

Magnetic Resonance Imaging in degenerative disease of the lumbar spine: Fat Saturation technique and contrast medium

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Summary. *Purpose:* To examine both anterior and posterior elements of the lumbar spine in patients with low back pain using MRI T2-weighted sequences with Fat Saturation (FS) and contrast enhanced T1-weighted sequences with FS. *Materials and methods:* Two thousand eight hundred and twenty (2820) patients (1628 male, 1192 female, mean age 54) presenting low back pain underwent MRI standard examination (Sagittal T1w TSE and T2w TSE, axial T1 SE) with the addition of sagittal and axial T2w Fat Sat (FS) sequences. Among all the patients, 987 (35%) have been studied adding Contrast Enhanced (CE) T1w FS sequences after administration of contrast medium. *Results:* Among 987 patients studied with contrast medium, we found: active-inflammatory intervertebral osteochondrosis in 646 (65%) patients; degenerative-inflammatory changes in facet joints (facet joint effusion, synovitis, synovial cysts) in 462 (47%); spondylolysis in 69 (7%); degenerative-inflammatory changes of the flava, interspinous and supraspinous ligaments in 245 (25%); inflammatory changes of posterior perispinal muscles in 84 (8%) patients. *Conclusions:* In patients with suspected no-disc-related low back pain, the implementation of T2w FS and CE T1w FS sequences to the standard MR protocol could allow a better identification of degenerative-inflammatory changes more likely associated to the pain. (www.actabiomedica.it)

Key words: MRI, Fat Saturation, contrast medium, lumbar spinal degenerative disease

Introduction

Low back pain (LBP) is one of the most common reasons why individuals seek medical care. According to the Global Burden of Disease 2010 Study, LBP causes more global disability than any other condition (1-10).

In most cases, the aetiology of the pain stems from the degenerative state of the intervertebral disc, with subsequent disc-root conflict. However, numer-

ous structural inter-segmental changes of the spine may be the cause of patients' signs and symptoms.

Degenerative disease of the spine is highly prevalent in the general population, and its incidence increases with age. Among those individuals, pain is the most commonly associated symptom with this pathological 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, dysplasia), and metabolic factors (e.g., diabetes) (11-20).

The intervertebral disc is generally the main component of the spinal column that undergoes degenerative phenomena: its progressive thickening induces alterations of the bony vertebral endplate and bone marrow of the contiguous vertebral bodies (21-31). The alterations in the disco-vertebral complex also affect the corresponding posterior osteoarticular elements of the spinal column, resulting in narrowing of the vertebral canal and/or the adjacent spinal neural foramina, or instability of the spinal column (21, 32-40).

Computed tomography (CT) and magnetic resonance imaging (MRI) have been widely adopted for the degenerative spine disease. Indeed, MRI represents a very sensitive technique for the assessment of early degenerative changes of the spine (13, 20, 32, 33, 35, 41-45).

However, standard MRI protocol without fat suppression and contrast media injection in patients with low back pain does not always enable clear identification of pain aetiology (46).

In our work we verified if the addition of T2-weighted sequences with Fat Saturation (FS) and, when indicated, T1-weighted sequences with FS after intravenous administration of contrast medium, were able to provide a more sensitive picture of the degenerative changes of the spinal column and occasionally disclose pathologic conditions unsuspected during a "standard" MRI examination (30, 43-45, 47-50).

Materials and methods

From January 2006 to December 2011 we performed MR scans in 2820 patients (1628 male and 1192 female, 14-83 years old, mean age 54) presenting low back pain sometimes radiating to the legs. Physical examination before MR has been performed for all patients aiming to identify those with "mechanical" and radicular pain; other inclusion criteria were: known degenerative disease of lumbar spine previously revealed on X-ray and/or MRI exams. Exclusion criteria were: history of trauma, tumours, known infective spinal disease, vertebral fractures and previous spine surgery.

The study has been conducted according to the declarations of Helsinki, and written informed consent has been obtained from all subjects.

Patients were examined with a 1.5 Tesla MR scanner (Siemens Symphony, Erlangen, Germany) using the study protocol described in table 1.

In case of failure to disclose the cause of pain and/or in case of T2-hyperintensity of osteoarticular and muscular-ligamentous structures on sagittal sequence, axial TSE T2-FS sequences on suspected pathologic area have been added; patients who benefitted of sagittal and/or axial TSE T1-w FS (Fat Sat) sequences after contrast medium administration (Dotarem-Guerbet -0.5 mmol/ml, 0.2 ml/kg dose) were selected following these criteria:

- presence of osteoarticular or muscular-ligamentous oedema detected on sagittal and axial T2-FS;
- presence of clinical findings such as "mechani-

Table 1. MR scan protocol (1.5 Tesla). Axial T2-FS and CE T1-FS sequences have been adopted on selective pathological areas previously identified on basic scan

	TR	TE	Matrix	Averages	Thickness	Acquisition Time
Basic Sequences						
Sagittal TSE - T1	567	10	192x256	1	4	2.57 m
Sagittal TSE - T2 - Fat Sat	4200	113	157x256	1	4.5	4.16 m
Axial (intervertebral discs) TSE - T1	600	15	256x256	1	4	3.34 m
Additional Sequences						
Axial (pathologic area) TSE - T2 - Fat Sat	4000	105	144x256	5	5	3.26 m
Sagittal T1 + Gadolinium	529	11	257x384	3	3.5	2.47
Axial T1 + Gadolinium	570	9.3	256x320	2	4	3.14

cal" pain, radicular pain, trigger points on palpation, even if the previous sequences failed to disclose oedematous lesions

All non-disc-related findings have been classified

as:

- Vertebral body changes (active/inflammatory endplate changes)
- Facet joints changes (facet joint effusion, synovitis, subchondral cysts, synovial cysts)
- Spondylolysis
- Ligament changes (Ligamentum Flavum, interspinous and supraspinous ligaments)
- Muscular changes (posterior perispinal muscles)

All the MR exams have been reported by consensus by two neuroradiologists with 15 years of experience in spine imaging.

Results

In 1833 out of 2820 patients (65%) no osteoarticular and/or muscular-ligamentous oedema have been detected with standard MR protocol and patients symptoms were variously associated to common causes of low back and sciatic pain: disc disease (1438 cases), degenerative spondylolisthesis (372 cases), and vertebral canal stenosis (567 cases).

987 out of 2820 patients (35%) received intravenous contrast medium because the T2-weighted sequences with FS displayed oedema in the osteo-articular and muscular-ligamentous structures (879 patients) or because the basic scan failed showed no alterations pertinent with the cause of pain (108 cases).

In these 987 cases, CE (Contrast-Enhanced) T1-FS sequences allowed the identification of a single or a combination of these entities:

- active/inflammatory endplate changes in 646 (65%) patients (Fig. 1, 3);
- facet joints changes in 462 (47%) patients (Fig. 2, 3, 4): facet joint effusion, synovitis (Fig. 2, 4, 7), osteoarthritis, synovial cysts (Fig. 9);
- spondylolysis in 69 (7%) patients (Fig. 5, 9);
- degenerative-inflammatory changes of the flava, interspinous and supraspinous ligaments in 245 (25%) patients (Fig. 4, 5, 7);

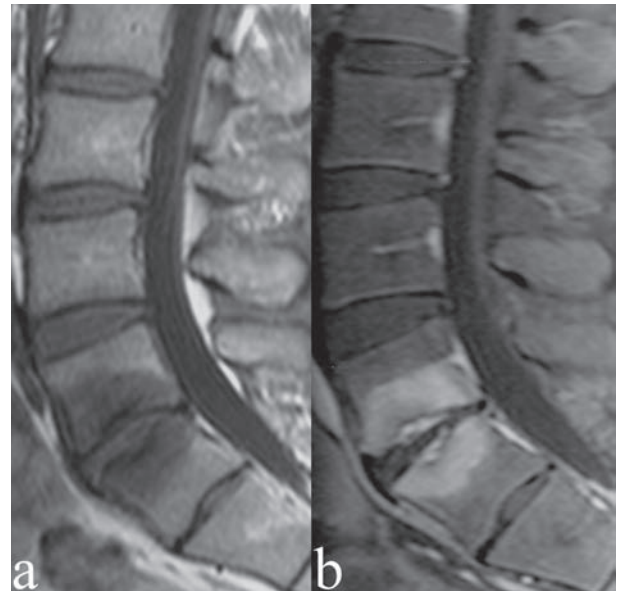


Figure 1. Patient with chronic low back pain. Severe osteochondrosis at L5/S1 with fibro-vascular transformation of bone marrow (Modic I). **a)** Sagittal T1-weighted image; **b)** sagittal T1-weighted image with Fat Saturation following contrast medium administration. Contrast enhancement of the endplates and subchondral cancellous bone, indicating active-inflammatory stage of the degenerative process and its extension

- changes of the posterior perispinal muscles (oedema, inflammation) in 84 (8%) patients (Fig. 3, 6, 8).

Among the 879 patients with T2-FS findings, CE T1-FS sequences allowed a better definition and extent evaluation of the lesions in 789 patients; in the remaining 90 patients, no substantial benefit have been provided by contrast medium administration.

In 108 out of 987 patients (11%) CE T1-FS allowed the identification of ligamentous lesions (29), synovitis (38), and discal/peridiscal inflammation (41), which have been missed on both standard and additional non-enhanced sequences.

Among all 987 subjects who have shown pathological findings on CE T1-FS images, 908 (92%) revealed a possible association with the clinical examination, 79 (8%) presented no clear agreement between MR findings and clinical examination. All 79 patients showed T2-hyperintensity and contrast enhancement of the lesions.

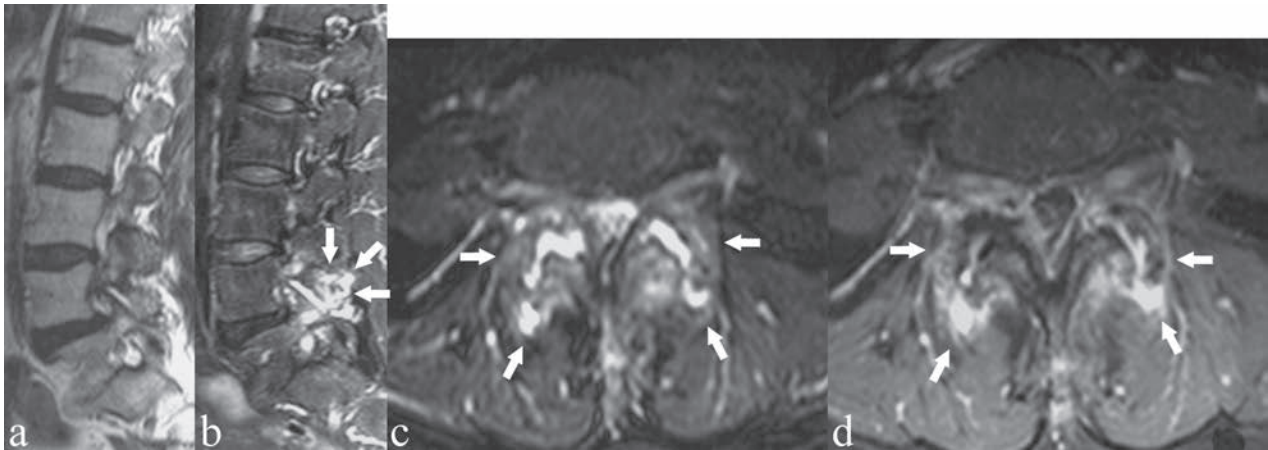


Figure 2. Patient with bilateral low back pain. **a)** Sagittal T2-weighted image; **b, c)** sagittal and axial T2-weighted images with Fat Saturation; **d)** axial T1-weighted image with Fat Saturation after contrast medium administration. Bilateral facet joint osteoarthritis and synovitis at L4/L5 (arrows). Hyperintensity on T2-weighted images and contrast enhancement of the bone marrow of posterior spinal facet joint articular processes (osteoarthritis) and within the facet joint space (synovitis); the same articular space is enlarged (**c**). Note the differences between **a)** and **b)**: “standard” T2-weighted image without fat saturation (**a**) fails to show the above mentioned pathological findings that are clearly showed on T2-weighted image with fat saturation (**b, c**)

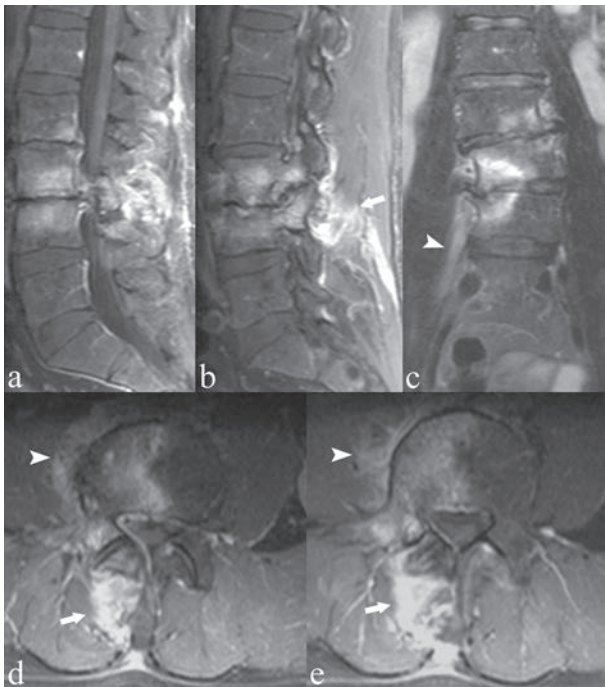


Figure 3. Patient with right-sided low back pain. Sagittal (**a, b**), coronal (**c**), and axial (**d, e**) T1-weighted images with Fat Saturation following contrast medium administration. Right-sided osteochondrosis at L3/L4, with contrast enhancement of the subchondral bone, indicating active inflammatory component of the degenerative process. Note the extension of the inflammation to the adjacent peridiscal soft tissue and psoas muscular fibers (arrowheads). There is also right facet joint osteoarthritis and inflammation of the right perispinal posterior muscular fibres (arrows)

Discussion

All the patients included in our study protocol suffered of LBP, occasionally radiating to the lower limbs. This work demonstrated that T2 weighted sequence with FS and/or T1 weighted sequence with FS after contrast media injection can improve the diagnostic performances of standard MR examination in patients with LBP. Moreover these sequences in the study presented population, permitted the complete and better definition of some pathological entities causing LBP.

It must be stressed that painful symptomatology can be the result of pathological changes involving several anatomic elements of the spine, with only a limited number of cases related to radicular compression. These various components capable of producing painful sensations are represented by spinal nerves, the osteoarticular elements of the spinal column (i.e., the periphery of the intervertebral disc, the posterior spinal facet joints, spinal ligaments) and the perispinal muscles.

The spinal column can be divided into two anatomic compartments – anterior and posterior – each corresponding to its respective intersomatic joint or disc and joints of the posterior vertebral arch. These regions can be further subdivided according to biome-

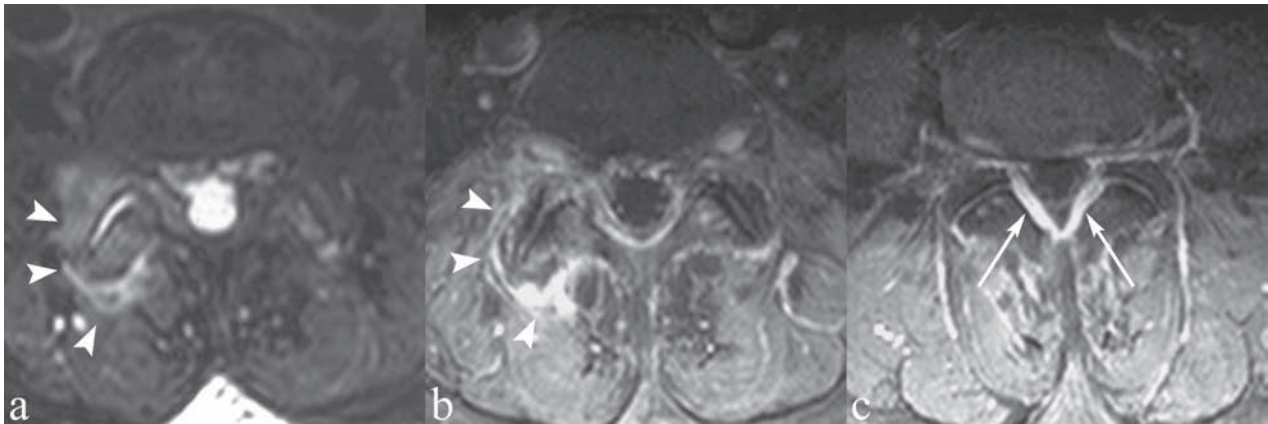


Figure 4. Patient with right-sided low back pain. **a)** Axial T2-weighted image with Fat Saturation; **b, c)** axial T1-weighted images with Fat Saturation following contrast medium administration. Facet joint osteoarthritis and mild synovitis on the right-side, with periapophyseal contrast enhancement indicating soft tissue inflammatory reaction (arrowheads). Note also contrast enhancement of the flava ligaments (**c**, arrows), indicating ligamentous degenerative-inflammatory changes

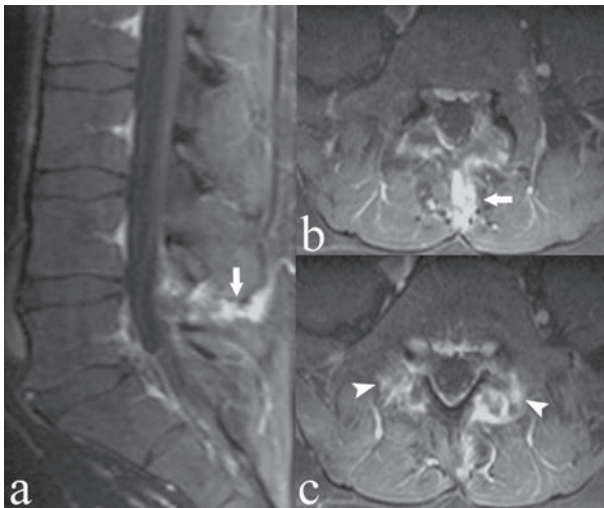


Figure 5. Young patient with low back pain and marked focal tenderness. Sagittal (**a**) and axial (**b, c**) T1-weighted images with Fat Saturation following contrast medium administration. The interspinous ligament at L4/L5 enhances following contrast medium administration (arrows), indicating degenerative-inflammatory ligamentous changes. Note also bilateral contrast enhancement in the pars interarticularis of L5 and adjacent soft tissues, indicating bilateral spondylolysis and reactive inflammation of the adjacent soft tissues (arrowheads)

chanical studies and the concept of “anatomic functional unit” of the spinal column (51-54). Morphostructural changes of one region have repercussions on another entire region or a part of that. These individual regions must be carefully examined by the radiologist.

The dense, complex regional innervation of spinal column structures correlates with the origin of pain, spreading from the spinal nerves via its ventral and dorsal roots which, in turn, generate a smaller series of ramifications. The different type of involved innervation determines the potential nature of the pain (radicular, local, referred) (33, 35, 55-57). Radicular pain depends to a lesser extent on the direct compression of the nerve root than on physiologic factors (altered tropism of the nerve due to compression on arterial afferent vessels and/or from venous stasis) and on neural and perineural inflammatory reactions (4, 20, 39, 42, 58, 59), which produces mechano- and chemosensitivity within the neural radicle. We can postulate that similar inflammatory factors are also involved in the pathogenesis of the other types of back pain (19, 24, 36, 49, 60-70).

The majority of the patients in our study protocol presented degenerative phenomena of the intervertebral discs on standard MR scan, consistent with the observation that discs are commonly the primary element of the spinal column that manifest degenerative alterations (29, 33). 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

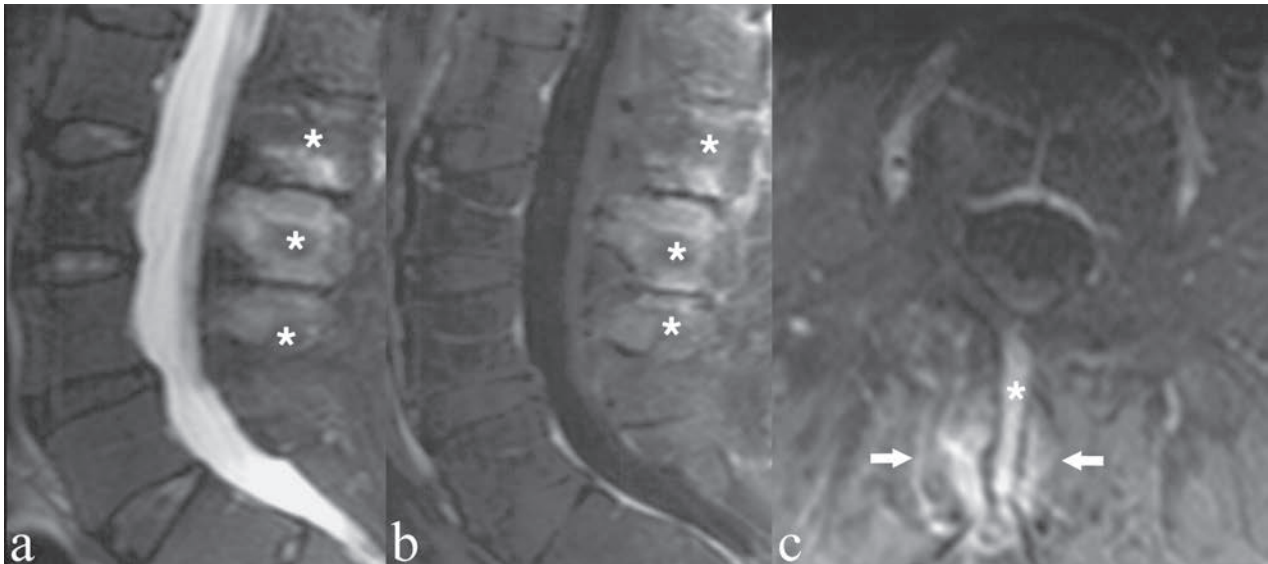


Figure 6. Patient with persistent low back pain; physical examination showed limited lumbar motion, exacerbation of pain by hyper-extension and tenderness on palpation of the interspinous spaces. **a)** Sagittal T2-weighted image with Fat Saturation; **b, c)** Sagittal and axial T1-weighted images with Fat Saturation following contrast medium administration. Lumbar hyperlordosis is present, associated with collision of the spinous processes at L2-L3-L4 (i.e. Baastrup's phenomenon). The bone marrow of the above-mentioned opposing spinous processes (asterisks) reveals hyperintense areas on T2-weighted image (**a**) and contrast enhancement (**b, c**); these findings represent stress-related bony reactive-degenerative changes. The corresponding L2/L3 and L3/L4 interspinous spaces are collapsed. The adjacent muscular fibers (i.e., interspinalis muscles) show contrast enhancement, indicating degenerative-inflammatory muscular alteration (arrows)

of the annulus fibrosus, and disc herniated; moreover, degenerative disc alterations are typically accompanied by morpho-structural changes of the adjacent vertebral bodies (e.g. osteochondrosis and spondylosis).

MRI remarkably documents such degenerative phenomena to a superior degree than other imaging modalities (32, 33, 43-45). 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 fully collapses and may undergo cystic changes, gaseous degeneration and calcification.

Discal degenerative processes are usually accompanied by bone marrow changes in the supra- and sub-adjacent vertebral bodies, which are currently classified as the following (71, 72):

Type I – fibrovascular replacement of the bone marrow;

Type II – proliferation of the fatty marrow;

Type III – osteosclerosis.

In our study patients, such phenomena, although usually manifested on standard MRI sequences, were more evident on T2-weighted images with FS; in particular, type I alterations were more evident, since the suppression of the fat signal enhances the fibrovascular transforming phenomena and underlying bone marrow edema. In addition, the use of T1-weighted sequences with FS after administration of contrast medium showed enhancement of this same bone marrow in parallel with the bone oedema (Fig. 1, 3) (this event is normally masked in non-FS T1-weighted sequences after contrast medium administration).

In 16 cases (1,6%) we found contrast enhancement of the 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 (43, 45).

In the study of patients with LBP, it is also necessary to carefully examine the osteoarticular, ligamentous, and muscular elements of the posterior aspect of the spinal column.

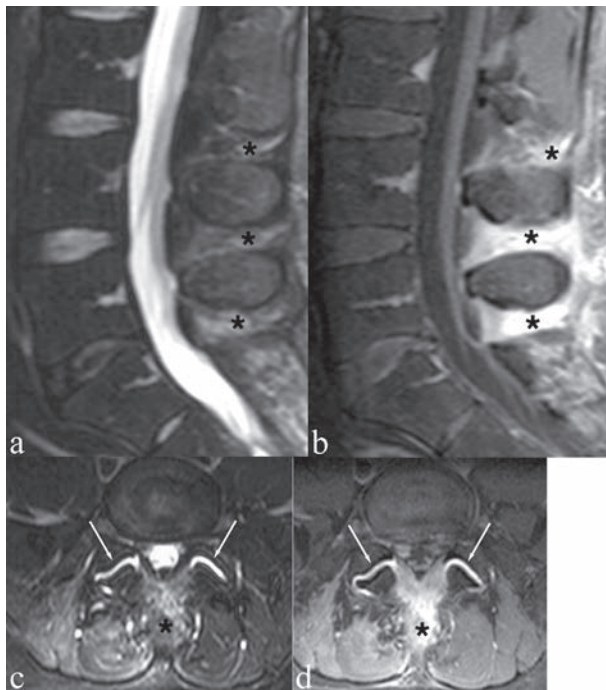


Figure 7. Patient with low back pain and focal tenderness on the midline. **a, c**) Sagittal and axial T2-weighted images with Fat Saturation; **b, d**) Sagittal and axial T1-weighted image with Fat Saturation following contrast medium administration. The interspinous ligaments at L3/L4, L4/L5 and L5/S1 (asterisks) show hyperintensity on T2-weighted images (**a, c**) and marked contrast enhancement (**b, d**). These findings indicate degenerative-inflammatory ligamentous changes. Note also bilateral facet joint synovitis (**c, d**, arrows)

The posterior spinal facet joints may undergo degenerative arthrosis, presenting with a narrowing or loss of the joint space (occasionally accompanied by

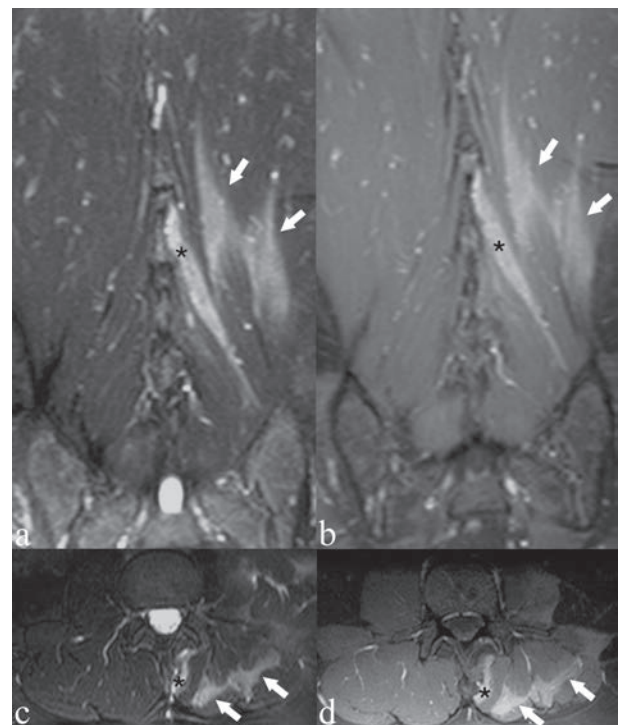


Figure 8. Patient with left-sided low back pain. There was a precise correlation between the MRI findings and the patient's focus of low back pain. **a, c**) Coronal and axial T2-weighted images with Fat Saturation; **b, d**) coronal and axial T1-weighted images with Fat Saturation following contrast medium administration. Hyperintensity on T2-weighted images (**a, c**) and contrast enhancement (**b, d**) within the perispinal muscle fibers on the left side: multifidus muscle (asterisk), longissimus and iliocostalis muscles (arrows)

intra-articular vacuum), joint surfaces erosions, subchondral sclerosis, para-articular osteophytosis, calcification of the joint capsule and ligamenta flava, and

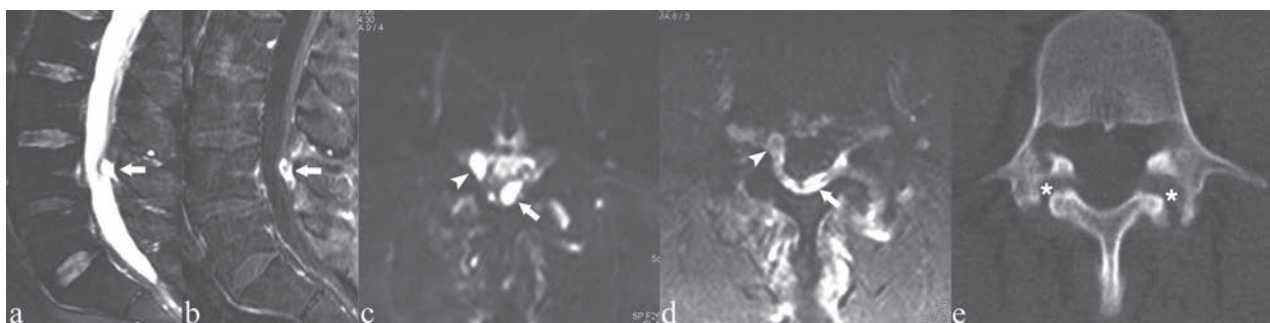


Figure 9. Patient with low back pain. **a, c**) Sagittal and axial T2-weighted images with Fat Saturation; **b, d**) Sagittal and axial T1-weighted images with Fat Saturation following contrast medium administration; **e**) axial CT image on L4. Mild anterolisthesis of L4 over L5 (**a-b**), with spondylolysis of L4 (**e**, asterisks). Note also the intraspinal synovial cyst (**c, d** arrowheads) and the neo-/pseudocyst in the epidural space of the posterior aspect of the central spinal canal (**a-d**, arrows)

apophyseal hypertrophy (29, 32, 33, 73). Such events generally occur secondary to degenerative phenomena of the corresponding intervertebral disc that progressively affect the posterior spinal elements, resulting in facet joint subluxation, remodelling of facet joints, and progressive stenosis of the contiguous spinal neural foramen. Phenomena contributing to spinal facet joint degeneration include axial plane facet joint orientation anomalies or lumbosacral hyperlordosis.

From a clinical perspective, the "facet joint syndrome" is characterized by a deep ill defined lumbar pain, which may be referred to the buttocks and lower limbs; the pain is typically induced or increased by hyperextension and rotation of the spinal column, which remains limited in its range of motion (74, 75).

MRI plays an eminent role in the identification of degenerative phenomena of the spinal facet joints, thereby revealing alterations otherwise unsuspected in other imaging techniques (76, 77). T2-weighted sequences, including those with FS, may show hyperintense signal within the joint space, reflecting joint effusion. In our series, 260 patients (26%) showed facet joint effusion. In 161 cases (16%) the same joint space revealed contrast enhancement on CE T1-FS as for facet joint synovitis (Fig. 2, 4, 7). T2-hyperintensity and contrast enhancement of the subchondral bone marrow, in association with the typical degenerative facet alterations, was found in 279 cases (28%). This phenomenon may be termed as degenerative "facet joint osteoarthritis" (Fig. 2, 3, 4). Periapophyseal contrast enhancement, found in 146 cases (15%) in this study, may be defined as sterile degenerative "periapophyseal soft tissue inflammatory reaction" (Fig. 4).

An additional consequence of such degenerative phenomena of the facet joint is represented by the formation of periarticular synovial cysts, resulting from the hydraulic forces during kinetic, weight bearing maneuvers within a closed joint space with an underlying expanded/balooned joint effusion (78). Synovial cysts can extend toward the central spinal canal (resulting in spinal stenosis) or into the contiguous neural foramen (in which case they may cause spinal nerve compression) or toward the retrospinal periapophyseal soft tissues (in which case, pain may be related to degenerative-inflammatory changes of the corresponding facet joint and periarticular soft tissues).

Synovial cysts present a signal similar to cerebrospinal fluid, peaking in hyperintensity on T2-weighted sequences with FS; in 89 out of 128 cases the wall of the cyst enhanced after contrast medium administration (Fig. 9).

In 69 patients with low back pain we found spondylolysis, with or without spondylolisthesis. Spondylolysis is a fracture of the pars interarticularis (79). The pathogenesis of this alteration seems to be related to repeated microfractures of the pars interarticularis, eventually resulting in a permanent defect. In 19 cases, especially those without spondylolisthesis (13 on 19 cases), visualization of the pars defect may be difficult to detect on MRI. T2-weighted images with FS and contrast medium-enhanced images with FS in these 19 cases allowed clear recognition of these "hidden" or occult fractures (44), otherwise not detectable by standard MR sequences. In particular, in the active-inflammatory phase, areas of T2-hyperintensity and contrast enhancement appeared in the region of the pars interarticularis defect, the contiguous pedicle and occasionally within the immediately adjacent soft tissues (Fig. 5, 9). These changes persist for months to years, presumably a reaction of the bone marrow and related soft tissues to increased local mechanical stresses.

In 245 patients affected by low back pain we found degenerative-inflammatory phenomena of the interspinous and/or supraspinous and/or flava ligaments (Fig. 4, 5, 7). As reported in literature, the fundamental factors responsible for chronic-progressive interspinous soft tissue injury include the following: hyperlordosis of the lumbosacral spine with consequent bony collision of the opposed vertebral spinous processes (i.e. Baastrup's phenomenon) and injury of the intervening interspinous ligament; collapse of the respective intervertebral discs, with consequent interspinous space narrowing with bony collision and traumatic injury of the corresponding interspinous ligament. Any form of ligamentous stress may, however, cause degenerative-inflammatory alterations in the ligaments themselves, in the absence of disc collapse or interspinous narrowing at the same level. Once again, in 245 cases FS techniques allowed the discernment of ligamentous changes that cannot otherwise be visualised. In 226 patients T2-weighted sequences with FS

showed hyperintensity of the above-cited ligaments and contiguous tissues. Following contrast medium administration, in 181 cases, T1-weighted sequences with FS showed areas of enhancement within and surrounding the ligaments themselves. We underline that in 19 cases, we found contrast enhancement of the ligaments without abnormal findings in T2-weighted images. Moreover, in 37 cases we found interspinous ligamentous pseudocysts also presenting as hyperintensity on T2-weighted sequences with FS, with peripheral contrast enhancement.

Degenerative-inflammatory changes may involve not only the interspinous and supraspinous ligaments but also the flavae ligaments (Fig. 4); in many instances several or all of these structures are involved, at one or at multiple levels, on one or both sides.

In 84 patients with low back pain, MRI detected alterations of the intrinsic posterior perispinal lumbosacral muscles (e.g. interspinalis, multifidus muscles) (3-8). It is possible to postulate that three-dimensional lumbar intersegmental hypermobile instability may induce various muscular alterations, including: spasm, inflammation, sprain, rupture and eventual degeneration/atrophy. These changes can either be caused by one or both of two mechanisms related to neuromuscular auto-trauma: rupture/avulsion of the intrinsic spinal muscles or traumatic denervation of the nerves supplying the same muscles (i.e. rupture-avulsion of the medial branch of the dorsal ramus of the spinal nerve).

MRI clearly visualized the above-mentioned muscular changes. In particular, T2-weighted images with FS showed hyperintensity of the posterior perispinal muscular tissues, otherwise not identifiable with standard imaging sequences without FS; typically, these same muscles showed enhancement after intravenous administration of contrast medium (Figure 3, 6, 8).

Such muscular changes may be directly or indirectly (e.g. muscular sprain, rupture, muscular spasm) involved in the pathogenesis of low back pain. In these cases, it was possible to find a close clinical-radiological correlation between the anatomic distribution of pain and the perispinal muscle alterations as visualized by MRI. In 33 patients with low back pain, these changes were the only abnormal findings visualised on MRI.

MRI of patients with low back pain demands careful examination not only of the discal-radicular complex but of the entire osteoarticular apparatus of the spinal column as well as the perispinal soft tissues, to search for clinically relevant degenerative-inflammatory processes. Without a careful imaging analysis of the non-discal and non-radicular tissues of the spine and perispinal tissues, the true clinical source of the patient's back pain may be overlooked, missed. In addition: 1) the true pain locus will likely go untreated, or 2) the clinician may treat a spinal finding that is not a cause of the dominant pain syndrome leading to a form of the failed therapy syndrome.

We emphasize that the imaging observations sought in this evaluation were intended to stand alone primarily as observational ones (i.e., associated with clinical findings but not unequivocally invoked as causal factors). Additional studies are needed to clearly correlate MRI findings with pain symptomatology and response to treatment.

In conclusion, it has been found that integrating MRI with T2-weighted images with FS and, when indicated, with T1-weighted images with FS after intravenous contrast medium administration, increases diagnostic sensitivity in patients with low back pain, particularly in those cases whose standard MR examination does not disclose a clear cause of pain. In such cases FS MRI enables a focused therapeutic regimen to be generated, and allows critical re-evaluation over time on specific therapy.

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