Degenerative Spinal Disease

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Introduction

Back pain is one of the most common disorders worldwide. A global burden of disease study from 2010 [1] ranks it sixth between HIV and malaria in terms of its impact on disability-adjusted life years. Degenerative disease of the spine is considered the most common etiologic cause. Mechanical, traumatic, nutritional, and genetic factors all play a role in the cascade of disk degeneration. The presence of degenerative change is by no means an indicator of symptoms, and there is a very high prevalence in asymptomatic individuals. The etiology of pain as the symptom of degenerative disease is complex and appears to be a combination of mechanical deformation and the presence of inflammatory mediators. The role of imaging is to provide accurate morphologic information and influence therapeutic decision making. A necessary component, which connects these two purposes, is accurate natural history data. This is critical because the justification of an intervention, whether diagnostic or therapeutic, requires the intervention to have a more favorable outcome than the untreated natural history of the disease process. In order to fully understand the value of imaging findings on therapeutic thinking, the following five considerations are critical: first, the reliability and reproducibility of imaging findings; second, the prevalence of findings in asymptomatic and symptomatic populations; third, the natural history and behavior over time; fourth, the prognostic value of the findings; and fifth, the treatability of the condition.

In terms of the reliability and reproducibility of the imaging findings, standard nomenclature is crucial and has been much discussed in the literature [2]. The morphologic changes one can identify in imaging are myriad and variable. These include degenerative disk changes such as narrowing, signal intensity loss on T2-weighted images, fissures, vacuum phenomena, annular disruption, bulge, and herniation. Adjacent changes in the soft tissues, bone, and ligament are also important as are morphologic changes such as canal and foraminal narrowing, nerve root compression, etc. Facet changes are also considered to be important. Even in the presence of standardized nomenclature, there is significant variability between and within readers. For instance, the reliability of interpretation based on interobserver reliability is quite good for morphology and kappa of .81 [3], yet only fair for the degree of stenosis, the presence of spondylolisthesis, marrow change, or facet disease [4].

Any study looking at the natural history of degenerative disk disease, prognostic value of imaging, or its effect on therapeutic decision making will be confounded by the high prevalence of morphologic change in the asymptomatic population [5–7]. 20–28 % of asymptomatic patients demonstrate disk herniations and the majority have evidence of additional degenerative disk disease [5–7]. These findings are not only non-predictive in the moment, but prospectively as well. In a 7-year follow-up of a patient group with back pain [8], the original MR findings were not predictive of the development or duration of low back pain.

The natural history and behavior of degenerative changes over time are important to appreciate. Degenerative disk space narrowing, facet disease, and stenosis tend to slowly progress over time. Eventual stabilization of the three-joint discovertebral complex is thought to be part of the natural history of degenerative disease, and it is assumed to be accompanied by a decrease in pain. These impressions,

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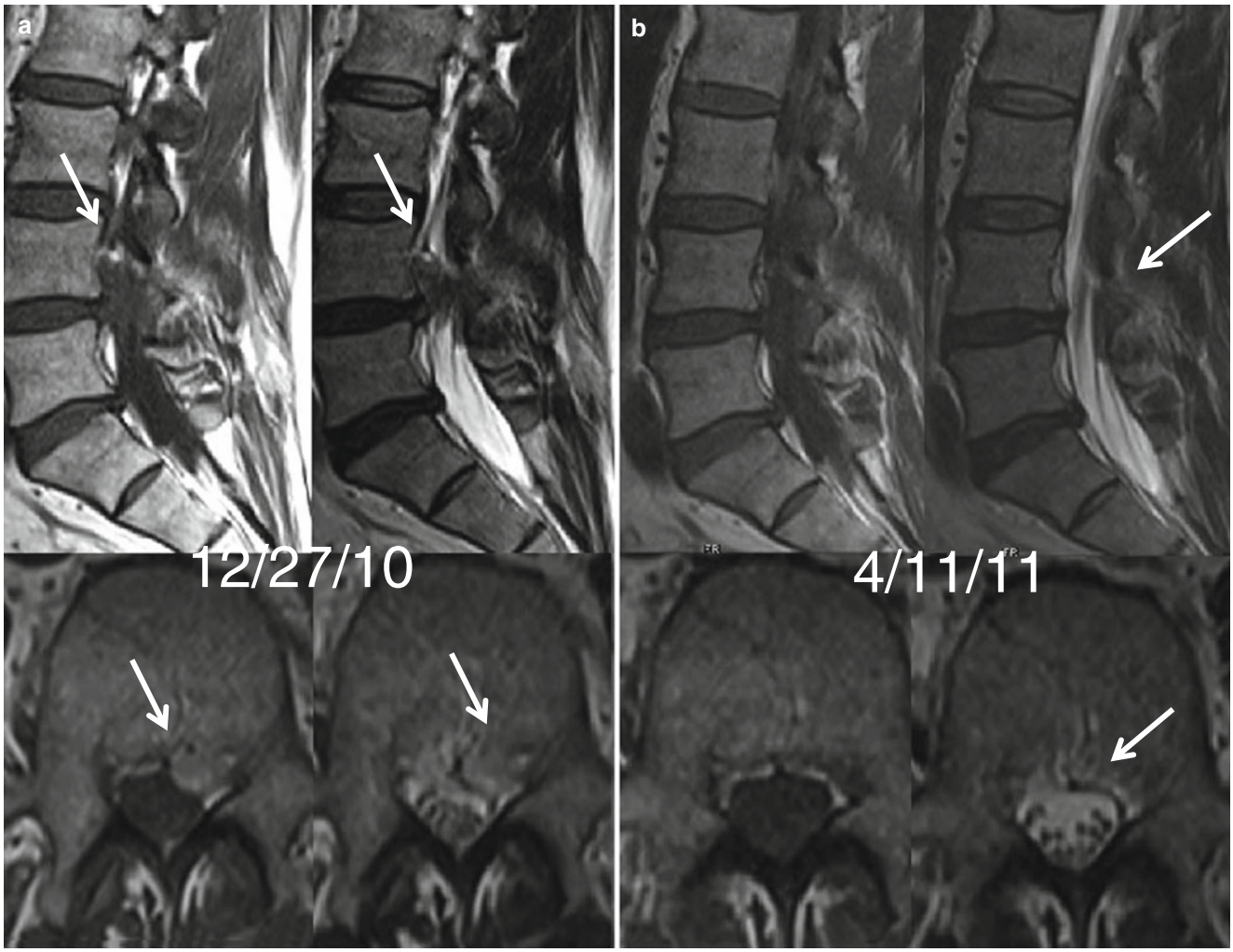

Fig. 1 46-year-old male with left leg radicular symptoms. (**a**) is a composite of sagittal and axial T1 and T2 images of the lumbar spine. (**a**) is from the initial MR performed on 27 December 2010. This study demonstrates a large disk extrusion (*arrows*) in the left anterior epidural space at the L4/L5 level. (**b**) is a composite of the follow-up MR performed 12 weeks later and demonstrates complete resolution of the previously described disk extrusion (*arrows*)

however, are anecdotal and have not been tested by a formal natural history study. Some findings, such as disk herniation and degenerative marrow changes, are known to change. Multiple studies in which computed tomography or MR imaging has been used have shown that the size of disk herniations, especially larger ones, can reduce dramatically in patients undergoing conservative treatment [9, 10].

The prognostic value of these findings is important in computing this information's effect on therapeutic thinking. In a study of symptomatic patients, the prevalence of disk herniation in patients with low back pain and those with radiculopathy at presentation was similar [11]. There was a higher prevalence of herniation, 57 % in patients with low back pain and 65 % in patients with radiculopathy, than the 20–28 % prevalence reported in asymptomatic series [6, 7]. In general, one-third of patients with disk herniation at presentation had significant resolution or disappearance by 6 weeks and two-thirds by 6 months (Fig. 1) [10, 11]. The type, size, and location of herniation at presentation and changes in herniation size and type over time did not correlate with outcome. Knowledge of imaging findings did not affect outcome or impact treatment. In a similar study, by Gilbert et al., earlier imaging did not affect conservative management. A systematic review and meta-analysis by Chou [12] showed that routine lumbar spine imaging in patients with low back pain and no features suggesting serious underlying conditions did not improve clinical outcomes compared with usual clinical care without immediate imaging. The reason these considerations are important is that the rates of spinal surgery are increasing, and there is a moderate to strong correlation between changes in the rates between CT and MR use and spine surgery [13]. This lack of prognostic value also appears to apply to the conservative management of spinal stenosis. There do not appear to be reliable

prognostic imaging findings that would correlate with surgical success or even whether patients would benefit from surgery and spinal stenosis [14, 15].

Interestingly, one imaging variable that did have positive predictive value was the presence of disk herniation at presentation. Patients who presented with a disk herniation were three times more likely to do well than those without a discernible disk herniation [11]. The reason for this is thought to be related to the favorable natural history of patients with disk herniations. That is, the overwhelming majority of these patients recover without significant intervention, and in fact we know from the morphologic data that the majority of these disk herniations regress or disappear over time. Therefore, the presence of a herniation is actually a good sign, that is, likely to have a more favorable natural history.

Intervertebral Disk

Intervertebral disk pathology is thought to be one of the causative factors of low back pain [16]. Studies that demonstrate innervation to the intervertebral disk provide evidence that may account for instances of discogenic low back pain [17]. It was revealed that innervation of the inner disk was observed only in painful disks, not in normal control disks [18, 19]. Based on these observations, nerve ingrowth into the inner disk may be a cause of nonspecific discogenic low back pain. MR imaging findings that correlate with painful disks on discography are those typical for disk degeneration, mainly signal loss of the disk on T2–WI, but also loss of disk height, the presence of a hyperintensity zone (HIZ), and modic changes [20].

The hyperintensity zone (HIZ) is a localized region of high signal intensity on T2–WI within the annulus fibrosus. Histopathologically these lesions represent replacement of the normal lamellar structure by a disorganized, vascularized granulation tissue consisting of small round cells, fibroblasts, and newly formed blood vessels around tears that extend from the nucleus pulposus to the outer region of the annulus fibrosus [21]. Originally the presence of an HIZ was strongly correlated with a painful disk on discography [22]. This correlation was confirmed in multiple later studies, but was also questioned in a few other studies. In general, the association between an annular tear on MR images and low back pain is unclear.

Bone Marrow Changes

Signal intensity changes of the vertebral body marrow adjacent to the end plates of degenerated disks are a long recognized and common observation on MR images of the lumbar spine [23, 24]. However, despite a growing body of literature on this subject, their clinical importance, etiology, and relationship to symptoms remain unclear [25]. These marrow changes appear to take three main forms on MR imaging. Type I changes demonstrate decreased signal intensity on TI-weighted images and increased signal intensity on T2-weighted images. They have been identified in approximately 4 % of patients scanned for lumbar disease [17], approximately 8 % of patients after diskectomy [26], and in 40–50 % of chymopapain-treated disks, which may be viewed as a model of acute disk degeneration [27]. Histopathologic sections of disks with type I changes show disruption and fissuring of the end plate and vascularized fibrous tissues within the adjacent marrow, prolonging T1 and T2. Enhancement of type I vertebral body marrow changes is seen with administration of gadolinium that at times extends to involve the disk itself and is presumably related to the vascularized fibrous tissue within the adjacent marrow. Type II changes are represented by increased signal intensity on T1-weighted images and isointense or slightly hyperintense signal on T2-weighted images. They have been identified in approximately 16 % of patients at MR imaging. Disks with type II changes also show evidence of end plate disruption, with yellow (lipid) marrow replacement in the adjacent vertebral body resulting in a shorter T1. Type III changes are represented by decreased signal intensity on both T1- and T2-weighted images and correlate with extensive bony sclerosis on plain radiographs. The lack of signal in the type III change no doubt reflects the relative absence of marrow in areas of advanced sclerosis. Unlike type III, types I and II changes show no definite correlation with sclerosis at radiography [28].

This is not surprising when one considers the histology; the sclerosis seen on plain radiographs is a reflection of dense woven bone within the vertebral body, whereas the MR changes are more a reflection of the intervening marrow elements. While the aforementioned histologic changes appear to describe the underlying anatomic substrate for the MR signal changes, they by no means describe the etiology of the underlying causative process. The marrow changes are likely epiphenomena and are a consequence of the biomechanical, cellular, and immunological factors that are primarily responsible for symptomatology.

Similar marrow changes have also been noted in the pedicles. While originally described as being associated with spondylolysis, they have also been noted in patients with degenerative facet disease and pedicle fractures [29, 30]. We do not know the exact mechanism by which these marrow changes occur. Their association with degenerative disk disease, facet changes, and pars and pedicle fractures suggests they are a response to biomechanical stress. This then suggests the first and likely most common etiology – mechanical.

Of these three types, type I changes appear to be more fluid and variable, a reflection of some ongoing underlying

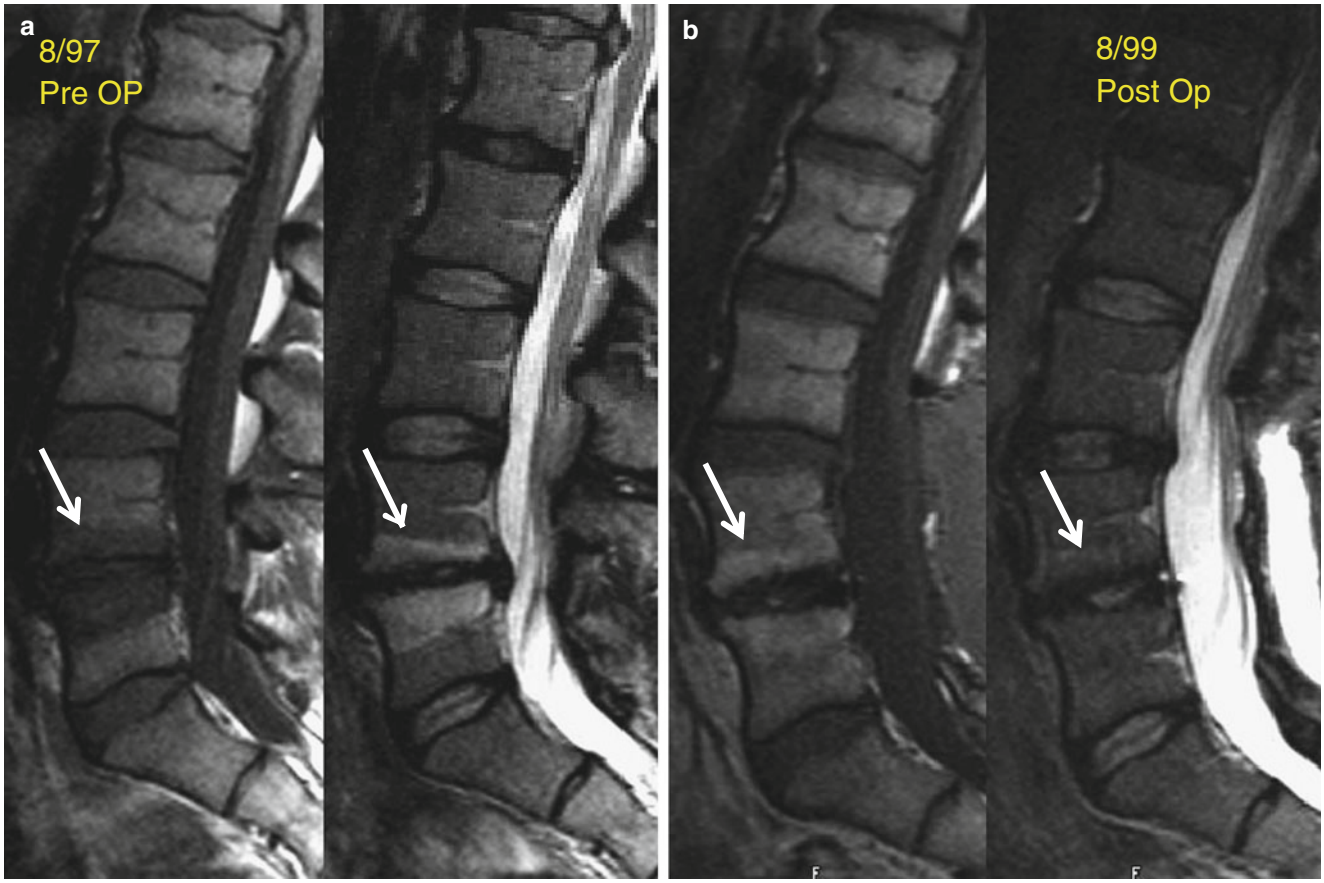

Fig. 2 (**a**) is a composite of sagittal T1- and T2-weighted images in a patient with severe low back pain. (**a**) (*arrows*) demonstrates degenerative type I marrow changes at the L4/L5 level with decreased signal intensity on T1 and increased signal intensity on T2 of the adjacent vertebral body margins. (**b**) is a composite of sagittal T1- and T2-weighted images obtained 2 years later. The patient had undergone a posterior lateral fusion in the interim. Note the laminectomy defect posteriorly. The type I degenerative marrow changes at L4/L5 have now converted to type 2 marrow changes with increased signal intensity on the T1 and normal signal intensity on T2 (*arrows*)

pathological process such as continuing degeneration with resulting changing biomechanical stresses. Of the three types, type I is most often associated with ongoing low back symptomatology [31–35]. In most cases, type II degenerative changes appear to be associated with a more stable state. Type II changes, however, are not always permanent and conversion between type II and I has been demonstrated. In general, when type II marrow changes convert to type I, there is usually a superimposed process such as continued or accelerated degeneration or vertebral osteomyelitis.

Some authors have suggested that mixed lesions are more common than originally thought and indicative of overlap and progression of one type to another [26, 36, 37]. In most studies of marrow changes, type II is the most prevalent and the prevalence increases with age [26].

The available data would support type I marrow changes are more strongly associated with symptomatology than type II and more fluid, and their resolution or change is more common and associated with clinical improvement. The greatest support for suggesting these marrow changes, particularly type I, is related to biomechanical instability which is based on observations following fusion (Fig. 2). Chataigner [38] has suggested that type I marrow changes have much better outcomes with surgery than those with isolated degenerative disk disease and normal or type II marrow changes. In addition, resolution of type I marrow changes to either normal or type II was associated with higher fusion rates and better outcomes. As further support for these fluid marrow changes reflecting biomechanical stress, we have seen similar marrow conversion in the pedicles of vertebral bodies associated with symptomatic pars and pedicle fractures as well as severe degenerative facet joint disease (Fig. 3). Self-reported pain scores tended to improve over time with concordant resolution of marrow signal intensity.

While the data is strong that there is a mechanical etiology to many of these marrow changes, there is a growing body of literature that suggests that in some there is a true infectious or inflammatory cause [39]. In patients with the low back pain and type I marrow changes, an important differential consideration is vertebral osteomyelitis. While

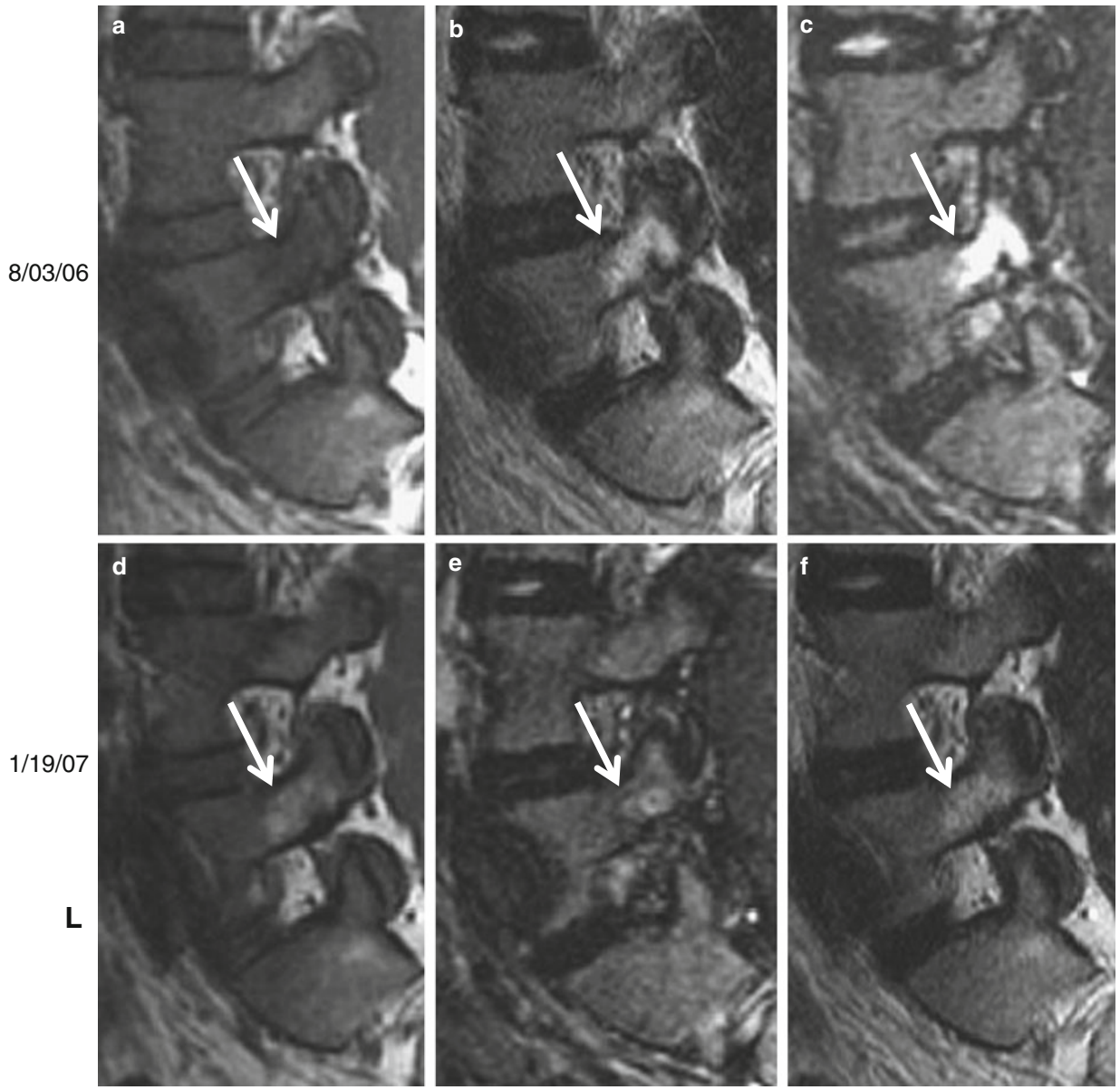

Fig. 3 (**a**–**c**) Left parasagittal T1, T2 and STIR weighted sequences obtained at the time of presentation with back pain. (**d**–**f**) are parasagittal T1-, T2-, and STIR-weighted images obtained 14 weeks after the initial study. Note the conversion of the type I marrow signal intensity change on the previous examination to a type 2 marrow signal. The decreased signal intensity on T1 has converted to an increased, more lipid marrow signal. The high signal intensity on T2 and STIR has for the most part resolved. The *arrows* denote the pedicles

classic pyogenic and fungal osteomyelitis may, in their earliest stages, overlap in appearance on MR with type I marrow degenerative changes, classic osteomyelitis has a distinctly different clinical and more rapidly changing imaging picture. More recently, it has been proposed that some type I marrow changes which heretofore have been presumed to be degenerative may in fact be secondary to a low virulent anaerobic bacterial process [40]. The authors hypothesize that the marrow changes are a side effect of the cytokine propionic acid production from the bacteria entering the adjacent marrow space, presumably through degenerative changes related to disk herniation and underlying degenerative disk disease.

Degenerative Facet Disease

The zygapophysial joint aka "facet" joints in the spinal column is located posterior to the vertebral body. Each vertebra has two facet joints. They are surrounded with a fibrous capsule and connect the superior and inferior articular facets of the vertebrae. Unlike the intervertebral disk, they are true

synovial joints. The joint produces synovial fluid, the prime lubricant for the joint and the nutritional source for the joint surface cartilage. Facet joints are an important part of the posterior column and provide structural stability to the vertebral column. The posterior ligamentous complex (facet joint capsule, ligamentum flavum, interspinous ligament, and supraspinous ligament) keeps the facet joints and the vertebrae in a fixed position with each other. Injury of this complex can result in subluxation or dislocation of the facet.

Most literature focuses on the intervertebral disks; however it is increasingly apparent that facet joints also play a major role in low back pain. Degenerative facet disease is the most frequent form of facet pathology, but degenerative disk and degenerative facet disease often go along [41]. Like in all synovial lined joints, arthrosis in facet joints is a continuum between loss of joint space narrowing, loss of synovial fluid, and cartilage and bony overgrowth. High-grade cartilage necrosis arises quite rapidly in facets. It is mainly a disease affecting the elderly population, present in virtually everyone after the each of 60 and in varying degrees affecting the majority of adults. No sex difference is noted. It is probably related to mechanical loading, minor repetitive trauma, and/or a form of predisposition [42]. The L4–L5 facet joints are more prone to degeneration than any other level, because of their more horizontal position in the sagittal plane. Facet joint osteoarthritis is intimately linked to the distinct but functionally related condition of degenerative disk disease and disk degeneration usually proceeds facet joint osteoarthritis [41].

Diagnosing pain as deriving from the facet joints can be challenging. History and physical examination may suggest, but cannot confirm, the facet joint as the source of pain [43]. Although radiologists are commonly asked by clinicians to determine the degree of facet joint osteoarthritis, the published radiological studies report no correlation between the clinical symptoms of low back pain and degenerative spinal changes observed on radiological imaging studies [44]. Specifically, the association between degenerative changes in the lumbar facet joints and symptomatic low back pain remains unclear and is a subject of ongoing debate. Current standard criteria for the diagnosis of facet joint pain are reduction in symptoms following the direct introduction of local anesthetic into the facet joint or block of local innervation [45]. The procedure is considered diagnostic if there is pain relief of more than 50 %.

In imaging studies more and more the emphasis lies on the visualization of inflammation of the facet joint and the surrounding soft tissues. It is believed that this inflammation is the cause of local, i.e., non-irradiating pain. Not all changes are inflammatory, especially bony overgrowth is a protective reaction to inflammation, diminishing inflammatory response. However bony overgrowth can be an important cause of neuroforaminal narrowing, giving rise to irradiating pain.

Table 1 Meyerding classification for spondylolisthesis

1	<25 % displacement of vertebral body
2	25–50 % displacement of vertebral body
3	50–75 % displacement of vertebral body
4	>75 % displacement of vertebral body
5	Spondyloptosis (vertebral body displaced completely anteriorly, with inferior displacement to level of vertebral body below)

Adult degenerative scoliosis (spinal deformity or curvature in the coronal plane) and degenerative spondylolisthesis (displacement of one vertebra relative to another in the sagittal plane) are also thought to be related to facet joint degeneration and failure of the motion segment. In degenerative scoliosis, asymmetric deformity and asymmetric loading lead to asymmetric degeneration, which in turn leads to more scoliotic deformity and further increased force transmission through the facet joint on the concave side of the curve. In degenerative spondylolisthesis, progressive loss of cartilage and articular remodeling lead to subluxation of the facet joint. Facet joints at spinal levels affected by degenerative spondylolisthesis have been found to be more sagittally oriented than those at levels without spondylolisthesis. Spondylolisthesis most often occurs at L4–L5, the same level that is most often affected by arthrosis [34].

Plain radiographs are of only limited use in investigating chronic back pain. Arthrosis of the facet joints is a frequent radiographic finding, particularly among the elderly. Oblique radiographs are the best projections to demonstrate the facet joints of the lower lumbar spine because of the oblique position and curved configuration of the facet joints. Even on oblique views, however, only the portion of each joint that is oriented parallel to the X-ray beam is clearly visible.

Typical findings in facet joint degeneration on plain radiographs include joint space narrowing, sclerosis, bone hypertrophy, and osteophytes. Intra-articular gas ("vacuum phenomenon") may be present and spondylolisthesis is not uncommon. Conventional radiography is insensitive in the detection of mild facet joint disease and becomes slightly more sensitive for detecting severe disease. The degree of degeneration tends to be underestimated. The literature reports a 55 % sensitivity and 69 % specificity in identifying the presence of degenerative change in the L3–4 and L5–S1 facet joints on plain radiography [46]. Therefore, standard radiographs can best be used for screening for facet joint osteoarthritis and grading spondylolisthesis according to the Meyerding classification (table 1) [47]. It is particularly useful for evaluating motion-related abnormalities in flexion or extension. This can be very important for assessing instability in case of spondylolisthesis. As mentioned before, the clinical relevance of detecting osteoarthritis of the facet joints remains unclear and controversial [39, 48]. They also have little value in being able to predict response to facet joint interventions.

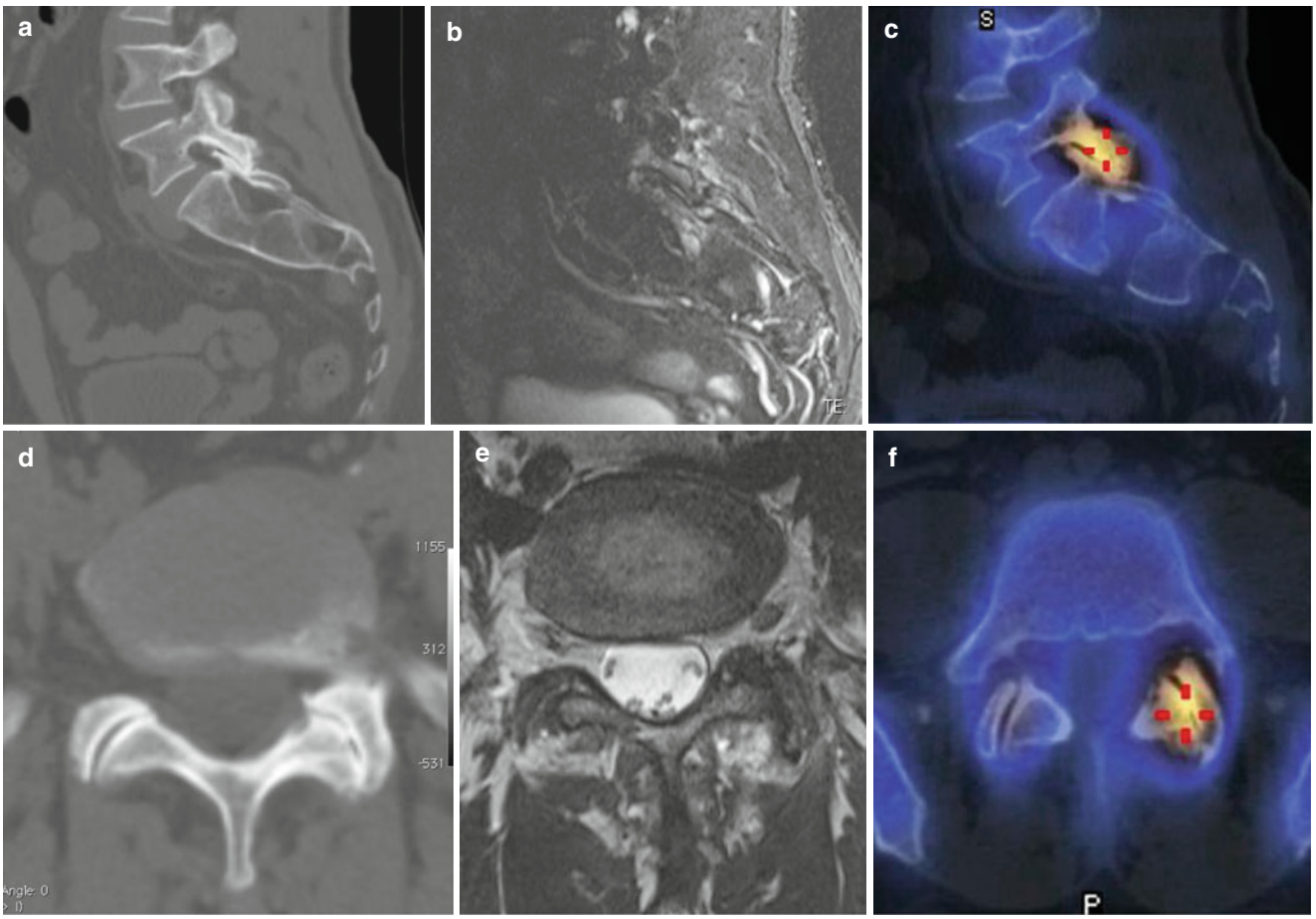

Fig. 4 Grade 3 facet degeneration (see also Table 2) and grade 4 facet joint synovitis (see also Table 3). Note the good correspondence of severe degenerative changes on CT (**a**, **d**) with narrowing of the joint, large osteophytes, severe hypertrophy of the articular process, and severe subarticular bone erosions and subchondral cysts, with inflammatory changes on STIR T2-weighted MRI (**b**) with extensive bone edema, which is not visible on regular T2-weighted imaging (**e**). Same facet joint shows marked increased uptake on SPECT (**c**, **f**). The *red cross* denotes the center of SPECT activity

In comparison with plain radiographs, CT is better in delineating the facet joints due to its capability to image the joint in multiple planes and the high contrast between bony structures and the surrounding soft tissue. Therefore, CT has the ability to detect degenerative changes in the facet joints earlier than plain radiographs. On CT scan we can see articular joint space narrowing with subchondral sclerosis and erosions, osseous overgrowth, and/or hypertrophy of the ligamentum flavum, causing impingement of the foramina (Fig. 4). Also secondary signs including intra-articular gas, joint effusion, and spondylolisthesis can be detected. Synovial cysts can arise, extending posterior of the facet joint but also anterior in the spinal canal or neuroforamen. Joint traction during subluxation may produce intra-articular gas (vacuum). These abnormalities associated with arthrosis can be categorized by CT [49]. Four grades of osteoarthritis of the facet joints were defined by Weishaupt, adapting the criteria published by Pathria (grade 0, normal; grade 1, mild degenerative disease; grade 2, moderate degenerative disease; and grade 3, severe degenerative disease) (table 2) [39, 41]. This grading system aids objective assessment of disease severity and progression. On the other hand, CT has a poor differentiation of soft tissues within the spine, and it is not that good in demonstrating cartilage abnormalities which may indicate early facet degeneration. In the presence of an MR examination, CT is not required for the assessment of facet joint degeneration due to relative good interobserver agreement [41]. But once again, abnormal morphology may not necessarily reflect underlying pathology.

Table 2 Grade criteria for facet degeneration (Pathria, adapted by Weishaupt)

0	Normal facet joint space (2±4 mm width)
1	Narrowing of the facet joint space (<2 mm) and/or small osteophytes and/or mild hypertrophy of the articular process
2	Narrowing of the facet joint space and/or moderate osteophytes and/or moderate hypertrophy of the articular process and/or mild subarticular bone erosions
3	Narrowing of the facet joint space and/or large osteophytes and/or severe hypertrophy of the articular process and/or severe subarticular bone erosions and/or subchondral cysts

Table 3 Grade criteria for facet joint synovitis

0	No signal abnormality
1	Signal abnormality confined to joint capsule
2	Periarticular signal abnormality involving less than 50 % of the perimeter of the joint[a]
3	Periarticular signal abnormality involving more than 50 % of the perimeter of the joint[a]
4	Grade 3 with extension of signal abnormality into the intervertebral foramen, ligamentum flavum, pedicle, transverse process, or vertebral body

[a]Signal abnormality may extend into the articular pillar or lamina, but does not contribute to the definition of the grade

Magnetic resonance imaging is a noninvasive investigation that is not associated with exposure to ionizing radiation. MRI is the preferred imaging technique for the diagnosis of most spinal diseases as it has a superior delineation of soft tissues compared to other imaging modalities. T2-weighted sequences are useful in identifying fluid in facet joint effusions, periarticular cysts, and also better delineate cartilage defects. As mentioned before CT and MR are consistent in demonstrating morphologic aberrances of the facet joint, but MRI is better to demonstrate compression of the thecal sac and the fat-filled neuroforamen, compressing the nerve roots. However, MRI is less sensitive for evaluating cortical anatomy, calcified structures, and subchondral sclerosis [41, 42]. The role of MR imaging in the evaluation of facet joint degeneration, however, is not that clear. Osteoarthritis of these joints may be demonstrated in patients who present with back pain with or without pain irradiating into the legs [50], but is also a frequent observation in a large percentage of asymptomatic patients. Moreover facet joint arthropathy defined anatomically on MRI and CT does not seem to be a significant predictor for the outcome of patients undergoing facet joint blocks [51]. Recent studies suggest that the facet joint (unlike the intervertebral disk) is perhaps better examined in the context of the scientific literature on other synovial joints. Normal facet joints with intact capsules may hold between 1 and 2 ml of fluid. A larger effusion may indicate a loss of capsular function with subsequent abnormal facet joint motion. A positive correlation is found between the amount of facet joint fluid present and the degree of lumbar instability [52]. Chronic degenerative processes in facet joints involve active synovial inflammation, which can be detected using MRI with a fat-saturation technique. Facet synovitis can be graded, using a grading system (Table 3). Facet synovitis appears to correlate with the patient's pain [53]. Moreover synovial abnormalities seem to correlate with SPECT findings [54] (Fig. 4).

The detection of inflammation in the facet joint may be more useful than imaging of joint morphology. Radionuclide bone scintigraphy can depict bone areas with increased osteoblastic activity, and it can depict synovial changes caused by inflammation or hyperemia. Bone scintigraphy also can depict degenerative changes, particularly those that demonstrate a high degree of remodeling. The induced radiopharmaceutical uptake can vary from subtle to pronounced, depending on the metabolic activity and size of the lesions. Osteophytes that are in the process of growing exhibit a high uptake, whereas mature osteophytes tend to have a normal or slightly increased uptake. Abnormalities can be detected earlier with bone scintigraphy than they can be with radiographic methods, and joints observed as abnormal at scintigraphy eventually show the most progressive radiographic changes. Joints that are radiographically abnormal but normal at bone scintigraphy do not show additional deterioration.

Anatomic co-localization with computed tomography (SPECT/CT) is important because facet joints are anatomically juxtaposed, the number of vertebral bodies is variable, and transitional lumbosacral vertebral bodies are present in 4–30 % of patients (Fig. 4).

Several studies show that strictly targeting facet joints with increased 99mTc MDP activity instead of using clinical localization for percutaneous treatment is predictive of a positive response and that use of bone scans can decrease the number of treated facet joints [55–57].

Thus SPECT/CT is emerging as an ideal modality for imaging the facet joint due to the detail of information it provides, the ability to accurately localize the site of pain, and the possibility to differentiate pars defects or other degenerative changes from facet joint disease. However, its use as an appropriate imaging modality should be considered carefully given the increased radiation dose in young individuals with the benign disease and altered low-dose CT protocols should be considered.

Radicular Pain

Acute lumbar disk herniations are the most common cause of acute radicular leg pain. After excluding emergent causes, such as cauda equina syndrome, epidural abscess, fracture, or malignancy, a 6-week trial of conservative management is indicated [58]. Patients should be advised to stay active. If symptoms persist after 6 weeks, or if there is worsening neurologic function, imaging and invasive procedures may be considered. Most patients with lumbar disk herniations improve over 6 weeks.

If a disk herniation is identified that correlates with physical findings, surgical discectomy may improve symptoms more quickly than continued conservative management. Epidural steroid injections can also provide short-term relief [58].

Herniated disks are more easily detected with MRI than with CT for a number of reasons. Firstly, MR imaging allows visualization of the complete lumbar (or cervical or thoracic)

spine in one examination. Secondly, sagittal images also depict the spinal canal in between intervertebral disk spaces. It is not unusual for a disk fragment to migrate (or extend) into the area behind the vertebral body. Some of these migrated disks can be missed on CT if axial slices are limited to the intervertebral disk spaces examined. Finally, the intrinsic tissue contrast is usually better on MR. Especially the lumbosacral region can be hard to assess on CT due to beam hardening, especially in larger patients.

Chronic radicular pain can be caused by a disk herniation, but also vertebral osteophytic spurs, degenerative osteophytic facet spurs and facet hypertrophy, and degenerative foraminal stenosis are an important cause of nerve root irritation. Foraminal nerve root entrapment is best visualized on T1-weighted MRI where the high contrast between fat tissue and the nerve root sheath is of great help. Usually a combination of hypertrophic degenerative facets with osteophytic spurs posteriorly, and vertebral osteophytes and/or disk herniation anteriorly, diminishes the anteroposterior diameter of the foramen. Foraminal height is lessened by degenerative disk disease and subsequent disk height loss. Whenever the normal rounded (oval) appearance of the nerve root sheath is lost in combination with loss of the surrounding fat tissue, nerve root compression should be considered.

References

1. Murray CJL, Vos T, Lozano R et al (2012) Disability-adjusted life years (DALYs) for 291 diseases and injuries in 21 regions, 1990–2010: a systematic analysis for the Global Burden of Disease Study 2010. Lancet 380:2197–2223
2. Fardon DF et al (2014) Lumbar disc nomenclature: version 2.0: Recommendations of the combined task forces of the North American Spine Society, The American Society of Spine Radiology and the American Society of Neuroradiology. Spine J 14(11):2525–2545
3. Lurie Jon D MD, MS (2008) Reliability of Magnetic Resonance Imaging Readings for Lumbar Disc Herniation in the Spine Patient Outcomes Research Trial (SPORT). Spine 33:991–998
4. Carrino JA, Lurie JD, Tosteson AN, Tosteson TD, Carragee EJ, Kaiser J, Grove MR, Blood E, Pearson LH, Weinstein JN, Herzog R (2009) Lumbar spine: reliability of MR imaging findings. Radiology 250(1):161–170
5. Wiesel SW, Tsourmas N, Feffer HL, Citrin CM, Patronas N (1984) A study of computer-assisted tomography. I. The incidence of positive CAT scans in an asymptomatic group of patients. Spine 9:549–551
6. Boden SD, Davis DO, Dina TS, Patronas NJ, Wiesel SW (1990) Abnormal magnetic-resonance scans of the lumbar spine in asymptomatic subjects: a prospective investigation. J Bone Joint Surg Am 72:403–408
7. Jensen MC, Brant-Zawadzki MN, Obuchowski N, Modic MT, Malkasian D, Ross JS (1994) Magnetic resonance imaging of the lumbar spine in people without back pain. N Engl J Med 331:69–73
8. Borenstein DG, O'Mara JW Jr, Boden SD et al (2001) The value of magnetic resonance imaging of the lumbar spine to predict low back pain in asymptomatic subjects: a 7-year follow-up study. J Bone Joint Surg Am 83-A:1306–1311
9. Saal JA, Saal JS, Herzog RJ (1990) The natural history of lumbar intervertebral disc extrusions treated nonoperatively. Spine 15: 683–686
10. Modic MT, Ross JS, Obuchowski NA, Browning KH, Cianflocco AJ, Mazanec DJ (1995) Contrast-enhanced MR imaging in acute lumbar radiculopathy: a pilot study of the natural history. Radiology 195:429–435
11. Modic MT, Obuchowski NA, Ross JS et al (2005) Acute low back pain and radiculopathy. Radiology 237:597–604
12. Chou R, Fu R, Carrino JA, Deyo RA (2009) Imaging strategies for low-back pain: systematic review and meta-analysis. Lancet 373(9662):463–472
13. Verrilli D, Welch HG (1996) The impact of diagnostic testing on therapeutic interventions. JAMA 275(15):1189–1191
14. ECRI. Treatment of degenerative lumbar spinal stenosis. I. Evidence report. Agency for Healthcare Research and Quality publication no. 01-E048 #32. Plymouth Meeting, Pa: ECRI, 2001
15. Benoist M (2002) The natural history of lumbar degenerative spinal stenosis. Joint Bone Spine 69(5):450–457
16. Takahashi K, Aoki Y, Ohtori S (2008) Resolving discogenic pain. Eur Spine J 17(Suppl 4):428–31
17. Troyanovich SJ, Harrison DD, Harrison DE (1999) Low back pain and the lumbar intervertebral disk: clinical considerations for the doctor of chiropractic. J Manipulative Physiol Ther 22(2):96–104
18. Coppes MH, Marani E, Thomeer RT, Oudega M, Groen GJ (1990) Innervation of annulus fibrosus in low back pain. Lancet 336: 189–190
19. Freemont AJ, Peacock TE, Goupille P, Hoyland JA, O'Brien J, Jayson MIV (1997) Nerve ingrowth into diseased intervertebral disc in chronic back pain. Lancet 350:178–181
20. O'Neill C, Kurgansky M, Kaiser J, Lau W (2008) Accuracy of MRI for diagnosis of discogenic pain. Pain Physician 11(3):311–326
21. Peng B, Hou S, Wu W, Zhang C, Yang Y (2006) The pathogenesis and clinical significance of a high-intensity zone (HIZ) of lumbar intervertebral disc on MR imaging in the patient with discogenic low back pain. Eur Spine J 15(5):583–587
22. Aprill C, Bogduk N (1992) High-intensity zone: a diagnostic sign of painful lumbar disc on magnetic resonance imaging. Br J Radiol 65:361–369
23. DeRoss A, Kressel H, Spritzer C et al (1987) MR imaging of marrow changes adjacent to end plates in degenerative lumbar disc disease. AJR Am J Roentgenol 149:531–534
24. Modic MT, Steinbert PM, Ross JS et al (1988) Degenerative disc disease; assessment of changes in vertebral body marrow with MR imaging. Radiology 166:193–199
25. Rahme R, Moussa R (2008) The modic vertebral endplate and marrow changes: pathologic significance and relation to low back pain and segmental instability of the lumbar spine. AJNR 29:838–842
26. Ross JS, Obuchowski N, Zepp R (1998) The postoperative lumbar spine: evaluation of epidural scar over a 1-year period. AJNR Am J Neuroradiol 19:183–186
27. Masaryk TJ, Boumphrey F, Modic MT, Tamborrello C, Ross JS, Brown MD (1986) Effects of chemonucleolysis demonstrated by MR imaging. J Comput Assist Tomogr 10:917–923
28. Modic MT, Masary TJ, Ross JS et al (1988) Imaging of degenerative disk disease. Radiology 168:177–186
29. Ulmer JL, Elster AD, Mathews VP, Allen AM (1995) Lumbar spondylolysis: reactive marrow changes seen in adjacent pedicles on MR images. AJR Am J Roentgenol 164:429–433
30. Morrison JL, Kaplan PA, Dussault RG, Anderson MW (2000) Pedicle marrow signal intensity changes in the lumbar spine: a manifestation of facet degenerative joint disease. Skelet Radiol 29:703–707, 2002;69:450–457
31. Toyone T, Takahashi K, Kitahara H et al (1994) Vertebral bone marrow changes in degenerative lumbar disc disease: an MRI study of 74 patients with low back pain. J Bone Joint Surg (Br) 76:757–764

32. Mitra D, Cassar-Pullicino VN, McCall IW (2004) Longitudinal study of vertebral type-1 end-plate changes on MR of the lumbar spine. Eur Radiol 14:1574–1581
33. Kuisma M et al (2006) A three-year follow up of lumbar spine endplate (Modic) changes. Spine 31(15):1714–1718
34. Albert HB, Manniche C (2007) Modic changes following lumbar disc herniation. Eur Spine J 16:977–982
35. Modic MT (2007) Modic type I and type 2 changes. J Neurosurg Spine 6:150–151
36. Karchevsky M, Schweitzer ME, Carrino JA et al (2005) Reactive endplate marrow changes: a systematic morphologic and epidemiologic evaluation. Skelet Radiol 34:125–129
37. Boos N, Weissbach S, Rohrbach H et al (2002) Classification of age-related changes in lumbar intervertebral discs: 2002 Volvo Award in basic science. Spine 27:2631–2644
38. Chataigner H, Onimus M, Polette A (1998) Surgery for degenerative lumbar disc disease: should the black disc be grafted (in French)? Rev Chir Orthop Reparatrice Appar Mot 84:583–589
39. Albert H et al (2008) Modic changes, possible causes and relation to low back pain. Med Hypotheses 70:361–368
40. Albert H et al (2013) Antibiotic treatment in patients with chronic low back pain and vertebral bone edema (Modic type 1 changes): a double-blind randomized controlled trial of efficacy. Eur Spine J 22:697–707
41. Fujiwara A, Tamai K, Yamato M, An HS, Yoshida H, Saotome K et al (1999) The relationship between facet joint osteoarthritis and disc degeneration of the lumbar spine: an MRI study. Eur Spine J 8:396–401
42. Gellhorn AC, Katz JN, Suri P (2013) Osteoarthritis of the spine: the facet joints. Rheumatology 9(4):216–224
43. Hancock MJ, Maher CG, Latimer J, Spindler MF, McAuley JH, Laslett M, Bogduk N (2007) Systematic review of tests to identify the disc, SIJ or facet joint as the source of low back pain. Eur Spine J 16:1539–1550
44. Schwarzer AC, Wang SC, O'Driscoll D, Harrington T, Bogduk N, Laurent R (1995) The ability of computed tomography to identify a painful zygapophysial joint in patients with chronic low back pain. Spine 20:907–912
45. Makki D, Khazim R, Zaidan AA, Ravi K, Toma T (2010) Single photon emission computerized tomography (SPECT) scan-positive facet joints and other spinal structures in a hospital-wide population with spinal pain. Spine J 10:58–62
46. Pathria M, Sartoris DJ, Resnick D (1987) Osteoarthritis of the facet joints: accuracy of oblique radiographic assessment. Radiology 164:227–230
47. Meyerding HW (1932) Spondylolisthesis. Surg Gynecol Obstet 54:371–377
48. Weishaupt D, Zanetti M, Boos N, Hodler J (1999) MR imaging and CT in osteoarthritis of the lumbar facet joints. Skeletal Radiol 28(4):215–219
49. Carrino JA et al (2008) Lumbar spine: reliability of MR imaging findings. Radiology 250:161–170
50. Resnick D, Niwayama G (1995) Degenerative diseases of the spine. In: Resnick D (ed) Diagnosis of bone and joint disorders. Saunders, Philadelphia, pp 1396–1462
51. Gorbach C, Schmid MR, Elfering A, Hodler J, Boos N (2006) Therapeutic efficacy of facet joint blocks. AJR Am J Roentgenol 186:1228–1233
52. Rihn JA, Lee JY, Khan M, Ulibarri JA, Tannoury C, Donaldson WF et al (2007) Does lumbar facet fluid detected on magnetic resonance imaging correlate with radiographic instability in patients with degenerative lumbar disease? Spine (Phila Pa 1976) 32:1555–1560
53. Czervionke LF, Fenton S (2008) Fat-saturated MR imaging in the detection of inflammatory facet arthropathy (facet synovitis) in the lumbar spine. Pain Medicin 9:400–406
54. Kim KY, Wang MY (2006) Magnetic resonance image-based morphological predictors of single photon emission computed tomography-positive facet arthropathy in patients with axial back pain. Neurosurgery 59:147–156
55. Pneumaticos SG, Chatziioannou SN, Hipp JA, Moore WH, Esses SI (2006) Low back pain: Prediction of short-term outcome of facet joint injection with bone scintigraphy. Radiology 238:693–698
56. Lehman VT, Murphy RC, Kaufmann TJ, Diehn FE, Murthy NS, Wald JT et al (2014) Frequency of discordance between facet joint activity on technetium Tc99m methylene diphosphonate SPECT/CT and selection for percutaneous treatment at a large multispecialty institution. AJNR Am J Neuroradiol 35:609–614
57. Matar HE, Navalkissoor S, Berovic M, Shetty R, Garlick N, Casey AT et al (2013) Is hybrid imaging (SPECT/CT) a useful adjunct in the management of suspected facet joints arthropathy? Int Orthop 37:865–870
58. Gregory DS, Seto CK, Wortley GC, Shugart CM (2008) Acute lumbar disk pain: navigating evaluation and treatment choices. Am Fam Physician 78(7):835–842