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Review Article: MRI of the postoperative lumbar spine

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Abstract Imaging assessment of the lumbosacral spine following surgery is complex and depends upon several factors, including the anatomy of the patient, the surgical procedure and the disease process for which it was performed, the age of the patient, the biomechanical condition of the underlying cortical and cancellous bone, intervertebral disc and musculoligamentous tissues, the time since surgery procedure and the duration and nature of the postsurgical syndrome. Depending upon these factors, one or a combination of complementary imaging modalities may be required to demonstrate any clinically relevant abnormality, to assist the surgeon in deciding if repeat surgery is necessary, its nature and at which vertebral level(s) it should be directed. This review stresses the important role of MRI following lumbar discectomy, intervertebral fusion and/or instrumentation in achieving the most beneficial and timely outcome in the patient presenting with an acute, subacute or chronic failed back surgery syndrome.

Keywords Lumbar spine · Surgery · Magnetic resonance imaging

Introduction

Low-back and radicular pain are widespread and in part adverse effects of today's lifestyles [1]. Genetic factors also play an important role in the development of back pain. Low-back and radicular pain are a leading cause of disability and cause substantial loss of productivity. Related health-care expenditure was estimated at $24 000 000 000 in the United States alone in 1990 [2]. The prevalence of low-back pain varies from 7.6% to 37% in different populations [3]. Most episodes of low-back pain are mechanical in origin and resolve within a 12-week period [4]. Recent studies, however, suggest that it may persist for longer periods in a large number of patients who eventually stop seeking medical help. The overwhelming majority of patients with low-back pain therefore probably undergo nonoperative self-treatment.

Acute episodes of back pain are associated with musculoligamentous strain or rupture and/or tears of the annulus fibrosus with or without intervertebral disc herniation and radiculopathy. The smaller proportion of individuals in whom back pain lasts more than 12 weeks may have a variety of mechanical or nonmechanical spinal disorders [5]. Low-back pain may also be associated with conditions unrelated to the spine, such as peripheral vascular, renal or gynaecological disorders.

The diagnostic and therapeutic management of low-back pain lacks consistency between individual physicians, treatment centres and regions, and clinical guidelines may help improve management and outcome [6]. Several imaging techniques are available, all of which may be helpful in the investigation of low-back pain. However, some are to be preferred, depending on several factors. Although costly diagnostic imaging should be undertaken only with a clear indication, advanced imaging studies, i.e., procedures other than conventional radiography, can play an important role in optimal selection of treatment in patients with persistent low-back pain who are suspected of having disc herniation [7]. Some investigators suggest that surgery should not be performed

unless diagnostic imaging demonstrates nerve-root compromise [8]. Others have concluded that surgical success is likely only in patients in whom symptoms, physical findings and imaging are congruent [9].

Surgery for lumbar disc herniation relieves pain in most patients, producing a good long-term outcome in almost 90% [10]. Repeat surgery, however, is less successful: only 60 to 82% of patients with recurrent disc herniation improve after surgery [11, 12, 13]. In patients who have only epidural scar tissue on serial imaging studies, the success rate of repeat surgery is as low as 17 to 38% [12, 14]. The obvious solution is to avoid where possible an initial operation which may lead to a less than satisfactory result, and thereby not create a clinical situation which necessitates repeat surgery.

MRI technique

Patient positioning

MRI of the lumbar and sacral spine is performed with the patient supine, and if possible feet first, since this diminishes feelings of claustrophobia in closed-bore magnets. We do not use a knee support, since this reduces the lumbar lordosis, which may lead to underestimation of the size and presence of disc herniation in the supine patient. Newer magnets allow for upright, sitting or even standing, lumbar spine imaging, and their first results are very promising [15].

In all examinations one should try to match the centre of the coil(s) to the centre of the region of interest, and in turn to the centre of the bore of the magnet. In addition, the patient should be positioned as parallel as possible to the long axis of the bore of the magnet, in order to minimise inadvertent oblique positioning and to reduce the distorting effects of any underlying scoliosis.

Protocols

Both sagittal and axial images are obtained in routine spine imaging. In the sagittal plane, T1- and T2-weighted images offer different, complementary information.

On T2-weighted images normal intervertebral discs are bright (i.e., give relatively high signal). With ageing and/or degeneration of the disc, water loss and collagen deposition occur, T2 relaxation time shortens and the discs gradually become darker (i.e., low-signal degenerative or 'black-disc disease'). However, in fast spin-echo (SE) acquisitions with longer echo trains (ETL), i.e., more echoes sampled after each 90° pulse, normal discs also become somewhat darker due to certain physical effects. Therefore, sagittal T2-weighted images with a relatively short ETL (< 10) are preferable to diagnose degenerative disc disease [16]. Sagittal and axial T2-weighted images are also excellent for showing the spinal cord and the nerve roots of the cauda equina. Central spinal canal stenosis and impressions on the thecal sac are most easily recognised on sagittal and axial T2-weighted images.

Sagittal SE T1-weighted images are more sensitive than conventional non-fat suppressed turbo- (fast) SE (TSE/FSE) T2-weighted images to bone marrow disease (e.g. degenerative endplate change, infection or vertebral metastases) [16], but short-tau inversion-recovery (STIR) or fat-suppressed T2-weighted images are also sensitive to many bone-marrow diseases [17]. The difference between osteophytes and soft disc material is usually better appreciated on T1-weighted images.

The normal epidural fat in the lumbar spine is very bright on T1-weighted images and contrasts well with the dural sac and the adjacent normal or pathological intervertebral disc. This is why axial T1-weighted images are preferred in the lumbar region, although axial FSE T2-weighted images without fat suppression are also sometimes acquired.

Additional axial SE T1-weighted images after intravenous gadolinium-containing contrast medium (referred to hereafter simply as contrast-enhanced images) should be obtained in patients who have undergone prior lumbar disc surgery [18, 19, 20]. One should monitor the examination to acquire these images as quickly as possible after injection; the imaging acquisition should be completed within 5 min of injection. The most important contribution of contrast medium is in differentiating scar tissue from recurrent disc herniation [18, 19], since the latter is generally accepted to be a possible indication for further surgery [21]. Assessment of the enhancement pattern of nerves, meninges, posterior spinal (zygo-apophyseal) joints and paraspinal soft tissues is important in some patients, as will be discussed. Some workers prefer to use FSE or fluid-attenuated inversion-recovery (FLAIR) T2-weighted images in addition to or instead of contrast-enhanced T1-weighted images in the differentiation of recurrent disc herniation from epidural fibrosis [22, 23].

In the lumbar spine, fat-suppression techniques can be used to assist in differentiating enhancing scar from epidural fat on contrast-enhanced T1-weighted images. However, abnormal postoperative nerve-root enhancement may be more difficult to differentiate from the normal slight pial-root enhancement usually seen on fat-suppressed images. In rare cases, fat suppression can be helpful for distinguishing between postoperative blood and normal epidural fat.

Metallic implants used for spinal fusion are not a contraindication to MRI [24]. Superparamagnetic materials such as steel, however, create severe magnetic-susceptibility artefacts. One should try to increase

bandwidth and shorten the echo time (TE) to lessen these [25]. In general, FSE sequences have less magnetic susceptibility than conventional SE sequences, which are in turn less susceptible than gradient-echo acquisitions. Metals which are not superparamagnetic, such as titanium, produce primarily radiofrequency artefacts, which are less marked [26], but may still obscure the neural foramina in the presence of pedicular screws.

Although spinal stimulators and other electronic implant devices such as cardiac pacemakers are normally a contraindication to MRI, some are MRI-compatible. In these cases the device must be switched off before entering the room containing the magnet - and switched on again upon exiting.

MRI myelography without contrast medium can be helpful in addition to standard imaging sequences. Although 3D-TSE sequences have been suggested, the acquisition time significantly adds to the overall duration of the examination. Therefore, single-shot wide-slab T2-weighted sequences with a very long TE are usually preferred. Although these give only one view of the thecal sac per acquisition, imaging time is very short, making it possible to obtain different views by running the sequence in different orientations (e.g., coronal, sagittal and two oblique views). A sequence of this type has the added advantage of eliminating postprocessing; no maximum-intensity projection processing is necessary.

MRI following discectomy/herniectomy

The normal postoperative spine

Interpretation of images of the lumbosacral spine in the immediate postoperative period, i.e., the first 6–8 postsurgical weeks, must be undertaken with caution. Normal, or at least expected, postoperative changes occur within the bones as well as the soft tissues, and vary in part depending on the type and extent of surgery and the time since the operation [27].

Surgical techniques

The most common surgical approach is a posterior midline incision which provides access to the posterior elements, spinal canal and intervertebral disc. The term *laminotomy* refers to removal of only the inferior margin of the lamina (i.e., a partial laminectomy) and is often used in cases of microdiscectomy. In *unilateral laminectomy*, the lamina on one side of the spinous process is more or less completely removed. Total or *bilateral laminectomy* involves removal of the lamina on both sides, plus the spinous process. In discectomy at the L5-S1 level, typically only the ligamentum flavum is incised and the posterior bony hemiarches are left intact. However, at L4-L5 and particularly at L3-L4, part of the hemiarch above has to be removed to obtain adequate access to the spinal canal. If the nerve root has to be accessed, the medial border of the inferior articular process of the posterior spinal joint has to be resected at L4-L5 or L3-L4. It is therefore important to know the precise radial location of the herniated disc material in advance, to preserve as much of the posterior spinal facet joints and the related posterior spinal bony elements as possible. Overzealous laminectomy or facetectomy may contribute to spinal instability and result in progressive spondylolisthesis.

Imaging findings

On MRI, the postsurgical absence of bone is sometimes difficult to assess, but can be best demonstrated on axial T1-weighted images. There is often asymmetry in the paraspinal muscle fat planes posteriorly. The margins of the paraspinal musculature may also be temporarily indistinct secondary to oedema in the subacute phase after surgery. The posterior border of the dural sac may expand posteriorly towards the surgical site and laminectomy defect, reflecting relative bony insufficiency. This is an expected, clinically irrelevant finding and does not represent a pseudomeningocele (see below).

On unenhanced images immediately after surgery, postdiscectomy changes can mimic the preoperative appearance of disc herniation in the epidural space because of disruption of the annulus fibrosus and oedema of the epidural tissues (Fig. 1). These render the outline of the dural sac and the posterior margin of the intervertebral disc margin and may efface the anterior border of the thecal sac. Homogeneous contrast enhancement of this epidural reaction may be observed (Fig. 1). The cause of these findings is granulation tissue and/or fibrosis, which explains the mild local epidural mass effect commonly seen in postoperative imaging of a clinically successful lumbar discectomy (Fig. 2).

In one study of patients asymptomatic following intervertebral disc surgery, all showed evidence of enhancing epidural fibrosis [28]. Enhancing lumbosacral vertebral endplates have been observed between 6 and 18 months after surgery in 19% of patients, and enhancement of the posterior annulus fibrosis has been reported in the majority of asymptomatic patients [29]. These are due to an aseptic reaction, although they can mimic early postoperative disc infection. Another study of asymptomatic patients showed residual or recurrent disc herniation at the operated level in 24% of patients within 6 weeks of surgery [27]. In 16% this was associated with mild to moderate mass effect on the dural sac and/or nerve roots or sleeves, and 5% had severe compression of the dural sac. In 78% of these patients with

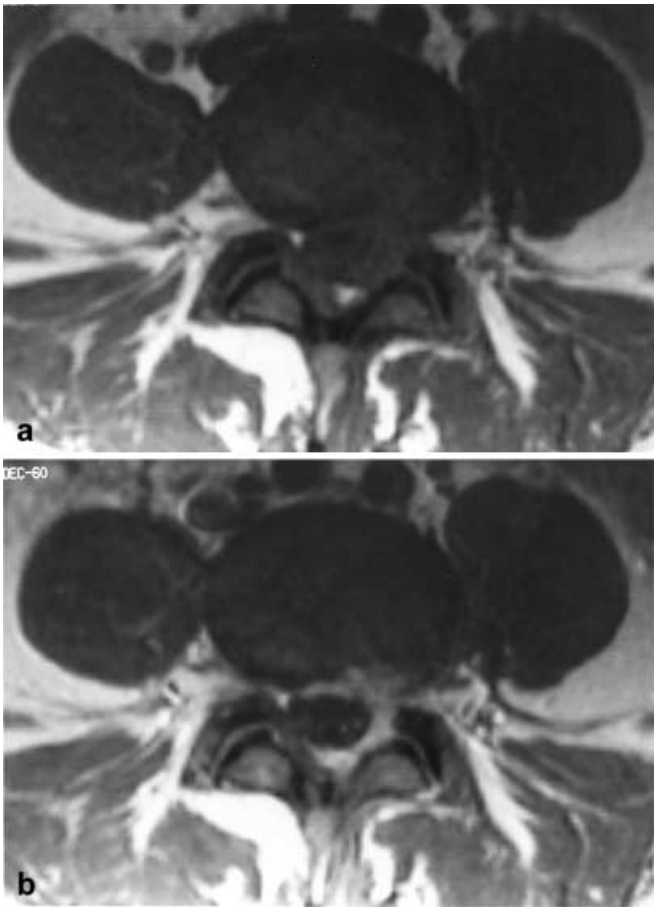


Fig. 1a, b. Normal postoperative T1-weighted images **a** before **b** after contrast enhancement. The axial T1-weighted image shows effacement of the normal high fat signal in the epidural space on the left, resembling the preoperative state. However, there is generalised enhancement in this region indicating (normal or at least expected) epidural fibrosis. Two small enhancing structures are seen within the thecal sac, representing nerve roots, indicating aseptic radiculitis. This is to be interpreted as normal or expected in the early postoperative phase, i.e., within 6 months of disc surgery

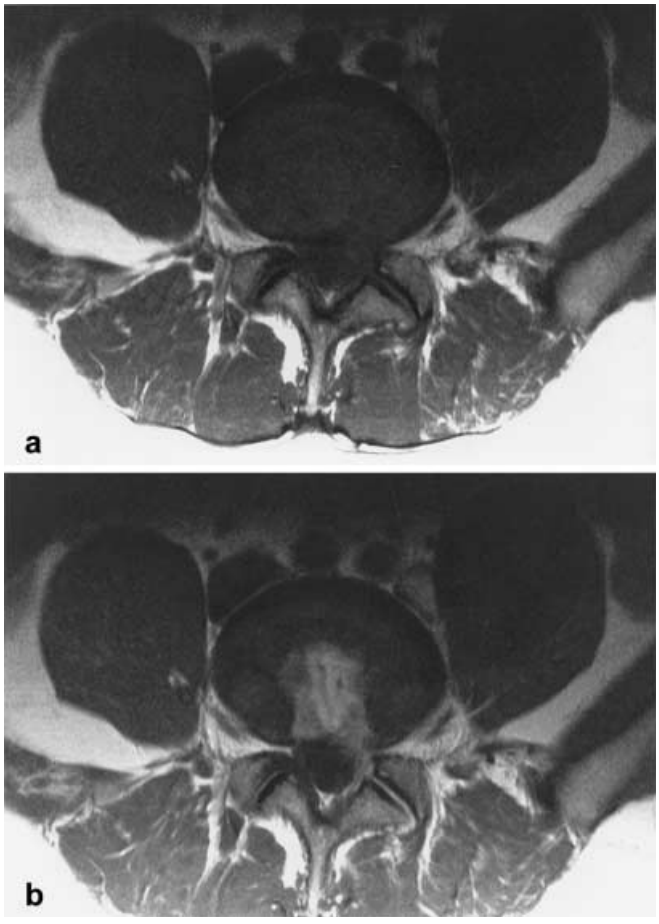


Fig. 2a, b. Normal postoperative T1-weighted images **a** before **b** after contrast enhancement. In this patient enhancement is more extensive, especially in the intervertebral disc space, than in Fig. 1. The slight deformation of the thecal sac anterolaterally on the left, is within normal limits. The enhancement in the disc space represents the surgical curettage tract; its size depends on the surgical procedure and it is normal, or expected, in this situation

residual or recurrent herniation, there was no progression in the shape of the herniated disc on imaging, nor had the mass effect resolved on MRI after 6 months as compared with 6 weeks after surgery.

The failed back surgery syndrome

Despite the relatively loose application of criteria for surgical success, lumbosacral spinal surgery has been so often unsuccessful in the past (in 10–40% of cases) that failed back surgery is now regarded as a syndrome: the failed back surgery syndrome (FBSS) [29]. FBSS is characterised by intractable pain and various degrees of functional incapacity following spinal surgery. The major identifiable causes include recurrent or residual disc herniation, arachnoiditis, radiculitis, spinal or spinal neural foraminal stenosis and failure to correctly identify the structural source(s) of pain [30].

The severity of recurrent symptoms has not been shown to correlate with the amount of epidural scar tissue [31, 32]. Management of patients with FBSS remains difficult [33], but the presence of recurrent or residual disc herniation is generally is thought to be an indication for further surgery [34, 35, 36]. The differentiation of scar tissue and recurrent or residual disc herniation may be achieved with relatively high accuracy on contrast-enhanced CT, but is even better made on IV contrast-enhanced MRI [36, 37].

When a residual disc herniation is present, one should keep in mind that it is not necessarily responsible for the patient's complaints [38]. Herniated disc fragments, especially when extruded into the spinal canal, as is often

the case in the postoperative phase, can regress spontaneously, chiefly via phagocytosis [38, 39].

Late nerve root enhancement (more than 6–8 months after surgery) should be considered abnormal. Good imaging correlation with radicular pain or deficits make it a very specific indicator of continuing underlying sterile radiculitis.

One should be aware of the possibility of changes in the size and shape of the spinal structures which may develop as a result of discectomy. One consequence can be secondary stenosis of a neural foramen, probably an important but under-recognised cause of the FBSS. The dimensions of the foramina are best assessed on parasagittal images.

Recurrent disc herniation

The imaging differentiation between recurrent or residual disc herniation and epidural fibrosis can usually be made using existing criteria [29, 34, 36, 37], including on the one hand obliteration of the epidural fat by uniformly enhancing epidural fibrosis in the anterior, lateral and posterior epidural space in epidural fibrosis (Fig. 1, 2) or, on the other, early central nonenhancement in recurrent or residual disc herniation (Fig. 3). We think the latter most important in differentiating the two conditions. Since MRI is more sensitive than CT to abnormal contrast enhancement, it is the imaging method of choice for investigation of the FBSS.

Epidural fibrosis

As mentioned above, the lack of early central contrast enhancement in cases of recurrent disc fragments and the homogenous enhancement pattern of scar tissue have been claimed to be the major differentiating criteria. The capillaries in granulation tissue are abundant and demonstrate scanty micropinocytotic vesicles, a high frequency of luminal occlusion (7% patency rate), a loose network of pericytes, and leaky endothelial vascular junctional complexes [40]. By comparison, mature scar tissue, which may be identified within 5 weeks of injury, has fewer patent capillaries that show a thin basal lamina, a layer of pericytes, micropinocytotic vesicles and endothelial vascular junctional complexes that are more tightly joined. The vascularised granulation tissue surrounding and often penetrating into the substance of disc herniations represents a normal reaction, the body's attempt to destroy or resorb the disc material, and can complicate the differentiation of disc herniation and isolated scar formation.

The association of scar tissue with mass effect, manifest as minor deformity of the dural sac, does not necessarily imply excess or pathological scar formation (Fig. 2) [27, 41]. The deformity of the dural sac usually diminishes within 6 months of surgery. However, deformity of the dural sac, with more than 10% loss of cross-sectional area of the central spinal canal in the axial plane, accompanying epidural scar is to be considered relatively abnormal when 6 months or more after surgery (Fig. 4). Nevertheless, what effect this epidural scarring has on signs and symptoms is not known, and many researchers believe there is no clear relationship between epidural fibrosis and the patients' complaints [31, 32].

Fig. 3a, b. Recurrent disc herniation: T1-weighted images **a** before **b** after contrast enhancement. The images show a large, centrally nonenhancing mass in the anterior epidural space, a typical recurrent or residual disc herniation. The rim enhancement, which represents normal or expected postoperative fibrous granulation tissue, is almost always seen

Complications of surgery

Haematoma

Although uncommon, symptomatic postoperative haemorrhage typically presents hours to days following surgery. MRI shows mixed blood-breakdown products,

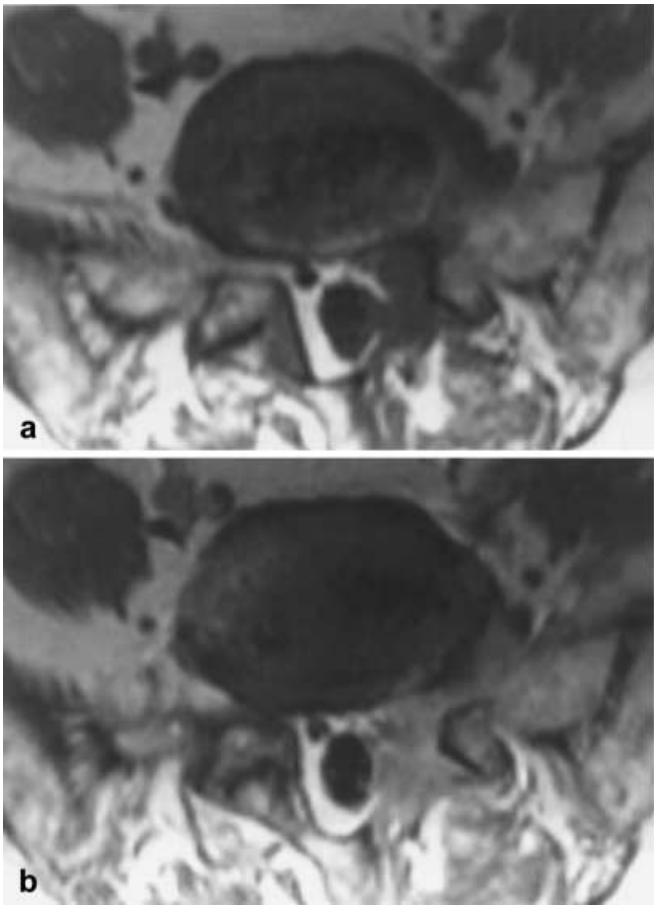


Fig. 4a, b. Abnormal postoperative scar formation: axial T1-weighted images **a** before **b** after contrast enhancement. Comparing the two images one can appreciate extensive deformity of the thecal sac and the left S1 nerve root-sheath complex. These appearances are to be considered abnormal when seen more than 6 months after disc surgery

and is more sensitive than CT to the presence of a haematoma as well as better for demonstrating its extent. Some haematomas may become large and can extend into the central spinal canal to compress the cauda equina (Fig. 5). This occurrence potentially constitutes a medical crisis, requiring emergency surgical evacuation.

Spondylodiscitis

Spondylodiscitis, or discitis combined with vertebral osteomyelitis, is a relatively uncommon complication of lumbar disc surgery. It is encountered not only after surgery or chemonucleolysis [42], but also following diagnostic procedures such as discography [43] and rarely myelography [44]. Postoperative spondylodiscitis occurred in 0.1–3% of patients in reported series [45, 46]. Although its frequency may be decreasing due to better technical and prophylactic measures, postoperative infection has not been completely eliminated. Disc space infection is probably due to direct intraoperative contamination [47]. Pre- or perioperative infection elsewhere in the body and compromised patient immunology may be predisposing factors. Infection is most frequently caused by *Staphylococcus epidermidis* or *Staphylococcus aureus* [48]. Spondylodiscitis is a serious complication, which may lead to long-lasting and sometimes permanent morbidity [49, 50]. It is commonly accepted that early appropriate treatment is capable of shortening the course and reducing the severe sequelae of spondylodiscitis. The diagnosis depends on a combination of clinical, laboratory and imaging findings. Clinically, severe low-back pain with or without sciatica typically appears 7–28 days (average 16 days) after surgery [51]. Clinical findings and classical screening methods such as white blood cell count, erythrocyte sedimentation rate and fever are, however, not reliable and are often fallible in indicating spondylodiscitis [49, 52, 53]. C-reactive protein (CRP) determination has proved a much more reliable screening test for infectious complications of lumbar disc surgery [54].

Although diagnosing spondylodiscitis with the help of MRI in the unoperated patient can be straightforward, it is typically more challenging following surgery. The operated level always shows more or less extensive changes due to the surgical intervention itself and the accompanying postoperative aseptic inflammatory response [27, 41, 48]. These changes may include type 1 changes [55], such as oedema of the vertebral marrow adjacent to the disc. Contrast enhancement can be normal in the intervertebral disc and along the vertebral endplates postoperatively [56]. Disappearance of the 'intranuclear cleft' sign [57] is not usually reliable, since the surgeon may have resected the nucleus pulposus.

Of available imaging techniques, only MRI contributes significantly to establishing the diagnosis of postoperative spondylodiscitis [45]. MRI may assist in several ways (Fig. 6) [58]. Absence of low signal on T1- and high signal on T2-weighted images in the marrow adjacent to the disc makes septic spondylodiscitis highly unlikely; the same holds true for absence of contrast enhancement of the intervertebral disc space. An enhancing soft tissue mass surrounding the affected spinal level in the paravertebral soft tissues and epidural space is highly suggestive of septic spondylodiscitis, and indicates further investigation.

However, some workers have not found MRI reliable by itself in diagnosing (septic) postoperative spondylodiscitis [48, 59]. While one study suggested that a combination of MRI findings alone was characteristic [60], these findings were not fully confirmed in a more recent study [48].

If, in any given case of suspected postoperative infection, MRI does not enable one to exclude or confirm septic spondylodiscitis, one should attempt to confirm

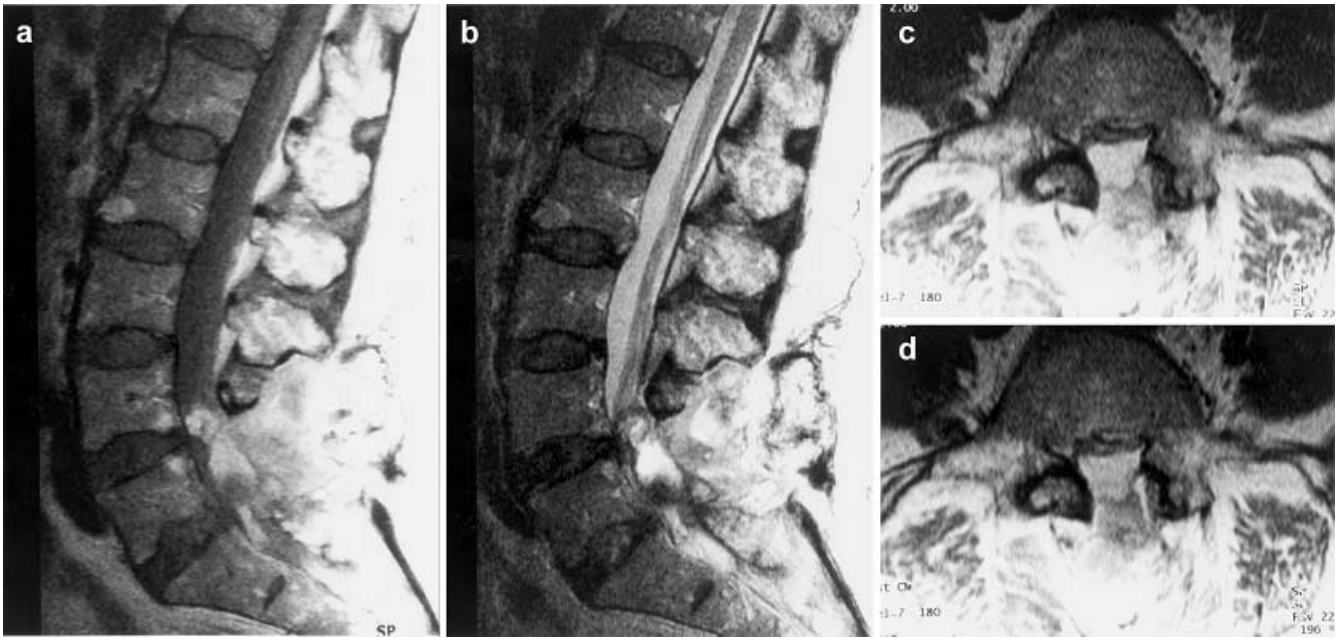
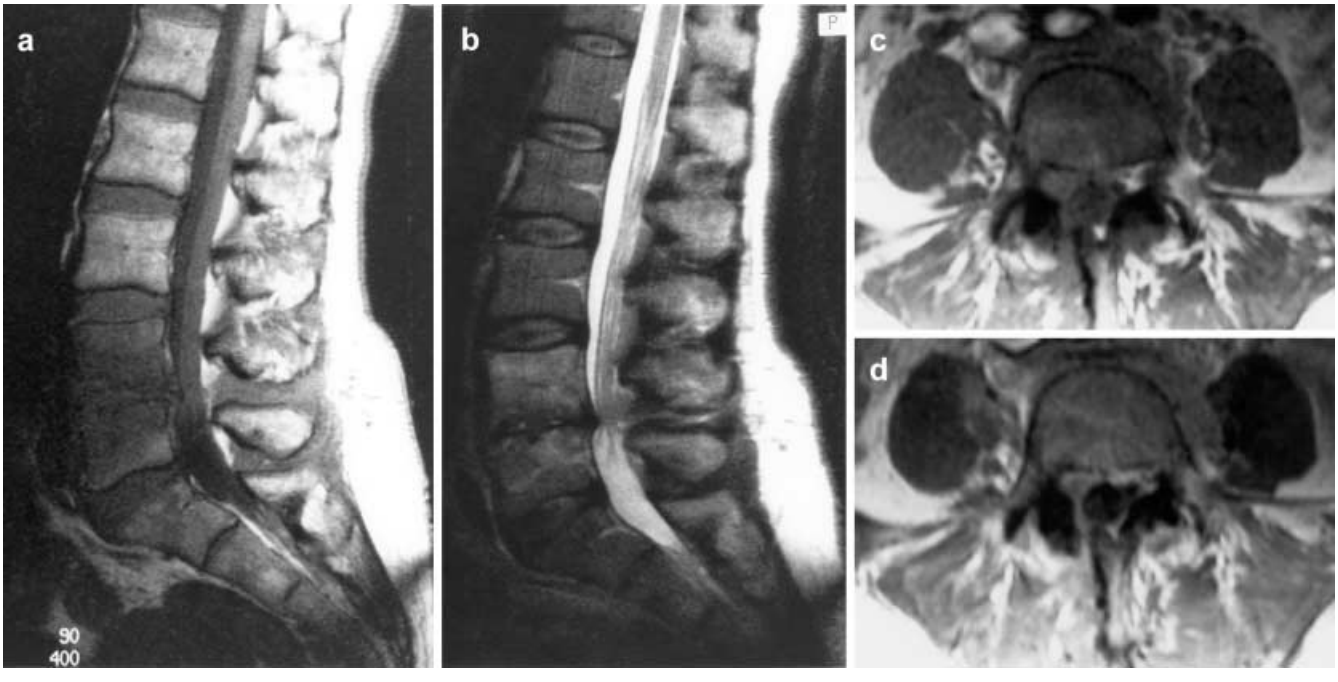


Fig. 5a–d. Large postoperative haematoma. Sagittal **a** T1- and **b** T2-weighted images; **c, d** axial T1-weighted images before and after contrast enhancement. The sagittal images show a large, partially intraspinal, mass at the operative level. It gives high signal on both T1- and fast spin echo T2-weighted images, indicating fat or haemorrhagic content. The peripheral low signal regions on the T2-weighted images suggest a large haematoma. The axial images confirm that the lesion does not enhance. Note marked compression of the dural sac and cauda equina by this large postoperative haematoma

the diagnosis by percutaneous biopsy. Biopsy of the disc space and successful isolation of the organism can yield a definitive diagnosis [49]. Since fine-needle aspiration is

Fig. 6a–d. Postoperative spondylodiscitis. Sections as in Fig. 5. The sagittal T1- and T2-weighted images show low and high signal, respectively, in the marrow of the vertebral bodies adjacent to the operated disc. Although they can be seen in the normal postoperative spine, these changes are usually less extensive in asymptomatic patients. The contrast enhancement around the disc is more typical of infectious spondylodiscitis, as in this proven case of staphylococcal infection



often negative in septic spondylodiscitis [48, 61], biopsy with a larger bore nucleotome is recommended [62].

Pseudomeningocele

Pseudomeningoceles are collections of cerebrospinal fluid (CSF) extending from the spinal canal into the posterior paraspinal soft tissues. These cystic lesions typically develop after inadvertent surgical laceration of the dura mater during surgery, or following incomplete closure of the meninges in cases of intradural surgery. They usually protrude through a surgical bone defect in the posterior spinal elements to form a cyst with MRI signal intensities comparable to CSF (Fig. 7). They are called pseudomeningoceles because they have no true archnoid lining, but instead walls of reactive fibrous tissue.

Pseudomeningoceles are sometimes incidental findings on imaging, causing no symptoms. However, partly because of mass effect, they may also be responsible for low-back pain, and even radiating mono- or polyradicular leg pain [29].

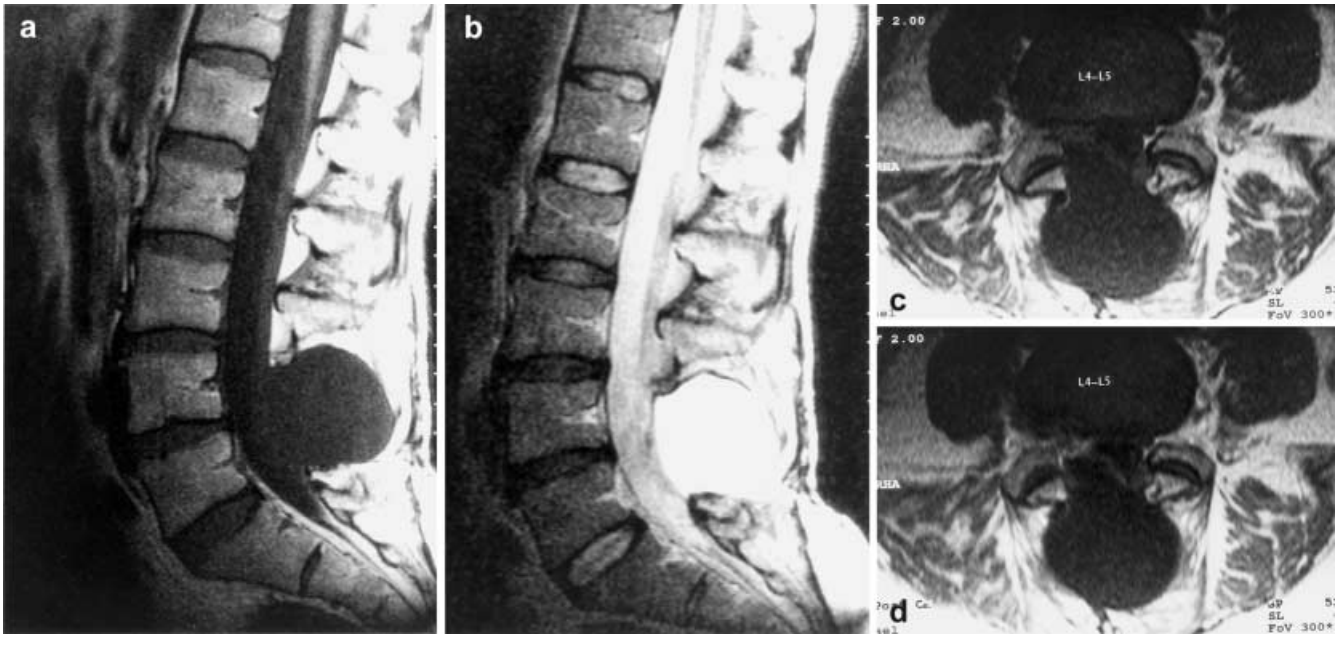
Sterile radiculitis

Contrast enhancement of the intrathecal spinal nerve roots of the cauda equina following a conventional dose of 0.1 mmol/kg (0.2 cc/kg) gadolinium contrast medium is not normal (Fig. 8). Spinal nerve roots normally have a visually intact blood-nerve barrier (BNB) on standard dose contrast-enhanced T1-weighted images. On the other hand, there is little or no BNB within the spinal dorsal root ganglia, which explains their intense enhancement. With frank compression injury, such as by a posterolateral disc herniation, to spinal nerves and nerve roots, however, this otherwise relatively intact BNB may break down. The complex, poorly understood sequelae of chronic neural trauma and ischaemia are believed to be the cause of the abnormal neurophysiological changes resulting in clinical radiculopathy that may continue long after the disc herniation has been surgically removed [63].

In one study, intrathecal nerve root enhancement was seen in 20% of patients who were asymptomatic 6 weeks after disc surgery, but in only 2% after 6 months [27]. In a study of symptomatic patients, contrast enhancement of spinal nerve roots was demonstrated at the surgical site, and extending craniad and caudad, in the chronic postoperative period, more than 6–8 months after surgery [63]. Almost certainly, the instances of nerve-root enhancement physically remote from the site of injury, i.e., distant from the level of the primary disc herniation and surgery, are associated with ongoing degenerative and regenerative phenomena within injured nerves [63].

From a practical standpoint, chronic postoperative nerve-root enhancement correlates well with a radicular clinical presentation. However, during the first 6–8 months after intervertebral disc surgery, nerve-root enhancement can be seen in asymptomatic patients, apparently reflecting transient sterile inflammation

Fig. 7a–d. Postoperative pseudomeningocele. Sections as in Fig. 5 show a large cyst protruding from the spinal canal through the surgical bone defect into the posterior soft tissues. This large pseudomeningocele causes posterolateral compression of the dural sac. The enhancing structure between it and the subarachnoid space is the dura mater; hence the name pseudomeningocele, since the cyst has no true meningeal lining



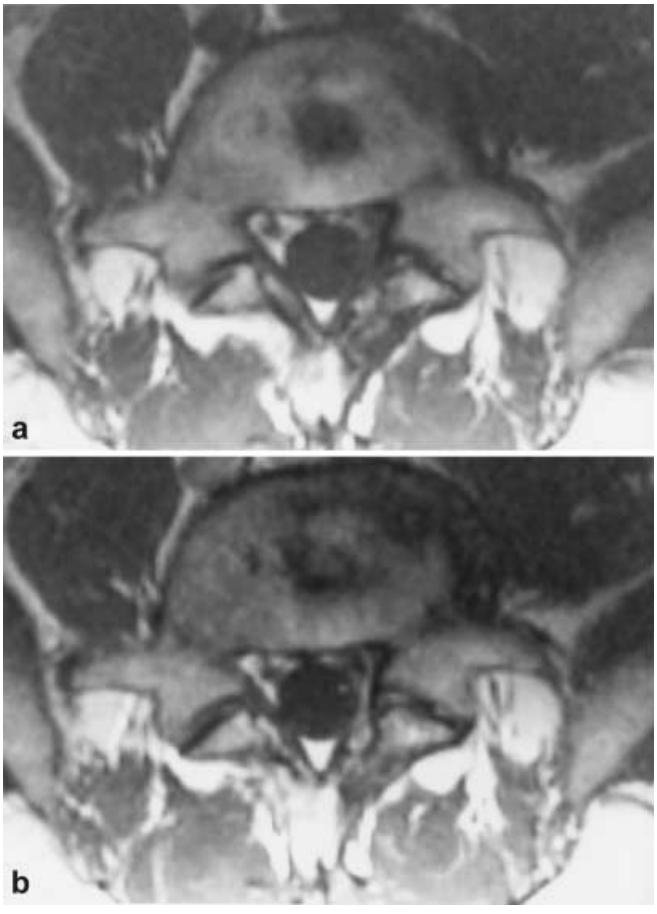


Fig. 8a, b. Postoperative sterile radiculitis: axial T1-weighted images **a** before and **b** after contrast enhancement. The enhancement of a nerve root, 1 year after surgery, with a standard dose of intravenous gadolinium-containing contrast medium (0.1 mmol/kg) is abnormal, representing sterile radiculitis. Note the expected enhancing postoperative epidural fibrosis

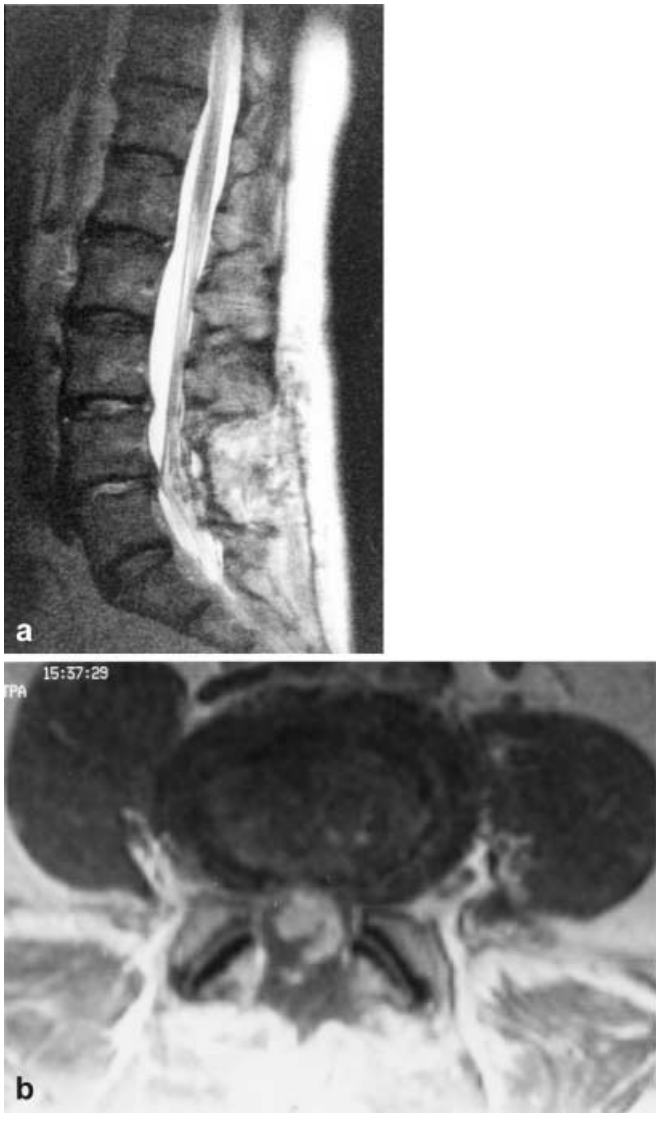


Fig. 9a, b. Sterile arachnoiditis: sagittal **a** T2- **b** contrast-enhanced axial T1-weighted images. There is clumping of the roots of the cauda equina at the operative level. After contrast medium, a large intrathecal ''mass'', representing the matted roots, is clearly discernible

within the nerve root undergoing repair. Nevertheless, in the proper clinical setting, that of the chronic FBSS, nerve-root enhancement may be used to regarded as a clinically relevant imaging finding in aetiologically uncertain cases of postoperative radiculopathy [63].

Sterile arachnoiditis

The potential factors inciting chronic sterile spinal arachnoiditis are much debated but include the surgical procedure itself, the presence of intradural blood following surgery, diagnostic lumbar puncture, treated perioperative spinal infection, the previous use of myelographic contrast media (especially older oil-based preparations) and prior intraspinal injection of anaesthetic, anti-inflammatory or chemotherapeutic agents (e.g., steroids, methotrexate). Chronically persistent lumbosacral signs and symptoms in 6–16% of postsurgical patients have been attributed to sterile arachnoiditis.

The three MRI patterns described in adhesive arachnoiditis include: scattered groups of matted or ''clumped'' nerve roots; an ''empty'' thecal sac caused by adhesion of the nerve roots to its walls; and an intrathecal soft tissue ''mass'' with a broad dural base, representing a large group of matted roots, which may obstruct the CSF pathways (Fig. 9) [29]. These changes may be focal or diffuse. Contrast enhancement of the thickened meningeal scarring and underlying intrathecal roots may or may not be observed [63].

The degree of severity of the enhancing and nonenhancing imaging findings in patients with chronic sterile arachnoiditis does not appear to correlate well with the degree of signs and symptoms [63]. When present, the symptoms usually indicate involvement of multiple roots, with pain and paraesthesiae being perceived in both legs.

Textiloma

A surgical sponge or ''cottonoid'', accidentally left behind in a surgical wound eventually becomes a textiloma [64]. Strictly speaking the term should be reserved for a surgical sponge consisting of organic material; synthetic material has replaced cotton, and the definition ''textiloma'' should be reconsidered. The term ''gossybipoma'' was used in older literature to denote a mass composed of a cotton matrix [65]. The foreign body is made of synthetic cotton-like (''cottonoid'') fibre (''rayon'') with a barium sulphate marking filament, visible on radiographic examinations. The pseudotumour consists of the foreign body itself with perilesional reactive changes, forming a foreign-body granuloma.

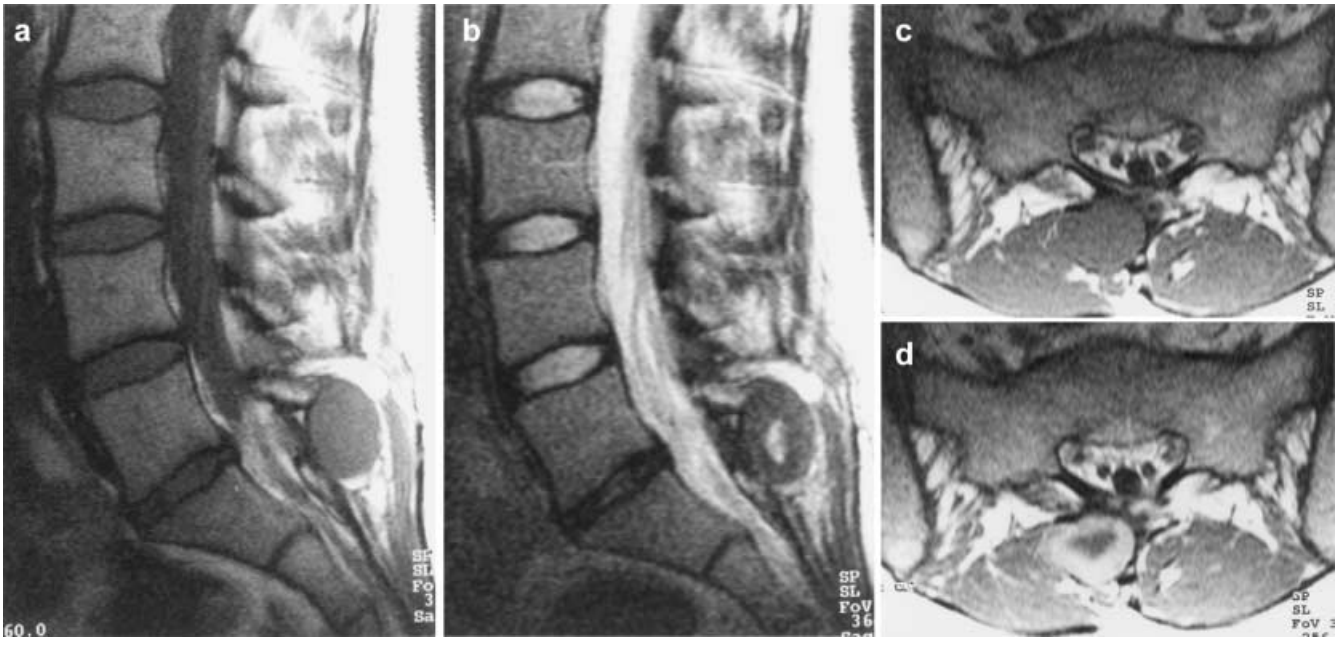
MRI can be confusing and misleading because the most typical radiographic sign of a forgotten ''cottonoid'', the filament, is not visible on MRI [66]. Indeed, this filament, consisting of barium sulphate, which is neither magnetic nor paramagnetic, causes no visible magnetic trace on MRI. Furthermore, the filament contains very few free protons and thus does not yield a significant MRI signal. The lesion shows a moderate degree of peripheral contrast enhancement on T1-weighted images, believed to be related to an inflammatory foreign-body reaction. On T2-weighted images these lesions give low signal, presumably reflecting a dense fibrous tissue reaction peripherally, and central foreign body material lacking mobile protons (Fig. 10) [67]. This also explains the centrally nonenhancing area on contrast-enhanced T1-weighted images.

Since the typical barium-impregnated filament is not visible on MRI, the differential diagnosis should include other postoperative changes such as hypertrophic scar surrounding a seroma and paraspinal abscess formation. The history also can help to differentiate between these entities, since the onset of complaints is usually much earlier in cases of postoperative infection than with a foreign-body granuloma.

Stenosis

Stenosis of the central portion or lateral recesses of the central spinal canal, and one or more neural foramina may be a cause of the FBSS. These forms of spinal stenosis may predate or follow spinal surgery. When the stenosis follows surgery, it may present years after the operation, as a result of accelerated degeneration of spinal segments above or below a single or multi-level bony segmental spinal fusion. These changes occur because of increased stresses placed on the segments

Fig. 10a–d. Textiloma: sections as in Fig. 5. A cottonoid left in the operation wound initiates a marked aseptic inflammatory reaction and is seen as a rounded low-signal mass on both T1- and T2-weighted images. The centre may give high signal on T2-weighted images due to central cystic or necrotic degeneration. The periphery shows marked contrast enhancement representing the extensive granulomatous reaction



above and below the fused segment(s) as load-sharring is shifted away from the solidly fused levels. Although published studies have methodological biases and small sample sizes, it appears that the sensitivity to and specificity for spinal stenosis of MRI and CT are similar: true-positive rates of approximately 90%, false-positive rates of approximately 10% [29].

Lumbosacral neural foramen narrowing is best imaged with direct sagittal T1-weighted images (Fig. 11). Foraminal narrowing with nerve root impingement, due to loss of intervertebral disc height after complete discectomy is not uncommon. Therefore, some surgeons resect only the visibly herniated or sequestered disc material after blunt enlargement of the site of annular rupture in an attempt to preserve the biomechanics of the disc. Unfortunately, this also leaves behind a significant amount of nuclear and annular material, which may lead to further herniation.

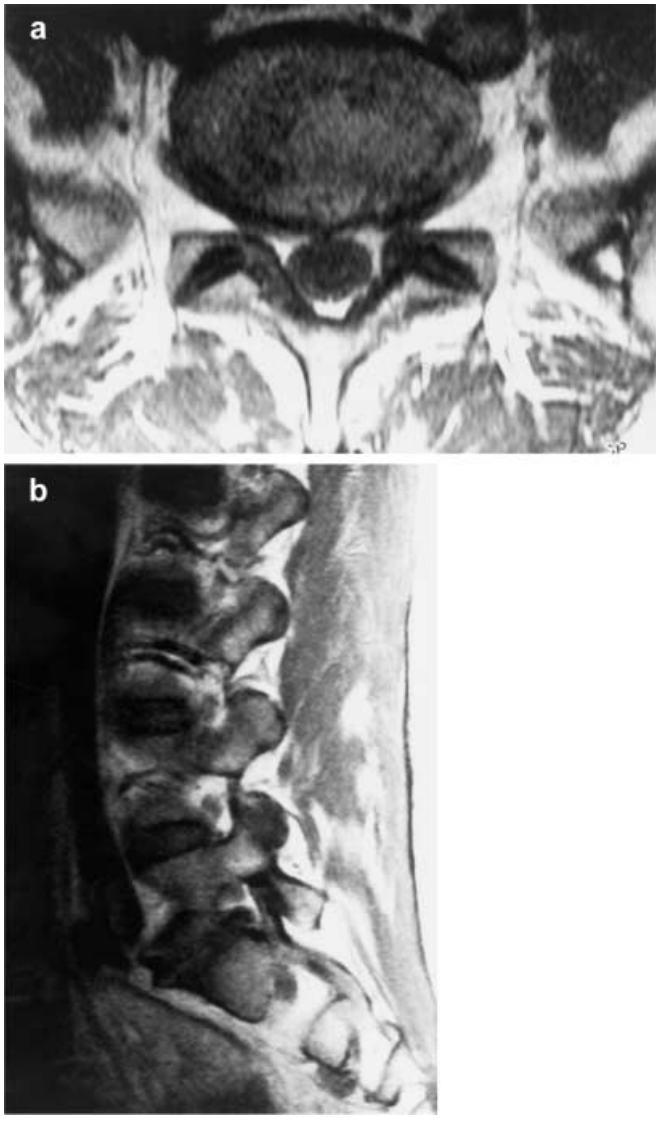


Fig. 11a, b. Postoperative spinal neural foramen stenosis: **a** axial **b** parasagittal T1-weighted images. Although the axial images show bilateral foraminal narrowing, the relationship of the nerve roots to the margins of the foramen is not clear. The left parasagittal image **b** clearly demonstrates spinal nerve root impingement due to foraminal encroachment caused by a loss of intervertebral height and osteophytic spur formation around the disc. The right parasagittal image demonstrated no encroachment on the nerve root in the opposite neural foramen

MRI following intervertebral fusion

Techniques

Intervertebral fusion cage surgery

Posterior lumbar interbody fusion (PLIF) is a vertebral fusion technique using a posterior access to the intervertebral disc space. This has the advantage of a shorter surgical route, which nevertheless can be more complicated, since it traverses the central spinal canal. It is preferred in cases in which intraspinal surgery, such as discectomy, is also necessary. This technique has grown in popularity over the last decade. The fusion itself is usually accomplished with metal or carbon "cages" filled with bony material harvested from the patient. In a PLIF the posterior margin of the cages should be aligned with the posterior margins of the vertebral bodies, to distribute the axial load in part over the stronger cortical vertebral border. In this way the risk for vertebral collapse or subsidence around the disc implant is minimised. Another possible major complication is posterior migration of the cage with compression of the dural sac and cauda equina (Fig. 12).

The anterior lumbar interbody fusion (ALIF), as it name suggests, is comparable to the PLIF, but access is gained anteriorly. This can be technically challenging, according to the specific anatomic relationships with the prevertebral vessels, and is usually performed only at the L5-S1 level.

Intervertebral bony fusion

Several techniques are available for obtaining bony fusion. All are based on gaining primary temporary mechanical functional stabilisation, so as to subsequently achieve secondary permanent intervertebral bony fusion.

Radiographic signs of fusion

The value of serial conventional radiographs of the spine following fusion is unclear [68]. It has been suggested that radiography underestimates the rate of pseudarth-

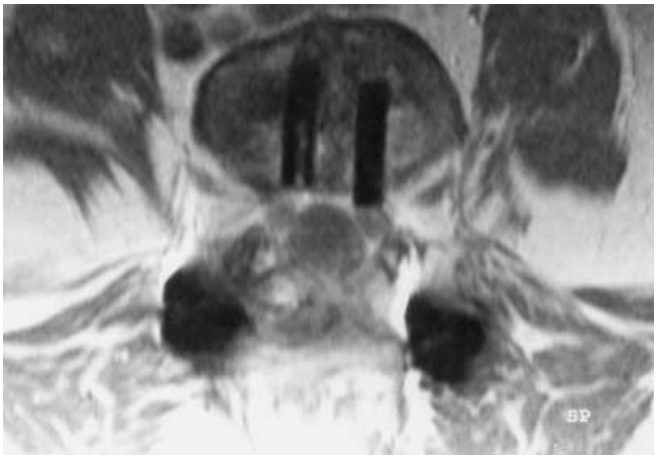


Fig. 12. Intervertebral disc cage migration after posterior lumbar interbody fusion (PLIF): axial contrast-enhanced T1-weighted image after laminectomy, pedicle screws and PLIF with two carbon disc cages. Extensive bilateral epidural fibrosis is a normal or expected postoperative finding and is related to the procedure. The cage on the patient's left has migrated posteriorly and slightly displaces the nerve root-sheath complex posteriorly. Normally the posterior edge of the cage should be aligned with the posterior vertebral margin, as on the patient's right

rosis (i.e., ununited intersegmental bony fusion) as compared with direct observation at surgical re-exploration, particularly when a hairline pseudarthrosis is present [69].

Conversely, other workers contend that conventional radiographs may underestimate the degree of fusion. The referring clinician is sometimes faced with the puzzling contradiction that static and dynamic radiological examinations do not show evidence of bony intersegmental fusion in a patient who is doing well clinically. The reason for this is believed to be that premineralised osteoid may be functionally fused, but is nevertheless radiolucent on conventional radiographs [70]. Calcification of osteoid typically takes many months to complete. It is accepted that at least 6–9 months from the time of surgery are necessary for solid intersegmental fusion to be seen radiographically [71, 72].

After mineralisation of the osteoid, the bone in the fusion area may appear more dense than the adjacent otherwise normal vertebral bone. As mature bony trabeculae develop, they bridge the fusion area between the adjacent bones. This leads to the cortical vertebral endplates becoming invisible, and thus to a loss of the so-called 'graft-host' interface between the implanted bone and the vertebra. In some instances, a dense line of sclerotic bone may be an indicator of fusion between the graft and the host vertebra. A well-documented observation indicating solid intervertebral bony fusion, is resorption of pre-existing spondylotic spurs around the disc, although this may take several months or years. Bony fusion across the posterior intervertebral joints is also a reassuring sign of functional bony fusion of two segments.

Pseudarthrosis and other complications

There are several causes of complications encountered on imaging in patients with fixation devices or interbody bone grafts. An unfortunate cause of unsatisfactory outcome is erroneous preoperative diagnosis of the clinically relevant pathology or inaccurate identification of the spinal level of the pathology responsible for the symptoms and signs. The latter error can occur, for example, in patients with transitional lumbosacral vertebrae, in whom mislabeling of vertebral segments may result in surgery at an incorrect level.

As noted, complications in the immediate postoperative period include haematoma and infection [72]. Late postoperative complications are migration, complete dislodgement or fracture of implants, which may contribute to complications such as failure of bony fusion, intersegmental instability caused by failure of fusion, and neurological injury [72]. An intermediate-signal gap between vertebral body and bone graft on T1-weighted images is an indicator of pseudarthrosis [69]. An overview of the imaging signs of pseudarthrosis is given in Table 1 (Fig. 13) [69, 72, 73]. Bone-graft material can migrate or hypertrophy, encroaching on the spinal canal or neural foramina. Rarely, the vertebra(e) adjacent to the operated level(s) can fracture, especially in osteoporotic patients.

A promising application of MRI is postoperative assessment of patients with interbody fusion grafts to document possible posterior or posterolateral extrusion into the spinal canal or neural foramina, or failure of bony interbody fusion (Fig. 12) [73]. In large published

Table 1. Imaging signs of pseudarthrosis, failed intersegmental fusion [68]

Collapse of the surgical construct with loss of disc space height, despite apparent rigid posterior fixation with pedicle screws and plates
Increasing vertebral slip – progressive spondylolisthesis
Posterior or posterolateral bone graft extrusion, migration, into central spinal canal
Broken screws, due to pseudarthrosis with continued stress on surgical implant
Resorption of the bone graft; decrease of bone density within graft over time
Major lucency or gap visible surrounding the fusion area: 2 mm or more around the entire periphery of the graft, metallic screw or disc cage
Intermediate signal on T1-weighted images in the junction between the vertebral body and the bone graft

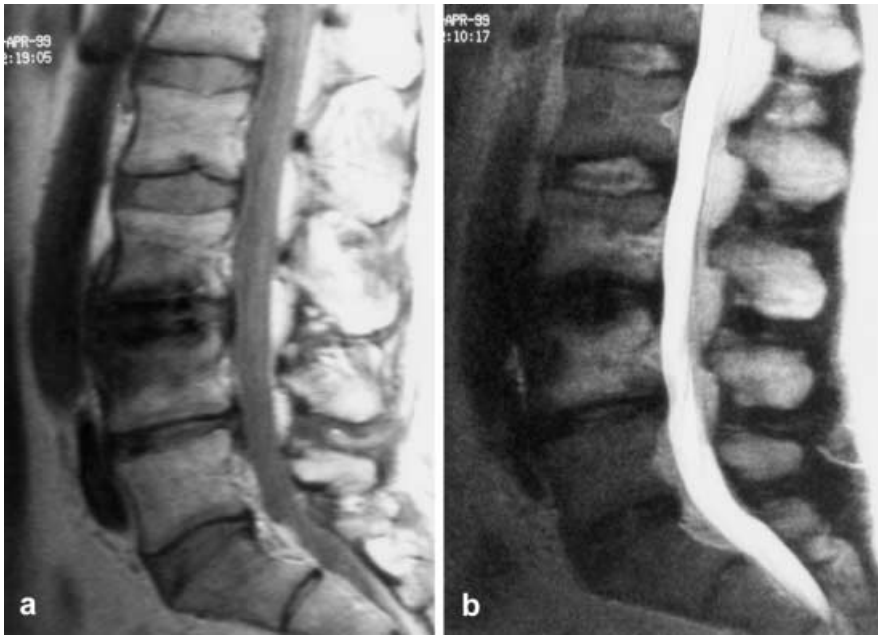


Fig. 13a, b. Pseudarthrosis after attempted surgical fusion: sagittal **a** T1- **b** T2-weighted images 1 year after attempted intervertebral fusion at L3-L4. Extensive signal change is seen in the marrow of the vertebral bodies adjacent to the operated disc, low on T1- and high on T2-weighted images, and representing oedema. If present more than 6 months after surgery these changes are evidence of probable nonfusion or pseudarthrosis

series, the graft extrusion is estimated to occur in 2% of cases [73]. Posterolateral extrusion of a graft can result in direct nerve-root compression, usually characterised by immediate, severe radicular pain. Axial sections may yield confusing and even contradictory results depending on the exact level and orientation of the slice relative to the bone graft or cage. Thanks to its multiplanar imaging ability, MRI provides direct sagittal, axial and coronal images, which define the exact position of the bone graft or cage. Moreover, MRI is useful for showing associated pathology in the central spinal canal. Accurate assessment of the degree and direction of bone-graft dislocation is important, because they may be directly related to the symptoms and signs and determine the approach for further intervention.

Conclusions

In the assessment of the patient after lumbar fusion, comparison with previous imaging examinations is desirable, to detect subtle changes which may indicate successful bony fusion, or alternatively herald an impending complication such as progressive spondylolisthesis [68]. Subtle changes on conventional radiography may become more evident on dynamic flexion/extension views or on thin-section CT coupled with multidimensional reformation. It is important for the clinician to inform the radiologist performing the examination about the type of surgical procedure(s) and the current clinical syndrome. These may affect the imaging strategy, and are important when interpreting the images and in the consideration of supplementary imaging. In the patient with a normal postsurgical clinical course, routine follow-up examination of the spine is obviously unnecessary [70, 74]. However, in problem patients, such as those with unexplained persistent pain, or symptoms suggesting pseudarthrosis, surgical re-exploration may be justified [74].

Radiologists should be familiar with the procedures and the surgical implants used by the surgeons in their institutions. Meaningful postoperative radiological assessment can be accomplished only when the indications for specific surgical techniques, their postoperative radiological appearances and their possible complications are known. Radiologists face continual changes in both surgical techniques and instrumentation, and should be knowledgeable about the devices available and the biomechanical principles directing their use. A working knowledge of evolving spinal fixation devices and surgical techniques is required in order to identify specific complications. The radiological findings should be discussed with the relevant surgical colleagues.

MRI following instrumentation

Types of instrumentation

There is a plethora of instrumentation systems, sometimes having only small variations in design and function. Choosing the appropriate implant depends in part on the pathology and the desired outcome, as well as the experience of the specific surgeon.

Pedicle screws

Pedicle screws are the most rigid construction device and have the highest frequency of successful fusion. They can be used with plates, hooks or rods. Since adjacent neural structures can easily be inadvertently injured, correct placement of the screws is imperative. Use of computer-assisted surgery can be helpful. In the lumbar spine pedicle screws are typically used for spinal instability associated with trauma, and for degenerative disease with instability, with or without spondylolisthesis.

Harrington rods

Harrington rods are designed to obtain and maintain spinal distraction. The system consists of at least one upgoing and one downgoing hook, which are usually placed under the respective laminae, and are connected with a rod. Harrington rods are primarily used to correct scoliosis, but are prone to induce a loss in lumbar lordosis, with eventual secondary back pain and/or gait disturbances. Some traumatic spinal injuries can also be effectively treated with Harrington rods.

Coutrel-Dubousset instrumentation

This newer device distributes forces more evenly, can serve for distraction and compression using the same rod and is able to manipulate the spine in three dimensions. Its rod is more flexible than the Harrington rod and allows use of pedicle screws or hooks. Crosslinks can also be used to create a more stable and more rigid torsional system. The indications for this device are primarily correction of scoliosis and kyphosis. The system can also be used in selected cases of trauma and for reconstructive procedures in patients with neoplastic invasion of the vertebral column.

Anterior instrumentation

Anterior fusion techniques are more recent developments. Here also, several instrument types are available. The indications are progressive idiopathic scoliosis with a curve of more than 40–50 ° which cause significant imbalance of the trunk. The systems are also used for spinal instability due to trauma, neoplasia or severe spinal degeneration.

Spinal fixation devices are used to stabilise the spine, reduce deformities and fractures, and replace abnormal vertebrae [68, 71, 72, 75]. Bony fusion is usually attempted along with placement of metallic surgical instrumentation materials [71, 75]. The spine is unstable in such patients and early operative intervention allows rapid mobilisation and rehabilitation. Various methods of fusion have evolved and are currently in use. Surgical procedures usually consist of posterior (posterior bony elements) and/or anterior (vertebral body) fixation. However, persistent lumbar instability is a potential clinical problem [76]. It is often believed to be the cause of recurrent low-back pain in patients who have undergone lumbar fusion [77].

Imaging of spinal implants

Determining the solidity of a fusion is a difficult problem. For a long time, it was widely accepted that the only way of determining the solidity of lumbar fusion was by surgical exploration [74]. This principle, known as ''Bosworth's dictum'' [70, 74, 78], was undoubtedly inspired by the limitations of imaging techniques half a century ago, and by the inconsistent and often unreliable results provided by conventional radiography. Routine re-exploration for the purposes of determining the status of a surgical fusion is, however, impractical because of the expense and morbidity involved [74]. Therefore, it is important to find an imaging technique which will give reliable information about the status of a lumbar intersegmental fusion.

A variety of radiological methods have been used. These include radiographs (conventional plain films, tomograms, biplane stress bending films, stereophotogrammetry, dynamic flexion/extension myelography and CT-myelography), CT, radionuclide bone scans and MRI. These can be divided into two categories, depending on whether they can be used to assess functional or structural integrity of spinal fusion. Most, such as radiographs, conventional tomograms, CT and MRI, give data on structural integrity. The purpose of using these techniques is essentially to identify the bony continuity of the fusion mass. Studies which assess functional integrity include any type of dynamic stress films. The purpose here is to demonstrate the presence or absence of motion between previously fused vertebral segments. These studies depend heavily on patient cooperation and may fail to show abnormal motion because of muscle guarding, spasm or internal fixation [74]. This section will focus on imaging strategies and findings, and underscore the need for radiologists to familiarise themselves with the surgical procedures performed at their institution.

Magnetic resonance imaging

In the preoperative assessment of the patient undergoing fusion, MRI is useful for detection of disc degeneration at levels above or below the intended fusion [79]. Foreign

ferromagnetic metal objects such as spinal fixation devices gives rise to local distortion of the magnetic field. This metal artefact is explained by the occurrence of a local gradient which is non-negligible compared with the frequency-encoding gradient. When the implants are made of materials which are not superparamagnetic materials, such as titanium or tantalum, distortion of the magnetic field is less severe, but these materials may still obscure normal regional anatomy. In clinical practice, large ferromagnetic metallic spinal fixation devices render MRI of the involved region of the spine virtually impossible. Even in the absence of metallic fusion devices, in patients who have had simply a discectomy or corporectomy, tiny fragments of metal sheared off instruments such as drills, suckers and other devices during surgery can give rise to areas of signal-void artefact which obscure the operated region [80]. These may be anything from small, mimicking a small anterior extradural defect, to large, obscuring a fusion mass and the contents of the spinal canal. They are often seen with MRI, even when conventional radiography or CT do not show metal in the same region.

Imaging of posterior fixation devices such as pedicle screws is limited by the metal artefacts they produce. As outlined earlier, gradient echo images are more susceptible to ferromagnetic metal artefacts than conventional or fast SE acquisitions; and metal artefacts are also more pronounced at higher field strengths.

Conclusions

Depending upon the factors mentioned above, one or a combination of complimentary medical imaging modalities may be required in a given patient following lumbosacral spinal surgery, to diagnose the clinically relevant abnormality and to assist the surgeon in deciding if repeat surgery is necessary, and if so, of what type and at which vertebral level(s). A clear understanding of the indications, limitations, and alternatives available to the imaging specialist will assist the referring physician in achieving the most efficacious triage as well as promoting the most beneficial and timely outcome in the patient presenting with the FBBS.

References

1. Fengler H, Wagner W (1997) Backache – orthopedic diagnosis and special therapeutic possibilities. Z Arztl Fortbild Jena 90: 677–685
2. Jinkins JR (1997) Posttherapeutic neurodiagnostic imaging. Lippincott-Raven, Philadelphia
3. Borenstein DG (1997) Epidemiology, etiology, diagnostic evaluation, and treatment of low back pain. Curr Opin Rheumatol 9: 144–150
4. Borenstein DG (1996) Chronic low back pain. Rheum Dis Clin North Am 22: 439–456
5. Van Goethem JWM, Biltjes IGGM, Van den Hauwe L, Parizel PM (1999) Imaging in low back pain. Pain Digest 9: 262–266
6. van Tulder MW, Koes BW, Bouter LM, Metsemakers JF (1997) Management of chronic nonspecific low back pain in primary care: a descriptive study. Spine 22: 76–82
7. Ackerman SJ, Steinberg EP, Bryan RN, BenDebba M, Long DM (1997) Persistent low back pain in patients suspected of having herniated nucleus pulposus: radiologic predictors of functional outcome – implications for treatment selection. Radiology 203: 815–822
8. Butt W (1989) Radiology for back pain. Clin Radiol 40: 6–10
9. Junge A, Dvorak J, Ahrens S (1995) Predictors of bad and good outcomes of lumbar disc surgery: a prospective clinical study with recommendations for screening to avoid bad outcomes. Spine 20: 460–468
10. Davis RA (1994) A long-term outcome analysis of 984 surgically treated herniated lumbar discs. J Neurosurg 80: 415–421
11. Bernard TN (1993) Repeat lumbar spine surgery. Factors influencing outcome. Spine 18: 2196–2200
12. Fandino J, Botana C, Viladrich A, Gómez-Bueno J (1993) Reoperation after lumbar disc surgery. Results in 130 cases. Acta Neurochir 122: 102–104
13. Herron L (1994) Recurrent lumbar disc herniation. Results of repeat laminectomy and discectomy. J Spinal Disord 7: 161–166
14. Jönsson B, Strömqvist B (1993) Repeat decompression of lumbar nerve roots. A prospective two-year evaluation. J Bone Joint Surg 75B: 894–897
15. Jinkins JR, Green C, Damadian R (2001) Upright, weight-bearing, dynamic-kinetic MRI of the spine: pMRI/kMRI. Riv Neuroradiol 14: 135-142
16. Van Goethem JWM (1999) Magnetic resonance imaging of the spine. In: Reimer P, Parizel PM, Stichnoth FA (eds) Clinical MR-imaging, a practical approach. Springer, Heidelberg, pp 129–156
17. Plecha DM (2000) Imaging of bone marrow disease in the spine. Semin Musculoskelet Radiol 4: 321–327
18. Jinkins JR, Van Goethem JW (2001) The postsurgical lumbosacral spine. Magnetic resonance imaging evaluation following intervertebral disk surgery, surgical decompression, intervertebral bony fusion, and spinal instrumentation. Radiol Clin North Am 39: 1–29
19. Bradley WG (1999) Use of contrast in MR imaging of the lumbar spine. Magn Reson Imaging Clin N Am 7: 439–457
20. Henk CB, Brodner W, Grampp S, et al (1999) The postoperative spine. Top Magn Reson Imaging 10: 247–264
21. Suk KS, Lee HM, Moon SH, Kim NH (2001) Recurrent lumbar disc herniation: results of operative management. Spine 26: 672–676
22. Barrera MC, Alustiza JM, Gervas C, Recondo JA, Villanua JA, Salvador E (2001) Post-operative lumbar spine: comparative study of TSE T2 and turbo-FLAIR sequences vs contrast-enhanced SE T1. Clin Radiol 56: 133–137

23. Mullin WJ, Heithoff KB, Gilbert TJ, Renfrew DL (2000) Magnetic resonance evaluation of recurrent disc herniation: is gadolinium necessary? Spine 25: 1493–1499
24. Malik AS, Boyko O, Aktar N, Young WF (2001) A comparative study of MR imaging profile of titanium pedicle screws. Acta Radiol 42: 291–293
25. Viano AM, Gronemeyer SA, Haliloglu M, Hoffer FA (2000) Improved MR imaging for patients with metallic implants. Magn Reson Imaging 18: 287–295
26. Rupp RE, Ebraheim NA, Wong FF (1996) The value of magnetic resonance imaging of the postoperative spine with titanium implants. J Spine Disord 9: 342–346
27. Van Goethem JWM, Van de Kelft E, Biltjes IGGM, et al (1996) MR findings after successful lumbar discectomy. Neuroradiology 38: S90–S96
28. Jinkins JR, Osborn AG, Garrett D Jr, Hunt S, Story JL (1993) Spinal nerve enhancement with Gd-DTPA: MR correlation with the postoperative lumbosacral spine. AJNR 14: 383–394
29. Shafaie FF, Bundschuh C, Jinkins JR (1997) The posttherapeutic lumbosacral spine. In: Jinkins JR (ed) Posttherapeutic neurodiagnostic imaging. Lippincott-Raven, Philadelphia, pp 223–243
30. Van Goethem JWM, Parizel PM, van den Hauwe L, De Schepper AMA (1997) Imaging findings in patients with failed back surgery syndrome. J Belge Radiol 80: 81–84
31. Cervellini P, Curri D, Volpin L, et al (1988) Computed tomography of epidural fibrosis after discectomy: a comparison between symptomatic and asymptomatic patients. Neurosurgery 23: 710–713
32. Coskun E, Suzer T, Topuz O, Zencir M, Pakdemirli E, Tahta K (2000) Relationships between epidural fibrosis, pain, disability, and psychological factors after lumbar disc surgery. Eur Spine J 9: 218–223
33. Hudgins PA, Clare CE (1990) Radiographic evaluation of the patient with failed back surgery syndrome (FBSS). Cont Neurosurg 12: 23–28
34. Bundschuh CV, Modic MT, Ross JS, Masaryk TJ, Bohlman H (1988) Epidural fibrosis and recurrent disk herniation in the lumbar spine: MR imaging assessment. AJNR 9: 169–178
35. Frocrain L, Duvauferrier R, Husson J-L, Noel J, Ramee A, Pawlotsky Y (1989) Recurrent postoperative sciatica: evaluation with MR imaging and enhanced CT. Radiology 170: 531–533
36. Sotiropoulos S, Chafetz NI, Lang P, et al (1989) Differentiation between postoperative scar and recurrent disk herniation: prospective comparison of MR, CT, and contrast-enhanced CT. AJNR 10: 639–643
37. Bundschuh CV, Stein L, Slusser JH, Schinco FP, Ladaga LE, Dillon JD (1990) Distinguishing between scar and recurrent herniated disk in postoperative patients: value of contrast-enhanced CT and MR imaging. AJNR 11: 949–958
38. Weber H (1994) The natural history of lumbar disc herniation and the influence of intervention. Spine 19: 2234–2238
39. Komori H, Okawa A, Haro H, Muneta T, Yamamoto H, Shinomiya K (1998) Contrast-enhanced magnetic resonance imaging in conservative management of lumbar disc herniation. Spine 23: 67–73
40. Kischer WC, Shetlar MR (1979) Microvasculature in hypertrophic scars and the effects of pressure. J Trauma 19: 757–764
41. Van De Kelft E, Van Goethem JWM, De La Porte C, Verlooy J (1996) Early postoperative gadolinium-DTPA-enhanced MR imaging after successful lumbar discectomy. Br J Neurosurg 10: 41–49
42. Fraser RD, Osti OL, Vernon-Roberts B (1986) Discitis following chemonucleolysis: an experimental study. Spine 11: 679–687
43. Guyer RD, Collier R, Stith WJ (1988) Discitis after discography. Spine 13: 1352–1354
44. Scherbel AL, Gardner WJ (1960) Infections involving the intervertebral disks: diagnosis and management. JAMA 174: 370–374
45. Lindholm TS, Pylkkanen P (1982) Diskitis following removal of intervertebral disk. Spine 7: 618–622
46. Bircher MD, Tasker T, Crawshaw E, Mulholland RC (1988) Discitis following lumbar surgery. Spine 13: 98–102
47. Tronnier V, Schneider R, Kunz U, Albert F, Oldenkott P (1992) Postoperative spondylodiscitis: results of a prospective study about the aetiology of spondylodiscitis after operation for lumbar disc herniation. Acta Neurochir 117: 149–152
48. Grane P, Josephsson A, Seferlis A, Tullberg T (1998) Septic and aseptic post-operative discitis in the lumbar spine – evaluation by MR imaging. Acta Radiol 39: 108–115
49. Fouquet B, Goupille P, Jattiot F, et al (1992) Discitis after lumbar disc surgery, features of "aseptic" and "septic" forms. Spine 17: 356–358
50. Rohde V, Meyer B, Schaller C, Hassler WE (1998) Spondylodiscitis after lumbar discectomy. Spine 5: 615–620
51. Dall BE, Rowe DE, Odette WG, Batts DH (1987) Postoperative discitis. Clin Orthop 224: 138–146
52. Grollmus J, Perkins RK, Russel W (1974) Erythrocyte sedimentation rate as a possible indicator of early disc space infection. Neurochirurgia 17: 30–35
53. Kapp JP, Sybers WA (1979) Erythrocyte sedimentation rate following uncomplicated lumbar disc operations. Surg Neurol 12: 329–330
54. Meyer B, Schaller K, Rohde V, Hassler W (1995) The C-reactive protein for detection of early infections after lumbar microdiscectomy. Acta Neurochir 136: 145–150
55. Modic MT, Steinberg PM, Ross JS, Masaryk TJ, Carter JR (1988) Degenerative disk disease. Assessment of changes in vertebral body bone marrow with MR imaging. Radiology 166: 193
56. Grand CM, Bank WO, Balériaux D, Matos C, Levivier M, Brotchi J (1993) Gadolinium enhancement of vertebral endplates following lumbar disc surgery. Neuroradiology 35: 503–505
57. Aguila LA, Piraino DW, Modic MT, Dudley AW, Duchesneau PM, Weinstein MA (1985) The intranuclear cleft of the intervertebral disk. Magnetic resonance imaging. Radiology; 155: 155
58. Van Goethem JWM, Parizel PM, Van den Hauwe L, Van de Kelft E, Verlooy J, De Schepper AMA (2000) Value of MR imaging in the diagnosis of postoperative spondylodiscitis. Neuroradiology 42: 580–585
59. Schulitz KP, Assheuer J (1994) Discitis after procedures on the intervertebral disc. Spine 19: 1172–1177
60. Boden SD, Davis DO, Dina TS, Sunner JL, Wiesel W (1992) Postoperative diskitis: distinguishing early MR imaging findings from normal postoperative disk space changes. Radiology 184: 765–771
61. Demaerel P, Van Ongeval C, Wilms G, Lateur L, Baert A (1994) MR imaging of spondylitis with gadopentate dimeglumine enhancement. J Neuroradiol 21: 245-251
62. Onik G (1996) Automated percutaneous biopsy in the diagnosis and treatment of infectious discitis. Neurosurg Clin North Am 7: 145-150
63. Jinkins JR (1993) Magnetic resonance imaging of benign nerve root enhancement in the unoperated and postoperative lumbosacral spine. Neuroimag Clin North Am 3: 525–541
64. Guiard JM, Bonnet JC, Boutin JP, Plane D, Guilleux MH, Delorme G (1988) Textile foreign body, "textiloma": CT features. Case report. Ann Radiol 31: 49–52

65. Williams RG, Bragg DG, Nelson JA (1978) Gossybipoma – The problem of the retained surgical sponge. Radiology 129: 323–326
66. Van Goethem JWM, Parizel PM, Perdieus D, Hermans P, de Moor J (1991) MR and CT imaging of paraspinal textiloma (gossybipoma). J Comput Assist Tomogr 15: 1000–1003
67. De Marco K, McDermott M, Dillon W, Bollen A, Edwards M (1991) MR appearance of postoperative foreign body granuloma: case report with pathologic confirmation. AJNR 12: 190–192
68. Parizel PM, Van Goethem JWM, van den Hauwe L, Deckers F, Gunzburg R, De Schepper AMA (1996) Imaging of spinal implants and radiologic assessment of fusion. In: Szpalski M, Gunzburg R, Spengler M, Nachemson A (eds) Instrumented fusion of the degenerative lumbar spine: state of the art, questions, and controversies. Lippincott-Raven, Philadelphia, pp 25–33
69. Ghazi J, Golimbu CN, Engler GL (1992) MRI of spinal fusion pseudarthrosis. J Comput Assist Tomogr 16: 324–326
70. Blumenthal SL, Gill K (1993) Can lumbar spine radiographs accurately determine fusion in postoperative patients? Correlation of routine radiographs with a second surgical look at lumbar fusions. Spine 18: 1186–1189
71. Slone RM, MacMillan M, Montgomery WJ, Heare M (1993) Spinal fixation. Part 2. Fixation techniques and hardware for the thoracic and lumbosacral spine. Radiographics 13: 521–543
72. Slone RM, MacMillan M, Montgomery WJ (1993) Spinal fixation. Part 3. Complications of spinal instrumentation. Radiographics 13: 797–816
73. Coughlan JD (1986) Extrusion of bone graft after lumbar fusion: CT appearance. J Comput Assist Tomogr 10: 399–400
74. Brodsky AE, Kovalsky ES, Khalil MA (1991) Correlation of radiologic assessment of lumbar spine fusions with surgical exploration. Spine 16: S261-S265
75. Slone RM, MacMillan M, Montgomery WJ (1993) Spinal fixation. Part 1. Principles, basic hardware, and fixation techniques for the cervical spine. Radiographics 13: 341–356
76. Parizel PM, Özsarlak Ö, Van Goethem JWM, van den Hauwe L, De Schepper AMA (1999) The use of magnetic resonance imaging in lumbar instability. In Szpalski M, Gunzburg R, Pope M (eds) Lumbar segmental instability. Lippincott-Raven, Philadelphia, pp 123–138
77. Lang P, Chafetz N, Genant HK, Morris JM (1990) Lumbar spinal fusion. Assessment of functional stability with magnetic resonance imaging. Spine 15: 581–588
78. Cleveland M, Bosworth DM, Thompson FR (1948) Pseudarthrosis in the lumbosacral spine. J Bone Joint Surg 30A: 302–312
79. Lang Ph, Genant HK, Chafetz NI, Hedtmann A, Norman D, Krämer J (1988) Magnetresonanztomographie bei Spondylolyse und Spondylolisthese. Z Orthoped 126: 651–657
80. Ross JS, Hueftle MG (1989) Postoperative spine. In: Modic MT, Masaryk TJ, Ross JS (eds) Magnetic resonance imaging of the spine. Year Book Medical Publishers, Chicago, 120–148