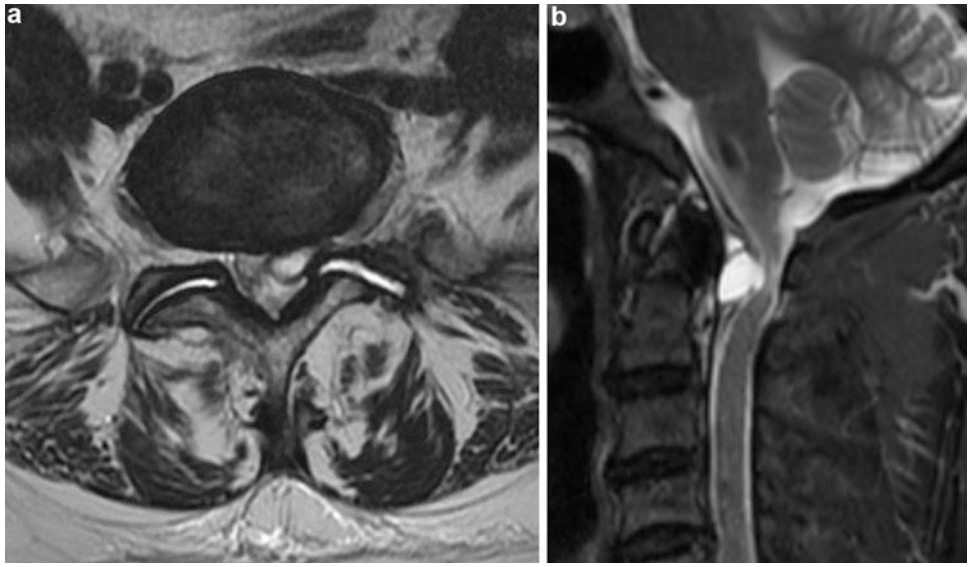


Fig. 22 (**a**) MR axial FSE T2-WI showing intra articular bilateral joint effusion and a large bilobate synovial cyst originating from L5-S1 left facet joint compressing the thecal sac. (**b**) MR midsagittal STIR image showing a synovial cyst of transverse ligament compressing the spinal cord

images, to hyperintense in case of hemorrhage, to hypointense for the presence of gas, encircled by a hypointense capsule which can enhance after contrast administration (Fig. 22).

CT shows a hypodense mass containing fluid or gas encircled by a wall which can contain calcifications (Fig. 23). The differential diagnosis regards the ligamentum flavum cyst (Fig. 24), a sequestered disc fragment, a cystic schwannoma.

Degenerative and Isthmic Spondylolisthesis

Pathology, Clinics, and Imaging

Spondylolisthesis refers to an anterior displacement of a vertebra in relation to the vertebra below (anterolisthesis). Wiltse classified lumbar isthmic and degenerative spondylolisthesis as type 2 and 3, respectively, along with dysplastic (type 1), traumatic (type 4), pathological (type 5), and iatrogenic (type 6) forms (Wiltse et al. 1976). Degenerative spondylolisthesis (DSL) is the most common cause of anterolisthesis after 50, with

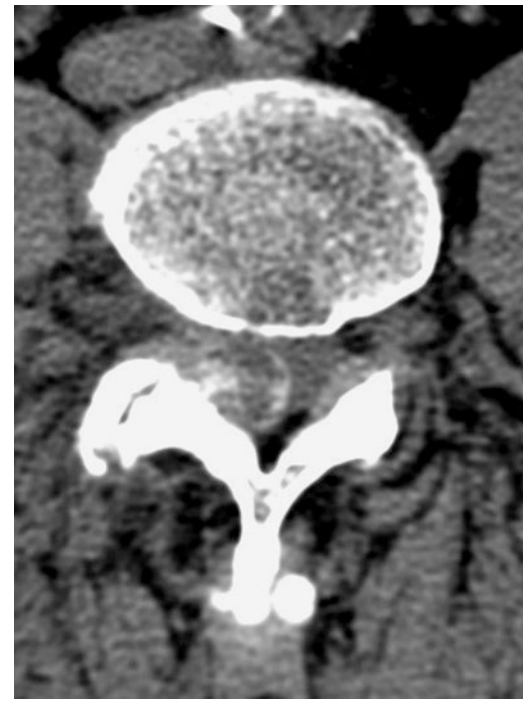


Fig. 23 Axial CT image of a large synovial cyst having calcified walls originating from degenerated right facet joint. Marked compression of thecal sac

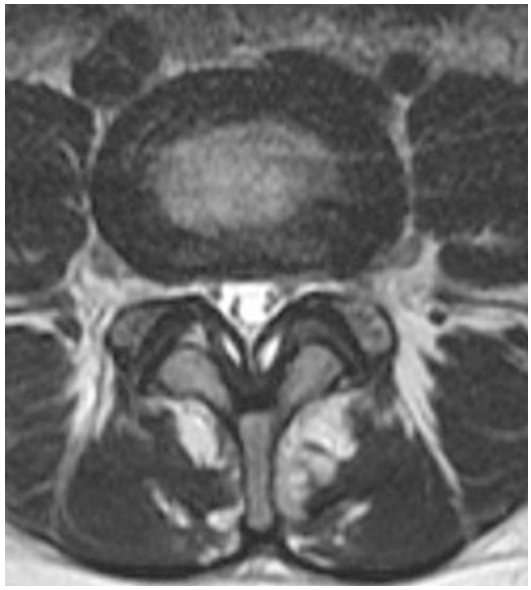


Fig. 24 MR axial FSE T2-WI. Double cyst inside flava ligament which appears hyperintense owing to mucoid degeneration and thickening

the prevalence raising in elderly subjects up to 14%. DSL prevails at L4-L5, mainly in case of L5 socialized, and in women. The cause of DSL is primarily the degenerative remodeling of facet joints associated to disc degeneration. The preserved integrity of the neural arc renders the slippage self-limited (not over one-third of AP diameter of endplate) with mild foraminal stenosis, whereas the stenosis of the central spinal canal and the lateral recesses may be severe, being often worsened by the ligament flava and joint capsules hypertrophy. While anterior subluxation is primarily a pathology of the posterior joints, retrolisthesis, the posterior translation of a vertebra is primarily a disorder of the disc space and prevails at L3-L4 (Fig. 25).

On imaging, the forward slip can be measured either in degrees from I to IV, by dividing the subjacent endplate in four quarters (Meyerding), or more accurately, as percentage of the AP diameter of the lower vertebra endplate (Taillard). On plain radiographs, the anterior displacement of the spinous process, concordant with that of the vertebral body, indicates the integrity of the neural arc consenting the differential diagnosis with the

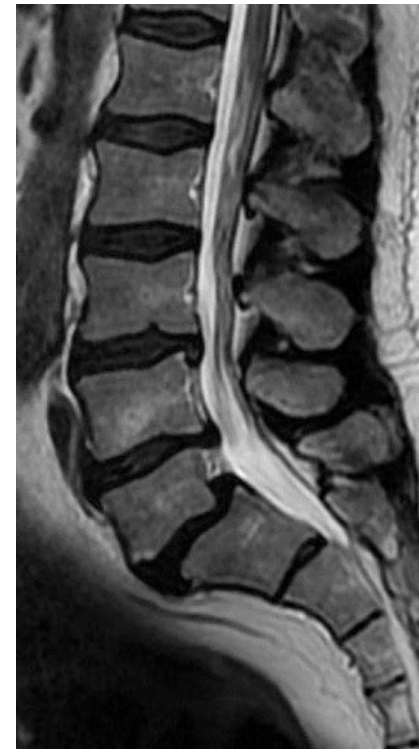


Fig. 25 Sagittal FSE T2-WI. Retrolisthesis of L4 and anterolisthesis of L5 on S1. Stenosis of L4-L5 foramina between dorsally displaced vertebral body and superior subjacent facets

spondylolysis. This finding can be useful also in MRI because of the difficulty in directly assessing the pars defect.

Disc degeneration can also lead to vertebral shift in the coronal plane (laterolisthesis). Lateral listhesis and angulation can be associated with lateral wedging of the vertebral body and asymmetric degeneration of the facet joints resulting in degenerative scoliosis. In case of retrolisthesis, the foraminal stenosis occurs because of relative forward displacement of the articular processes of the subjacent vertebra (Fig. 25). Anterolisthesis can be asymmetrical, with rotation of the slipping vertebra in the horizontal plane when the facet subluxation dominates on one side. The narrowing mainly involves the recess and the neuroforamen contralateral to rotational sense because of the greater slippage of the facet. The slipping vertebra can also rotate in the sagittal

plane, either in flexion or extension with foraminal stenosis prevailing in the latter form.

DSL is often an expression of instability and increases or appears with motion or standing (occult DSL) requiring dynamic radiographs in flexion-extension or dynamic MR. Occult DSL have to be suspected in case of joint effusions over 1 mm (Chaput et al. 2007). DSL not necessarily indicates actual instability: progression of slippage occurs only in 30% of cases, and 65% of patients do not worsen weather treated conservatively. With time, fibrosis of joint capsules, osteophytes, and disc collapse tend to re-stabilize the slippage (Matsunaga et al. 2000). Since disc collapse has proved to be a powerful stabilizing change, an instrumented fusion is recommended only when the preoperative disc height is greater than 2 mm.

DSL may be associated with axial low back pain but also with increased risk of neural element compromise for central canal, lateral, or foraminal stenosis. Unlike vascular claudication, pain in neurogenic claudication is also provoked by standing, appears after walking a variable distance, and is relieved by flexing the spine. Acquired ISL can also occur as a complication of degenerative processes (Fig. 25). Isthmic defects compromise the ability of the posterior elements to stabilize the MS, generating instability (Jinkins 2004). Because of vertebral splitting, the neural arc is pulled by ligaments and the spinous process recoils in relation to adjacent ones.

ISL preserves the largeness of the central spinal canal but deforms and narrows the neuroforamina whose changes have to be analyzed in the sagittal views because axial images tend to under-estimate the stenosis degree. The foraminal stenosis is often asymmetrical. ISL is also not necessarily painful being a frequent condition in asymptomatic patients. In a population of 32,600 asymptomatic individuals, a pars defect was detected in 7.2%. As a consequence, it is impossible to indicate it as a source of pain on the basis of radiographic findings alone. Pain could be generated by the excessive motion either at the fracture site or at the joints no longer connected to the vertebral body when they are pulled by muscles. Relief of pain after repeated infiltrations of an isthmic defect is a good predictor of successful fusion.

Spinal Canal Stenosis

Pathology, Clinics, and Imaging

Any disproportion in the spinal canal between the size of the neural elements and space available is called SCS (Fig. 26). The diagnosis describes not a simple anatomical condition but a complex pathophysiological entity not yet completely understood, having a series of clinical features only loosely correlated with imaging data.

Degenerative changes of bony elements, capsules, and ligaments may lead to SCS:

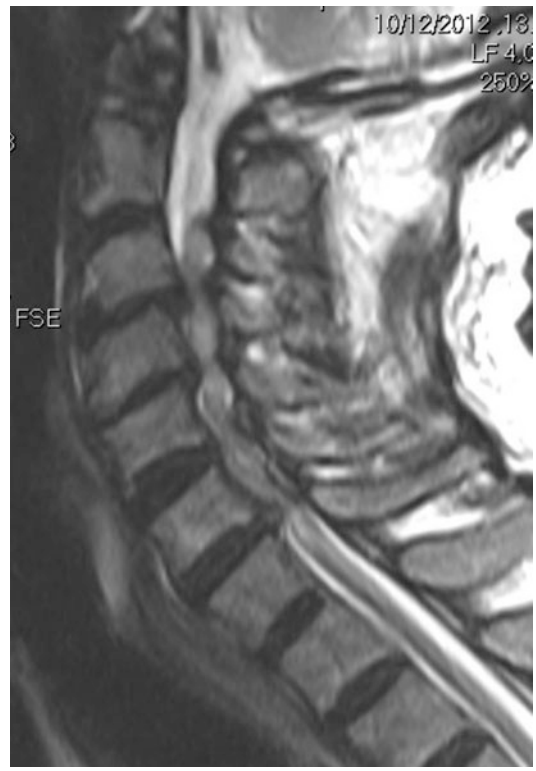


Fig. 26 MR midsagittal FSE T2-WI image. Severe cervical canal multilevel stenosis. The spinal cord is both ventrally and dorsally compressed between disc protrusions and thickened and redundant flava ligaments. Cord parenchyma hypersignal is expression of myelomalacia

– *Acquired* (developing in a normal spinal canal)
– *Mixed* (developmental worsening of a pre-existing congenital stenosis) (Fig. 26)

On the basis of location and severity, SCSs may be classified as:

– C*entral* (reduction of sagittal diameter)
– *Lateral* (narrowing of lateral recesses)
– *Foraminal*
– *Concentric* (reduction of all diameters)

Posteriorly and centrally located osteophytes and disc changes either in the form of herniation, protrusion, or bulging create central SCS. Facet hypertrophy and posterolateral osteophytes can generate stenosis of lateral recesses and foramina.

In *cervical spine stenosis,* degeneration of nucleus pulposus leads to bulging of the annulus fibrosus, redundancy of flava ligaments, disc-osteophyte bar formation, and hypertrophic facet and uncovertebral joints changes. This cascade of degenerative changes may result in cervical central, lateral and/or foraminal stenosis. Ossification of the posterior longitudinal ligament can associate to diffuse idiopathic skeletal hyperostosis.

Both static and dynamic factors contribute to spondylotic myelopathy with repetitive injury causing secondary ischemia, inflammation, and apoptosis until cystic cavitation and gliosis of the central grey matter and demyelination of the white matter along tracts. Patients with chronic mild myelopathy may be unaware of subtle changes in balance and fine motor dexterity (Cowley 2016). Nerve roots involved demonstrate sensory and/or motor dysfunction. Cervical pain may be local or radiate.

Plain radiographs give a first indication of cervical SCS by using the Torg ratio, determined by dividing the sagittal diameter of the spinal canal by the sagittal diameter of the vertebral body. A ratio of less than 0.80 indicates significant spinal stenosis and an increased risk for neurologic injury (Prasad et al. 2003). The Torg ratio has proved to be an accurate indicator of spinal stenosis because the ratio eliminates measurement differences caused by different target distances, object-to-film distance, and magnification errors common with radiographs. In any case, an AP diameter of the cervical canal below 14 mm indicates a critical stenosis (normal range 15–25 mm, on average 17 mm) (Gallucci et al. 2007).

MRI is the gold standard for the study of cervical spine and spinal cord. Short tau inversion recovery images (STIR) offer additional sensitivity for bony and intramedullary lesions. FSE or TSE T2-WI are better suited to detect degenerative disc-osteophyte bars and ligamentous abnormality. On MRI, the most used parameter in the assessment of the cervical spine is the SAC, obtained by subtracting the sagittal diameter of the spinal cord from the sagittal diameter of the spinal canal. The SAC is physiologically smaller at level of cervical enlargement, ranging from 2.5 to 10.4 mm and is greatest at C7.

Stenosis of the cervical foramen requires axial views because of its oblique orientation. Dynamic MRI of the cervical spine can detect the worsening of the SCS in extension because of the infoldings of the yellow ligaments. While both canal stenosis and cervical cord compression occur in asymptomatic people, severe stenosis well correlates with clinical features. A light ill-defined T2W hyperintensity corresponds to reversible edema whereas bright well-defined T2W hyperintensity likely represents definitive gliosis, cystic necrosis, and demyelination. T1-hypointensity represents irreversible myelomalacia.

Lumbar spine stenosis is the most common form of spinal stenosis, prevailing at L4 level, followed by L3, L5, and L1. The most important elements involved are the discs, facet joints, and flava ligaments. Owing to the number of proposed criteria for defining lumbar spinal stenosis, there is wide variation in the reported incidence. The normal AP dimension of the lumbar thecal sac ranges from 12 to 14 mm. The most accepted cutoff values are 12 mm for the AP diameter of the osseous spinal canal, and 3 mm for both the diameter of the foramen and lateral recess height. An AP <10 mm diameter indicates a frank stenosis. On both axial TC and MR images, a cross-sectional area less than 100 mm^2 indicates critical stenosis, while an area between 100 and 130 mm^2 an early stenosis (an area of sac 180 ± 50 mm^2 is considered normal).

Nerve root capillaries and venules can be occluded at pressures of 30–40 mm Hg, while intrathecal pressure can raise up to 100 mm Hg during the extension in case of lumbar stenosis. A critical value of stenosis in the lumbar canal has been measured at 77 mm^2, below which the intrathecal pressure raises until hampering the venous drainage of cauda equine with congestion edema and ischemia of nerve roots. However, no single quantitative measure has revealed to be diagnostic of a complex pathology such as lumbar stenosis.

Lumbar spine stenosis must be suspected in older patients with gluteal or lower limbs pain or discomfort worsened by walking or standing and improving or resolving with sitting or bending forward. While the hallmark of central canal stenosis is the neurogenic claudication, radicular symptoms may associate or prevail for lateral recess or foraminal stenosis.

Several studies have demonstrated the equivalence of MR imaging versus MDCT, among which Modic found the accuracy of MR, CT/CT-myelography, and myelography to be 82%, 83%, and 71%, respectively. Three signs have to be looked for on MR imaging.

1. *Redundant nerve roots*, consisting in elongated, enlarged, and serpiginous nerves on T2-weighted sagittal MR images, probably due to the traction of nerve roots through the stenosis during flexion-extension and failure to recover the normal position in neutral posture. Redundant nerve roots are present above the point of stenosis in 85% of cases (Poureisa et al. 2015).
2. *Sedimentation sign,* failure of cauda equina to lie into the inferior thecal sac during recumbency on either side of the stenotic level (Barz et al. 2010). Sedimentation sign is highly associated with severe stenosis.
3. *Nerve roots enhancement* in postcontrast MR imaging, expression of congestion, and blood-nerve barrier breakdown.

Both CT and MRI standard examinations are performed with the patient lying supine and with the knees bent, a position for the straightening of the lumbar lordosis; the symptoms caused by spinal stenosis tend to be less severe. If the patient lies supine with straight legs, the psoas muscle causes a lumbar lordosis similar to that in the standing position. This position can be more available for assessing with greater accuracy the SCS.

Lateral recess stenosis can be diagnosed when on axial images the subarticular zone is narrowed (normal diameter is 3–4 mm) by a superior facet, and/or posterolateral vertebral body osteophyte, and/or a disc protrusion, or by a synovial cyst. Lumbar and dorsal foramen stenosis is better evaluated on sagittal MR scans or sagittal reformatted CT scans (Fig. 27).

Axial loaded CT and MR as well as dynamic MRI of the lumbar spine can show an increase of a degenerative stenosis (Gallucci et al. 2007). In standing position, extension has the greatest effect on canal stenosis. The passage from lumbar flexion to extension reduced the cross-sectional area of the dural sac by 16%, the axial loading by 19%.

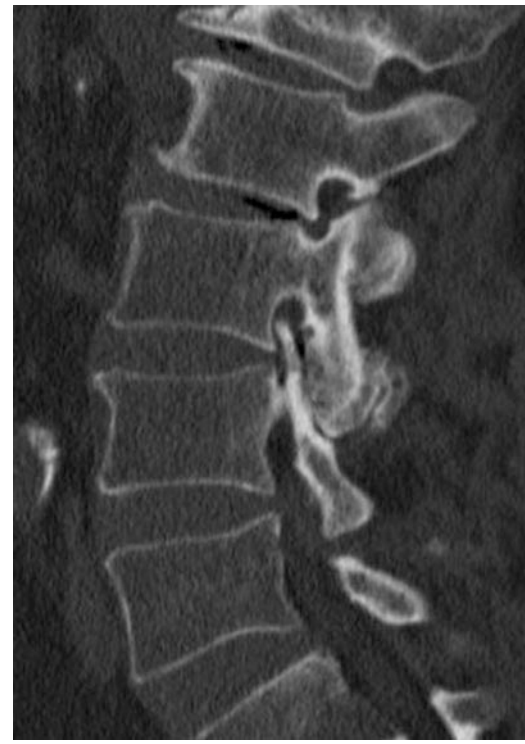


Fig. 27 CT parasagittal reformatted image. L3-L4 facet joint degenerative changes consisting in marked hypertrophy of L4 superior facet encroaching the foramen

In both cases, the yellow ligaments were the most important contributors of the dynamic stenosis. Dynamic studies can improve the sensitivity of static imaging by detecting dynamic changes. Lohman preconized before surgery for patients to undergo a dynamic study for better assess the levels and the degrees of stenosis (Lohman et al. 2006).

All measurements and imaging data have to be strictly related to clinical features. The relationship between clinical symptoms of spinal stenosis and its radiologic manifestations is in effect undefined. Despite a significant SCS, patients may have mild or no clinical symptoms at all: the prevalence in asymptomatic subjects varies according to the age from 7% to 21%. The opposite situation is also common. Furthermore, patients often do not have relief of symptoms after decompressive surgery.

Thoracic spine stenosis: Degenerative changes are less common in the dorsal spine in relation to reduced motion. Yellow ligaments hypertrophy and calcification/ossification are frequent cause of stenosis with myelopathy and/or radiculopathy.

Spine Instability

Pathology, Clinics, and Imaging

Spine stability is the basic requirement which ensures the protection of the nervous elements and prevention of the early deterioration of spinal components themselves. Degenerative spine instability is considered a major cause of pain and disability and is a frequent indication for surgery. Degenerative instability consists in an alteration of vector forces in the relations inside and between the motion segments (MS), generating abnormal, imbalanced, paradoxical movements. An initial degenerative change, generally involving the intervertebral disc, creates disorders of movement which increase the original bone and articular abnormalities and extend them to other joints of the same level (three-articular complex) and finally to those of adjacent segments transforming over time a segmental pathology in a regional one. Because of disc collapse, the annulus and ligaments become lax and redundant favoring spinal canal stenosis, subluxation of the vertebral bodies, and raising of the subjacent facets in the foramina until the neo-arthrosis with the peduncles. The neo-arthrosis promotes facet remodeling and osteophytosis and worsens the foraminal stenosis.

Kirkaldy-Willis and Farfan recognized three biomechanical and clinical phases of a so-called "degenerative cascade" during the evolution of degenerative instability: dysfunction, instability, and restabilization (Kirkaldy-Willis and Farfan 1982). During the dysfunction phase, intermittent nonspecific back pain appears along with initial changes in the discs and facet joints. During the instability phase, imaging may detect spondylolisthesis or retrolisthesis, but instability may only consist in a pure movement syndrome, with no apparent bony lesions (microinstability) when abnormal motion develops in one or several directions under an impaired muscle control, generating symptoms. Traction spurs are due to increased pulling by the anterior longitudinal ligament and Sharpey's fibers upon bone insertions in case of increased abnormal motion. Traction spurs typically develop 2–3 mm apart from vertebral edges and have a horizontal orientation.

Patients with degenerative spondylolisthesis have the largest facet joint effusions. The most significant association occurs in subjects with a mobile, intermittent, low-grade anterolisthesis in comparison to overt and advanced cases because the former cause increased movement of the articular processes between the standing posture and the recumbency. A facet effusion >1 mm should be an indication for dynamic radiographs or MR to diagnose an occult spondylolisthesis that can be missed with static imaging (Fig. 28).

As before mentioned, Modic type I changes are also associated to segmental instability and convert in type II changes after successful fusion surgery. As spinal degeneration further progresses, the fibrosis of the joint capsules, the formation of osteophytes, the marked disc collapse, and the expansive remodeling of vertebral bodies lead to an overall reduction of mobility and increased stiffness (Kirkaldy-Willis and Farfan 1982). During the restabilization, the end phase

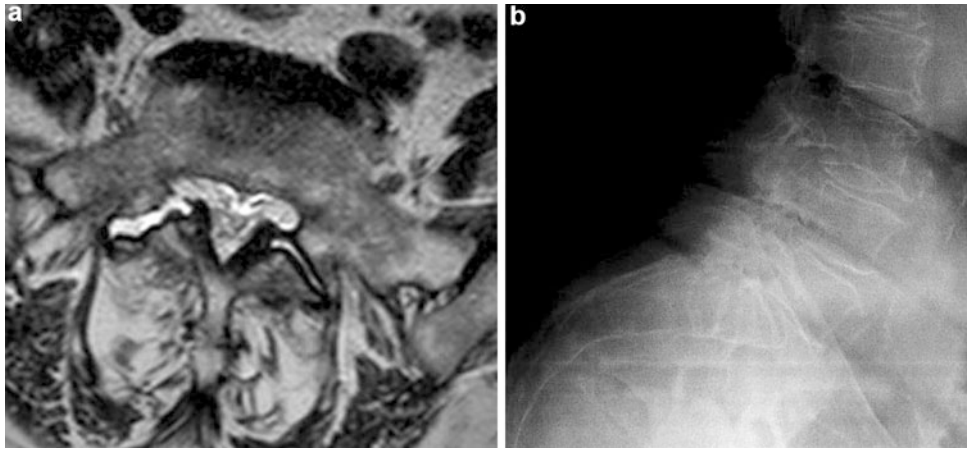


Fig. 28 (**a**) MR axial FSE T2-WI image. Bilateral joint effusion and degenerative hypertrophy of L5-S1 facet joints. A joint effusion enlarging the joint space by 1 mm or over must raise the suspect of occult intermittent anterolisthesis. (**b**) X-ray lateral projection, A dynamic study confirmed this hypothesis with appearance of listhesis in flexion

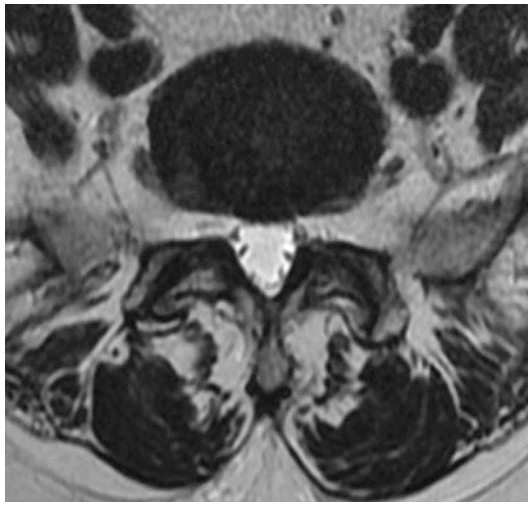


Fig. 29 During the late steps of instability, a marked hypertrophy and osteophytosis appears in order to restabilize the motion segment. Wrap around bumper osteophytes are one of these findings

of the degenerative process, functional limitation and stiffness appear, while spinal pain can eventually recede or persist due to the irritation or impairment of nervous structures. During the restabilization phase, imaging findings change and consist in diffuse disc collapse, radial remodeling of vertebral bodies, claw osteophytes, "wrap around bumper" osteophytes (Fig. 29) endplates and facet joints sclerosis, and neo-arthrosis between the spinous processes.

Role of Imaging

In the degenerative pathology of the spine, imaging data have to be always analyzed in the light of the clinical context. For the actual imaging techniques to be of value, they have to contribute to final diagnosis and correctly guide the treatment choices, excluding the small percentage of patients (approximately 5%) suffering an unknown systemic disease (Jarvik and Deyo 2002). Early imaging of acute spinal pain has not demonstrated to provide useful information to improve clinical outcome out of subjects in which unresponsive pain, persistent or worsening neurological deficits require intervention in acute setting.

In the case of typical or acute back pain or radiculitis, it is invariably accepted that patients have no theoretical risk in waiting 4–6 weeks before undergo imaging, having the possibility of a spontaneous regression of symptoms in the case of extraspinal diseases such as neuritis, muscular sprain or insertional inflammation, or even small acute disc herniations. Accordingly, Carragee assessed the usefulness of early imaging

in a prospective study on a large population of asymptomatic patients in which a control MR was performed at the appearance of back pain and showed new relevant findings in only less than 5% in comparison of an initial baseline MR. All other positive signs had been present in the first MR study, when the subjects were asymptomatic. In the absence of baseline MR study a number of these findings would have been considered relevant (Carragee et al. 2006).

In patients not responding to conservative therapy, imaging can offer data for diagnosis and to modify the therapy for eventual minimally invasive procedures or surgery. Conventional X-ray examination still plays an important role of preliminary modality, being cheap, universally available, safe, and able to offer a panoramic view of the spine, with good details on bone structures and the stability of the spine. The following modality of choice is MRI in the assessment of cervical and thoracic spine. In lumbar spine, CT is considered as sensitive as MR but for safety concerns is reserved for focus and adds information about known lesions.

All population-based studies demonstrate a prevalence of imaging degenerative findings in comparison with symptomatic disease prevalence. Studying by MR the lumbar spine of asymptomatic subjects, Jensen found completely normal discs in only 36% of cases, in the remaining two-third disc degenerated at single or multiple levels (Jensen et al. 1994). On imaging, a gold standard for diagnosing the spine instability does not exist; a clear correlation between clinical and imaging data is: abnormal movements are often present in asymptomatic individuals. Conventional imaging findings are only indirect signs of instability whose specificity and clinical relevance vary among the different reports and need to be established definitively. Open MR systems combines the benefits of conventional MRI and dynamic radiographs allowing positional-dynamic studies in either standing or seated positions that may disclose dysfunctional movements which worsen or uncover a stenosis, a disc protrusion, or extrusion of an intermittent or occult spondylolisthesis (Fig. 30).

However, dynamic MRI presents several drawbacks such as the reduced signal/noise ratio due to the lower field strength, and the difficulty of patient to hold a painful posture. Axial-loaded CT (AL-CT) and MR (AL-MR) both simulate the upright position and depict several findings referred to as elementary modifications which

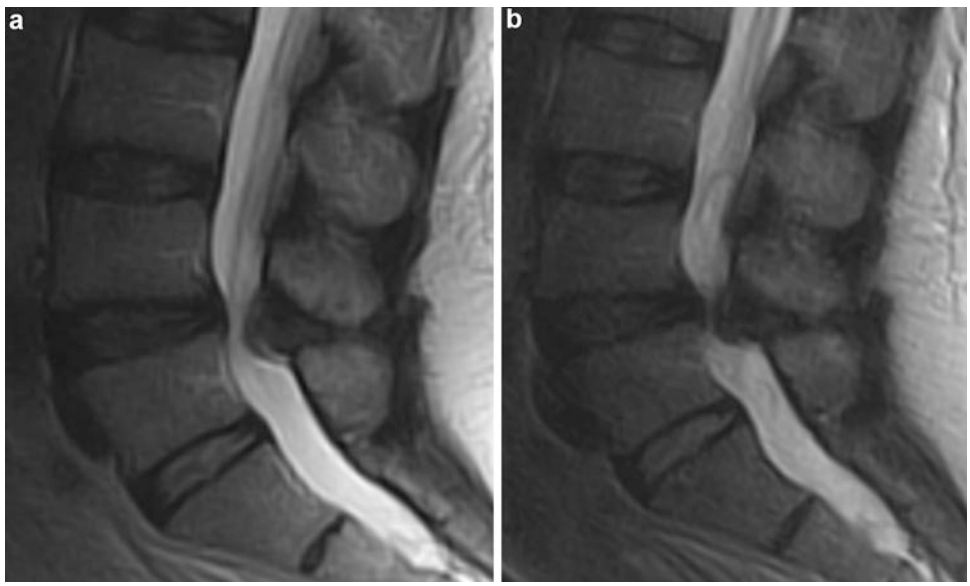


Fig. 30 (**a**) MR sagittal FSE T2-WI in recumbent position showing a slight anterolisthesis of L4 along with yellow ligaments hypertrophy. (**b**) MR sagittal FSE T2-WI in standing position showing the worsening of listhesis

can be observed alone or associated in various patterns referred to as complex dynamic modifications (Muto et al. 2016). Abnormal motion patterns tend to evolve in a quite stereotyped way up to degenerative listhesis. However, axial-loading cannot reproduce the postural changes related to muscles activity and the physiological loads that increase in the caudal direction along the spine. AL-CT is preferred to AL-MR in postoperative imaging and in the late stages of instability where abnormal movements are expected to be globally reduced and better depicted by using 3D reconstructions. AL-MR better shows fluid modifications under load and the changes occurring in the soft tissues, in particular, increased disc protrusion and thickening of yellow ligaments which often worse a spinal canal or foraminal stenosis. In case of microinstability, the stenosis appears only after AL.

Patients with suspected spinal canal stenosis may benefit from AL or dynamic studies, especially in view of surgery. Despite the advantages of AL and positional CT-MRI, dynamic radiographs remain the most commonly used technique because of the simplicity, wide availability, and lower cost. The value of plain functional radiographs remains debated due to the lack of any standardization in technical execution and measurement methods, and the wide overlap of motion patterns among symptomatic and normal subjects. The optimal patient position to disclose maximal motion of the lumbar spine is also debated; while many authors prefer using the sitting position, others use lateral decubitus in lieu of the classical standing position to maximize the abnormal motion. Functional plain radiographs finally just show the relations of FSUs in the positions of maximal flexion and extension, with no insights into the quality of ongoing movement.

Spine instability is often an over-diagnosed condition for which fusion surgery is not always the correct choice. Like other weight-bearing joints, in many cases spinal pain is due to an abnormal and irregular distribution of loads between joint surfaces. Pain may persist after technically successful fixations or unexpectedly resolves in cases of pseudarthrosis.

Postoperative Findings

The usefulness of surgery versus conservative therapy remains debated because despite surgery has proved to be superior to nonoperative management at middle term (1 year), the long-term effects (4–10 years) are similar. The role of surgery is essentially that of affording a more rapid relief of pain and functional recovery. For reaching this target, it is mandatory a rigorous selection of patient, considering the non-negligible rate of surgery failure, ranging from 10% to 40% of cases (Thomson 2013). Because of the high incidence, failed back surgery is considered a special disease, referred to as FBSS. FBSS has been defined as significant back pain and/or radicular pain and functional impairment either persisting or appearing after surgical intervention for spinal pain in the same topographical location as original pain.

Most of the cases of FBSS and the majority of postoperative studies regard patients who underwent surgery due to herniated lumbar disc, though a posterior approach. FBSS has both preoperative and postoperative risk factors. Preoperative conditions influencing the likelihood of success of spinal surgery include the accuracy of diagnoses, socioeconomic (workers' compensation), behavioral, and psychological factors (depression, anxiety) (Baber and Erdek 2016). Postoperative causes for the reappearance of symptoms are: epidural fibrosis, disc recurrences, segmental instability, segmental stenosis, wrong-level surgery (Fig. 31), insufficient decompression, or a combination of thereof. The workup of FBSS includes an accurate history and physical examination as well as imaging and diagnostic procedures. The first clinical insight regards the location of symptoms in comparison to presurgical site and the temporal delay of their onset on the basis of which the complications are distinguished in early or late ones (Table 5).

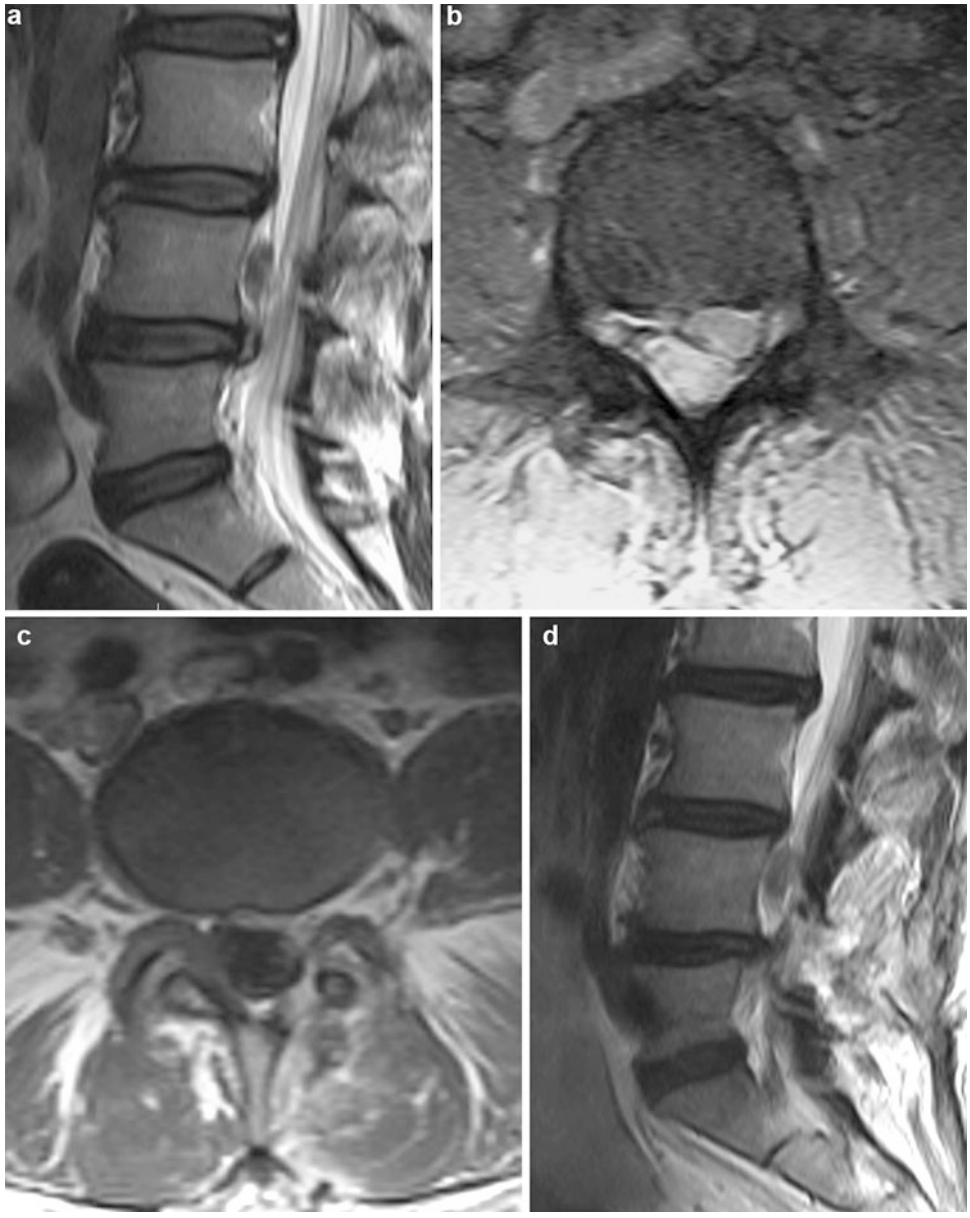


Fig. 31 FBSS due to wrong-level and incomplete surgery. 41-year-old woman complaining acute refractory radicular pain within L4 left dermatome and not L5 signs. After surgery, persistence of pain in the same distribution as prior the operation. (**a**, **b**) Presurgical MR. Left parasagittal and axial FSE T2-W images showing a sequestered disc fragment migrated into the left anterior epidural space of L4 and located at infrapedicular and pedicular levels. The fragment compresses the L3 nerve root against the pedicle before its exit trough the neuroforamen. In addition, the L3-L4 disc presents a little paracentral little disc herniation with not correlated symptoms. (**c**, **d**) left parasagittal FSE T2-WI and axial postcontrast FSE T1-WI of a new MRI showing the signs of a recent surgical access with diffuse contrast enhancement and imbibition of the anterolateral left epidural space. Removal of herniation at disc level, but, persistence of the intact disc sequestered fragment

Table 5 Early and late postsurgical complications

Early postsurgical complications	Late postsurgical complications
Hemorrhage,	*Arachnoiditis,*
Infection,	*Epidural fibrosis*
Pseudomeningocele,	*Recurrent disc herniation,*
Residual disc herniation.	*Spinal canal or foraminal stenosis, instability,*
	Textiloma (foreign body)

Role of Imaging in FBS

Conventional imaging does not convey any demonstration of soft tissue changes.

MDCT is suitable for assessing osseous canal and neuroforaminal stenosis secondary to a disc collapse, and for detecting an eventual textiloma, but it may have insufficient contrast resolution for reliably distinguishing residual/recurrent disc herniation from epidural fibrosis and for evaluating hemorrhagic or infective complications. Post-myelography CT is the modality of choice for researching postoperative CSF leakages.

MRI is the gold standard for visualization of the FBSS by its superior soft tissue contrast resolution, further improved in postcontrast imaging. The MR protocol should include FSE sagittal T1-T2-weighted and STIR images and axial T1 imaging before and after intravenous administration of gadolinium contrast medium. Fat-suppression acquisitions help in distinguishing hemorrhage from epidural fat on plain imaging and in differentiating residual epidural fat from enhancing fibrosis in post-contrast imaging (Van Goethem et al. 2002). MRI reveals both bone and soft tissue changes. Bone changes vary from the traditional laminectomy, with eventual facetectomy, to minimally invasive approaches, where the signs of previous microsurgery may be difficult to recognize. The soft tissue changes vary according to the type of surgery and time elapsed from.

Epidural Fibrosis Versus Disc Herniation

The assessment of the spine during the first 4–8 weeks after surgery is in general particularly challenging (Fig. 32). The presence of edema, blood, and debris within both an expanded and blurred epidural space and inside the posterior annulus may simulate the persistence of disc material in up to 80% of patients (Fig. 32). With time, when granulation and then the scarring develop, the mass effect recedes while it appears contrast enhancement. Contrast enhancement is initially strong in the hyper-vascularized reactive epidural granulomatosis and progressively fades over time with aging of the scar.

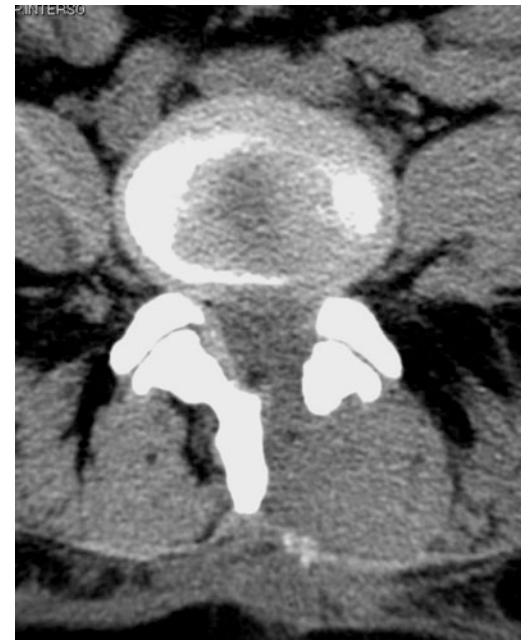


Fig. 32 Axial CT image of L4-L5 disc soon after surgery. Imbibition, enlargement, and blurring of both the epidural space and disc with suggestion of residual disc protrusion

Epidural fibrosis is a constant and expected feature of any postoperative spine, located in the anterior, lateral, posterior space, ipsilateral to surgical access, regularly present also in asymptomatic subjects (Fig. 31). According to some authors, epidural fibrosis is a radiological entity whose amount does not correlate with clinics and that should not be considered a complication of surgery when it does not associate to significant deformation of nervous elements. Epidural scarring is not always detectable by MR and may be found only during epiduroscopy which is indicated in case of symptomatic subjects having negative MR. Since some epidural fibrosis is

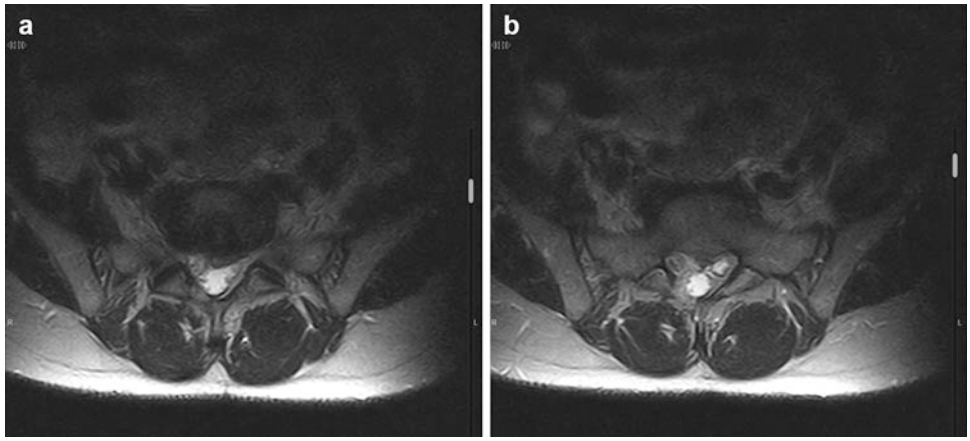


Fig. 33 Subject operated 1 month ago for L5-S1 disc herniation. Persistent low back pain. (**a**, **b**) MR axial FSE T2-WI trough the L5-S1 disc and the superior endplate of S1 showing an evident residual central disc herniation. Left epidural space ipsilateral to surgical access through a traditional laminectomy shows diffuse young scarring surrounding the S1 nerve root sleeve with an intermediate signal cleanly different from hypointense disc signal

constant, a residual/recurrent disc herniation, if present, always coexists with it (Fig. 33).

Recurrent disc herniation, defined clinically as pain recurrence at least after 6-month of remission since surgery, in one prospective study was found in 23% of the patients, half of whom symptomatic (Fig. 34) (Lebow et al. 2011).

While a disc extrusion appears in direct continuity with the parent disc, exerts a more evident mass effect, and has smoother margins, the epidural fibrosis is more irregular, provokes traction, and is only ipsilateral to surgery side and contiguous, not continuous, with the disc. Disc herniation exhibits dark-intermediate signal in T1-WI and dark signal in T2-WI (bright when it is recent), whereas scarring has intermediate signal in T1-WI and intermediate to bright signal in T2-WI. However, signal overlapping often occurs on unenhanced MR images, thus contrast imaging is mandatory for differentiating the immediate and uniform enhancement of granulation tissue of the epidural scar from disc tissue which lacks vascularization. Quick post-contrast imaging (within a few minutes) is, however, required because a slow contrast filling-in of the disc occurs with time by diffusion from surrounding reactive tissue. The differential diagnosis is important because while a residual or recurrent disc herniation may indicate another intervention, epidural fibrosis generally does not. Like epidural fibrosis, a residual or recurrent disc herniation is not necessarily the cause of patient complaints, being it present in up to 24% of asymptomatic individuals, even with significant deformation of the dural sac. However, a presurgical radicular pain that persists in the immediate postoperative period may indicate a wrong-site surgery or incomplete herniation removal (Fig. 31).

Septic Versus Sterile Discitis

The incidence of infection correlates with the extent of surgery, being greater in the open approaches. Open surgery has a reported risk of infection of 3–13%, provoked in most of cases by *staphylococcus aureus* and *staphylococcus epidermidis* and by direct inoculation. Focal intradiscal signal changes on imaging of asymptomatic subjects are the rule after surgery and consist in hypointensity on T1-WI and hyperintensity on T2-WI images by mechanical or chemical discitis, along with edema, vascular congestion, and focal little endplate irregularities due to surgical curettage. While the absence of these findings excludes infection, their presence has not to be confounded with a septic spondylodiscitis. Inflammation indices in the absence of a septic complication are always normal. A septic

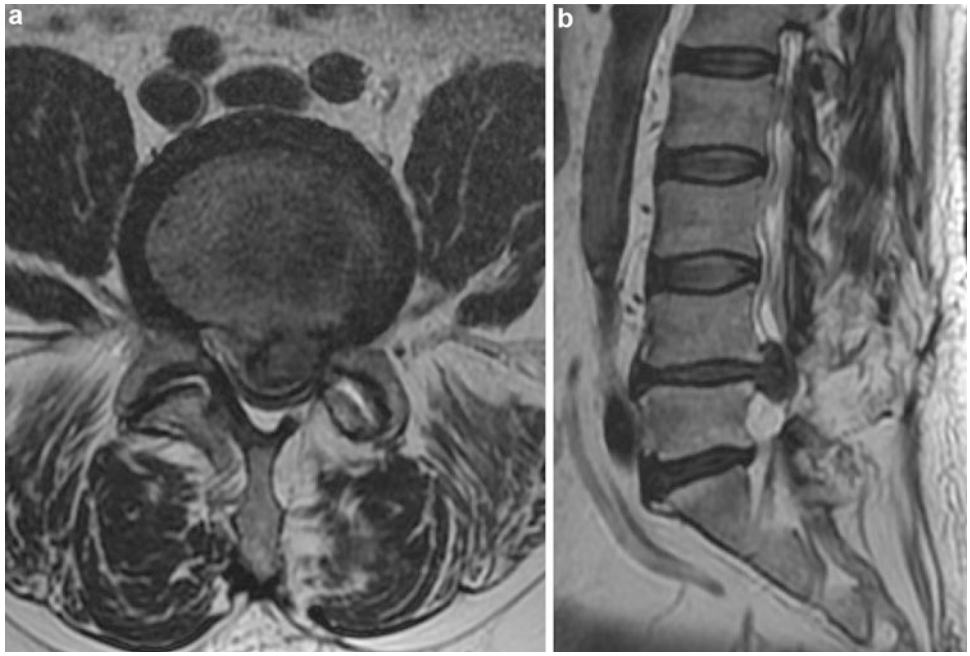


Fig. 34 Previous surgery with right laminectomy 9 months ago. New onset of left-sided sciatica refractory to therapy. (**a**, **b**) MR axial and sagittal FSE T2-Wi Large recurrent disc extrusion with severe compression of thecal sac

spondylodiscitis manifests with reappearance of progressive pain short after intervention. On imaging, disc and endplate changes are more marked and diffuse, with the latter in the form of frank destruction, flanked by paravertebral and epidural soft mass eventually abutting in abscess (Fig. 35). CRP has proved to be a useful screening test, while clinical findings, leucocytes count, and erythrocyte sedimentation rate are not reliable.

Sterile Radiculitis

Another expected postoperative finding is the sterile radiculitis. Normally at the conventional dosage of gadolinium the cauda equina roots do not enhance. A temporary superficial enhancement of intrathecal nerve roots may express a reactive alteration of blood-nerve barrier after surgery or a neural damage due to the persistent compression exerted by the disc herniation lasting sometimes after its removal. Nerve root enhancement can manifest in asymptomatic patients and must be considered pathological only if persists over 6 months after surgery.

Fluid Collections

Fluid collections can cause FBSS in acute postoperative setting. Seroma, pseudo-meningocele, hematoma, and abscess can all compress the thecal sac and/or the nerve roots. MR can evaluate the site and extension of collections and in particular their intraspinal extension and the compressive effects. The distinction between hematoma and seroma is better done by MR than by CT. Hematomas occur in less than 1% of patients and can become symptomatic within hours to days after surgery. Hematoma shows high T1 signal intensity, do not communicate with the dural sac, and do not associate with abnormalities inside the disc. MR contrast imaging can be used to distinguish infection from noninfectious fluid collections, but some reactive enhancing tissue is always present around every collection (Fig. 36). Hyperintensity of fluid content on DWI imaging and osseous involvement and destructive bony changes may guide the differential diagnosis.

Incidental durotomy can eventually result in pseudo-meningocele or CSF fistula. Pseudo-meningoceles are cystic collections of CSF due to

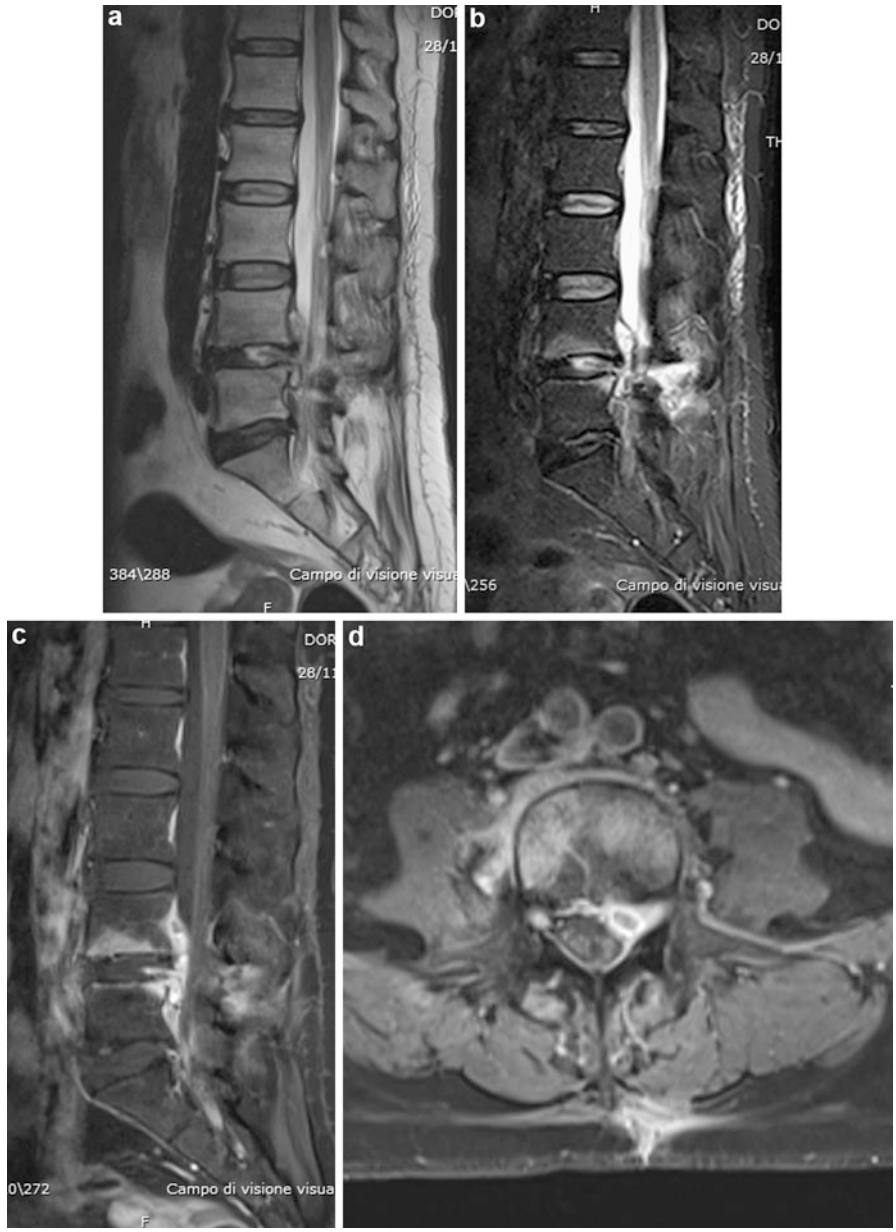


Fig. 35 A few days after surgery for L4-L5 disc herniation, reappearance of worsening pain, fever, and raise of CPR. (**a**, **b**, **c**, **d**) MR sagittal FSE T2, STIR, and post-contrast FSE T1 fat sat images showing an inflammatory soft mass within the anterior epidural space most of which corresponding to a flegmon but with initial abscess forming. The hypersignal and contrast enhancement inside posterior disc could be only normal expression of recent surgery

laceration of the dura mater during surgery or defective closure. They lack an arachnoid lining and are contained by a fibrous wall (Fig. 37). Small collections can remain asymptomatic, but large pseudocysts become compressive and can generate back or radicular pain or occasionally intracranial hypotension. Pseudo-meningoceles expand in the paraspinal space through the bone surgical opening and can be recognized by absence of mass effect, homogeneous T2 hypersignal, low T1 signal intensity, and communication with the thecal sac (Fig. 37).

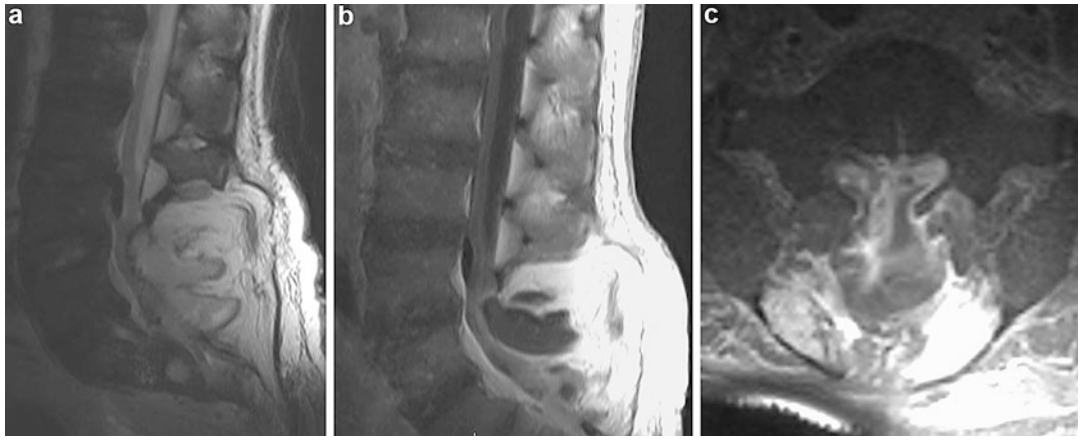


Fig. 36 (**a**, **b**) MR FSE T2-WI and FSE T1WI after gadolinium administration showing a large postoperative paravertebral collection with inhomogeneous internal signal surrounded by intense contrast enhancement, better detectable in the axial fat-saturated image (**c**). The collection extended from the surgical opening until subcutaneous fat plane. Postsurgical abscess

CSF fistulae have a reported incidence of 2% (Fig. 38). CSF leaks must be identified in order to prevent intracranial hypotension and meningitis. CT or MR-myelography can differentiate a communicating collection from a seroma. MR myelography is able to detect CSF leaks in 20% of negative CT myelography (Akbar et al. 2012).

Arachnoiditis

Sterile arachnoiditis is a late complication accounting for 6–16% of chronic symptoms after surgery, favored by intradural bleeding, previous infection, or intraspinal injections. Matted and clumped cauda equina nerve roots can either form little clusters or adhere to dural walls, creating an empty dural sac or fuse in an intrathecal amorphous soft tissue mass. The contrast enhancement of meninges and roots is variable.

Interbody Fusion Surgery

Indication, Clinics, and Complications

In the degenerative spine, most of the surgery not specifically addressing disc herniations is aimed to either decompressing neural elements by abnormal osteoligamentous structures or to fusing two or more levels in order to treat a segmental instability or a painful intervertebral disc. In view of a disc dissection, posterior instrumentation serves to distract the disc space, restoring normal interspace height, and decompressing the neural foramina. The evacuated disc space is filled with bone grafts or fusion cages containing autograft bone.

The goal of spinal fusion is to restore anatomic alignment and functional biomechanics as near to normality as possible. Implanted hardware must not replace the bony components but only serve to provide immediate stability and favor the integration of bone grafts. In case of non-union, any instrumentation is destined to fail. However, while lytic and degenerative spondylolisthesis are the only undiscussed indications for instrumented surgery, for all of remaining pathologies the real advantages of an additional implant have been debated. According to classic three-column concept of spinal stability by Denis, the stability requires the integrity of at least two columns. Instrumentation is deemed necessary if more than one column is disrupted by any pathologic event, including degenerative changes.

The surgical approach and the choice of devices depend on the clinical and anatomical contexts and the surgeon's preferences. The anterior approach is generally preferred in the cervical

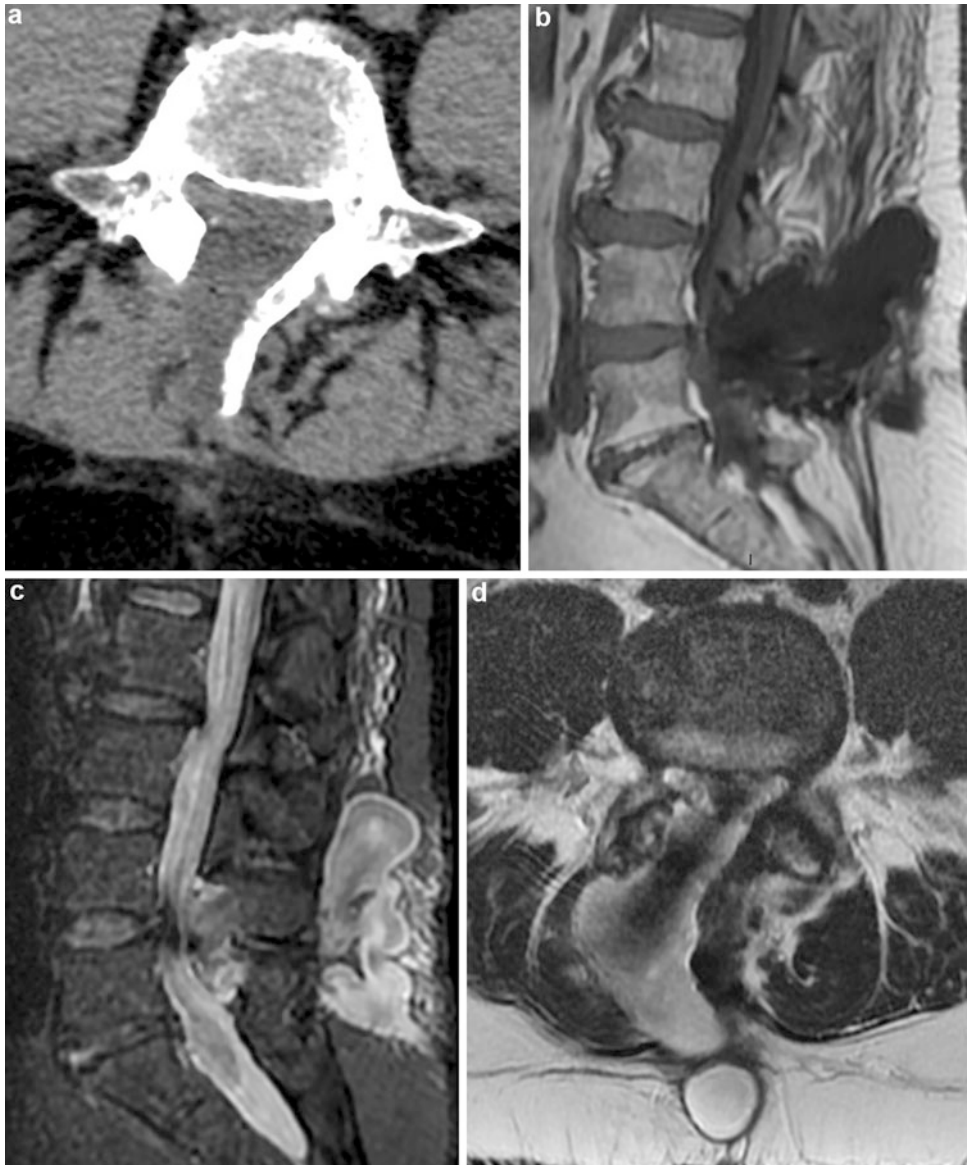


Fig. 37 (**a**) axial postsurgical CT at L5-S1 level showing a little fluid collection occupying the surgical breach. One month later appearance of intracranial hypotension syndrome and control MRI. (**b**–**d**) Sagittal FSE T1-WI and STIR-WI and axial FSE T2-WI detecting the intervening formation of a large paravertebral collection isointense to LCR, in large communication with the thecal sac, is also confirmed by CSF pulsation artifacts

spine because of the risk of cord manipulation during a posterior approach (trans-oral, anteromedial, anterolateral). Anterior instrumentation consists in rod-screw or plate-screw systems. Approaches to the thoracic spine can be anterior (trans-thoracic, trans-sternal, and thoracoscopic) and posterolateral. For the lumbar spine, the techniques include the PF, PLF, PLIF, ALIF, and XLIF, the latter being a transversal through the psoas muscle consenting the placement of rectangular wide implants providing a large interface improving fusion, leaving an intact posterior annulus. Anterior approaches can be transperitoneal or retroperitoneal. Anterior surgery for

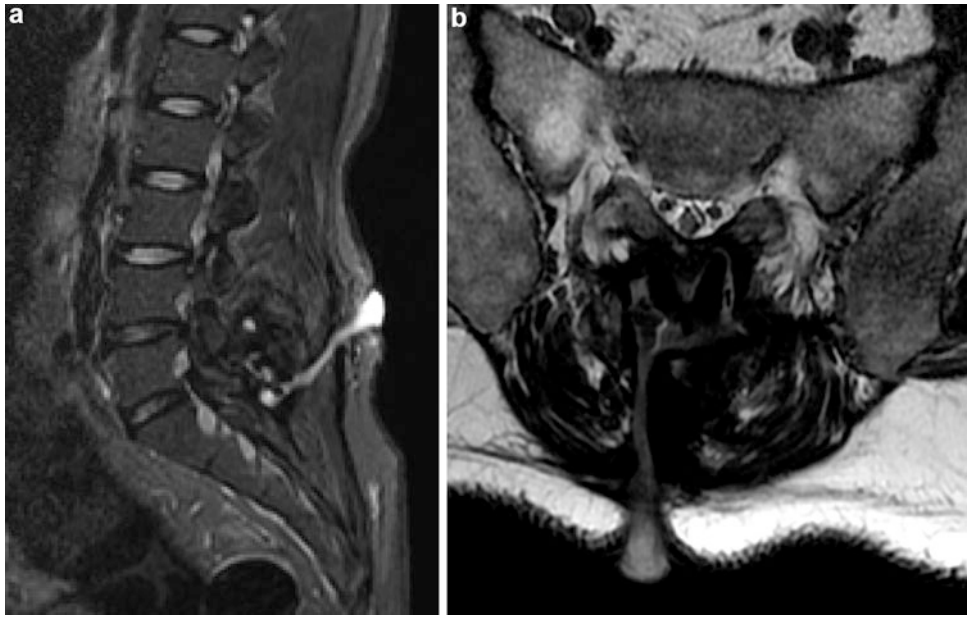


Fig. 38 (**a**) MR lumbar spine sagittal STIR image after surgery for disc herniation. (**b**) MR axial FSE T2-WI at L5-S1. Postoperative fistula extending from spinal canal to cutaneous plane

the lumbar spine has 20% complications rate for great vessel or viscera.

In the cases of discogenic pain, disc prostheses can also be used and placed through a posterior or anterior approach. It is an arthroplasty preserving the motion, but the placement of hardware requires great accuracy and is technically demanding. A posterior approach is often preferred in the lumbar spine owing to inferior morbidity. Posterior instrumentation can be of three types: rods with hook or wire systems, pedicle screw with rods or plaques, laminar or facet screw. Screws are inserted in the pedicles or facets and hooks are anchored on laminae and pedicles and interconnected rigidly or dynamically.

Role of Imaging in Spine Instability

Preoperative imaging has the challenging role to find the pathology responsible of spinal pain being a condition which has often demonstrated a multifactorial etiology (discogenic, facet-mediated, and myofascial) where it is complicated to find the prevalent component, also considering the high rates of abnormal findings in asymptomatic subjects. The high rates of degenerative changes in asymptomatic patients and their increase with age suggest that most of current imaging findings of degenerative disc disease (DDD) rather represent interbody joint natural aging findings.

Preoperative imaging should also be able to select patients for surgery, by predicting postoperative outcomes of spinal fusion in the case of DDD and refractory persistent pain. However, while some surgical series have demonstrated the efficacy of fusion surgery in selected patients suffering from refractory chronic axial LBP and advanced DDD, the long-term clinical outcomes of fusion for patients with chronic LBP without radicular symptoms has no significant difference with outcomes afforded by some conservative multidisciplinary programs, including intensive rehabilitation activities. The best scientific evidence available at the moment does not support the use of any preoperative test for selecting patients for fusion (Willems et al. 2013).

One recent study has proposed a surgically oriented grading system, the Lumbar Fusion

Outcomes Scale (LUFOS), based on three preoperative imaging findings, including nuclear medicine for tracer endplate uptake, presence of Modic changes, and advanced degenerative changes in Pfirrmann scale on MRI, which has been shown to be highly predictive of long-term surgical outcomes after lumbar fusion in patients suffering persistent refractory axial LBP with no radiculopathy.

Postoperative imaging is typically performed to assess the progression of osseous fusion, to confirm the correct positioning and the integrity of instrumentation, to detect suspected complications, and a new disease or disease progression. During the study of any instrumented spine, the radiologist should systematically analyze the integrity of the neuro-vascular components throughout the spine, as well as all adjacent structures such as great vessels, musculature, posterior mediastinum, and pre-paravertebral soft tissue. The modality and protocol used to image the postoperative spine depend on the location, the clinical question, and the type of instrumentation; however, a standard reference for noninvasive imaging evaluation of fusion is actually lacking. Knowledge of the surgical approaches (anterior, posterior, lateral) is necessary for assessment of postoperative spine.

Due to wide availability, conventional imaging is the ideal modality to check changes in spinal curves and sagittal balance, spinal deformities, segmental instability by using dynamic studies, and for the long-term surveillance of the correct positioning and integrity of the spinal constructs, even though it does not consent a 3D representation of the spine. MDCT is the best modality for assessing the instrumented spine, thanks to a detailed evaluation of spinal hardware and surrounding bone tissue. Starting by isotropic voxels, MDCT consents high resolution axial and reformatted images in every spatial plane, including curved reformats and imaging in volume rendering. Subtle fractures, screw loosening, and implant position can be detected by reducing beam hardening artifacts with appropriate technical options. Intraoperative CT studies allow immediate evaluation of hardware positioning preventing complications by misplacement (Splendiani et al. 2017).

Metallic streak artifacts on CT are related to hardware size and density. Titanium generates fewer artifacts than stainless steel and cobalt-chrome. They can be controlled with some acquisition parameters such as high tube voltage (120–140 Kvp rather 80 Kvp), lower pitch, and thinner sections, and, in post-processing, by using thicker sections, soft tissue instead of bony reconstruction kernels, and extended CT Hounsfield scale. Dual-energy-CT technology also may reduce metallic artifacts.

MRI still has a limited role in the assessment of hardware spine and is principally suited to exclude other complications. Open MRI systems allow studies in upright position or dynamic-positional to investigate a spinal canal or foraminal stenosis with increased specificity or to reveal an occult spondylolisthesis (Splendiani et al. 2016). Metallic artifacts remain a major limit to MRI application. Susceptibility artifacts may consist in a loss of signal in phase direction by intravoxel dephasing and in spatial misregistration in the frequency encoding and slice selection gradients.

In the instrumented spine, actual titanium alloy devices, being less magnetic, produce on MR imaging fewer artifacts than the traditional stainless steel. In addition, in the presence of metallic hardware, MR acquisitions can be optimized by using broader bandwidths and by preferring fast/spin-echo or turbo spin-echo sequences having elevated turbo factor, rather than conventional spin-echo and gradient-echo sequences, or by preferring STIR sequences rather frequency-selective fat saturation acquisitions. Whatever the sequence is, it is possible to limit the artifacts with the use of small field of view, high-resolution matrix, thin sections, and by shifting the phase encoding directions from the craniocaudal (projecting artifacts on the vertebral bodies and sparing the spinal canal) to anterior-posterior one (obscuring the canal and sparing bodies and discs) (Gallucci et al. 2005). Metallic artifacts on MRI can also arise from tiny metallic drill fragments even in the absence of any hardware. The progressive introduction of new cages, plates, and screws made of inert polymers will make MRI able to have more extended applications.

In posterior or posterolateral fusions, the radiographic appearance of the autografts placed along decorticated facets or laminae is much variable, with some forming coarse bony masses, other being ill-visualized even by CT. Intertransverse fusion masses are usually more evident. A solid fusion creates within one year and the final aspect of fusions varies from very compact and solid to irregular and fragmented units. In the interbody implants, whatever the type is, the appearance is well-defined short after surgery, and then the borders become increasingly blurred until to disappear. Fusion cages appear to float inside the intervertebral space on plain radiographs (Fig. 39), while CT thanks to superior resolution already demonstrates initial bridging bone traversing the openings of the cage.

The most frequent complication in hardware placement is the improper angulation or depth of screws. Posterior fixation screws must not violate the medial or inferior pedicle cortices and maintain a parallel course to the endplate without entering the anterior body cortex (Fig. 40).

Functional fusion, being defined as less than 3° of motion between flexion and extension views, is due to non-mineralized osteoid tissue and normally precedes evident fusion occurring only 6–9 months postintervention. Inadequate fixation and persistent motion can lead to bone graft resorption rather than incorporation. Ray defined six criteria for assessing the integration of grafts and devices on plain radiography which although not validated, have gained clinical acceptance (Ray 1997) (Table 6).

In dynamic studies, while the stiffness created by the construct renders motion not obvious despite pseudo-arthrosis, until 2–3° of motion can be due to the normal bone compliance. Interbody spacer position has to be assessed in both horizontal and vertical planes on serial imaging studies. New materials like carbon fibers eliminate streak artifacts on CT or susceptibility artifacts on MRI making easier the assessment of cages misplacement, extrusion, or subsidence (Fig. 41).

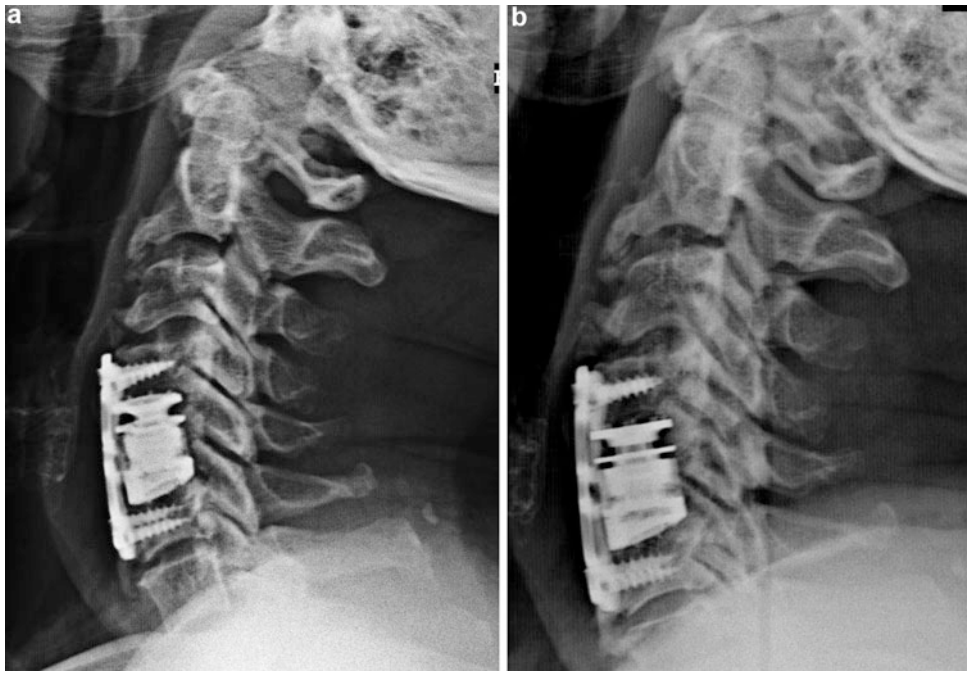


Fig. 39 Plain control radiographs after C5 corpectomy. For several months after surgery, the implant appears to float being completely surrounded by radiolucency. In the last control, it is possible to observe the reduction of radiolucent rim as sign of good integration of the device

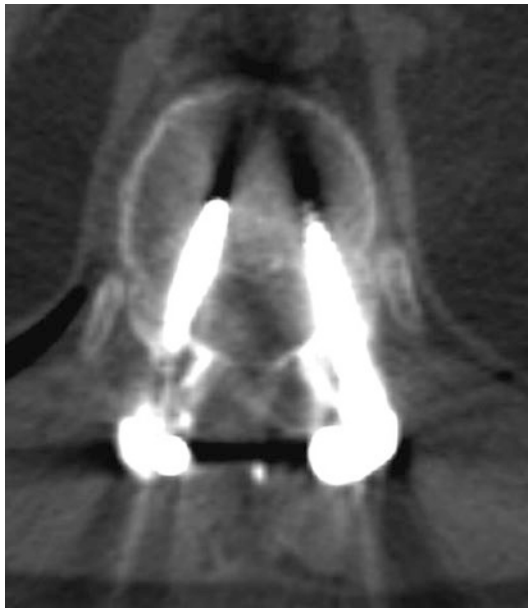


Fig. 40 CT axial image showing the misplacement of both screws violating the pedicle medial cortices

Table 6 Radiographic criteria for bony fusion

1. Less than 3° of position changes in dynamic studies
2. No lucency around the implant
3. Minimal loss of disc height
4. No fracture of device, graft, vertebra
5. No sclerotic changes
6. Visible bone formation in or around the graft

Posterior migration is defined when the posterior radiopaque marker of spacer is less than 2 mm ventral to the posterior vertebral endplate edge. Subsidence is the migration of a fusion cage into a bony endplate over 3 mm resulting in loss of height restoration and eventual neuroforamen stenosis. The spacer subsidence, like a discectomy without intervertebral height restoration, may result in foraminal narrowing owing to direct height loss and progressive subluxated facet degenerative hypertrophy and osteophytosis.

The failure of spinal fusion and the persistence of chronic instability and motion one year after surgery are defined pseudo-arthrosis, which consists in fibrous rather osseous union of the fusion complex, and represents itself a source of pain. Among the factors predisposing, there are surgical materials and techniques as well as risk factors of patient, such as scoliosis, osteoporosis, and corticosteroid use. The reported incidence of pseudo-arthrosis ranges from 3–25% to 3–46% for posterolateral and anterior interbody lumbar fusion, respectively. Pseudo-arthrosis, particularly in early stages, can only have a subtle appearance and imaging maintains low accuracy in the assessment of nonunion in comparison of surgical exploration. Clinical data deserve a prominent role: A number of subjects with non-union on imaging are asymptomatic.

On CT imaging, the preferred modality for this investigation, the diagnosis is made difficult by the large variability of findings, especially in posterior fusions. By studying the fusion site in multiple planes starting by CT volumetric acquisitions with the finest collimations available, it is possible to detect abnormal lucency and surrounding sclerosis or a corticated lucency at the graft–bone interfaces. However, a geometrically complex pseudo-arthrosis is hard to be assessed even by CT. Imaging can be extended to bone scan and SPECT. Radionuclide bone scanning offers higher sensitivity than plain radiographs, with a cold spine indicating good fusion, but false negative and false positive are frequent and only very intense focal activity has relevance in that ill-defined and intermediate uptake may only correspond to a normally increased bone turnover at the fusion site.

MRI has no significant role in the diagnosis of pseudo-arthrosis. In case of fusion, Modic type I changes fade or evolve in type II. In case of pseudo-arthrosis, MRI can reveal linear hypointensity in T1-WI, hyperintensity on T2-WI between vertebral bone and bone graft, deemed suggestive when persistent over 6 months and associated to reactive marrow changes in the form of Modic type I, bone sclerosis, and eventual contrast enhancement. The principal role of MRI is rather the exclusion of other complications.

Pseudo-arthrosis abuts in implant loosening and failure with fracture or displacement. Implant loosening manifests with a radiolucent rim of 2 mm or plus at bone-implant interface and even more with an enlarging lucency on sequential controls.

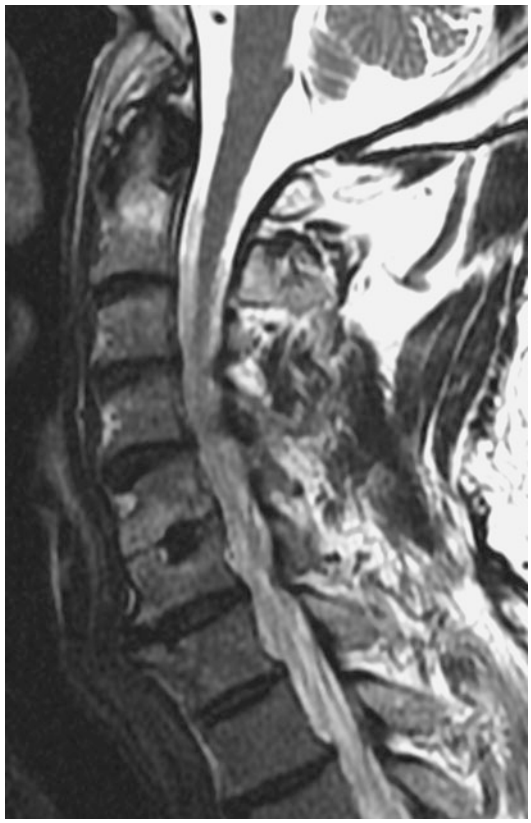


Fig. 41 MR FSE T2-W sagittal image showing a previous anterior C5-C6 discectomy with spacer completely subsiding into the C6 superior endplate with interspace collapse

FBSS can be a complication of both an unsuccessful spine fusion surgery, causing pseudoarthrosis, and a successful fusion that will causes increased biomechanical stress and motion in the adjacent motion segments resulting in accelerated degenerative changes. Increased stresses by adjacent fusion also can cause repeated microtrauma in the adjacent disc resulting in degeneration, internal disruption, or new disc herniation (Fig. 42). Awareness of potential degenerative alterations occurring with time in the adjacent segments and of coexistent comorbidities (obesity, osteoporosis, Parkinson disease) help in anticipating complications of fusion surgery and obtain an anticipated diagnosis.

Disc prostheses and dynamic stabilization devices able to limit abnormal segmental motion are developed to reduce the incidence of adjacent vertebral segment degeneration and may be used as an alternative to vertebral fusion procedures.

Sample Report 1

Patient History: 65-year-old man with back pain and neurogenic claudication for 3 months with recent right-sided radiculopathy

Clinical Diagnosis: Spinal canal stenosis and Right sciatica

Purpose of MR Study: Rule out spinal canal stenosis

Imaging Technique: MR scan with sagittal T1, T2, T2 STIR, and axial T2, no contrast administration

Full Findings: Diffuse degenerative changes of the evaluated spine segment with osteochondrosis and spondylosis signs. Subchondral low (T1 wi) and high (T2 and T2 STIR) signal intensity at L4 L5 level and, less evident, L5 S1, with associated R L4 L5 herniated disk with R foraminal extension (Modic type 1). The R L4 L5 nerve root is compressed showing edema, and there is a mild compression of the thecal sac at the same level. Median L3 L4 and L5 S1 protruded disk.

The conus-cauda region does not show signal intensity or morphological abnormalities.

The bone marrow signal intensity is normal at the remaining levels.

The is a narrowing of the spinal canal at L4 L5 level.

Interpretation: The radiological pattern together with the clinical findings suggested the diagnosis of degenerative spine abnormality with evidence of a spinal canal stenosis and median R Paramedian L4 L5 HNP.

Sample Report 2

Patient History: 63-year-old female with acute back pain resistant to medical treatment since 4 weeks without sciatica

Clinical Diagnosis: Back pain, no radiculopathy

Purpose of MR Study: Rule out spine abnormality related to back pain

Imaging Technique: MR scan with T1, T2, and T2 STIR sagittal wi (Fig. 43)

Full Findings: Diffuse degenerative changes of the evaluated spine segment with osteochondrosis and disk dehydration with

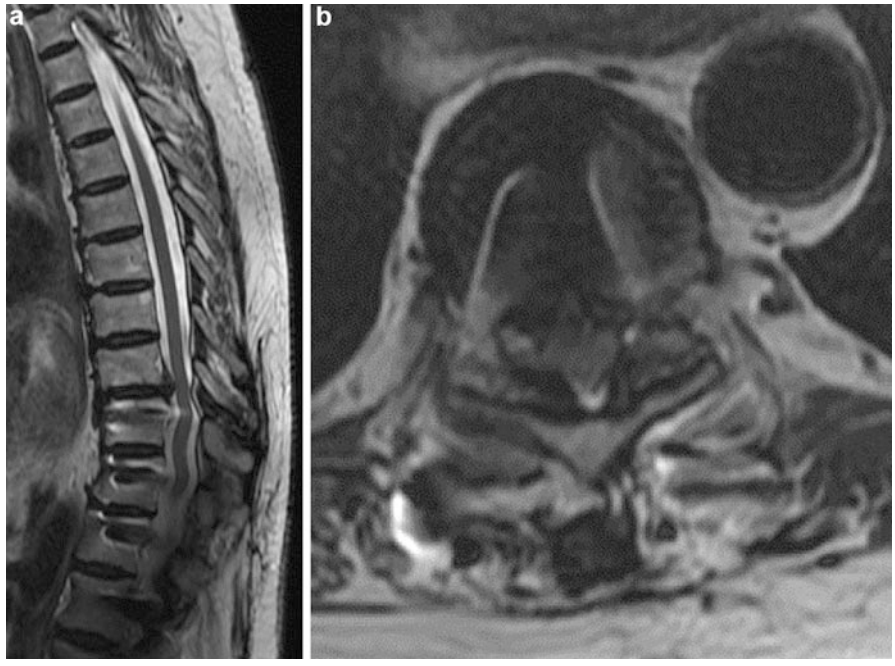


Fig. 42 (**a**, **b**) MR axial FSE T2-WI and sagittal midline FSE T2-WI showing a new disc herniation at D7-D8 compressing the spinal cord, formed in a hardwired spine 9 months after surgery

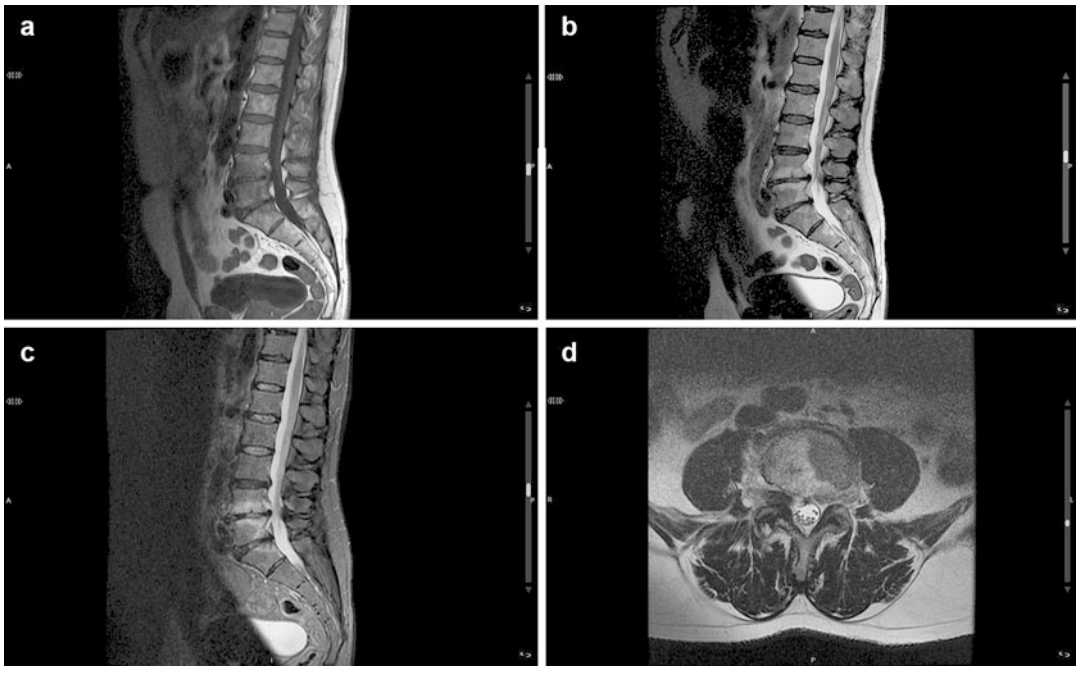


Fig. 43 (**a-d**). Diffuse degenerative changes involving the lumbar spine with osteochondrosis and spondylosis signs. Subchondral hypointense FSE T1-WI (**a**) and hyperintense FSE T2-WI (**b**) and T2 STIR (**c**) band is present at both L4-L5 and, less evident, L5 S1 endplates (Modic type 1), with associated right foraminal L4-L5 disc herniation. The right L4-L5 nerve root is compressed showing edema, and there is a mild compression of the thecal sac at the same level. Notice also median L3-L4 and L5-S1 disc protrutions

diffuse hypointensity in all sequences at all levels. Bone marrow edema with low (T1 wi) and high (T2 and T2 STIR) signal intensity at L1 and L2 Level with vertebral compression fractures with mild posterior wall displacement at L2 level.

Stabilized fracture of the superior T11 endplate without evidence of bone marrow abnormality.

Modic type 2 abnormality is visible at L4 L5 level.

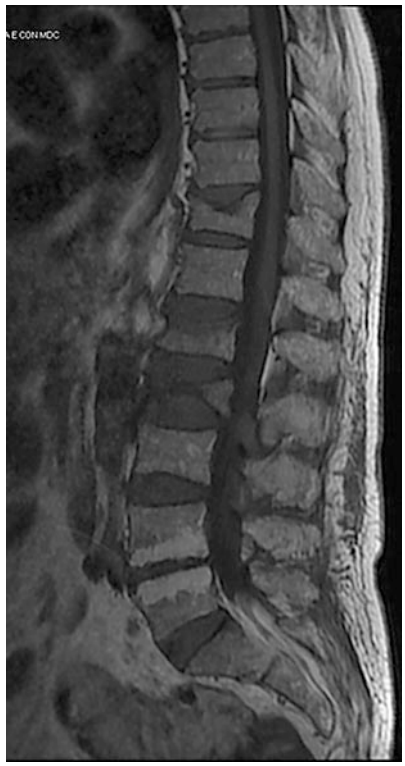
The conus-cauda region does not show signal intensity or morphological abnormalities.

The bone marrow signal intensity is normal at the remaining levels.

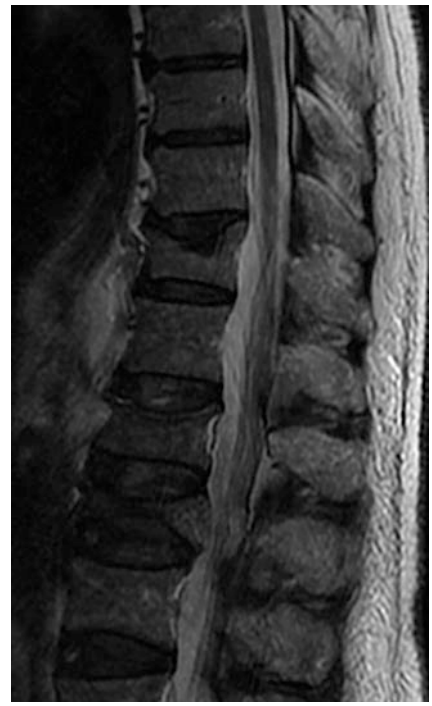
No evidence of herniated disk.

Interpretation: The radiological finding associated to clinical findings suggest the diagnosis of acute vertebral compression fracture at L1 and L2 (almost vertebra plana): Further clinical evaluation is suggested to consider vertebroplasty treatment.

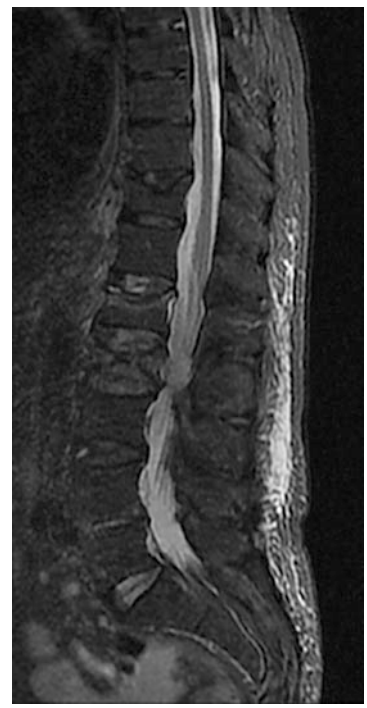
Sagittal T1 wi



Sagittal T2 wi



Sagittal T2 STIR



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Suggested Reading

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