MRI in Degenerative Disease of the Spine

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Degenerative spinal pathology is one of the most common causes of pain in society. Degenerative disease of the spinal column is highly prevalent in the general population, and its incidence increases with age.

Pain is the main symptom among those individuals most commonly associated with this pathologic condition; others are neurologic disorders (i.e. sensory, motor, neurovegetative dysfunctions).

Different factors, both individually and concomitantly, can result in degenerative processes of the spine, such as mechanical (e.g. postural anomalies, heavy weight-bearing, sports), anatomic (e.g. malformations, dysplasias) and metabolic factors (e.g. diabetes).

The lumbar spine is most frequently involved; up to 70–80 % of people have low back pain during their lifetime [1].

It is understood that only a certain number of patients with back pain will be found to have a disc herniation, spinal stenosis or well-known causes of pain; in the other cases it may be difficult to discover the source of pain. It is equally well understood that the syndrome may arise from the posterior elements/perispinal tissues of the lumbar spine, which are richly innervated [2–4].

The diagnostic techniques of computed tomography (CT) and magnetic resonance imaging (MRI) have made an important contribution to the study of degenerative diseases of the spinal column. In particular, MRI provides a very sensitive method for examining degenerative phenomena of the spine, even in the initial stages of the disease lifetime [1–3, 5–9].

However, traditional MRI of patients with low back pain does not always enable clear identification of pain aetiology. In our experience, T2-weighted sequences with fat saturation and, when indicated, T1-weighted sequences with fat saturation after intravenous administration of contrast medium provide a more sensitive picture of the degenerative changes of the spinal column and occasionally disclose pathologic conditions unsuspected during a "standard" MRI examination [1–3, 5].

All the patients in this series were examined with a 1.5 T MR system (Siemens Symphony TIM) by using the following basic study protocol:

- TSE/SE T1-weighted images on the sagittal plane (and axial plane on the lumbosacral spine)
- TSE T2-weighted images with fat saturation on the sagittal plane
- TSE T2-weighted images with fat saturation on the axial plane (eventually to be conducted on the pathologic area, for a better spatial characterization of the oedematous lesion)
- GE T2*-weighted images on the axial plane on the cervical spine
- TSE/SE T1-weighted images with FS on the sagittal and axial planes following administration of contrast medium (eventually to be conducted to identify the active-inflammatory stage of the disease)

An efficient spinal imaging protocol must comprise T2-weighted sequences with fat suppression, in particular with fat saturation technique or STIR sequences, in order to clearly visualize hyperintensity corresponding to oedematous lesions, otherwise not easily identifiable with "standard" imaging sequences without fat suppression (Fig. 2.1).

In the same way, administration of contrast medium must be followed by T1 sequences with fat saturation, in order to clearly identify the active inflammation.

The indication for contrast medium administration was based on evidence of osteo-articular or muscular-ligamentous oedema in T2-weighted images with fat saturation. In some selected cases, we administered contrast medium although the basic scan failed to disclose oedematous lesions on T2-weighted images (e.g. in cases of clinical-radiological discrepancy).

In this series we administered intravenous paramagnetic contrast medium (Dotarem – Guerbet – 0.5 mmol/ml, 0.2 ml/kg dose).

The fat saturation technique consists in a spectral saturation of the fat, by adding a selective radiofrequency (RF) impulse on the fat frequency. To determine the fat frequency,

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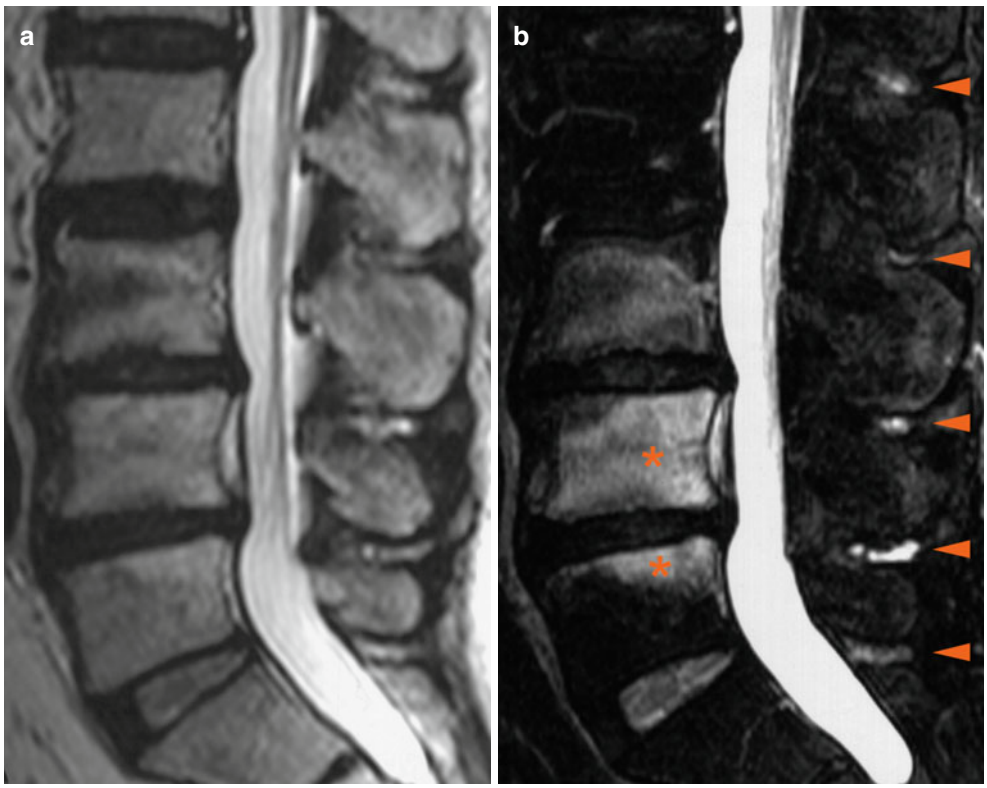

Fig. 2.1 (**a**, **b**) "Standard" sagittal TSE T2-weighted image without fat saturation (**a**) and sagittal TSE T2-weighted image with fat saturation (**b**). Hyperintense areas in fat suppressed T2-weighted image in the L3-L4-L5 vertebral bodies, indicating oedema of the bone marrow (**b**, *asterisks*). Note that the sequence without fat saturation does not detect the same lesions in the same way (**a**). Note also hyperintense lesions of the interspinous ligaments in T2-weighted images with fat saturation (**b**, *arrowheads*). The same lesions cannot be defined in T2-weighted image without fat saturation (**a**)

the machine checks for the presence of approximately a 130-Hz peak, as compared with the resonance frequency of water, at which point it emits the RF impulse. The effect of the impulse is to excite the fat over 180°, whereby it will be unable to return the signal. Such a technique enables the saturation of the fat signal on almost all of the available sequences, from spin-echo to gradient-echo, using any type of MRI sequence (T1, T2, T2*).

We have to remember that fat signal can be suppressed also by using STIR sequences, but, in our experience, this technique has a long acquisition time, whereas FS technique substantially does not change the relative acquisition time.

Today, the correct diagnosis is important in effecting improved aeration outcomes: it will be reflected in an elevated benefit/cost ratio with regard to MRI and will enable more accurate therapy of the specific source of painful symptoms (e.g. microinvasive percutaneous treatment).

In this text-atlas we deal with the following degenerative spinal changes, with particular attention on corresponding inflammatory aspects: disc degeneration, disc herniation, facet joint pathology (e.g. osteoarthritis, joint effusion, synovitis, and synovial cysts), spondylolysis, canal stenosis, spinal/perispinal ligamentous degenerative-inflammatory changes and perispinal muscular changes. Moreover, we show some unusual clinical cases.

2.1 Disc Degeneration

Discs are commonly the primary element of the spine that manifest degenerative alterations. As with every occurrence of arthrosis, such degenerative phenomena are largely due to genetics, long-term heavy weight-bearing and other complex factors.

From the morphological standpoint, disc degeneration results in loss of height, radial bulging, fissuring of the annulus fibrosus and disc herniation; moreover, degenerative disc alterations are typically accompanied by morpho-structural changes of the adjacent vertebral bodies (e.g. spondylosis and osteochondrosis).

These degenerative phenomena, and associated inflammatory component, are clinically important as they are associated with nonirradiating (discogenic) pain, caused by stimulation of the nerve terminals innervating the disc-vertebral body complex [4, 5] (Fig. 2.2).

MRI clearly depicts such degenerative phenomena to a superior degree than other imaging modalities. Among other findings, the degenerated disc demonstrates a reduction in signal intensity on T2-weighted images (principally due to dehydration processes and variations in proteoglycan composition) and a decrease in height; in the more advanced stages of degeneration, the disc collapses and may undergo cystic changes, gaseous degeneration and calcification.

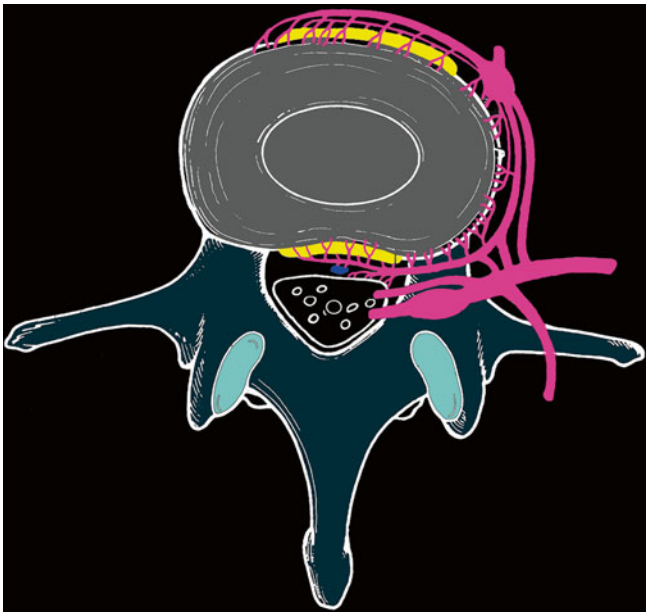

Fig. 2.2 Schematic figure depicting the rich innervation of the disc-vertebral body complex (modified from Jinkins)

Discal degenerative processes are usually accompanied by bone marrow changes in the supravertebral and subjacent vertebral bodies, which are currently classified as the following [10]:

Type I – fibrovascular replacement of the bone marrow
Type II – proliferation of the fatty marrow
Type III – osteosclerosis

In our experience, type I alterations are more evident on T2-weighted images with fat saturation, since the suppression of the fat signal enhances the fibrovascular transforming phenomena and underlying bone marrow oedema. In addition, the use of T1-weighted sequences with fat saturation after administration of contrast medium frequently shows enhancement of this same bone marrow in parallel with the bone oedema (this event is normally marked in "traditional" T1-weighted sequences after contrast medium administration).

It is sometimes possible to detect contrast enhancement of the adjacent intervertebral disc, with or without associated disc changes; in our opinion this finding can be named as "aseptic" or "sterile" discitis, of a degenerative nature [1, 5].

2.2 Disc Herniation

Disc herniation is one of the most common diseases, with very high social costs; it is the first cause for absenteeism from work and the main cause for surgery on the spine [11].

Herniated disc arises from the rupture of the annulus fibrosus and subsequent leakage of nucleus pulposus beyond the margins of the same annulus.

The aetiology of disc herniation includes degenerative disc disease, repeated trauma and genetic factors [9].

Symptoms of disc herniation originate from compression of nerve roots and/or spinal cord, depending on the level and position of the hernia.

In particular, radicular pain is caused by mechanical compression, inflammatory effects, vascular and biochemical modifications caused by the contact between the disc and nerve root [11].

Patients affected by herniated disc many times begin a diagnostic and therapeutic route involving neurosurgeons, orthopaedics, neurologists and physiatrists.

Diagnosis involves the collection of anamnestic data, physical examination, diagnostic imaging (MRI, CT) and electromyography.

MRI represents the gold standard diagnostic technique, exquisitely delineating disc herniation and the relationship to adjacent soft tissues.

It is important to describe the precise location of the herniated disc. The relationship of the disc material to surrounding structures should also be mentioned in describing a disc herniation.

The hallmark of a herniated disc is a focal contour abnormality along the disc margin, with a soft tissue mass displacing the epidural fat, thecal sac or nerve root. The herniated disc is usually contiguous with the corresponding disc by a narrow waist, which is the site of a radial tear in the annulus [9]. In some cases, a free disc fragment can develop, that is, no longer in continuity with the parent disc and can migrate inferior or superior to the parent disc level.

Unusual MR findings include atypical signal intensity or uncommon location. Some herniated discs present different signal intensity with respect to the disc parent, showing high signal intensity on T1- or T2-weighted images.

Unusual locations include extraforaminal or atypical migratory disc patterns. Sometimes disc herniation occurs completely outside the canal (e.g. the so-called far lateral herniation); rarely disc fragments are sequestered posterior to the thecal sac.

It is well known that contrast-enhanced MRI has a major role in the examination of the operated spine (i.e. diagnosis of recurrent hernia versus postoperative scar). Disc material usually does not enhance, showing peripheral contrast enhancement due to granulation tissue, whereas epidural scar demonstrates intense and homogeneous enhancement.

Contrast-enhanced MRI has a more limited use in the routine examination of the unoperated spine; however, a situation exists where the administration of contrast medium is valuable and should be considered. In particular, contrast-enhanced images are helpful in the following cases of differential diagnosis: foraminal herniated disc versus nerve

sheath tumour, intense peridiscal inflammatory response that can mimic infections and in some situations of clinical-radiological discrepancy.

In all cases, an efficient imaging protocol must comprise fat-saturated T2-weighted sequences (in order to detect oedematous lesions) and, in selected cases, contrast-enhanced T1-weighted sequences with fat saturation (in order to detect inflammatory components of the lesions or clarify diagnostic doubts).

2.3 Canal Stenosis

Canal stenosis consists in a narrowing of the central spinal canal, lateral recesses, neural foramina or a combination of these locations. This condition can be congenital or acquired or result from a combination of both factors.

Acquired (degenerative) causes are multiple: disc bulges or herniations, spondylosis, marginal osteophytes, facet joint arthrosis, calcification of the ligamenta flava and spondylolisthesis.

Stenosis is also documented after surgical procedures as a result of exuberant degeneration [11].

The lumbar and cervical regions are most commonly affected.

Symptoms of canal stenosis depend on the compression of the spinal cord or nerve roots, including pain, numbness, muscle weakness and problems with bladder or bowel function.

Typical symptoms of lumbar stenosis are neurogenic claudication, with inability of the patient to walk long distance and with sensation of heavy legs and progressive lack of strength [11].

CT and MRI with axial acquisitions allow to accurately measure the amplitude of the canal, both central and lateral.

2.4 Facet Joint Changes (Joint Effusion, Synovitis, Synovial Cysts, Osteoarthritis)

Typical degenerative changes of the zygapophyseal joints include erosion of the articular cartilage associated with joint space narrowing, periarticular hyperostosis with osteophyte formation, subchondral bone changes (e.g. eburnation, erosion, cysts), joint subluxation (i.e. with consequent joint space widening and anterior/posterior degenerative spondylolisthesis) and soft tissue changes (e.g. joint effusion, thickening/calcification of the ligamenta flava, facet joint synovial cyst formation) [2, 8, 9].

We underline that these posterior spinal structures are richly innervated primarily, although not solely, from the dorsal rami of the spinal nerves (Fig. 2.3) [4], so justifying the painful symptomatology.

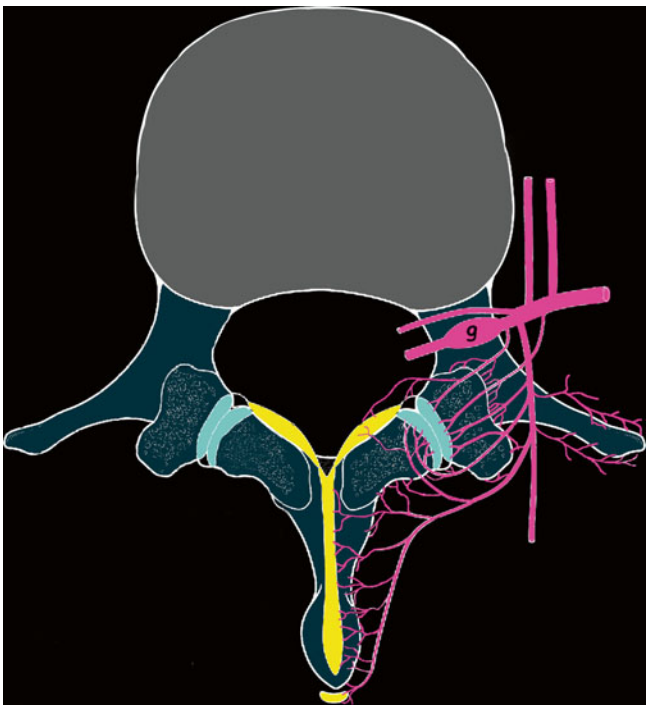

Fig. 2.3 Schematic figure depicting the rich innervation of the posterior vertebral compartment, arising from the dorsal branch of the spinal nerve (g = spinal ganglion) (modified from Jinkins)

Facet joint changes are easily recognized with standard MRI sequences. However, by using T2-weighted sequences with fat saturation and, when indicated intravenous contrast medium administration, it is possible to uncover additional otherwise occult potentially clinically pertinent findings. For example, T2-weighted sequences enable the clear, unambiguous visualization of joint effusions, subchondral bony changes (e.g. marrow oedema) and periapophyseal soft tissue changes (e.g. oedema/sterile inflammation). Contrast media administration further confirms the presence and extent of such inflammatory-degenerative phenomena. Isolated contrast enhancement of the synovial surfaces may be termed synovitis; contrast enhancement of the subchondral bone in association with the typical osteoarthritic degenerative alterations may be termed osteoarthritis; periapophyseal contrast enhancement may be defined as periapophyseal soft tissue degenerative-inflammatory reaction [2].

With regard to posterior spinal facet joint synovial cysts, such degenerative alterations may extend into the spinal canal or alternatively may have a posterior perispinal/extraspinal soft tissue location [2, 6–8]. Intraspinal synovial cysts can extend into the central spinal canal and/or into the spinal neural foramen, where they may respectively encroach on the thecal sac/cauda equina or upon the exiting spinal nerve and therefore can be the source of radicular pain or spinal claudication.

Extraspinal synovial cysts develop postero-inferiorly to the corresponding posterior spinal facet joint. Their clinical

manifestation is controversial; logically, the symptomatology probably arises from degenerative changes of the corresponding facet joint, including the neurologically competent synovium; these posterior perispinal/extraspinal soft tissue synovial cysts obviously will not impinge upon major neurologic structures [2].

Such synovial cysts may be not detectable if T2 sequences with fat saturation are not acquired. Synovial cysts may or not reveal a thin rim of enhancement after the intravenous injection of gadolinium, enhancement perhaps indicating the presence of an inflammatory component.

The pathogenesis of synovial cysts is linked to degenerative joint disease likely associated with joint effusion and elevation of intra-articular pressure; combined with kinetic-weight-bearing manoeuvres, the joint fluid theoretically creates a sudden- or chronic-progressive expansion of the synovial surface into the form of an aneurismal sac. Naturally, most or all of these synovial cysts communicate with the adjacent facet joint. Histologically these cysts may be lined by synovial tissue (i.e. true synovial cysts) or not lined with synovium (i.e. neocysts or pseudocysts lined by fibrous tissue) [2, 6, 7].

2.5 Spondylolysis

Spondylolysis is a fracture of the pars interarticularis. The pathogenesis of this alteration is believed to involve repeated microfractures of the pars, eventually resulting in a permanent defect. The pars lesion becomes bridged by a fibrocartilaginous bar and may develop into a pseudarthrosis; occasionally, bony union of a pars defect occurs [12].

In some cases, especially those without spondylolisthesis, visualization of the pars defect may be difficult to detect on T2-weighted imaging without fat saturation, and intravenous contrast medium-enhanced images permit clear recognition of these "hidden" fractures [2]. In particular, in the acute-inflammatory phase, areas of T2 hyperintensity and contrast enhancement appear in the region of the pars interarticularis defect, pedicle and occasionally within the immediately adjacent soft tissues. These changes persist for months to years and are presumably a reaction of the bone marrow and soft tissues to increased local mechanical stresses.

2.6 Spinal/Perispinal Ligamentous Degenerative-Inflammatory Changes

The ligaments of posterior vertebral compartment, and in particular the interspinous ligaments together with the related adjacent spinous processes and perispinous soft tissues, are thought to be a possible source of low back pain [2, 13–15]. These osseous and ligamentous tissues may undergo extensive degenerative-inflammatory alteration. Sudden trauma may induce an acute interspinous ligament sprain, with minor or major/complete tears of the ligamentous fibres; chronic repetitive microtrauma occurring during normal daily activities results in progressive degenerative changes of these structures [16].

According to the published literature, the fundamental factors responsible for chronic-progressive interspinous injury include the following: variable but normal hyperlordosis of the spinal segments at and suprajacent to the lumbosacral junction, with consequent bony collision of the opposed vertebral spinous processes (i.e. Baastrup's phenomenon) and injury of the intervening interspinous ligament [6, 7, 13, 14], and collapse of the intervertebral disc, with resultant narrowing of the respective interspinous space, collision of the spinous processes and injury of the interposed interspinous ligament [6, 7]. Such ligamentous degenerative changes may occur before, simultaneously with or following iso-segment intervertebral disc degeneration; furthermore, this may occur with only minor hyperlordosis.

Degenerative phenomena of the spinal ligaments has been evaluated histologically, biochemically and by MRI [15, 17–21]. Histologic examination may show varied degenerative changes in the interspinous ligaments: fragmentation and necrosis of the fibre bundle, proliferation of cells and vessels, fibrous inflammatory exudates, formation of pseudocystic cavities (i.e. formation of interspinous bursae), cartilaginous metaplasia and synovial metaplasia. These changes explain the variability of MRI signal in the region of the interspinous ligaments that may be either iso- or hyperintense on T1- and T2-weighted images and which frequently demonstrate contrast enhancement. In some cases, these ligamentous changes are not detectable on T2-weighted imaging using fat saturation, but only after the administration of intravenous contrast medium coupled with fat-saturated T1-weighted acquisitions [2].

Based upon published reports, there seems to be no specific terminology for these MRI/pathologic changes of the interspinous ligaments. Some authors use the definition of "interspinous bursitis" [8]. We think that the term bursitis should be applied only in presence of an interspinous *pseudobursae* or pseudocysts (i.e. lined by fibrous tissue), which are related to interspinous pseudarthrosis and which demonstrate rim enhancement after the administration of intravenous contrast medium within the fibrous walls of the pseudocyst cavity. Actually, it's possible to find bursae that do not show contrast enhancement and ligaments without bursae that show contrast enhancement, so we prefer to distinguish between degenerative (i.e. no enhancement) and degenerative-inflammatory (i.e. enhancement) changes of the spinal ligaments [2].

These ligamentous changes may present clinically with focal midline lumbar tenderness and pain on palpation as well as upon extension or flexion manoeuvres; such pain typically can be relieved by local anaesthetic/steroid

injections. However, although low back pain has been attributed to these interspinous alterations, clinical significance of the above-mentioned findings is controversial, as similar changes may be observed in asymptomatic individuals. In our experience, contrast enhancement of the ligaments corresponds generally to local painful symptomatology. Upright, weight-bearing and dynamic-kinetic MRI of the spine may be also helpful in the clinical-radiological correlation of such ligamentous pathology [22].

Chronic degenerative-inflammatory changes may involve not only the interspinous ligaments but also the supraspinous ligaments and the ligamenta flava; in many instances several or all of these structures are involved, at one level or at multiple levels.

2.7 Muscular Changes

Occasionally, in patients with low back pain, MRI detects alterations of the intrinsic posterior perispinal lumbosacral muscles (e.g. interspinalis, multifidus muscles).

It is possible to postulate that three-dimensional lumbar intersegmental hypermobile instability, exceeding the normal range of motion, may predispose and precipitate various pathologic degenerative muscular alterations, including spasm, sprain, inflammation and acute-subacute degeneration [2]. These changes can be caused by either one or both of two mechanisms related to neuromuscular autotrauma: rupture-avulsion of the intrinsic spinal muscles or traumatic denervation of the nerves supplying the intrinsic spinal muscles (i.e. rupture-avulsion of the medial branch of the dorsal ramus of the spinal nerve) [16, 23].

MRI clearly visualizes the above-mentioned muscular alterations. In particular, T2-weighted images with fat saturation show hyperintensity of the perispinal muscular tissue, otherwise not identifiable with standard imaging sequences without fat suppression; typically, these same muscle fibres showed enhancement after the intravenous administration of contrast medium.

Such muscular changes may be directly (e.g. intrinsic spinal muscle sprain/rupture) or indirectly (e.g. intrinsic spinal muscle spasm) involved in the pathogenesis of low back pain. It is possible to find a close clinical-radiological correlation between the anatomic distribution of pain and the perispinal muscle alterations as visualized by MRI. In some patients with low back pain, these changes may be the only abnormal finding on MRI.

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