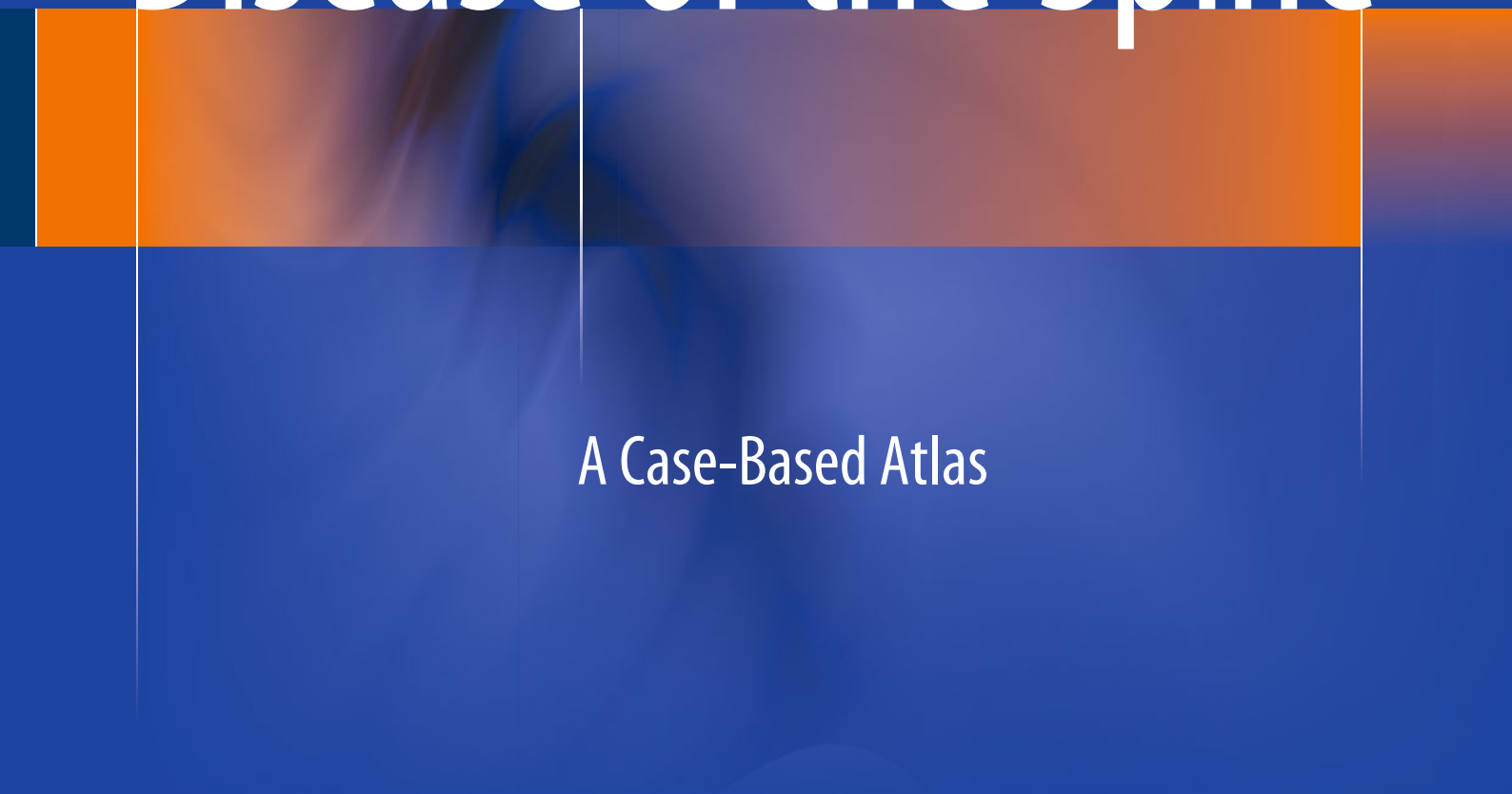
Paola D'Aprile

# MRI of Degenerative Disease of the Spine

## A Case-Based Atlas



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## A Case-Based Atlas

In collaboration with Alfredo Tarantino

Springer

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# Foreword

Medical knowledge has tremendously increased in the last decades. Molecular biology, genetics, and medical imaging have completely transformed our approach to diseases. In many fields, the quality of our patient's life has improved; the prognosis of dreadful diseases has dramatically changed. Back pain, however, and its principal cause, degeneration of the spine components, still remains one of the most diffuse painful conditions for mankind. Despite the dramatic improvement in diagnosis introduced first by CT in the 70s, followed by MR in the 80s, much remains to be discovered to understand biological bases of degeneration, and much remains to be done for its treatment. Understanding of basic biomechanical principles is well established, but the role of inflammation and degeneration still lacks a comprehensive explanation.

For this reason, any attempt to propose new modalities to increase the informative value of the available diagnostic tools is welcome as well as the effort to communicate the personal interpretation of the results to the medical community based on a wide daily clinical experience and verification. Paola D'Aprile and Alfredo Tarantino's book is such an attempt. Despite the existence of numerous books on the radiological examination of the spine, this laudable effort merits to be acknowledged and its diffusion encouraged.

The book is mainly an atlas with 65 clinical cases well documented and well interpreted. Emphasis is placed on the technical aspects of performing MR, with a strong suggestion to use contrast agents as well as STIR or fat suppressed sequences. An accurate analysis of images implies also to pay attention not only to disc profile, facet joints, and neural spaces but also to ligaments and paraspinal muscles, particularly post gadolinium, in search for signs of inflammation and oedema.

Our concepts of spine pathology should continuously be challenged by new diagnostic (and therapeutic) approaches. Paola D'Aprile's accurate and original study is such a challenge: combining imaging approach with clinical approach, the aim is to widen the interpretation of images from morphology to pathophysiology. New insights in the future might come from "metabolic" approaches such as spectroscopy and diffusion.

This book should be of interest not only to radiologists and neuroradiologists but also to neurosurgeons and orthopedic surgeons. All physicians, however, could take advantage from its reading since management of back pain is frequently dealt with by general practitioners.

Giuseppe Scotti
Humanitas Research Hospital
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# Preface

The passion for my work has been fed over time, in the belief that the diagnosis more fair and accurate helps the therapy and treatment of diseases of the spine and helps, so to ease the pain.

The pain is part of life, but to live and deal with, it helps to know the mind and soul to appreciate life more. I devote myself, always in the search for a precise and accurate diagnoses, cause I am convinced that the care and treatment of pain depends on the exact diagnosis and to be aware that you can help many people in search of the cure more appropriate and in pain relief, makes me love more and more the neuroradiology.

The spine degenerative disease includes a series of arthrosic-degenerative processes, which generally affect mechanical supports, resulting from a natural aging/worn process.

These processes can be localized in the anterior vertebral compartment (vertebral-disc unit) and in the posterior vertebral compartment (interapophyseal joints, spinal ligaments and paravertebral muscles).

The main clinical onset of this disease is pain which is often localised in the lumbar region and can also cause impaired mobility.

The Magnetic Resonance is the gold standard imaging for the study of degenerative disease of the spine for its accuracy and diagnostic efficacy.

The study protocol include RM fat saturation sequences T2, since more sensitive to the pattern of edema, also with administration of gadolinium in some cases; even the administration of gadolinium for more effective diagnostic as well as a more accurate assessment of the extent of the process and pathological involvement of the soft tissues.

In the diagnostic process with MR are very important to check carefully all anatomic elements of the spinal as well as muscular and ligamentous supports. It is very important to make a correlation between the clinical data and the results of the MR in order to find out the cause of the pain.

The radiologist must have a cultural “background” to identify the several aspects of the degenerative disease. The examination should also targeted in order to identify the real cause of the pain and consequently identify the therapeutic “targets”.

The disc-radicular conflict is one of the most common pathologies. Some aspects of degenerative diseases must be assessed carefully within the spinal functional unit.

This case-based atlas aims at showing less common aspects of the degenerative disease highlighted with the MR mainly in their active-inflammatory phase.

A correct diagnosis allows a specific therapeutic procedure, even in consideration of the many treatments analgesic we have today.

Bari, Italy — Paola D’Aprile

# Acknowledgements

Thank you with respect, gratitude and friendship Dr. Randy Jinkins for his teachings and for giving me the passion and interest to the spine.

I thank all those who will read this book with the hope that it will be a little help and comparison in the daily research of the diagnosis.

I thank my colleagues, the technical staff at the MRI, the administrative and nursing staff, without whose co-operation and dedication I would not have been able to work peacefully and in harmony, respecting the patient, always trying to give affirmative and appropriate answers to all those who have chosen to undergo neuroradiological examinations in our Department.

I would also like to thank Springer and in particular Antonella Cerri and Andrea Ridolfi for showing trust in my project.

Bari, Italy Paola D'Aprile

# Contents

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# Part I

## Clinical and Technical Aspects

# Biomechanics of the Spine

# 1

## 1.1 Spine Stability

The spine is a multiarticular structure composed by motion segments (MS) whose correct function has as prerequisite the stability.

There exist in literature a number of definitions of stability.

The American Academy of Orthopedic Surgeons defined it as "the capacity of the vertebrae of remaining cohesive in all physiological body movements" [1].

White and Panjabi considered the clinical stability as "the ability of the spine under physiologic loads to limit patterns of displacement to not damage or irritate the spinal cord and nerve roots and to prevent incapacitating deformity or pain caused by structural changes" [2].

Spinal stability is a fundamental property for the protection of nervous elements, the active generation of forces in the body trunk and the transfer of them between the upper and lower limbs, the prevention of the early biomechanical deterioration of its own components and the reduction of energy expenditure during muscle action.

The key feature of the biomechanics and stability of the spine is the highly nonlinear load/displacement ratio, the effort required for movement significantly changing in its various phases [3].

In every MS the initial part of range of motion (ROM), on either sides of neutral posture, meets a scarce resistance and requires a relatively low effort, because of the general laxity status of ligaments and joint capsules. This part of motion, referred to as neutral zone (NZ), is replaced by the elastic zone (EZ) where the ligaments, capsules, fascias and tendons going in tension require more effort per unit of displacement nearing the ends of ROM.

In this way two opposed needs are met, reducing the muscle effort near the neutral posture and ensuring stability at the end of joint excursion.

Each vertebra can perform with respect to adjacent vertebrae, three translations and three rotations in relation to each of the x-, y-, z- Cartesian axes of space and various combinations of main and coupled movements.

Each movement has its ROM, NZ and EZ.

Stability implies an appropriate relationship between NZ and EZ even within a normal ROM.

The stabilization of the spine is guaranteed by a stabilization system consisting of three closely related subsystems (Fig. 1.1) [3] including:

- The spinal bones and joints or passive subsystem
- The muscles forming the active subsystem
- A central control unit, the CNS

### 1.1.1 Spinal Bones Joints and Ligaments

The passive subsystem plays a structural role, ensuring spinal stability, namely, within the elastic zone. It also has a transducer function carried out from a number of mechanoreceptors located within the joint capsules, discs and ligaments which send a continuous flow of proprioceptive inputs about the load status, spatial position and movement from each MS to CNS. On the basis of this information flow, CNS replays through an appropriate and coordinate muscle activity [4].

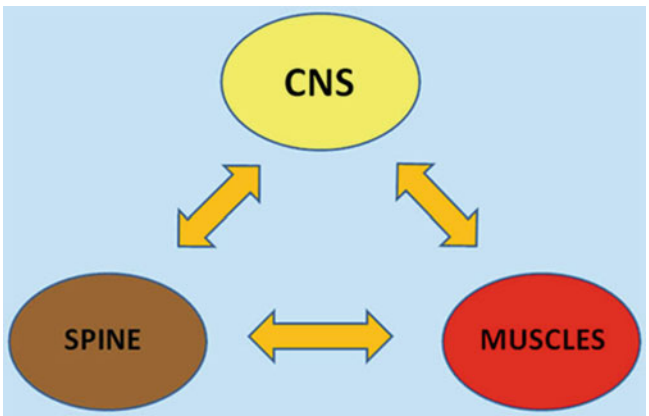


**Fig. 1.1** Three strictly related subsystems control the stability of the spine: the spinal column, the muscles and the central nervous system. In case of damage to one subsystem, more compensatory work is played by the others in order to preserve stability

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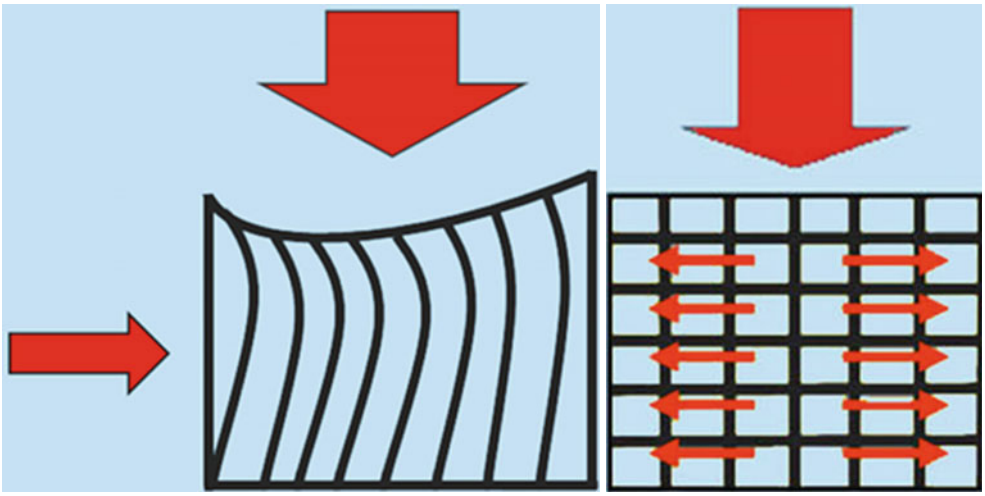

**Figs. 1.2 and 1.3** The vertical compressive loads are first accepted by vertical trabecular columns which transmit forces between the endplates. The intrinsic tendency to bow of isolated vertical columns (Fig. 1.2) is restrained by horizontal lamellae whose tension favours the radial dispersion of forces conferring resilience to the vertebral body (Fig. 1.3)

The vertebral body is the key element of the load-bearing system. It mainly consists of spongy trabecular bone which plays a crucial role in determining the strength and resilience of the vertebral body. Compressive loads are first accepted by vertical trabecular struts joining directly the vertebral endplates. Vertical columns, if isolated, would tend to bow and lose their load-bearing capacity if these were not restrained and maintained right by tension of horizontal trabeculae (Figs. 1.2 and 1.3). It is this conversion of vertical loads in transverse tensions that confers weight-bearing resistance to vertebral body.

A solid bony block would be heavy and, like all crystalline structures, unable to bear dynamic loads which could fracture it, whereas, conversely, a box of cortical shell would easily collapse under compressive forces because of scarce resilience of cortical bone. Cancellous bone guarantees the best load/resistance ratio.

The resistance of vertebral spongiosa either depends on trabecular architecture and bone density.

The vertebral spongiosa of any vertebra contains four main trabecular systems having a quite constant orientation: a vertical system between endplates which transmits axial loads, a curved system running in neural arc and two curved systems joining the endplates with the articular and spinous processes which anchor the neural arc to vertebral body resisting shearing forces (Fig. 1.4).

The bone loss in osteoporosis results in an exponential reduction of resistance. During early stages of osteoporosis, the elective reabsorption of transverse connections leads to a progressive relative elongation of vertical columns, whose resistance decreases by the square of length. Later, the thinning of columns themselves also contributes to a quadratic loss of strength with a summation of both effects.

Out of any pathological change, vertebral endplates can fail by fracturing following repeated stresses.

When a vertebral body is compressed, a part of the energy applied to deform it will be lost and, when the load is removed, less energy is available for it to restore the initial shape, which is so not immediately regained. This mechanical phenomenon, defined as *hysteresis*, expresses the amount of energy lost during the deformation of any solid structure. By virtue of hysteresis, a structure regains its original shape at a rate and extent different to that at which it was deformed. If forces are applied with many repetitions and sufficient frequency, the vertebral body, like any material, is unable to recover and accumulate small weaknesses, losing gradually stiffness, and, finally, fails under a stress much less than that could damage it if applied a single time [5].

Failure fractures of the vertebral endplates may occur after as few as 30–80 applications of forces corresponding to 50–80 % of ultimate compressive strength (normally between from 3,000 to 10,000 N). These forces are within the ranges occurring during normal daily activities [6].

Endplate fractures are now considered a possible cause of internal disc disruption and discogenic pain [7].

The annulus acts as the first ligament for restraining the complex 3-D movements a vertebra can perform.

The oblique alternating arrangement of collagen fibres between adjacent lamellae (with a degree of 65–70° to endplate plane) and the degree of obliquity of fibres within each lamella both optimize the capacity to restrain movements in all directions. A steeper orientation of lamellar fibres would increase the resistance to distraction, but it would compromise that to sliding and twisting; a flatter inclination would enhance control of twisting, but it would lessen that of distraction and bending.

Adjacent to vertebral endplates, the disc acts as *shock absorber* of the forces transmitted to the head and brain during walking and jumping.

Both nucleus and inner annulus are involved in weight-bearing. The nucleus pulposus mainly works in the NZ bearing low axial loads, while the stiffer annulus fibrosus accepts a larger proportion of the highest loads. The densely packed lamellae confer compression stiffness to annulus, but, over

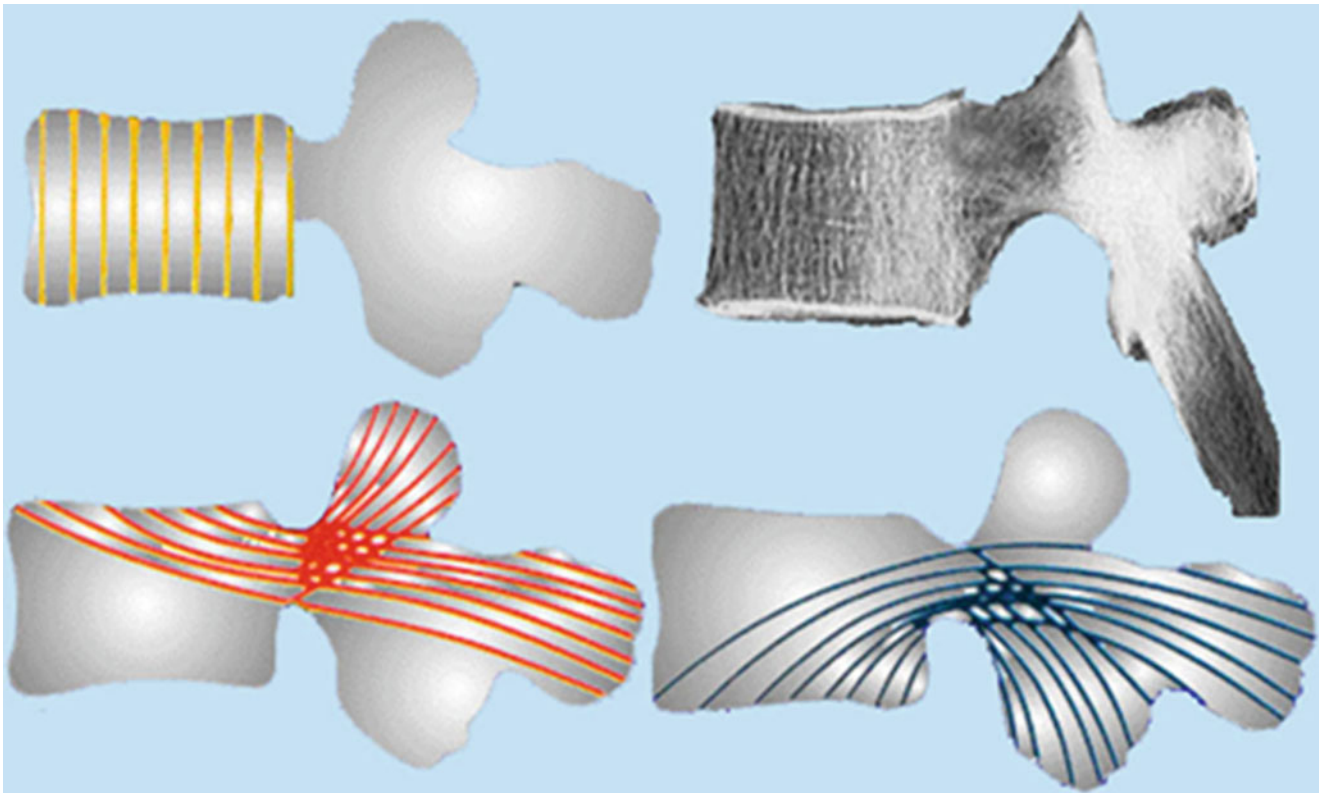

**Fig. 1.4** The load-bearing capacity of the vertebral body depends on trabecular architecture. The vertebral spongiosa contains four main trabecular systems including a vertical system between endplates, a curved system running in neural arc and two curved oblique systems joining the endplates with the articular and spinous processes which anchor the neural arc to vertebral body resisting shearing forces

time, an isolated annulus under sustained loads tends to buckle and collapse.

The nucleus provides an internal bracing mechanism. Like a ball of fluid, the nucleus can be deformed but not compressed. Under axial compressive load it expands radially stretching the annulus and pushing away the opposite endplates. The vertical pressure transmits part of load from one vertebra to the next, lessening the tension created in the annulus fibrosus. The radial pressure stretches and braces the annulus preserving the vertical orientation of lamellae, avoiding their buckling and increasing their capacity to transmit loads [8] (Fig. 1.5). The cooperative action of the nucleus and the annulus enhances the disc capacity of bearing higher and prolonged loads.

This mechanism is lost in case of degenerative depressurization of nucleus pulposus that leads to external and internal buckling of annular lamellae. Buckling accelerates and favours the further annulus disruption (Fig. 1.6).

In addition, under compression, a part of energy is used and stored to stretch the annulus and then released soon after the discharge of the spine. This momentary diversion of energy attenuates the speed of transmission of the force from a vertebra to another, preserving the adjacent endplates.

Under very high compressive loads, the first structure to fail is usually the endplate rather than the disc.

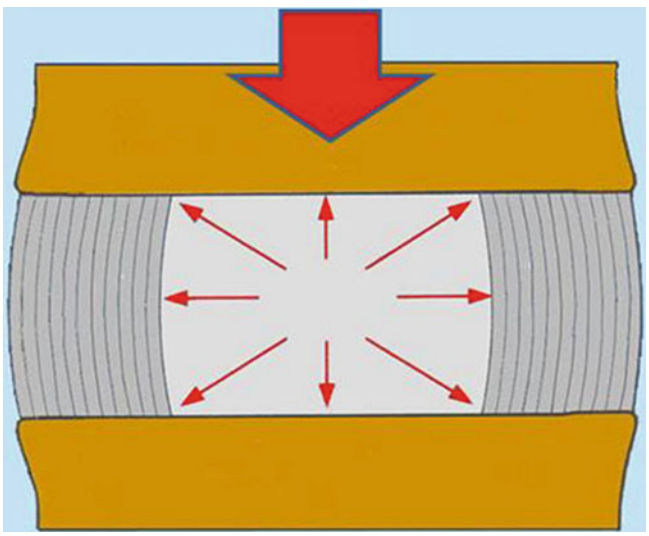

**Fig. 1.5** A well-pressurized hydrated nucleus pulposus provides an internal bracing to annulus lamellae aiding annulus to contribute to weight-bearing. The cooperative action consents to disc to bear sustained and higher loads

The VB is exclusively designed to bear longitudinal loads and has no features that confer stability to intervertebral joints and limit movements within the horizontal plane [8].

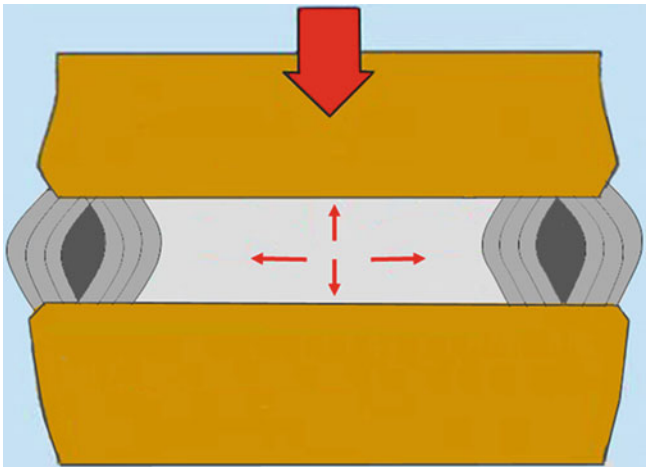

**Fig. 1.6** As a consequence of disc degeneration, the nucleus pulposus loses its internal pressure favouring the collapse, buckling and tearing of annulus

For the stability in the horizontal plane, the VBs are totally dependent on the posterior elements.

The facet joints fulfil two basic functions:

- Control of the direction and amplitude of movement
- Sharing of loads

Facet joints resist sagittal translation and limit extension and axial rotation protecting the discs from torsion injuries which normally occur over only 3° of rotation.

The spatial orientation and symmetry of the facets are essential requirements for correct functioning. Every significant asymmetry predisposes to instability and premature degeneration of the facets and discs.

With regard to acceptance of loads, according to the three-column model by Louis, the weight of the head and trunk is transmitted from C3 to L5 upon three columns arranged like a triangle, with the anterior column being composed of vertebral bodies and discs and the posterior columns of the vertical succession of the facet joints [9]. The columns have a balanced action for which the posterior facets, depending on the posture, born from 0 % up to 33 % of the load, but, in case of hyperlordosis, high and prolonged weight load and disc degeneration, the load can raise up to 70 %.

In the passive subsystem the ligaments and joint capsule act as passive stabilizers of MSs.

The biomechanical efficacy of any ligament depends not only on its intrinsic strength but also and more on length of lever arm by which acts, the distance between the bony insertion and the instantaneous axis of rotation (IAR) of the vertebra which locates in the posterior part of vertebral body.

Specifically, the ISL and SSL, being located far apart from the IAR, work by a long lever arm and resist the spinal flexion more than the FL and PLL which function with shorter lever arms.

### 1.1.2 The Muscles and Central Control Unit

The stabilizing role of facets and ligament is flanked by muscles.

The muscles stabilize the spine actively and mainly operate within the neutral zone where ligaments and capsule are relaxed.

Each of the back muscles is capable of several actions. No action is unique to a muscle and no muscle has a single action

While short segmentary muscles (multifidus, interspinous, intertransverse) act, namely, as force transducer, long muscles are mainly responsible for the genesis of movements.

The muscles contribute to stability in two ways.

By compressing spinal segments, they limit joints motion, decreasing either the ROM or NZ [10].

The muscles also pull directly displacing segments, but, by virtue of their longitudinal orientation, they resist much better sagittal rotation but do not resist with same efficacy to translations or twisting.

The central control unit, the CNS, receives extensive inputs from all of bones, joints and muscles and tendons in order to regulate and coordinate in time and space the muscular posture and movement.

In case of damage to soft tissues and to mechanoreceptors after a single or repetitive trauma, corrupted transducer signals are sent to the CNS causing an inappropriate motor response with reduced temporal and spatial coordination.

The altered muscle response, in turn, increases the mechanical stress of bony and joint components and elicits an abnormal feedback response by FSUs and muscles themselves [11].

A vicious circle finally creates that leads to the development of inflammation, muscle fatigue and activation of nociceptors with onset and perpetuation of pain.

Patients with chronic low back pain have a delayed muscle response and offset in performing movements as well as a reduced postural control compared to asymptomatic subjects [12].

While the transverse abdominis and multifidus muscles stabilize the spine before initiating movement or accepting a load, in patients suffering from chronic spinal pain, such contractions are delayed [12].

## 1.2 Spine Instability

Spine instability is a frequent and often misdiagnosed cause of neck and back pain and disability.

Like stability, it also lacks a general universally accepted definition.

White and Panjabi defined instability as "the loss of the ability of the spine, under physiologic loads, to maintain its patterns of displacement so there is no initial or additional neurologic deficit, no major deformity, and no incapacitating pain" [13].

Pope and Panjabi qualified instability as a loss of stiffness leading to abnormal and increased movement in the MS [14].

Even though the location and type of lesion in the MS determines the pattern of instability, as spinal movement is three-dimensional with coupled movements, tissue derangement tends to cause dysfunctional motions in more than one direction.

While the classical definitions of instability refer to a global increase of joint excursion, over the normal limits (terminal instability), abnormality can manifests not at the endpoints, but only during a step of range of motion, modifying the quality of motion.

In fact, the instability is not an all-or-nothing phenomenon and a complete instability is rare. Each spinal component concurs to stability, and a number of studies have tested the contribution of every single structure.

In many clinical and experimental situations, instability consists in an enlarged neutral zone with excessive displacement early in range under minor loads (looseness), despite a normal ultimate joint strength.

In the light of relative importance of the NZ, Panjabi redefined instability as the reduced ability by the stabilizing systems of the spine to maintain the neutral zones of the MSs within physiological limits so that deformity, neurological deficit or disabling pain does not occur [3].

When an MS undergo a loss of stiffness, owing to a reduction of one of its restraints that resists a given movement, under an external force it can have acceleration and a momentary lapse of control in early, middle or late range which is felt as instability [15].

Instability can stem from a mismatch between a pattern of motion programmed for a given usual movement and the proprioceptive feedback. The CNS relieves the mismatch and replays with a sudden muscle reflex experienced as a jerk or a catch [15].

### 1.2.1 Degenerative Instability

Degenerative instability is considered a common cause of acute and chronic spinal pain and disability as well as a frequent indication for surgery.

Degenerative instability has been defined as a change in vector forces relating the MSs generating abnormal, imbalanced and paradoxical movements.

A degenerative primum movens, involving generally the disc, primes disorders of movement which, in turn, increase the original bony, articular and biomechanical abnormalities and extends them to other joints of the same and adjacent segments transforming a segmental into a regional pathology.

Because of degenerative disc collapse, the annulus and ligaments become lax and redundant favouring anterior, posterior and vertical subluxation of the vertebral bodies.

During the evolution of degenerative instability, Kirkaldy-Willis and Farfan distinguished three main biomechanical and clinical phases forming a so-called degenerative cascade: painful dysfunction, instability and restabilization [16].

During the instability phase several radiological signs can be found including endplate oedema (Modic type 1 changes), peduncle and isthmus oedema, traction spurs [17], extended discal vacuum associated with mild disc space narrowing, joint effusion and facet joint gapping over 1 mm [18], synovial cysts, anterolisthesis and retrolisthesis [16].

All these signs of conventional imaging are indirect and their specificity and the clinical relevance for diagnosing instability are not consistent in the different reports and need to be established definitively.

Open MR systems allow positional-dynamic studies in either standing or seated positions to detect increased and abnormal intersegmental movements.

Positional-dynamic MRI studies may disclose dysfunctional movements which can worsen or uncover a central canal, lateral recesses and/or foraminal stenosis, disc protrusion or disc extrusion [19].

By comparing the results of traditional and functional MR in a small cohort of patients, Smith reported abnormal findings detected only by dynamic studies in 52 % of patients with appropriate successful treatment in all cases of LBP and sciatica [20].

Axial-loaded CT (AL-CT) and MR (AL-MR) simulate the weight-bearing upright position and depict several findings including hypermobility, appearance of or increase in listhesis and the appearance or increase of canal and/or foraminal stenosis [21]. These findings can be observed alone (elementary modifications) or coexist in various patterns referred to as complex dynamic modifications [21].

Abnormal motion patterns in axial-loading imaging tend to evolve in a quite stereotyped way up to degenerative listhesis [21]. A basic drawback of axial-loading imaging is the impossibility to reflect postural changes related to muscle tone and physiological loads that are not uniform at different levels but increase in the caudal direction along the lumbar spine, so upright/positional MR is considered to outweigh axial-loaded MR and CT.

When spinal degeneration further progresses, in the final phase of "restabilization", the fibrosis of the joint capsules, the formation of osteophytes, the marked discal collapse and the remodelling of vertebral bodies increase stiffness and favour an overall reduction of mobility.

The final phase of restabilization is associated with significant disc collapse, radial remodelling of vertebral bodies, claw and "wrap around bumper" osteophytes [22], Modic type 3 changes, facet sclerosis and neoarthrosis between the spinous processes [21].

A spontaneous restabilization may occur even in case of spondylolisthesis whose presence does not always imply actual instability [16]. Disc collapse and osteophytes may block the progression of slippage, often with secondary improvement of pain. In effect, despite widespread opinion, spondylolisthesis is not always associated with increased motion

A 10-year follow-up study found progression of slippage only in 30 % of cases and 65 % of patients who were initially neurologically normal did not worsen and could be treated conservatively [23].

The relation between instability and pain has been long debated and often is hard to be demonstrated. In a hypermobile segment pain can be aggravated by the movement because of excessive engagement of irritated restraints, not necessarily by instability which can be present as an independent and parallel phenomenon.

According to Mulholland, abnormal movements detectable in a degenerative spine are not necessarily the cause of pain and instability is often a myth [24]. In many cases spinal pain is due to an abnormal, irregular distribution of loads between joint surfaces. In fact, pain may persist after technically successful fixations or unexpectedly resolves also in cases of pseudarthrosis. Pain seems to be often elicited primarily from load stresses caused by the posture or powerful muscle contraction such as that of the erector muscles during lifting tasks rather than by movement itself.

As stressed by Bogduk, instability is a biomechanical term and a diagnosis too often abused.

The concept of enlarged neutral zone may contribute to justify the many cases of suspected but not ascertained instability.

Despite all efforts in this clinical topic, a definitive diagnosis remains often elusive.

**Acknowledgments** I would like to thank Dr. *Roberto Izzo* for his contribution in writing this chapter.

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# MRI in Degenerative Disease of the Spine

# 2

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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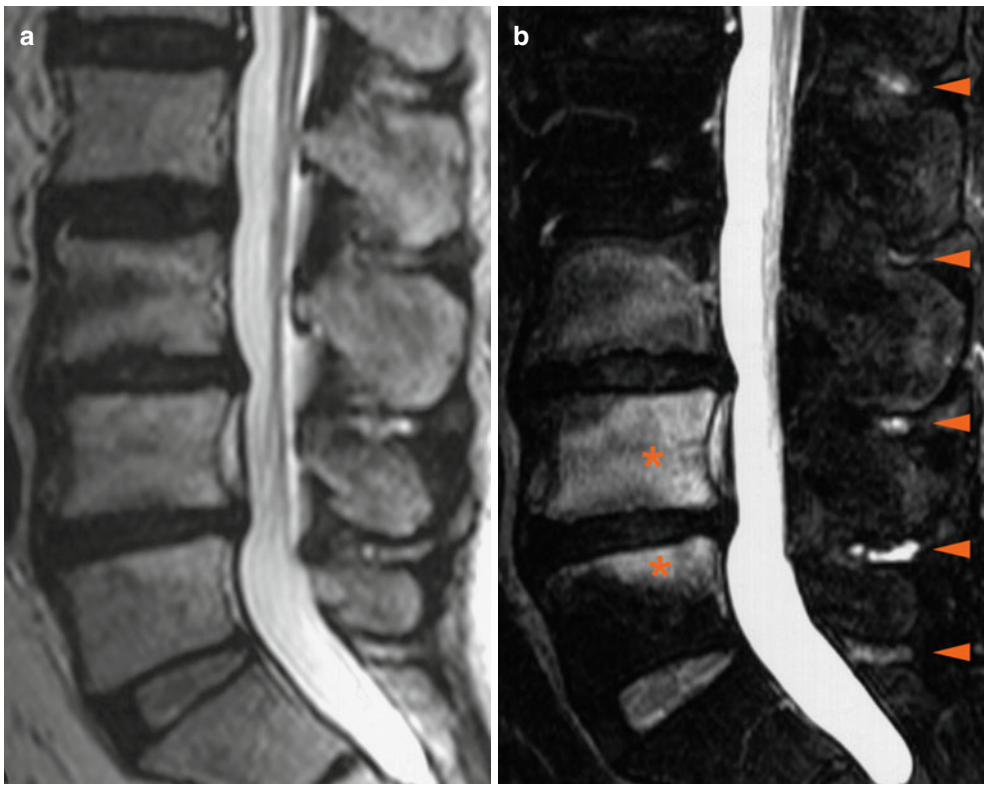


**Fig. 2.1** (**a**, **b**) "Standard" sagittal TSE T2-weighted image without fat saturation (**a**) and sagittal TSE T2-weighted image with fat saturation (**b**). Hyperintense areas in fat suppressed T2-weighted image in the L3-L4-L5 vertebral bodies, indicating oedema of the bone marrow (**b**, *asterisks*).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

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

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

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

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

## 2.1 Disc Degeneration

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

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

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

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

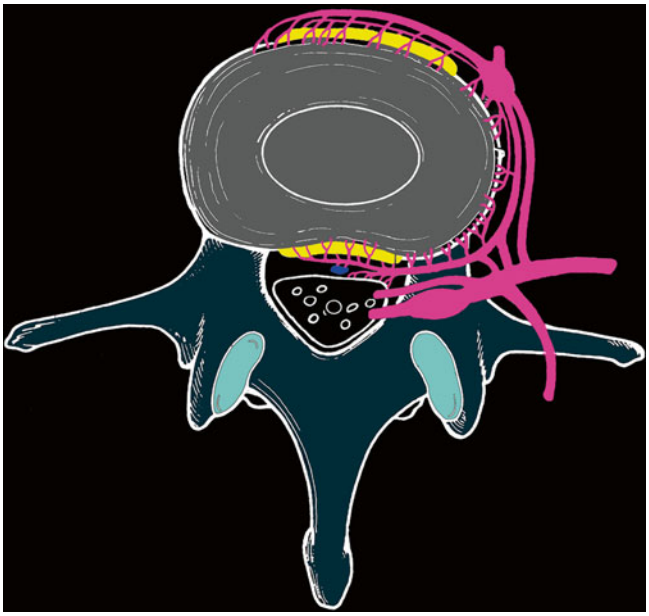

**Fig. 2.2** Schematic figure depicting the rich innervation of the disc-vertebral body complex (modified from Jinkins)

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

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

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

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

## 2.2 Disc Herniation

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

## 2.3 Canal Stenosis

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

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

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

The lumbar and cervical regions are most commonly affected.

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

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

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

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

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

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

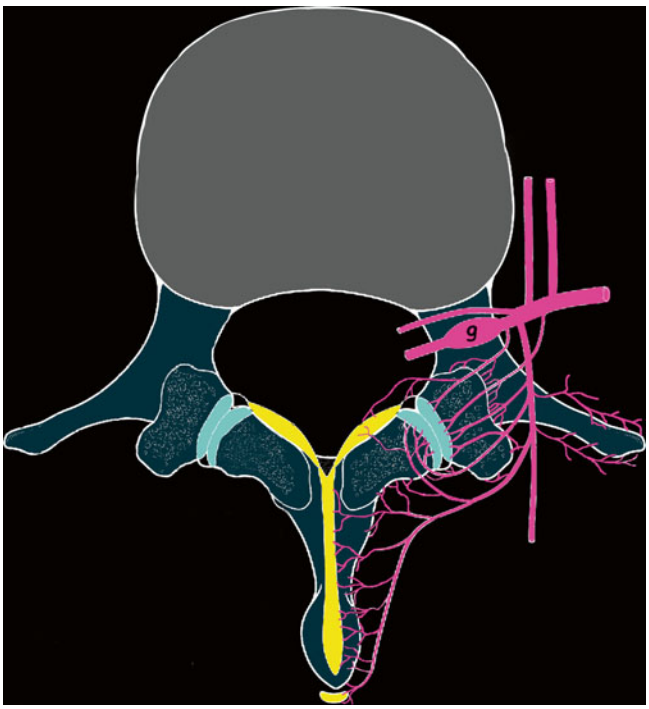


**Fig. 2.3** Schematic figure depicting the rich innervation of the posterior vertebral compartment, arising from the dorsal branch of the spinal nerve (g = spinal ganglion) (modified from Jinkins)

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

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

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

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

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

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

## 2.5 Spondylolysis

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

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

## 2.6 Spinal/Perispinal Ligamentous Degenerative-Inflammatory Changes

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

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

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

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

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

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

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

## 2.7 Muscular Changes

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

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

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

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

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# Part II

# Clinical Cases

- A 44-year-old male
- Persistent low back pain
- Limitation of motion of the spine

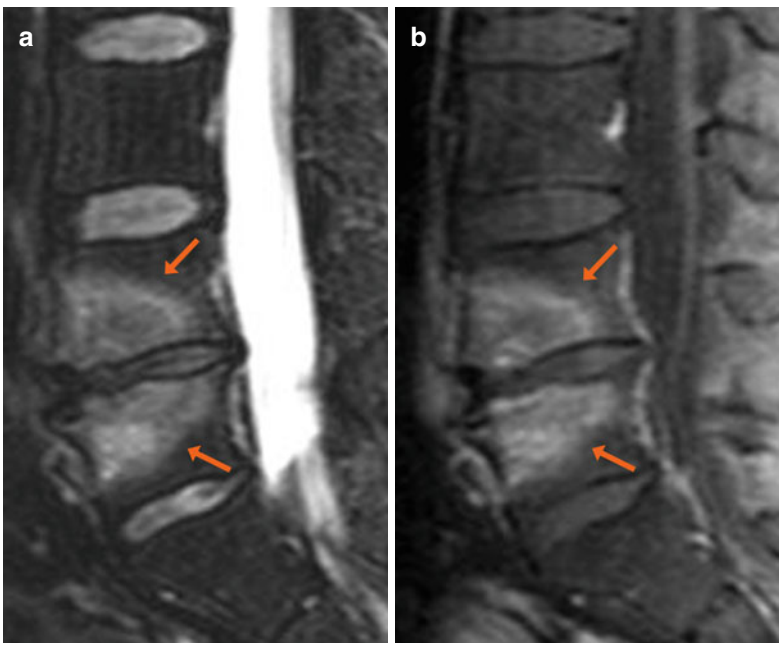


**Fig. 1** (**a**, **b**) Sagittal TSE T2-weighted image with fat saturation (**a**) and sagittal SE T1-weighted image with fat saturation following administration of contrast medium (**b**). The opposing L4 and L5 vertebral bodies present hyperintense areas on T2-weighted imaging (**a** *arrows*), with enhancement after contrast medium administration (**b** *arrows*). Reduction in height of the corresponding L4/L5 intervertebral disc. Diagnosis: osteochondrosic degeneration in active-inflammatory phase, with fibrovascular transformation of the bone marrow (Modic type I)

P. D'Aprile, *MRI of Degenerative Disease of the Spine: A Case-Based Atlas*,
DOI 10.1007/978-3-319-09447-2_3, 

# Osteochondritis

Case 2

- A 51-year-old-woman
- Chronic low back pain

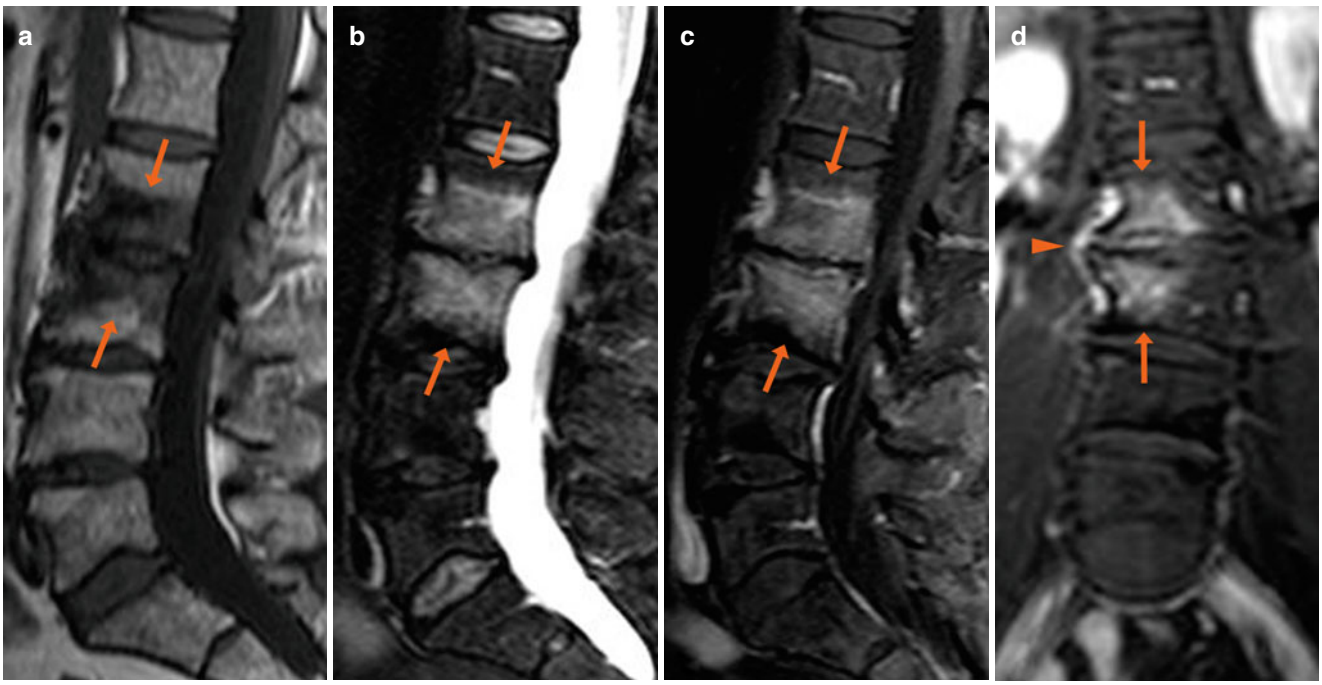


**Fig. 1** (**a**–**d**) Sagittal SE T1-weighted image (**a**), sagittal TSE T2-weighted image with fat saturation (**b**) , sagittal and coronal SE T1-weighted images with fat saturation after administration of contrast medium (**c**, **d**). Typical osteochondritis at L2/L3, with oedematous pattern of the subchondral bone, and contrast enhancement of the same areas and articular surfaces (**a**–**d**, *arrows*). Left-convex scoliosis (**d**). Note that signal changes develop on the right side of the intervertebral joint, corresponding to the point of greatest joint load in the scoliotic spine. Note also periarticular contrast enhancement on the right side at L2/L3 (**d**, *arrowhead*), indicating an aseptic reactive inflammation of the periarticular soft tissues

P. D'Aprile, *MRI of Degenerative Disease of the Spine: A Case-Based Atlas*,
DOI 10.1007/978-3-319-09447-2_4, © Springer International Publishing Switzerland 2015

- A 32-year-old woman
- Chronic low back pain
- Limitation of motion of the spine in the frontal and lateral planes

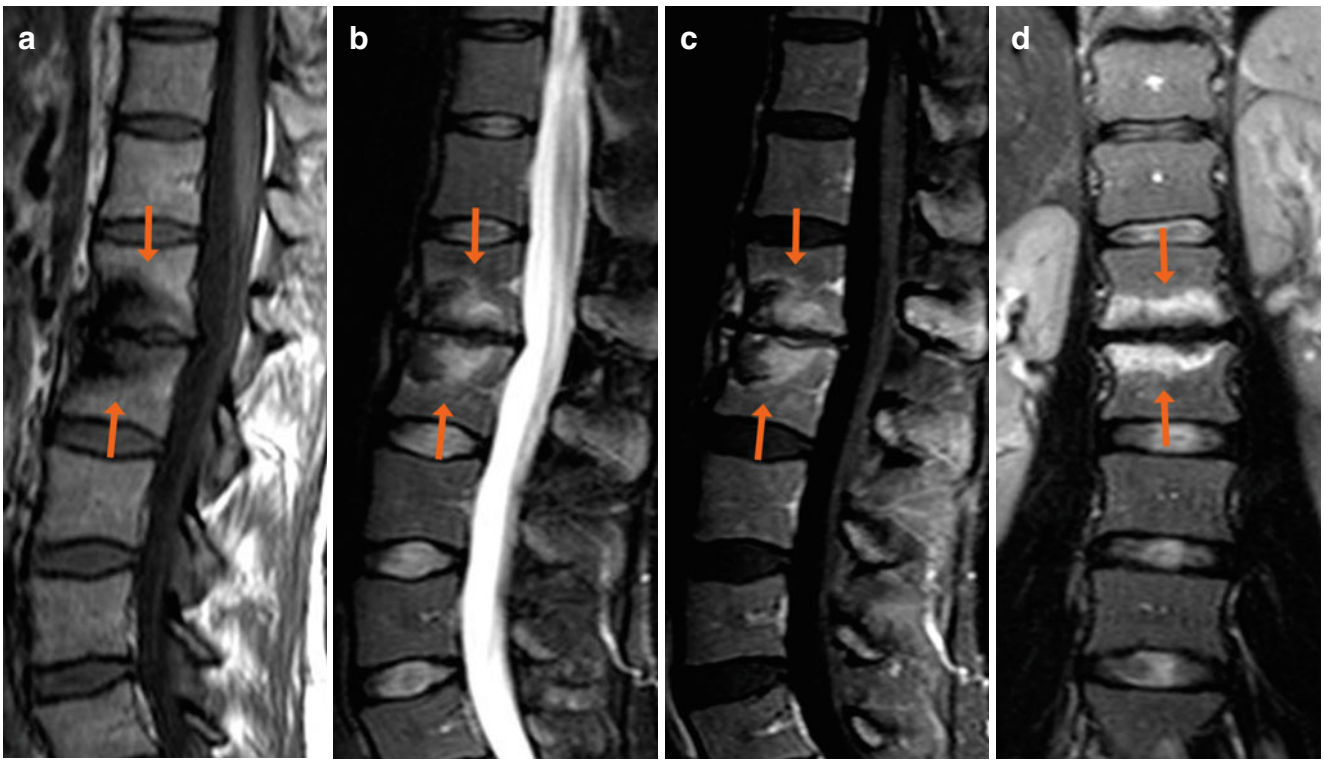


**Fig. 1** (**a**–**d**) Sagittal SE T1-weighted image (**a**), sagittal TSE T2-weighted image with fat saturation (**b**), sagittal and coronal SE T1-weighted images with fat saturation after contrast medium administration (**c**, **d**). The opposing L1/L2 vertebral bodies show hypointense signal in T1 (**a**, *arrows*) and hyposignal/hypersignal in T2 (**b**, *arrows*), with partial contrast enhancement (**c**, **d**, *arrows*). The corresponding L1/L2 intervertebral disc is collapsed. The spine presents a loss of the physiological curve at the same level. Diagnosis: osteochondrosis in active-inflammatory phase

P. D'Aprile, *MRI of Degenerative Disease of the Spine: A Case-Based Atlas*,
DOI 10.1007/978-3-319-09447-2_5, © Springer International Publishing Switzerland 2015

# Osteochondritis

Case 4

- A 48-year-old woman
- Chronic low back pain
- Improvement with rest

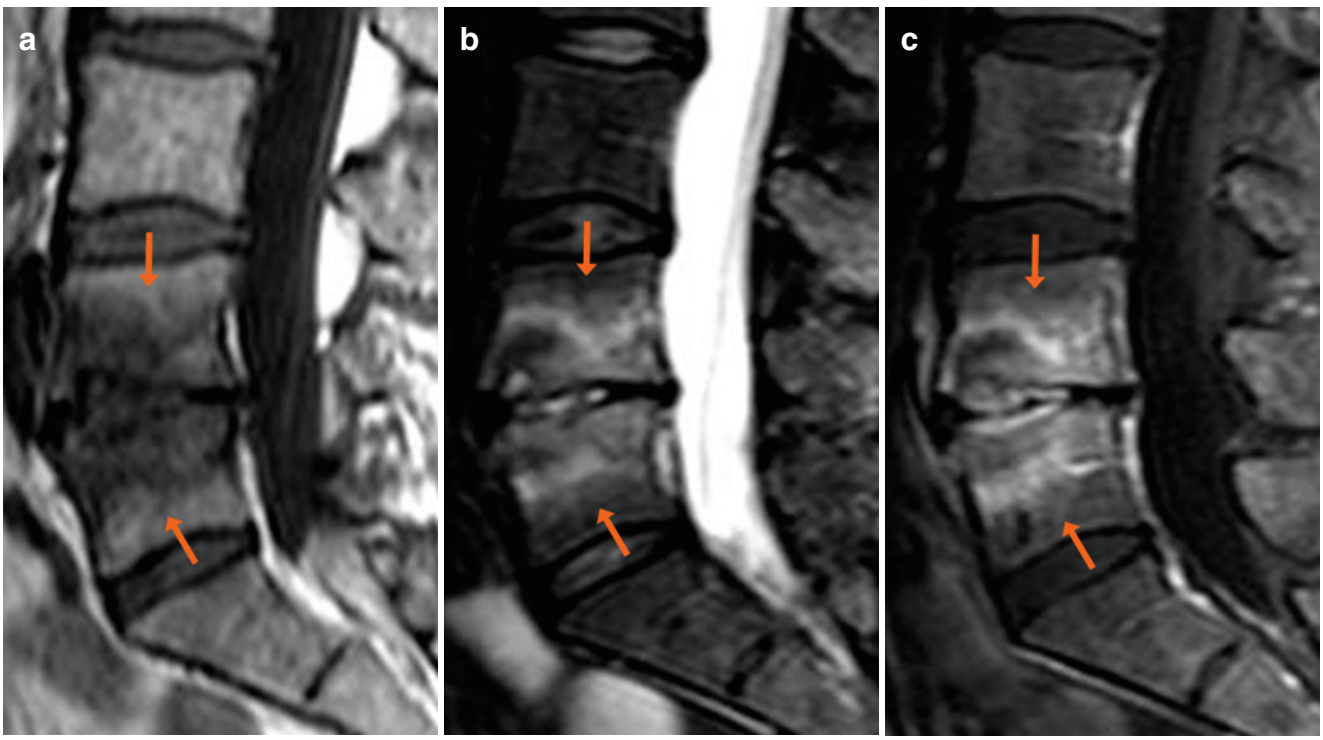


**Fig. 1** (**a**–**c**) Sagittal SE T1-weighted image (**a**), sagittal TSE T2-weighted image with fat saturation (**b**), sagittal SE T1-weighted image with fat saturation following administration of contrast medium (**c**). The opposing L4 and L5 vertebral bodies present a hypointense band in T1 (**a**, *arrows*), with hyperintense signal in T2 (**b**, *arrows*) and contrast enhancement (**c**, *arrows*). The L4/L5 intervertebral disc is collapsed. The same disc shows hyperintense signal in T2 (**b**) and marginal contrast enhancement (**c**), due to sterile inflammation. Diagnosis: osteochondrosis in active-inflammatory phase (i.e. osteochondritis)

P. D'Aprile, *MRI of Degenerative Disease of the Spine: A Case-Based Atlas*,
DOI 10.1007/978-3-319-09447-2_6, © Springer International Publishing Switzerland 2015

# Osteochondritis

# Aseptic Discitis

Case 5

- A 50-year-old female
- Chronic low back pain

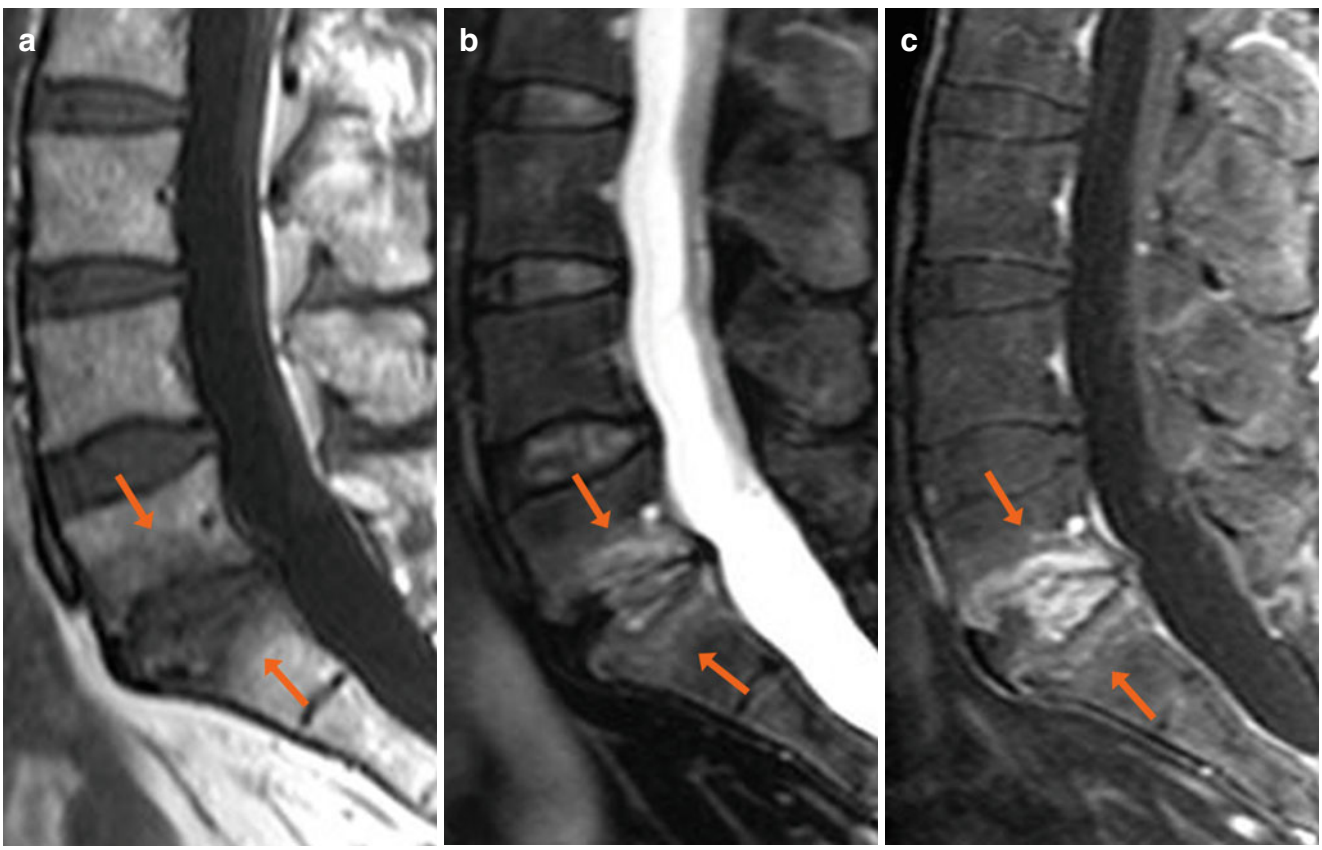


**Fig. 1** (**a**–**c**) Sagittal SE T1-weighted image (**a**), sagittal TSE T2-weighted image with fat saturation (**b**), sagittal SE T1-weighted image with fat saturation following administration of contrast medium (**c**). Signal changes (hypointense in T1 and hyperintense in T2) and marked contrast enhancement of the subchondral cancellous bone, chondral surfaces and disc at L5/S1 (**a**–**c**, *arrows*). Erosion of the articular surfaces. Typical osteochondritis and aseptic discitis in active-inflammatory phase

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DOI 10.1007/978-3-319-09447-2_7, © Springer International Publishing Switzerland 2015

- A 35-year-old female
- Chronic low back pain
- Improvement with rest

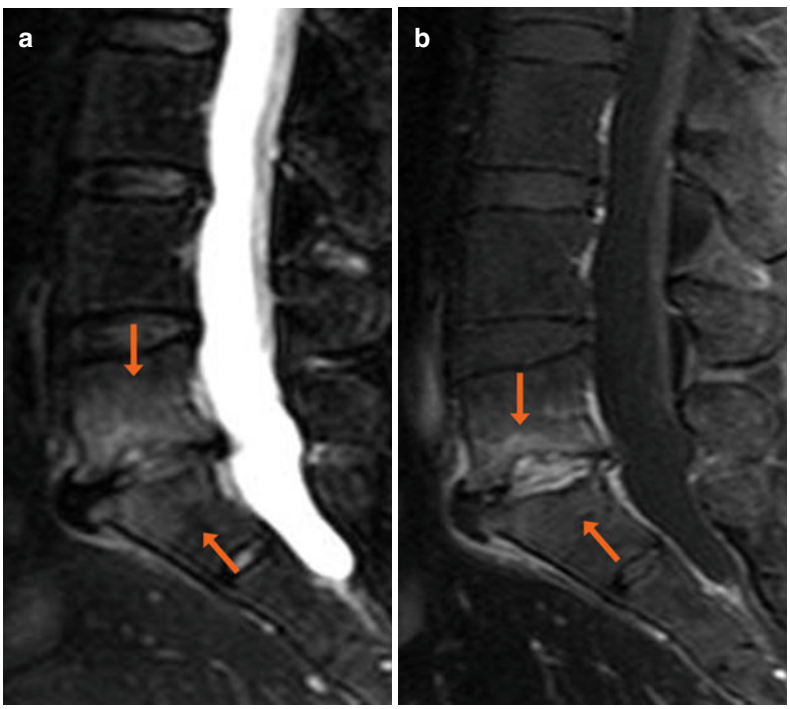


**Fig. 1** (**a**–**d**) Sagittal TSE T2-weighted image with fat saturation (**a**), sagittal and axial SE T1-weighted images with fat saturation after administration of contrast medium (**b**–**d**). T2 hyperintensity (**a**) and contrast enhancement (**b**) of the subchondral cancellous bone, chondral surfaces and intervertebral disc at L5/S1, indicating osteochondritis in inflammatory phase, with reduction in height of the same disc (**a**, **b**, *arrows*). Note also peridiscal contrast enhancement, indicating inflammation of the peridiscal soft tissue (**c**, **d** *arrowheads*)

P. D'Aprile, *MRI of Degenerative Disease of the Spine: A Case-Based Atlas*,
DOI 10.1007/978-3-319-09447-2_8, © Springer International Publishing Switzerland 2015

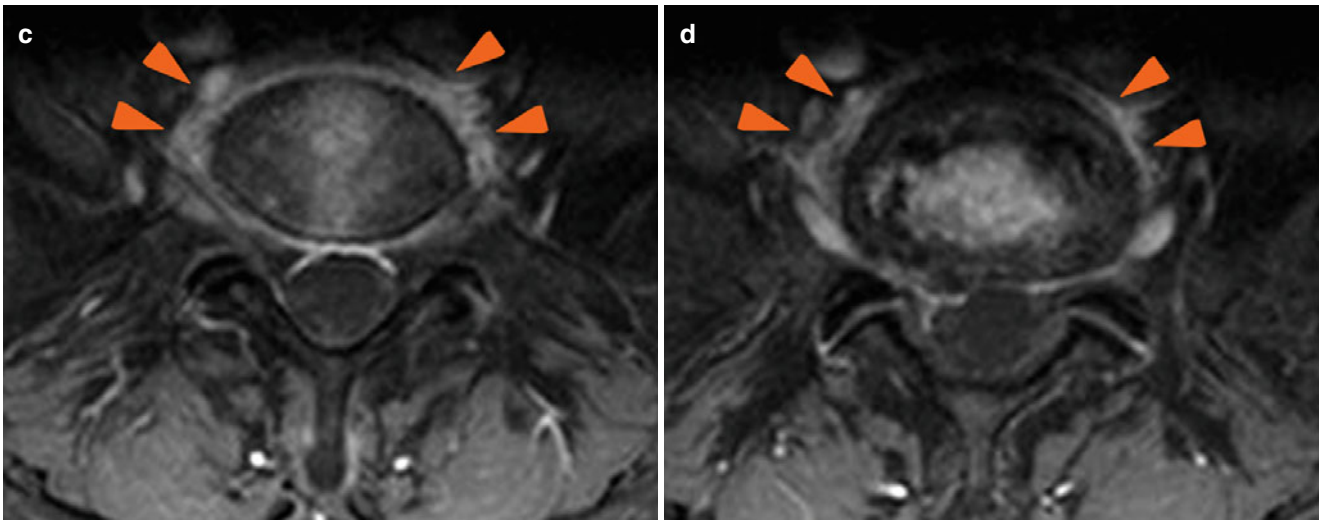


**Fig. 1** (continued)

- A 42-year-old male
- Back pain
- Limitation of motion of the thoracolumbar spine in the sagittal plane

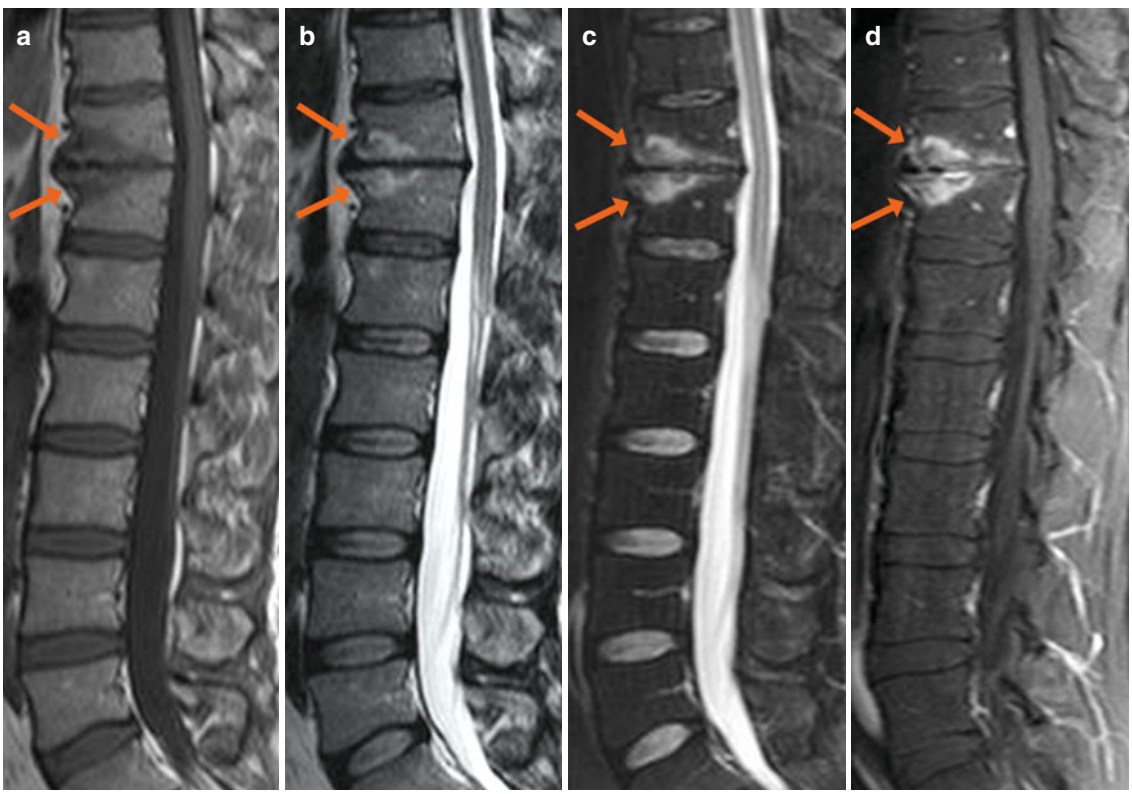


**Fig. 1** (**a**–**e**) Sagittal SE T1-weighted image (**a**), sagittal TSE T2-weighted image (**b**), sagittal TSE T2-weighted image with fat saturation (**c**), sagittal SE T1-weighted image with fat saturation after contrast medium administration (**d**), sagittal CT image – MPR (**e**). T1 hypointensity (**a**) and T2 hyperintensity (**b**, **c**) of the subchondral cancellous bone at T11/T12, indicating fibrovascular transformation of the bone marrow and oedema (**a**–**c,** *arrows*). The signal change is more diffuse in the anterior corners of the vertebral bodies. Note that oedematous changes are more evident in T2-weighted images with fat saturation (**c**) than in T2 images without fat saturation (**b**). The intervertebral disc is reduced in height. Marked contrast enhancement of the same areas and chondral surfaces (**d,** *arrows*), indicating osteochondritis in active-inflammatory phase. CT image showing osteosclerosis of the anterior edges of the same vertebral bodies, erosions of the articular surfaces and osteophytosis (**e**)

P. D'Aprile, *MRI of Degenerative Disease of the Spine: A Case-Based Atlas*,
DOI 10.1007/978-3-319-09447-2_9, 

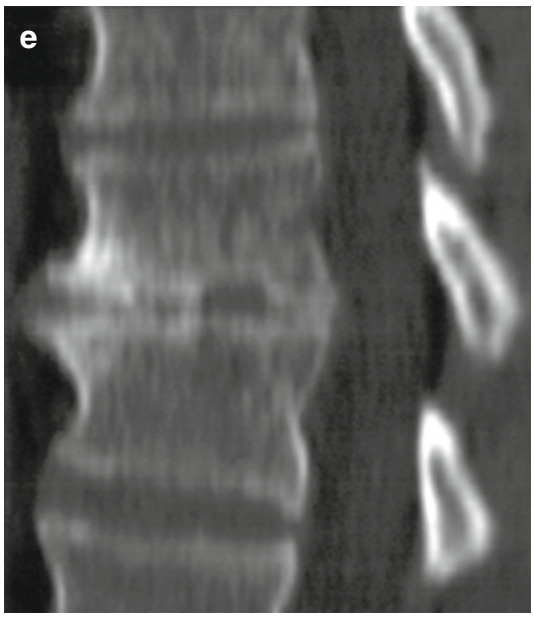


**Fig. 1** (continued)

# Facet Joint Effusion

Case 8

- A 46-year-old woman
- Patient with chronic back pain, exacerbated by flexion and extension

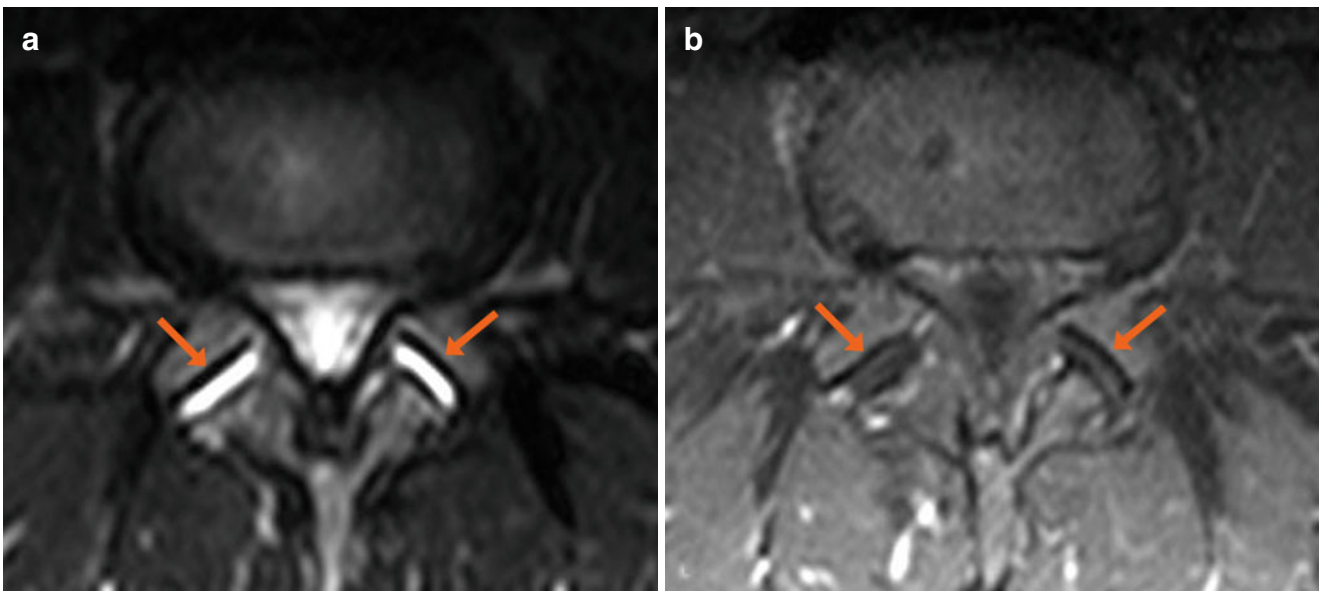


**Fig. 1** (**a**, **b**) Axial TSE T2-weighted image with fat saturation (**a**), axial SE T1-weighted image with fat saturation following administration of contrast medium (**b**). Facet joints show widening of the joint space bilaterally. This space is hyperintense in T2 (**a**, *arrows*), with no enhancement after contrast medium administration (**b**, *arrows*). These findings indicate joint effusion

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DOI 10.1007/978-3-319-09447-2_10, © Springer International Publishing Switzerland 2015

# Facet Joint Effusion

Case 9

- A 68-year old male
- Chronic low back pain, exacerbated by flexion and extension
- History of chronic dorsal and lumbar pain

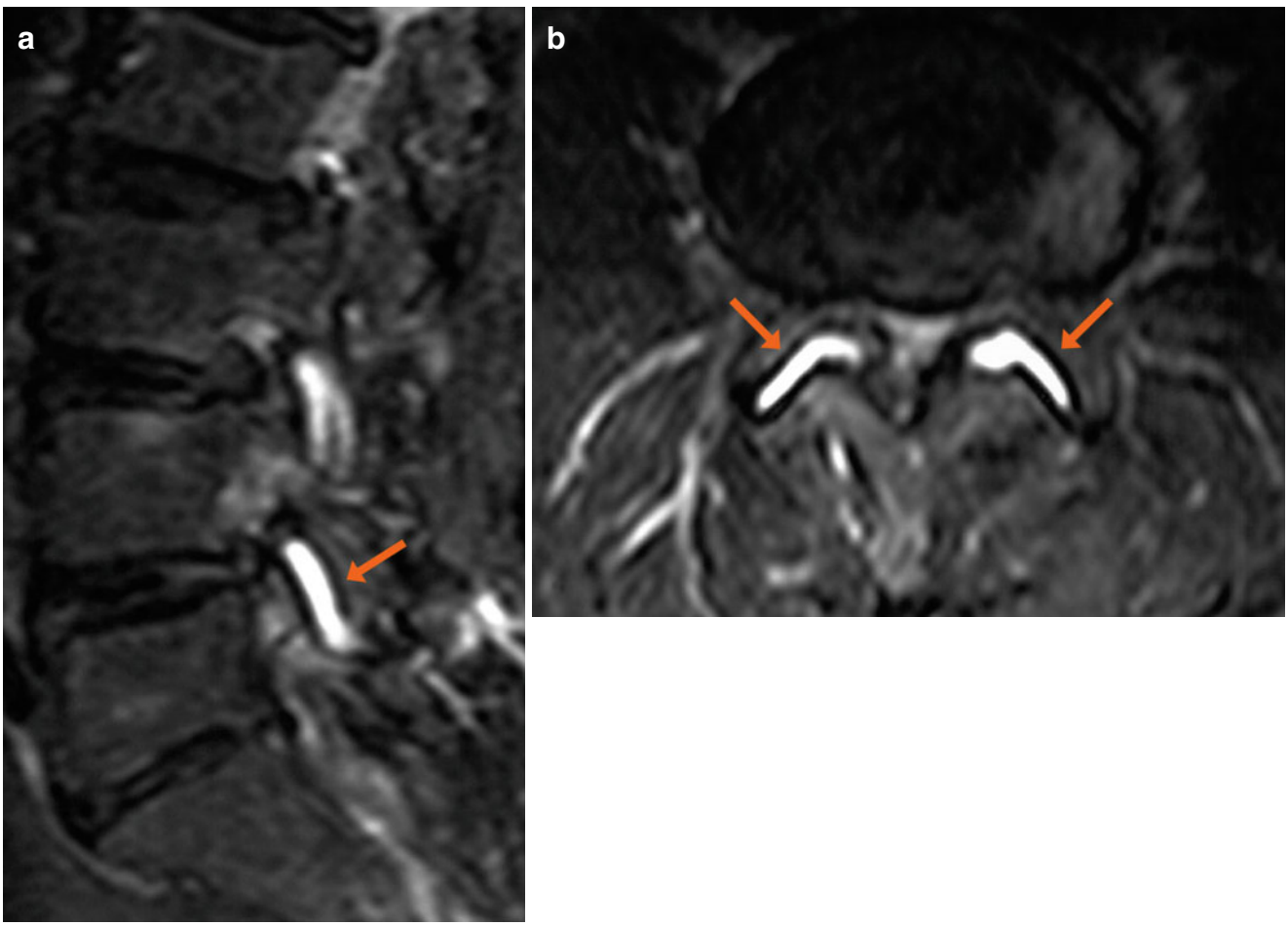


**Fig. 1** (**a**, **b**) Sagittal and axial TSE T2-weighted images with fat saturation (**a**, **b**). Facet joint effusion at L4/L5 bilaterally, with widening of the joint spaces (*arrows*)

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DOI 10.1007/978-3-319-09447-2_11, © Springer International Publishing Switzerland 2015

# Synovitis
# Radiculitis

Case 10

- A 36-year-old man
- Patient with chronic back pain, exacerbated by flexion and extension
- Radiculopathy on the right side

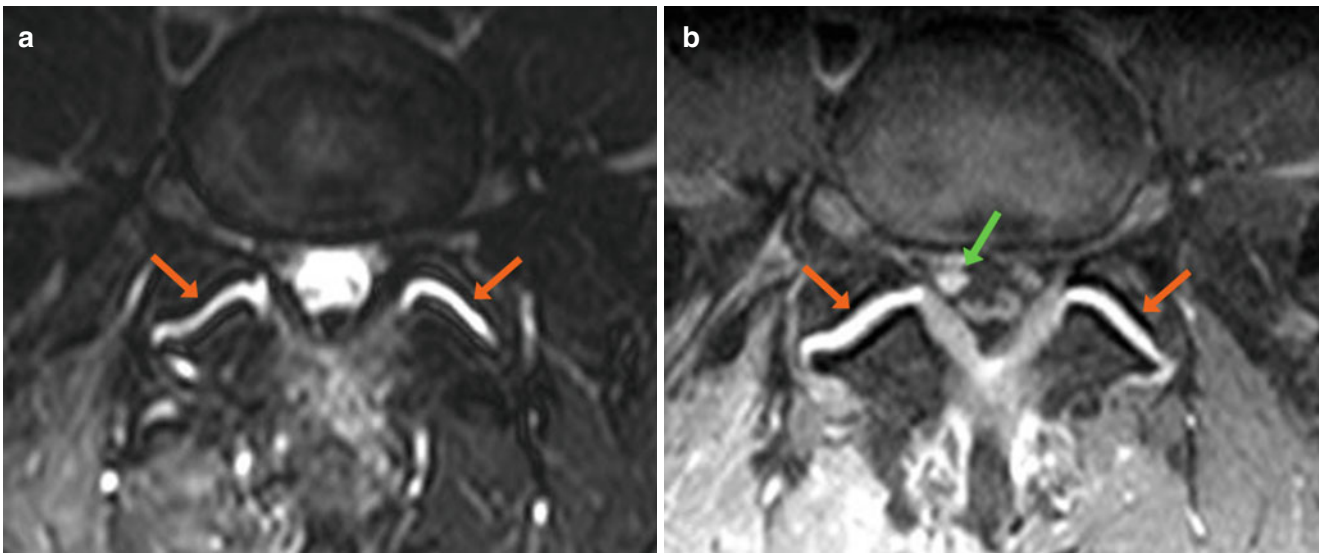


**Fig. 1** (**a**, **b**) Axial TSE T2-weighted image with fat saturation (**a**), axial SE T1-weighted image with fat saturation, following contrast medium administration (**b**). Facet joints at L5/S1. Widening of the joint space, with enhancement after contrast medium administration indicating synovitis (*red arrows*). Note also contrast enhancement of a nerve root on the right side, referable to radiculitis (*green arrow*)

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DOI 10.1007/978-3-319-09447-2_12, © Springer International Publishing Switzerland 2015

- A 62-year-old male
- Limitation of motion of the lumbar spine
- Low back pain, more marked on the left side

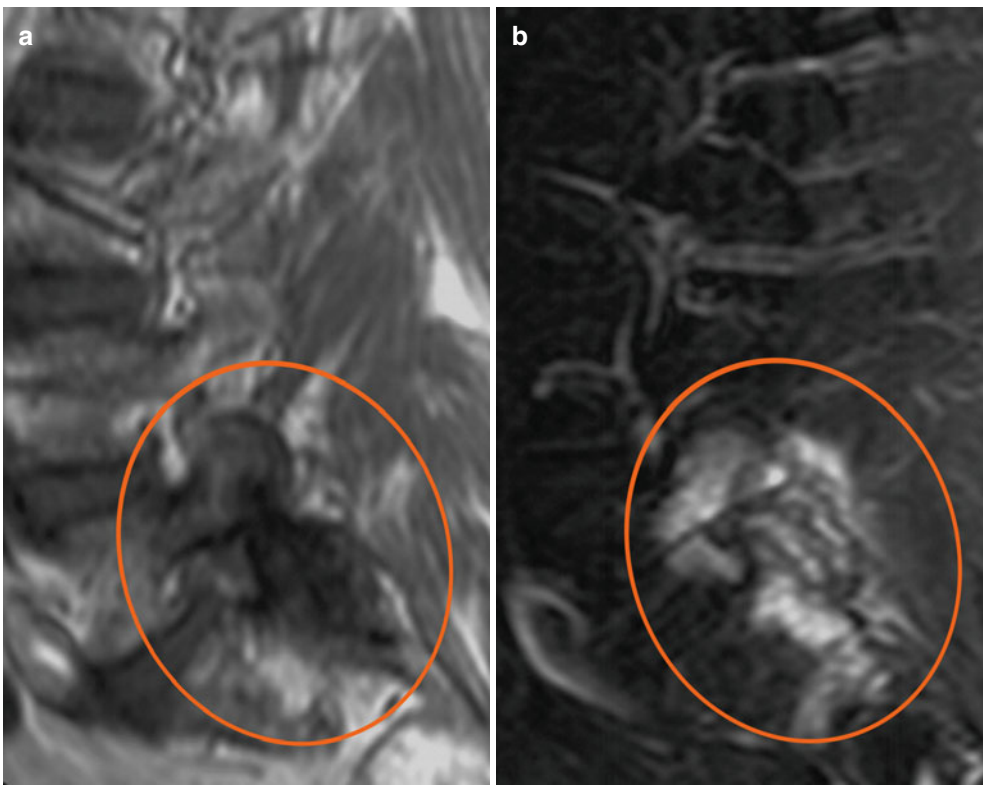


**Fig. 1** (**a**–**f**) Sagittal T1-weighted image (**a**), sagittal and axial T2-weighted images with fat saturation (**b**–**d**), axial T1-weighted images with fat saturation following administration of contrast medium (**e**–**f**). Structural and signal changes of the L5/S1 facet joint (**a**, **b** *circle*). The images on the axial plane show T2 hyperintensity (**b**–**d**, *arrows*) and contrast enhancement (**e**, **f**, *arrows*) of the articular apophyses and pedicles, referable to oedema-osteitis. It coexists periapophyseal soft tissue inflammatory reaction, more evident after contrast medium administration (**e**, **f**). Diagnosis: bilateral facet joint osteoarthritis at L5/S1, more marked on the left side, in active-inflammatory phase

P. D'Aprile, *MRI of Degenerative Disease of the Spine: A Case-Based Atlas*,
DOI 10.1007/978-3-319-09447-2_13, © Springer International Publishing Switzerland 2015

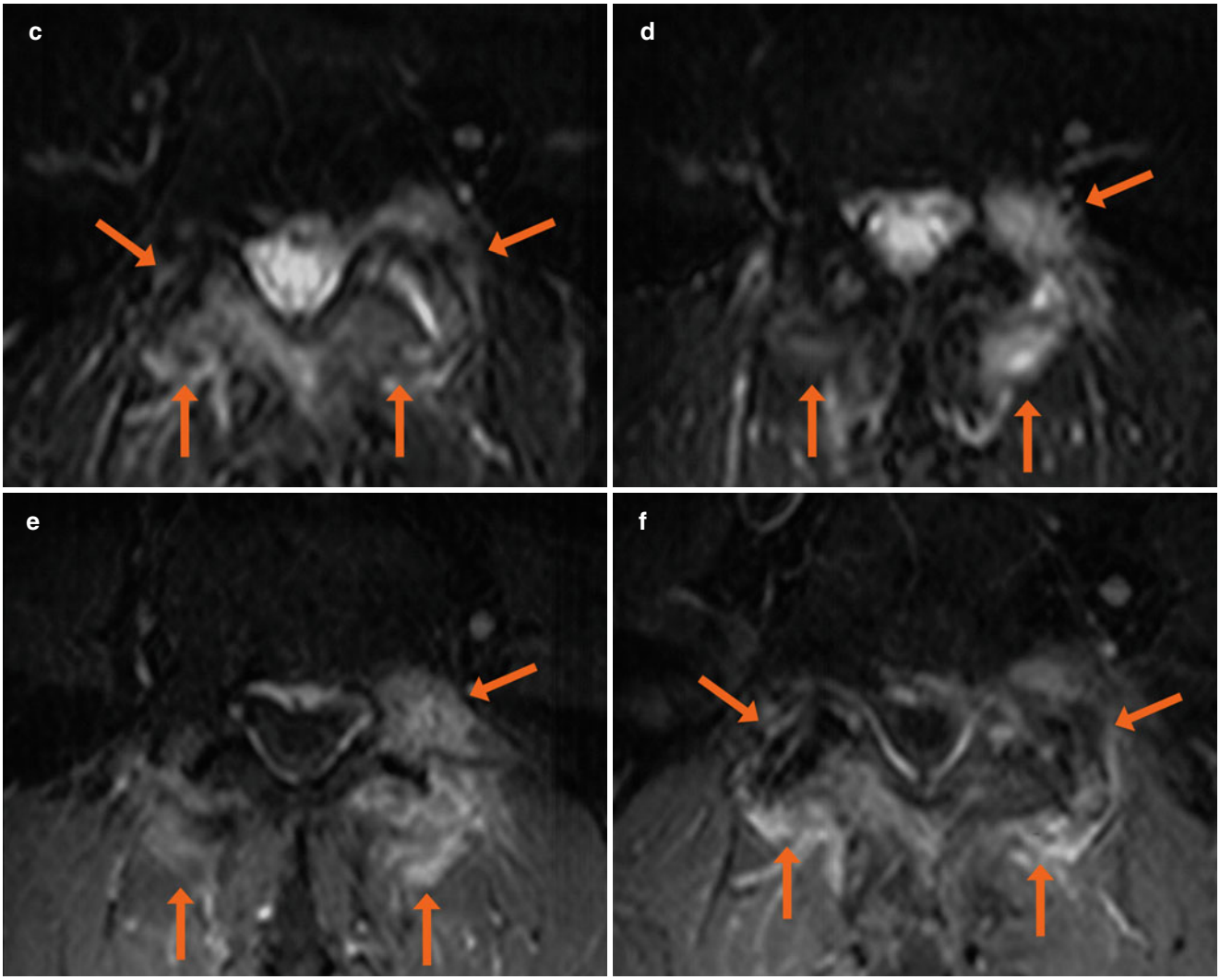


**Fig. 1** (continued)

# Facet Joint Osteoarthritis

Case 12

- A 67-year-old female
- Low back pain, more marked on the left side

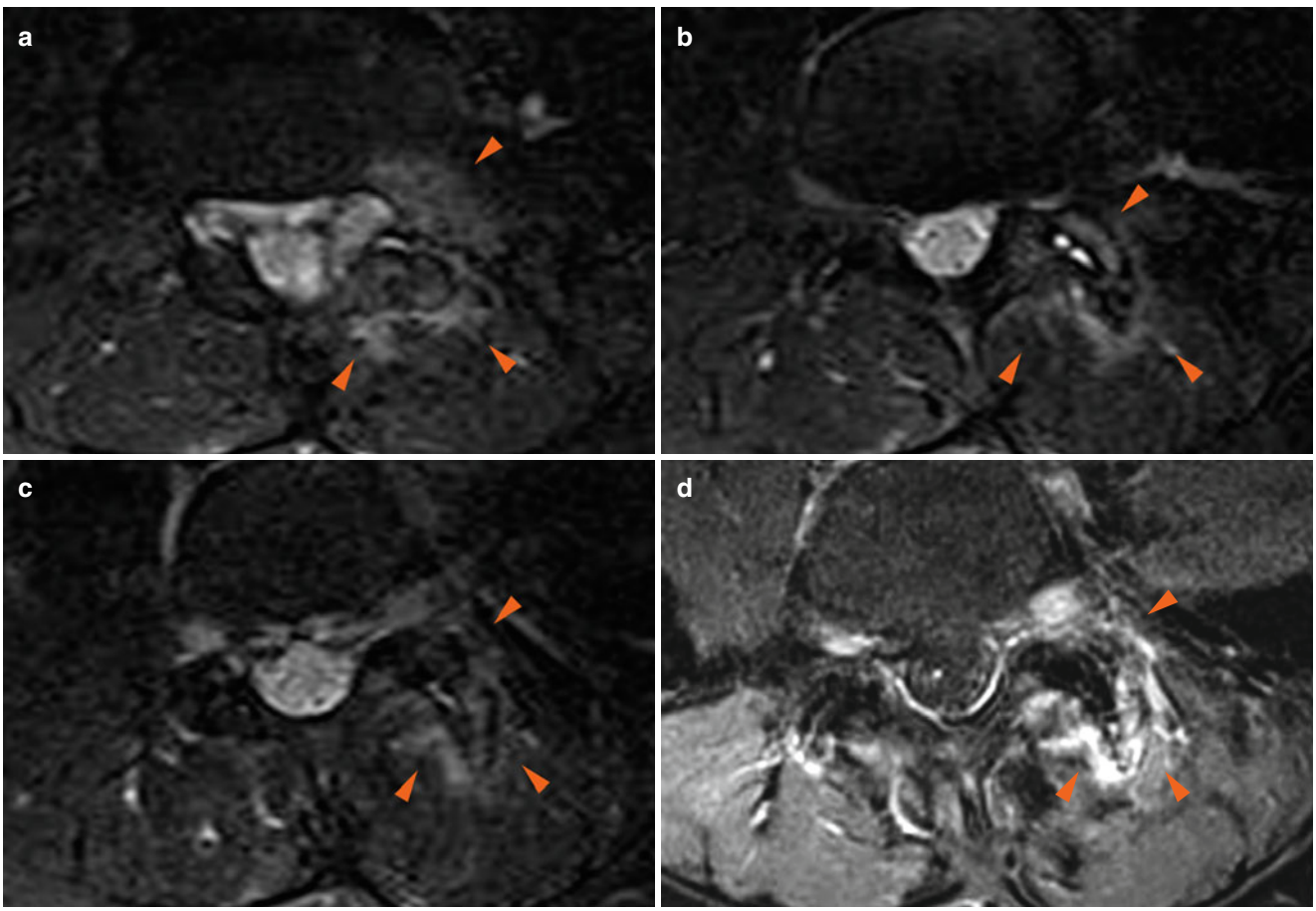


**Fig. 1** (**a**–**f**) Axial TSE T2-weighted images with fat saturation (**a**–**c**), axial SE T1-weighted images with fat saturation following administration of contrast medium (**d**–**f**). Left facet joint arthrosis with facet hypertrophy at L5/S1. Hyperintense signal on T2-weighted images in the articular facets and surrounding soft tissues (**a**–**c**, *arrowheads*). The same areas enhance on T1-weighted images after contrast medium administration (**d**–**f**, *arrowheads*). There is also contrast enhancement of the facet joint space (**e**). These findings indicate sterile facet joint osteoarthritis in active-inflammatory phase, with synovitis and inflammation of the periarticular soft tissues

P. D'Aprile, *MRI of Degenerative Disease of the Spine: A Case-Based Atlas*,
DOI 10.1007/978-3-319-09447-2_14, © Springer International Publishing Switzerland 2015

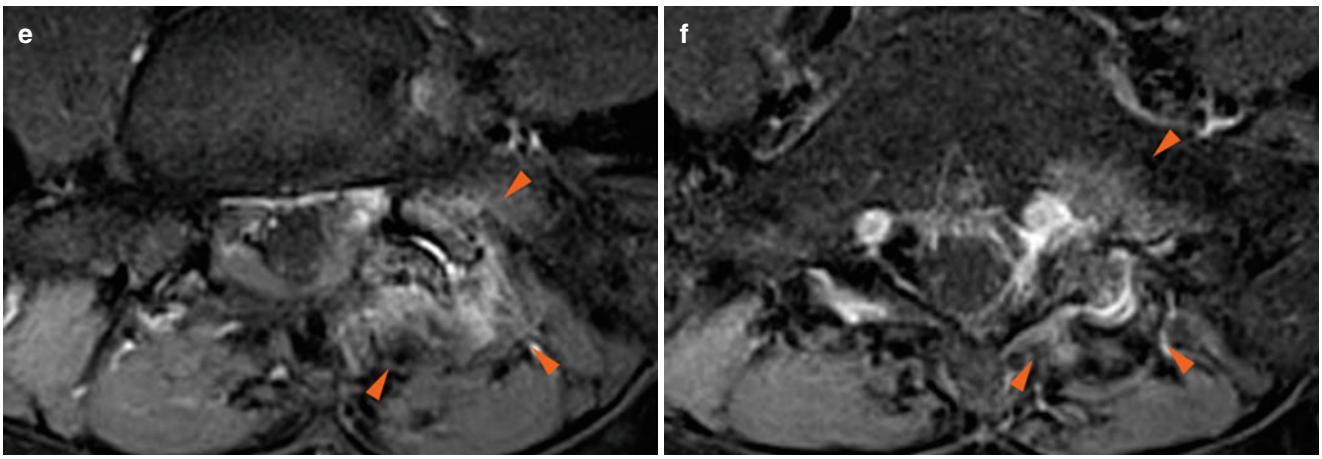


**Fig. 1** (continued)

- A 46-year-old male
- Persistent low back pain with focal tenderness on the right side at L5/S1.

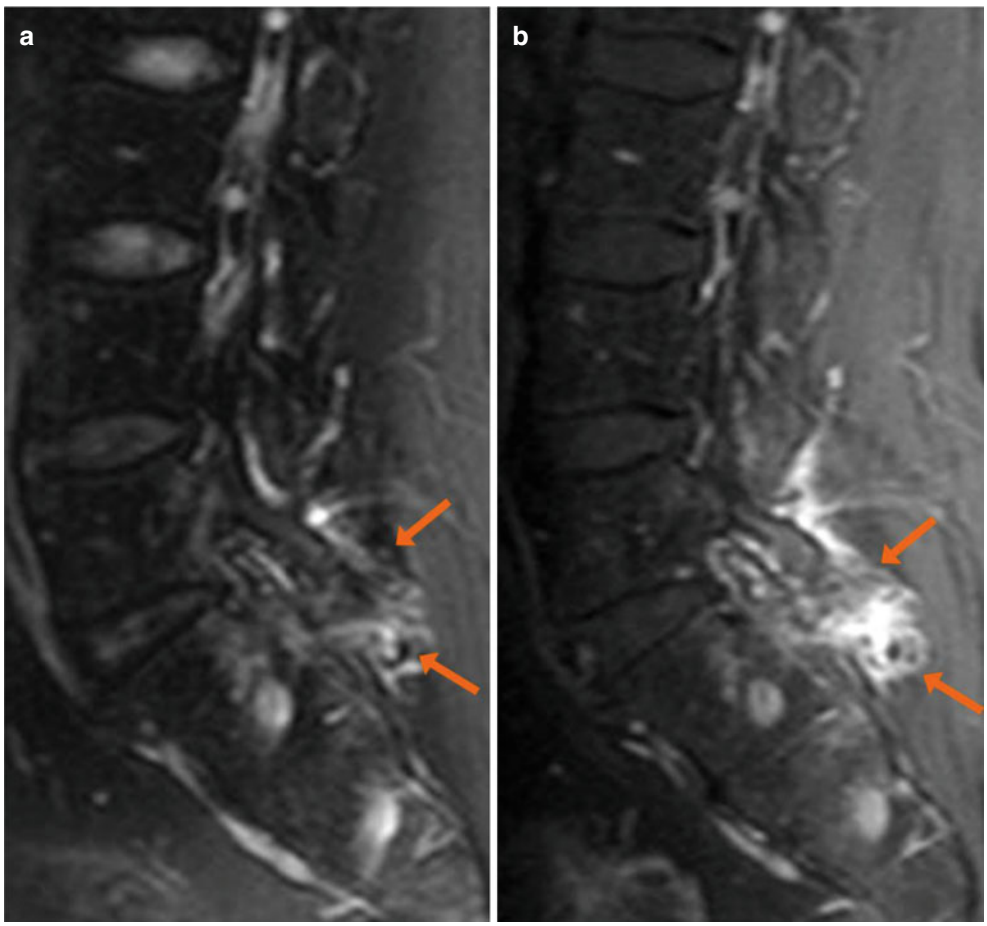


**Fig. 1** (**a–h**) Sagittal and axial TSE T2-weighted images with fat saturation (**a**, **d**, **e**), axial SE T1-weighted image (**c**), sagittal and axial SE T1-weighted images with fat saturation images (**b**, **f**, **g**), CT image (**h**). Hyperintense signal in T2 (**a**, *arrow*) and contrast enhancement (**b**, *arrow*) in the articular facets and surrounding soft tissues. Axial images allow a better spatial definition of the lesion, with oedema-osteitis of the articular facets, with small erosions of the articular surfaces and with periarticular inflammation (**c**–**g**, *circle*). CT image shows right facet joint arthrosis, with small articular erosions (**h**, *circle*). Diagnosis: sterile facet joint arthritis in active-inflammatory phase

P. D'Aprile, *MRI of Degenerative Disease of the Spine: A Case-Based Atlas*,
DOI 10.1007/978-3-319-09447-2_15, © Springer International Publishing Switzerland 2015

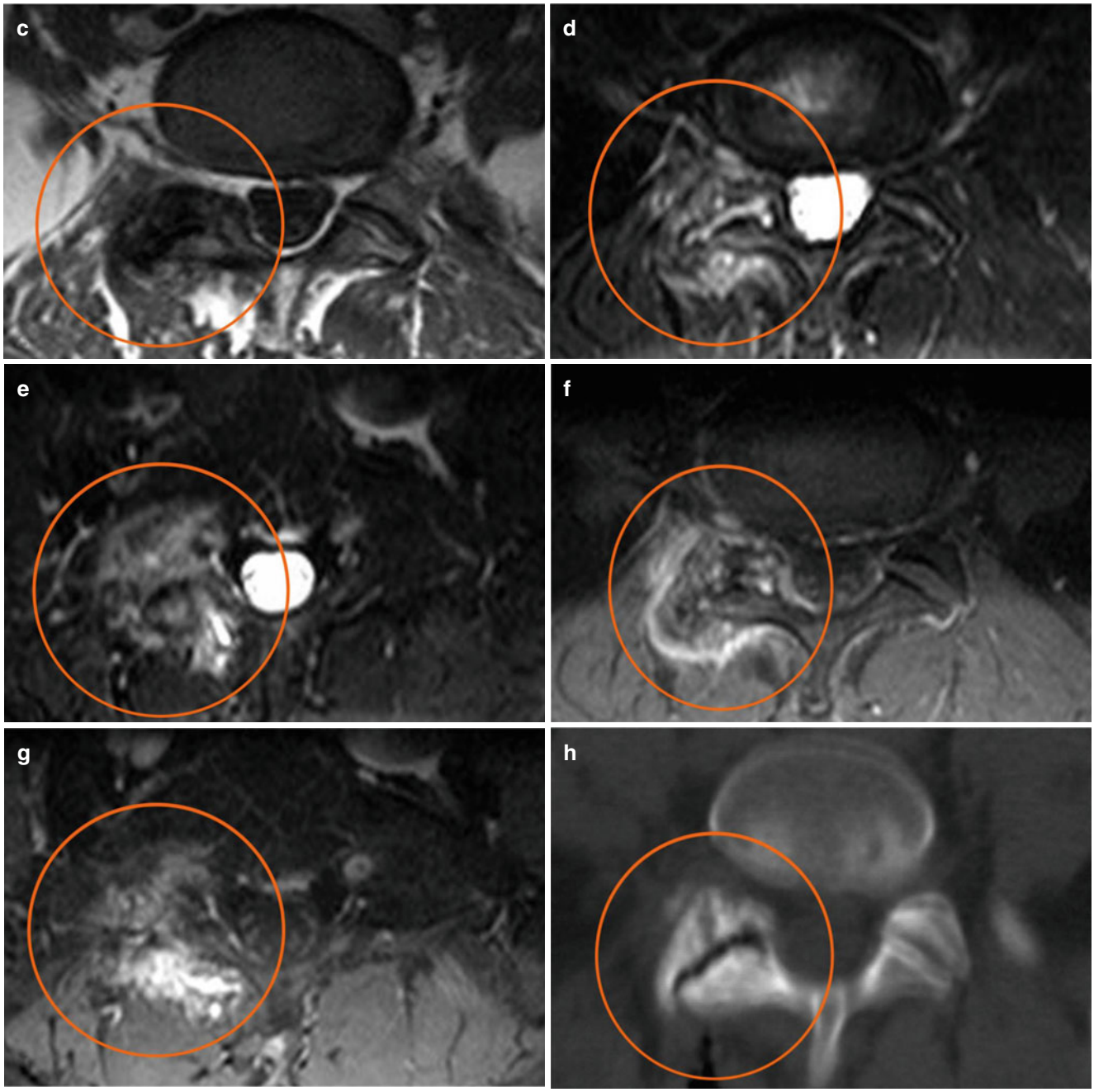


**Fig. 1** (continued)

# C1-C2 Osteoarthritis

Case 14

- A 70-year-old man
- Neck pain
- Limitation of motion of the cranio-cervical junction

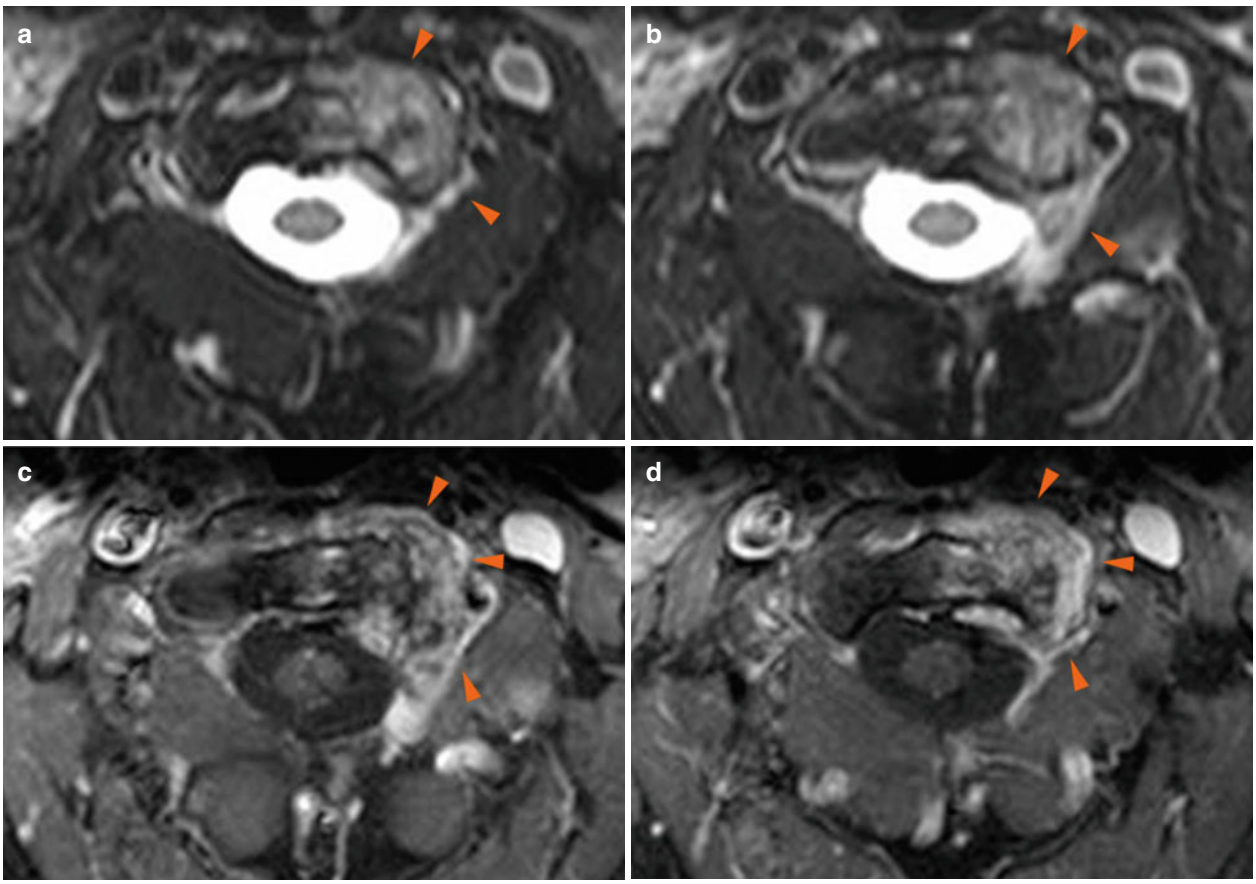


**Fig. 1** (**a**–**f**) Axial TSE T2-weighted images with fat saturation (**a**, **b**), axial and coronal SE T1-weighted images with fat saturation following administration of contrast medium (**c**–**f**). Marked atlanto-axial osteoarthritis on the left side. T2 hyperintensity (**a**, **b**, *arrowheads*), and contrast enhancement (**c**, **d**, *arrowheads*) of the left lateral mass of the atlas and axis, indicating oedema-osteitis. The images on coronal plane allow to better highlight contrast enhancement of the joint space, which is reduced in amplitude (**e**, **f**, *arrow*). Note also contrast enhancement of the adjacent periodontoid epidural space, due to inflammatory reaction (**f**, *arrowhead*)

P. D'Aprile, *MRI of Degenerative Disease of the Spine: A Case-Based Atlas*,
DOI 10.1007/978-3-319-09447-2_16, © Springer International Publishing Switzerland 2015

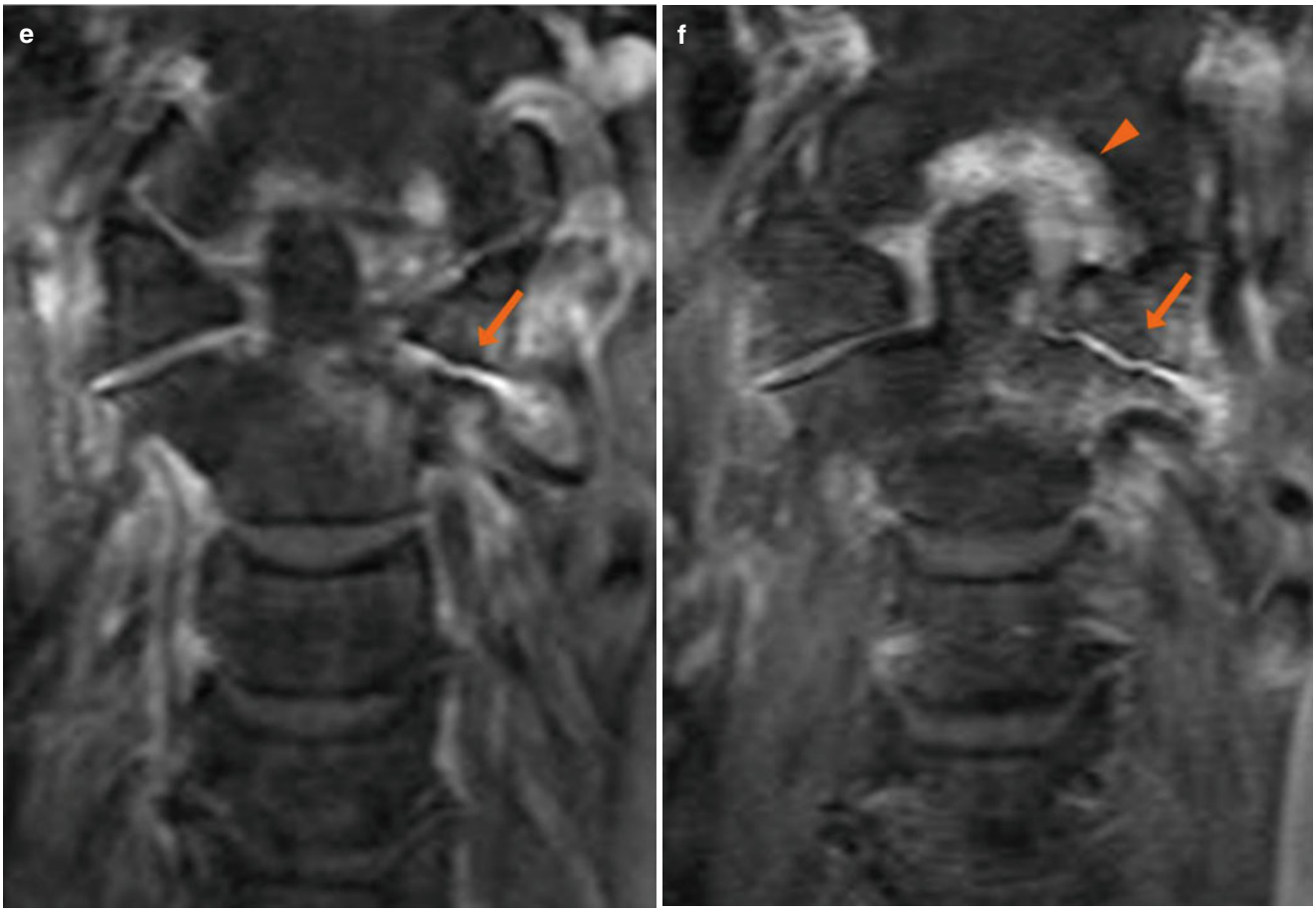


**Fig. 1** (continued)

- A 67-year-old female
- Neck pain more marked on the left side

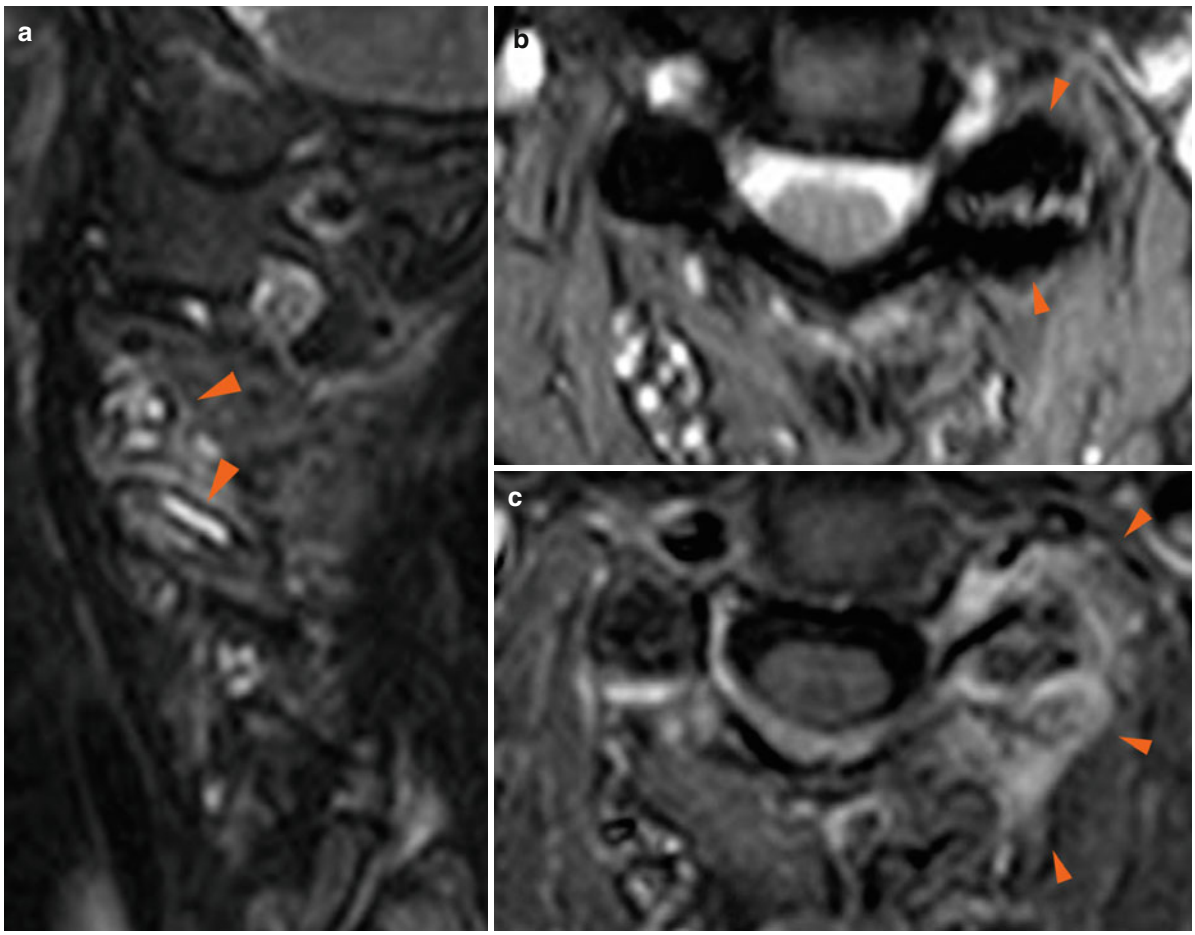


**Fig. 1** (**a**–**d**) Sagittal TSE T2-weighted image with fat saturation (**a**), axial GE T2*-weighted image (**b**), axial SE T1-weighted images with fat saturation following administration of contrast medium (**c**, **d**). Oedema of the facet joint at C3/C4, with joint effusion (**a**, *arrowhead*). The axial image confirms a mild joint effusion, with erosion of the articular surfaces (**b**, *arrowhead*). The administration of contrast medium shows enhancement of the facet joint (i.e. osteoarthritis) and periarticular soft tissues (i.e. periapophyseal inflammation) (**c**, **d**, *arrowhead*)

P. D'Aprile, *MRI of Degenerative Disease of the Spine: A Case-Based Atlas*,
DOI 10.1007/978-3-319-09447-2_17, © Springer International Publishing Switzerland 2015

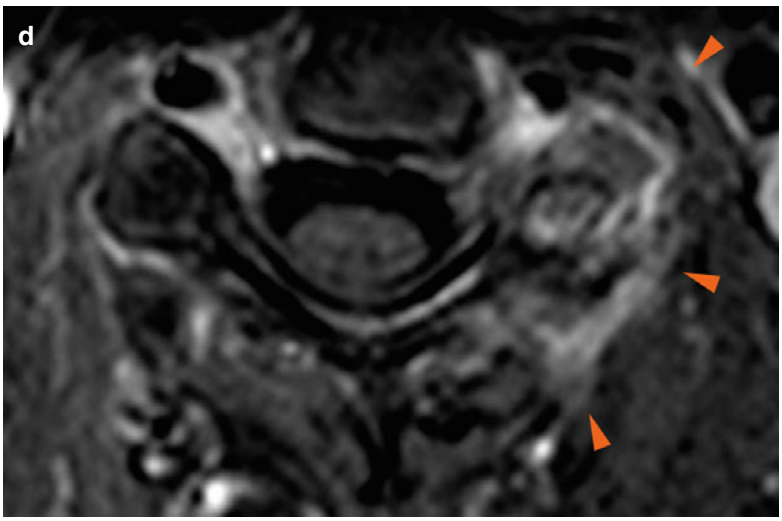


**Fig. 1** (continued)

# Facet Joint Osteoarthritis
# Ligamentous Inflammation

Case 16

- A 43-year-old man
- Neck pain more marked on the right side

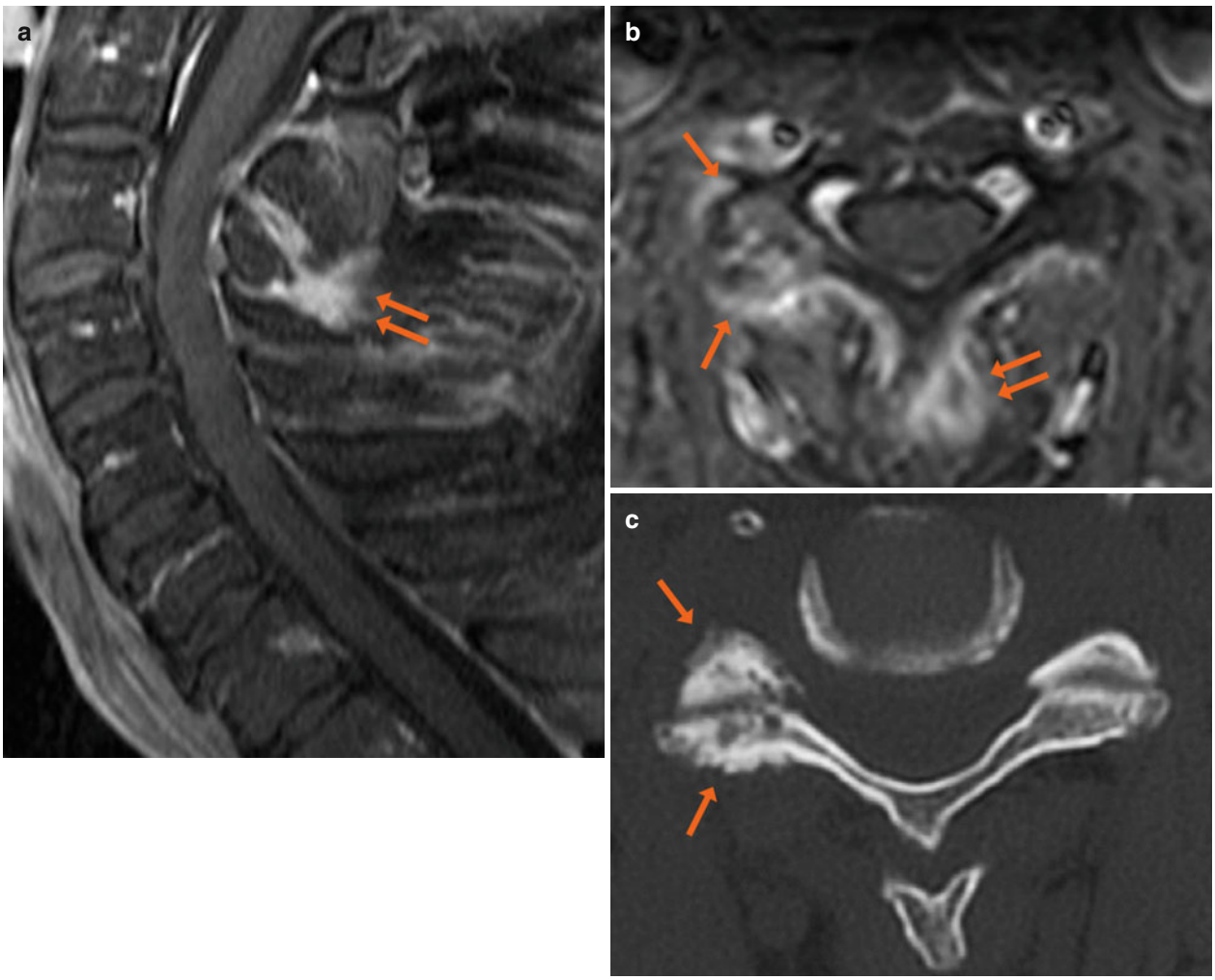


**Fig. 1** (**a**–**c**) Sagittal and axial contrast-enhanced T1-weighted images with fat saturation (**a**, **b**), axial CT image (**c**). Contrast enhancement of the interspinous ligaments at C2-C3-C4, due to ligamentous inflammation (**a**, **b**, *double arrow*). Contrast enhancement of the C3/C4 right facet joint and periarticular soft tissues, indicating osteoarthritis and periapophyseal inflammation (**b**, *arrow*). CT image confirms the osteoarthritis, with bone erosions, subchondral cysts and osteosclerosis (**c**, *arrow*)

P. D'Aprile, *MRI of Degenerative Disease of the Spine: A Case-Based Atlas*,
DOI 10.1007/978-3-319-09447-2_18, © Springer International Publishing Switzerland 2015

# Osteochondrosis

## Facet Joint Osteoarthritis

Case **17**

- A 70-year-old male
- Patient with chronic neck pain
- Limited motion
- Point tenderness corresponding to right facet joint at C4/C5

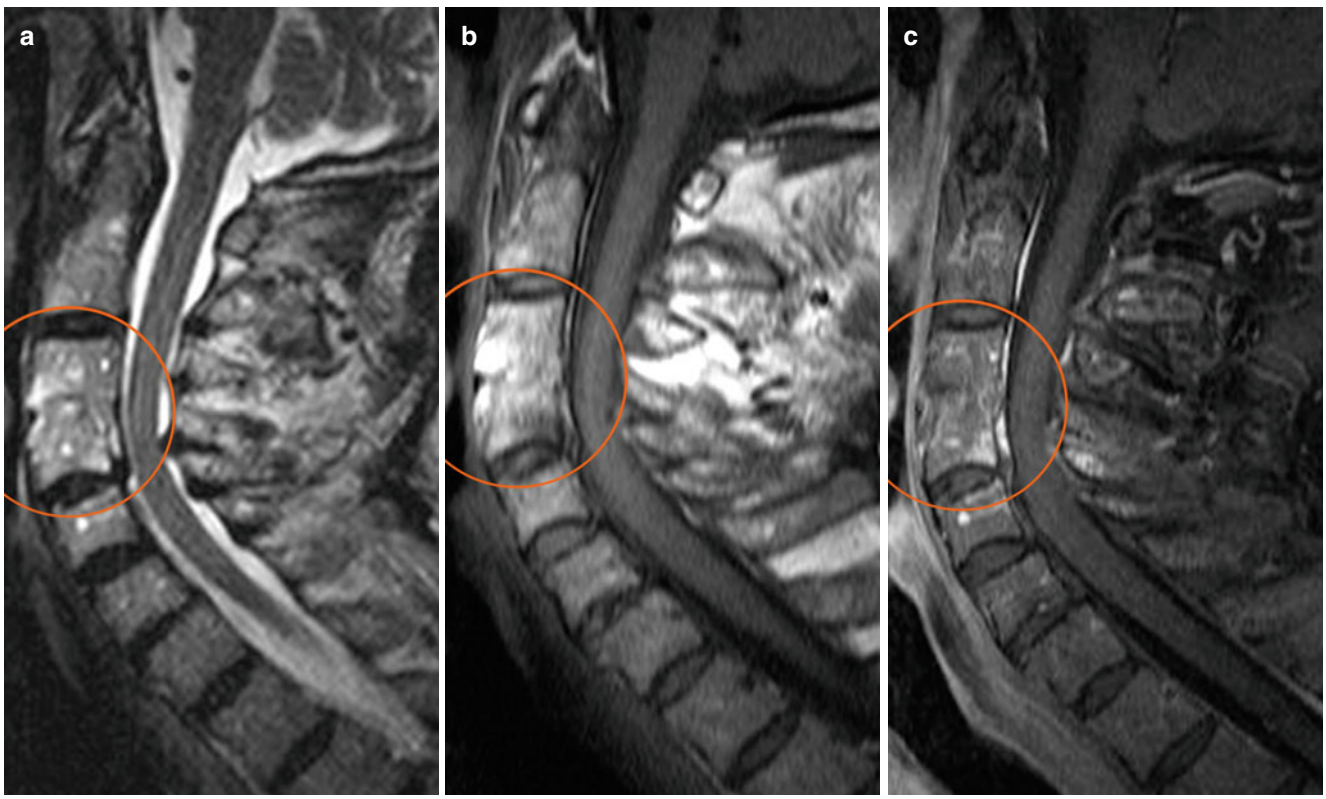


**Fig. 1** (**a**–**f**) Sagittal T2-weighted image (**a**), sagittal T1-weighted image (**b**), sagittal and axial T1-weighted images with fat saturation following administration of contrast medium (**c**–**f**). Marked osteochondrosic degeneration at C3/C4, with intervertebral fusion (**a**–**c**, *circle*). The same patient presented a clear C4/C5 facet joint osteoarthritis on the right side, coupled with periapophyseal soft tissue inflammation, well defined by intense contrast enhancement of the same structures (**d**–**f**, *arrowheads*)

P. D'Aprile, *MRI of Degenerative Disease of the Spine: A Case-Based Atlas*,
DOI 10.1007/978-3-319-09447-2_19, © Springer International Publishing Switzerland 2015

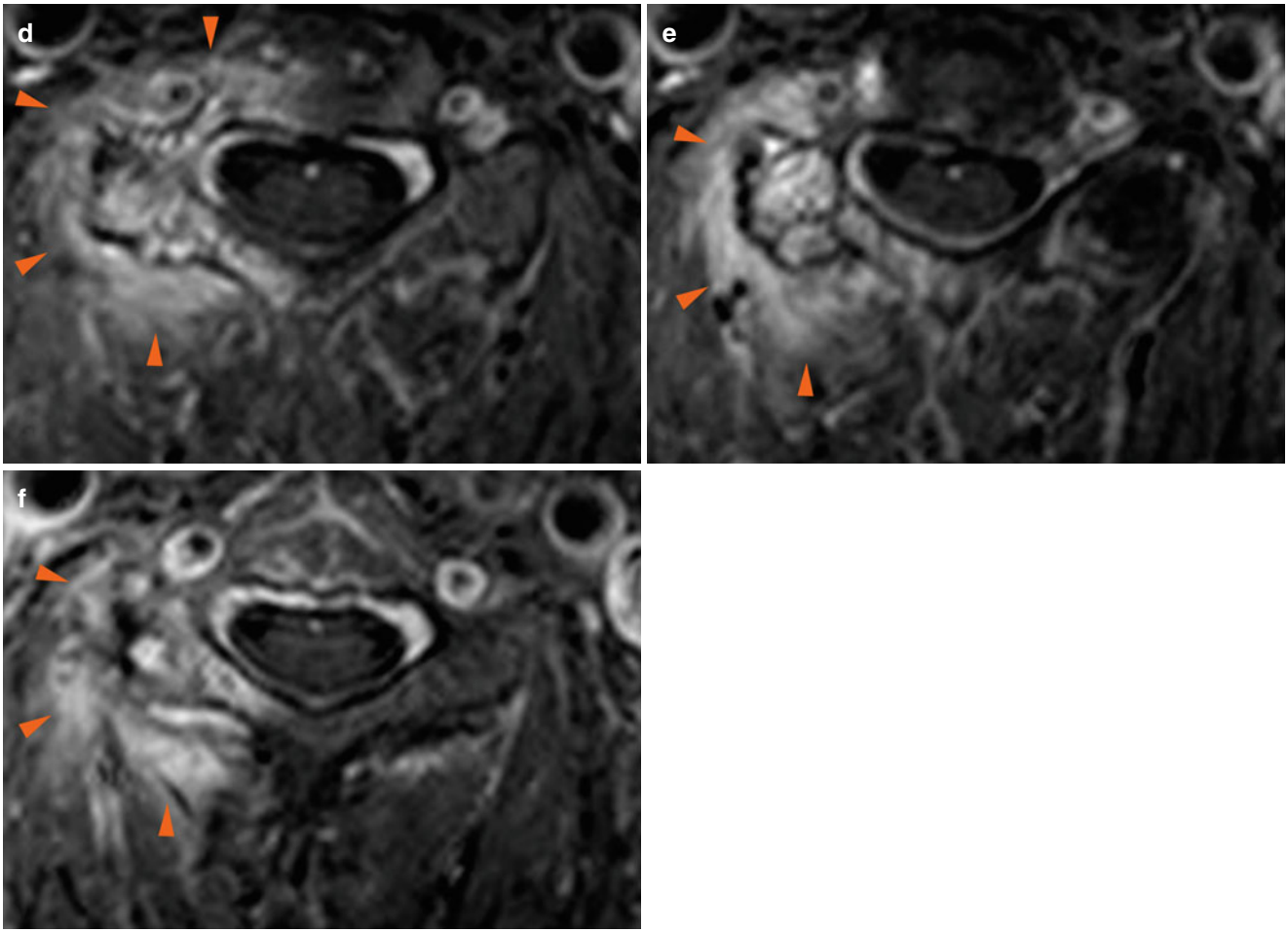


**Fig. 1** (continued)

# Facet Joint Osteoarthritis

## Synovitis

Case 18

- A 60-year-old female
- Chronic low back pain
- Limited motion of the lumbar spine
- Worsening of pain with motion and improvement with rest

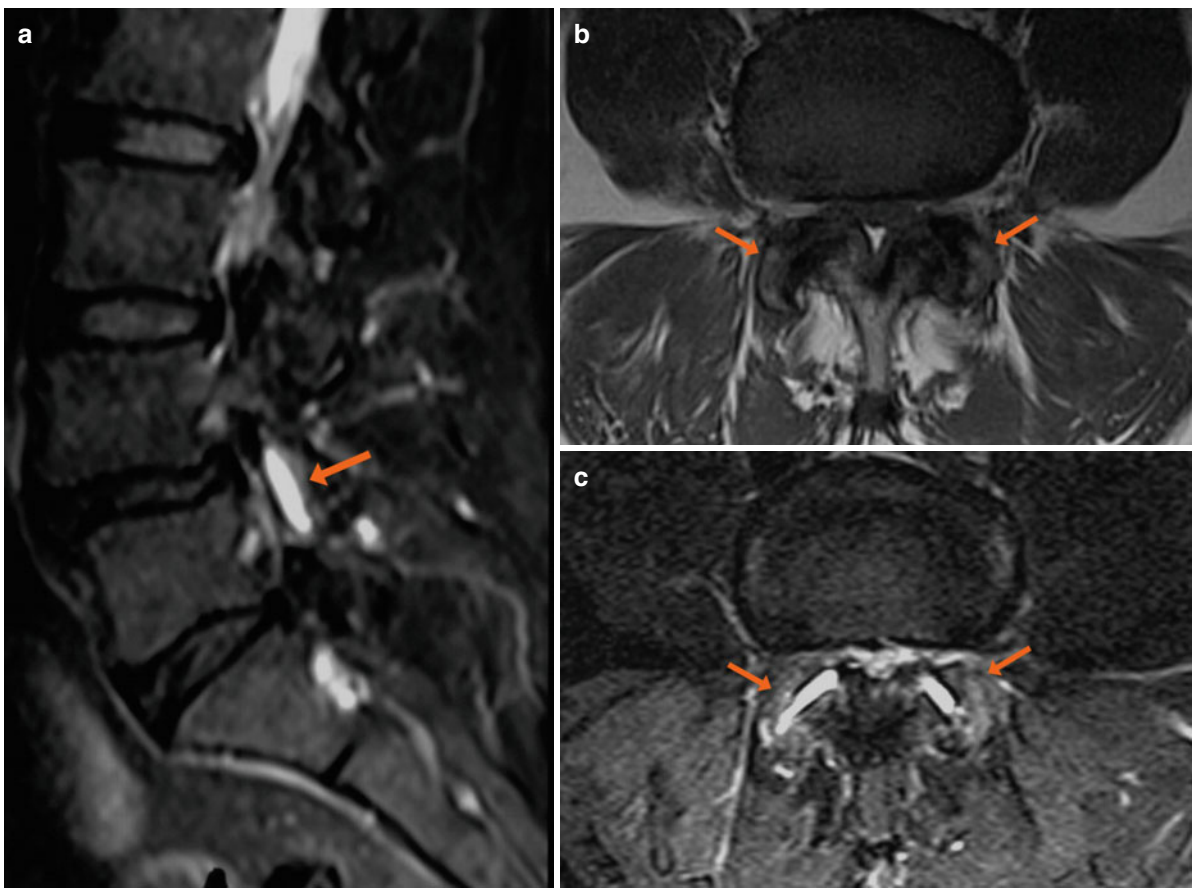


**Fig. 1** (**a**–**e**) Sagittal and axial T2-weighted images with fat saturation (**a**, **c**), axial T1-weighted image (**b**), sagittal and axial T1-weighted images with fat saturation following administration of contrast medium (**d**, **e**). Bilateral facet joint osteoarthritis at L4/L5 (**b**, **c**, **e**, *arrows*). Widening of the facet joint spaces (**a**, **c**, *arrows*). Marked contrast enhancement of the synovial membrane, especially postero-inferiorly to the facet joint, indicating synovitis with synovial hypertrophy (**d**, *arrows*). Stenosis of the central spinal canal (**b**, **c**, **e**)

P. D'Aprile, *MRI of Degenerative Disease of the Spine: A Case-Based Atlas*,
DOI 10.1007/978-3-319-09447-2_20, © Springer International Publishing Switzerland 2015

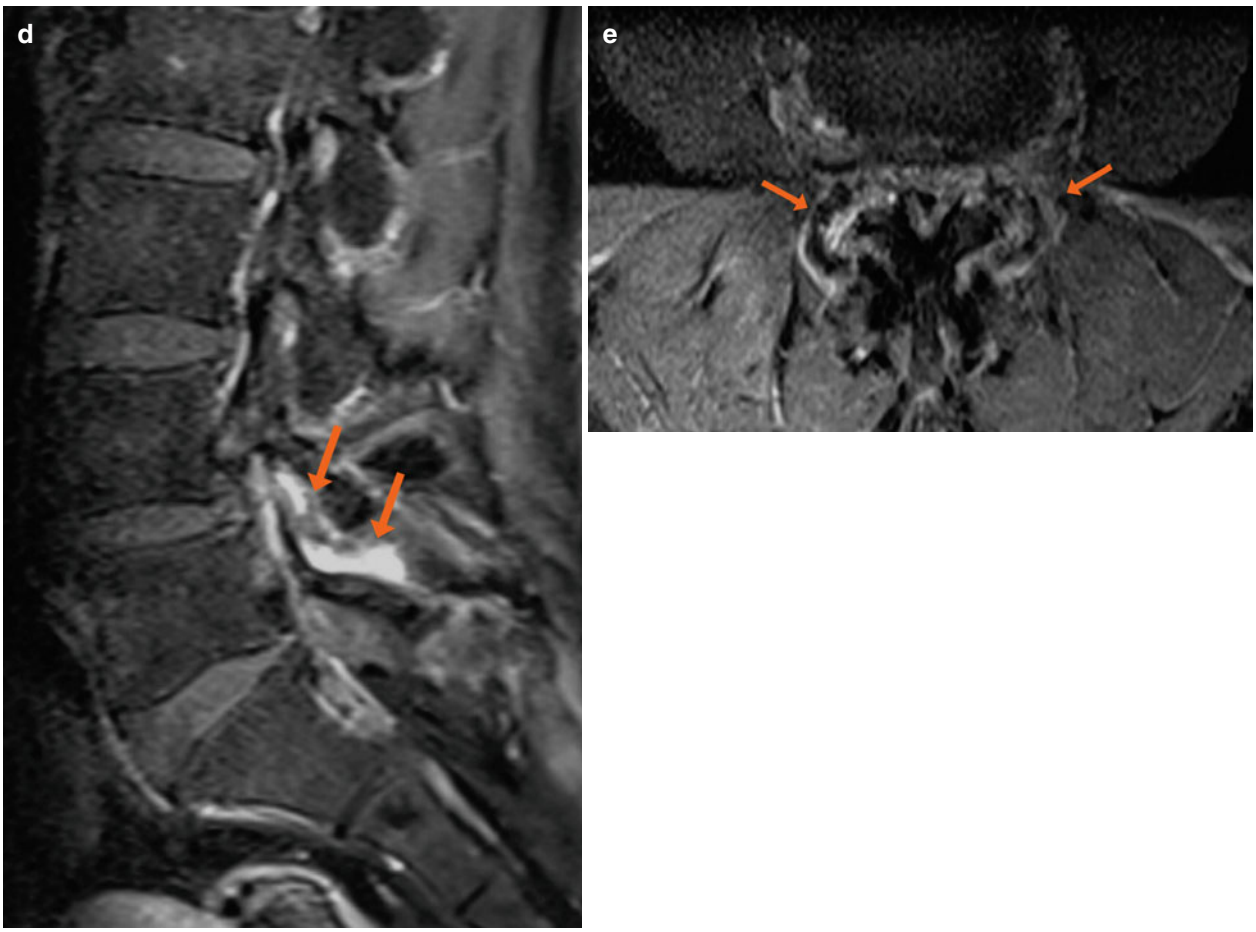


**Fig. 1** (continued)

# Facet Joint Osteoarthritis

## Synovitis

Case 19

- A 61-year-old man
- Chronic low back pain
- Exacerbation of pain by extension and flexion
- Point tenderness corresponding to L4/L5 right facet joint

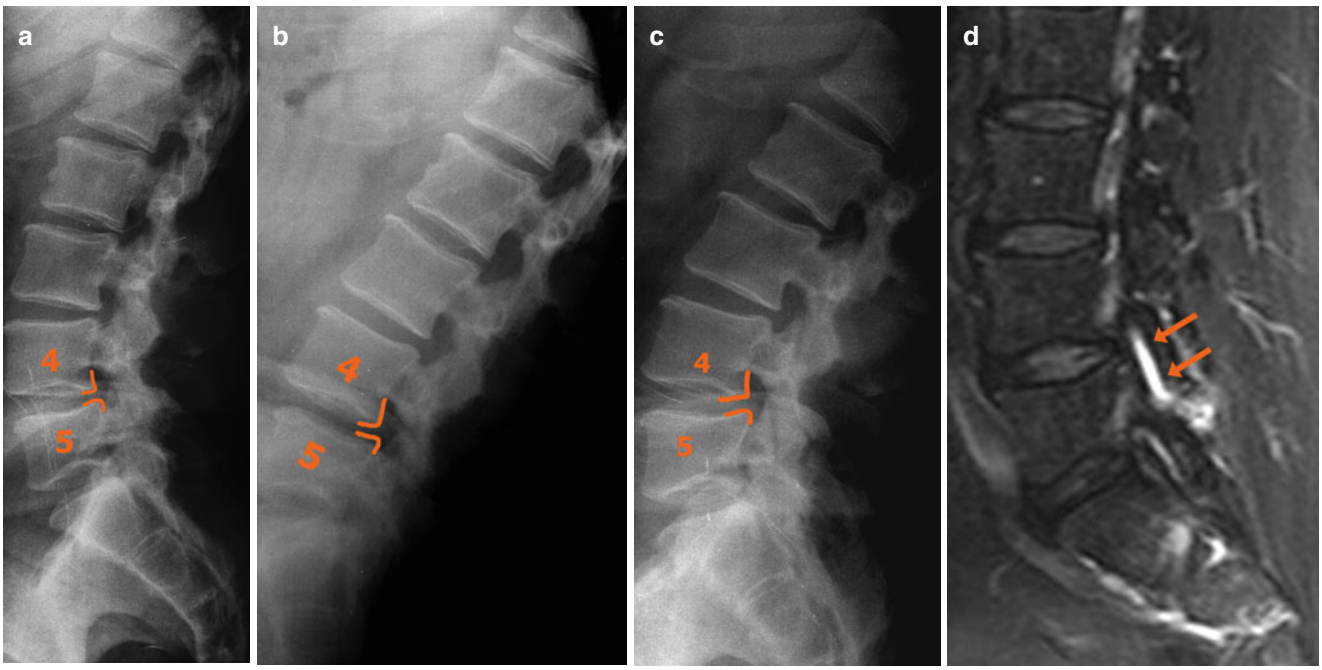


**Fig. 1** (**a**–**e**) Dynamic X-ray of the lumbosacral spine (**a**–**c**), sagittal T2-weighted image with fat saturation (**d**), axial SE T1-weighted image with fat saturation following administration of contrast medium (**e**). Mild anterolisthesis of L4 over L5 (**a**–**c**), more evident in hyperflexion (**b**). Right facet joint osteoarthritis, with T2-hyperintensity (**d**, *arrows*) and marked contrast enhancement of the joint space indicating synovitis (**e**, *circle*). The osteoarthritis is clearly an expression of the vertebral instability

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DOI 10.1007/978-3-319-09447-2_21, © Springer International Publishing Switzerland 2015

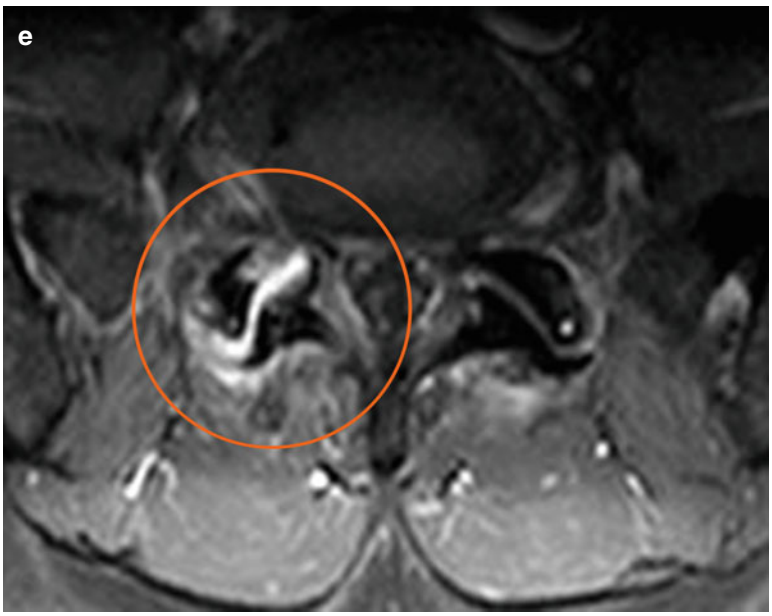


**Fig. 1** (continued)

- A 61-year-old female
- Deviation of the head
- Chronic cervical pain

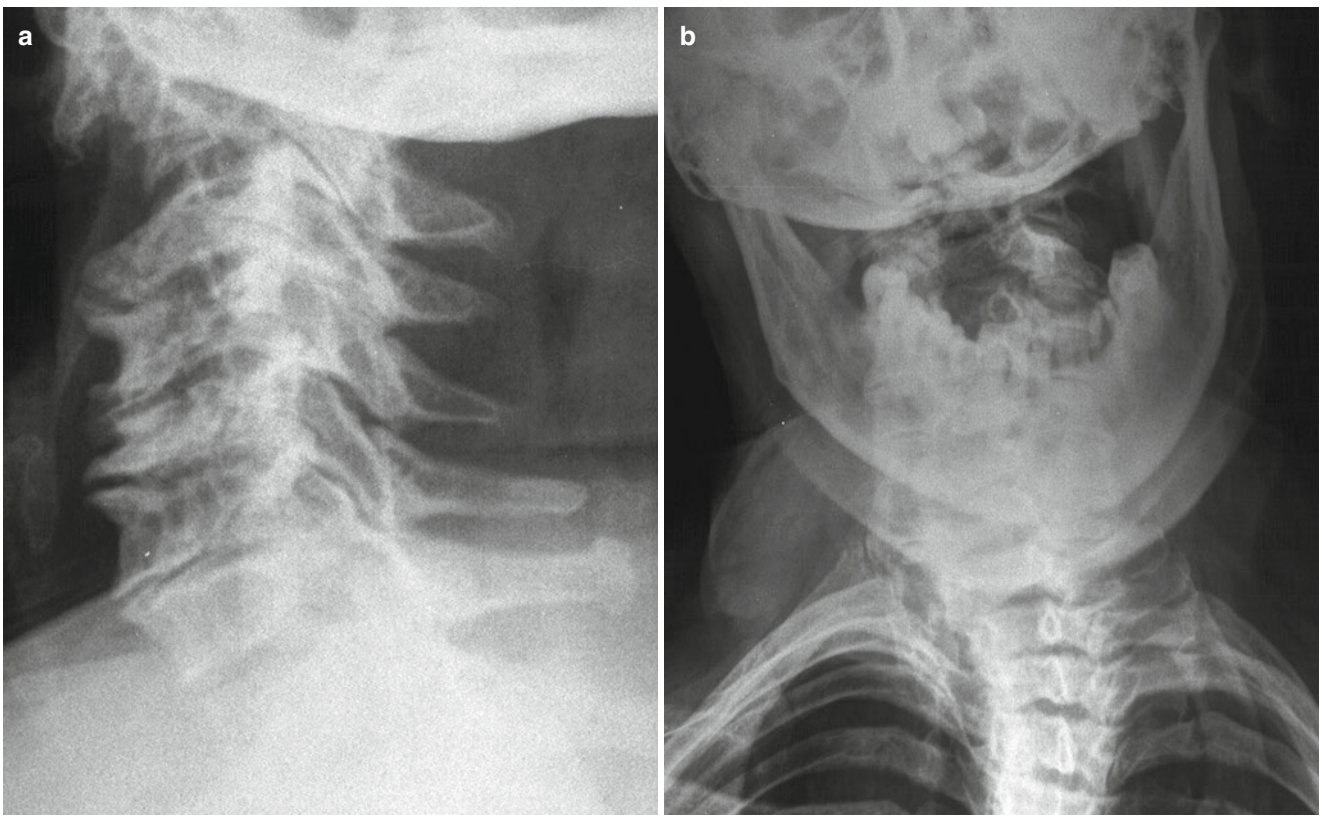


**Fig. 1** (**a**–**g**) Lateral and AP plain film radiographs (**a**, **b**), axial SE T1-weighted images with fat saturation after contrast medium administration (**c**–**e**) , CT images (**f**, **g**). Diffuse spondylotic and osteochondrotic degenerative changes (**a**). Right deviation of the head in malformation of the craniocervical junction (**b**). MRI shows marked contrast enhancement of the atlanto-axial and atlanto-odontoid joint on the right side, indicating osteoarthritis in active-inflammatory phase (**c**–**e**, *arrows*). Note also diffuse epidural contrast enhancement at the same level, indicating a reactive epidural inflammation (**e**). CT images show widening of the distance between the right lateral mass of the atlas and the odontoid process (**f**, **g**, *arrow*)

P. D'Aprile, *MRI of Degenerative Disease of the Spine: A Case-Based Atlas*,
DOI 10.1007/978-3-319-09447-2_22, © Springer International Publishing Switzerland 2015

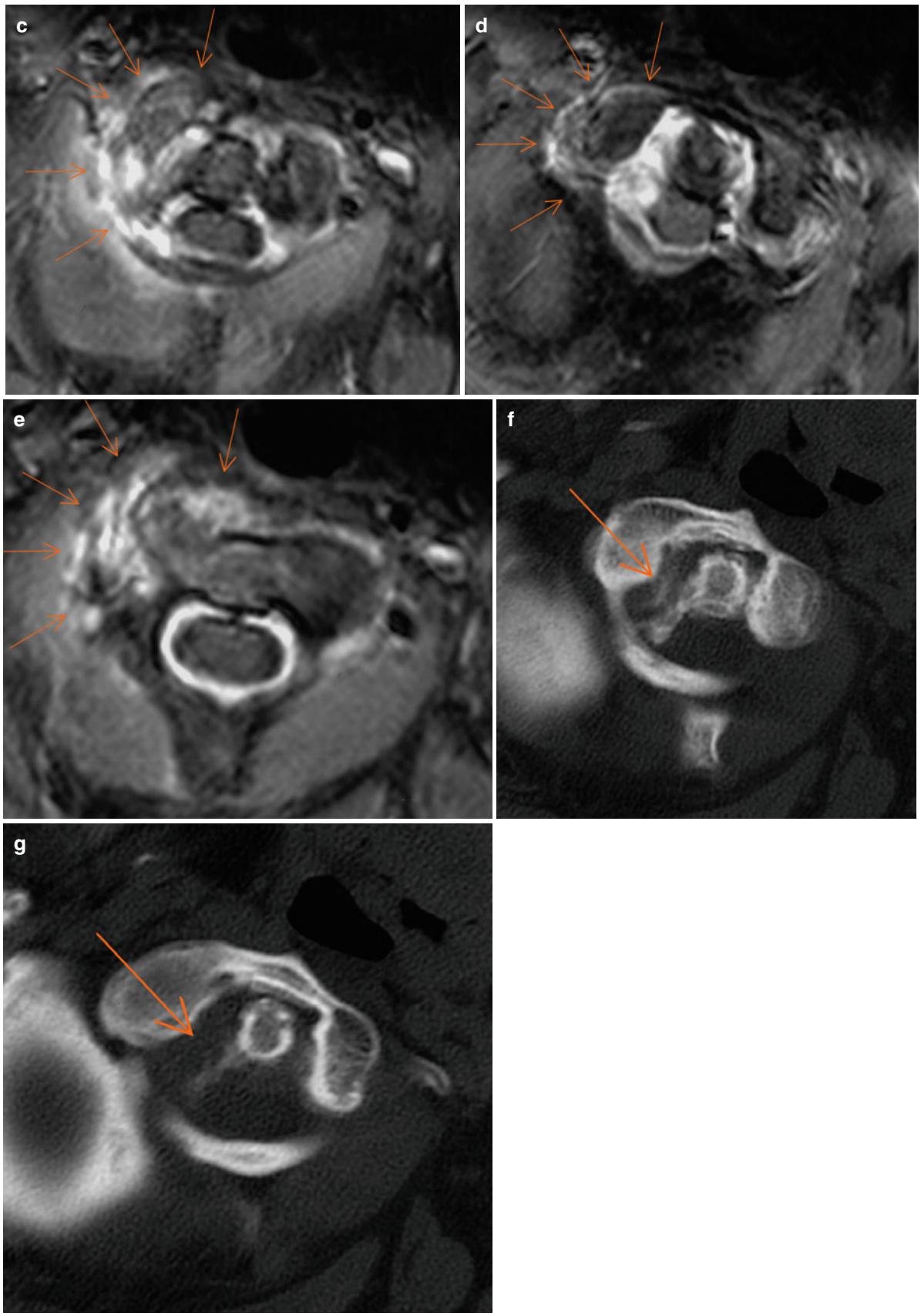


**Fig. 1** (continued)

# Peridiscal Inflammation

# Synovitis

# Bone Collision

Case 21

- A 74-year-old patient
- Chronic low back pain
- Radiculopathy on the right side
- Exacerbation of pain with lateral flexion on the right side

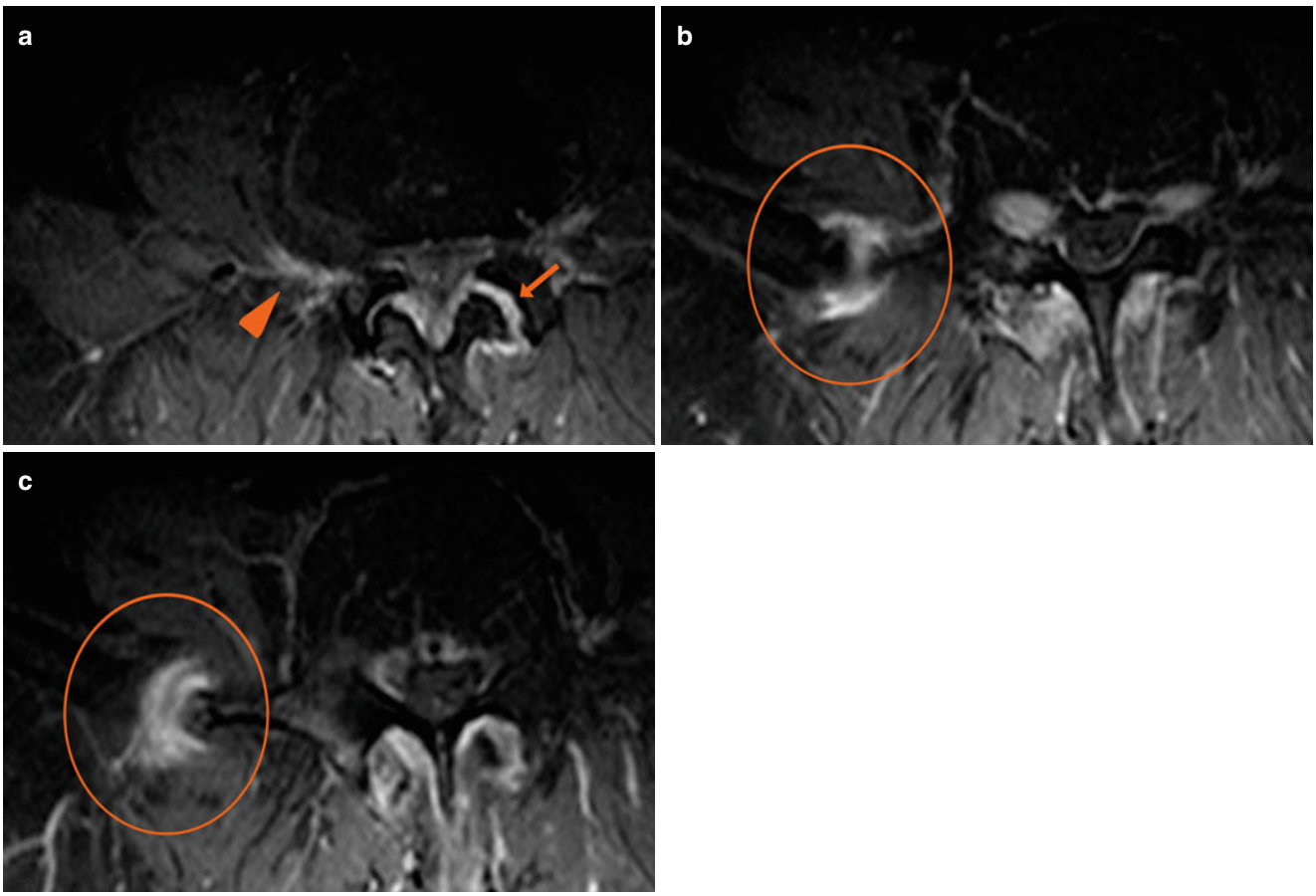


**Fig. 1** (**a**–**c**) Axial SE T1-weighted images with fat saturation following administration of contrast medium (**a**–**c**). Extraforaminal disc protrusion with surrounding contrast enhancement due to peridiscal inflammation (**a**, *arrowhead*). Contrast enhancement of the left facet joint space, indicating synovitis (**a**, *arrow*). Bone collision between the right transverse process of L4 and ipsilateral iliac wing, with contrast enhancement at the same level indicating reactive-inflammatory phenomena (**b**, **c**, *circle*)

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DOI 10.1007/978-3-319-09447-2_23, © Springer International Publishing Switzerland 2015

# Atlanto-Axial Osteoarthritis

Case 22

- A 68-year-old woman
- Cervical pain
- Limitation of motion of the head

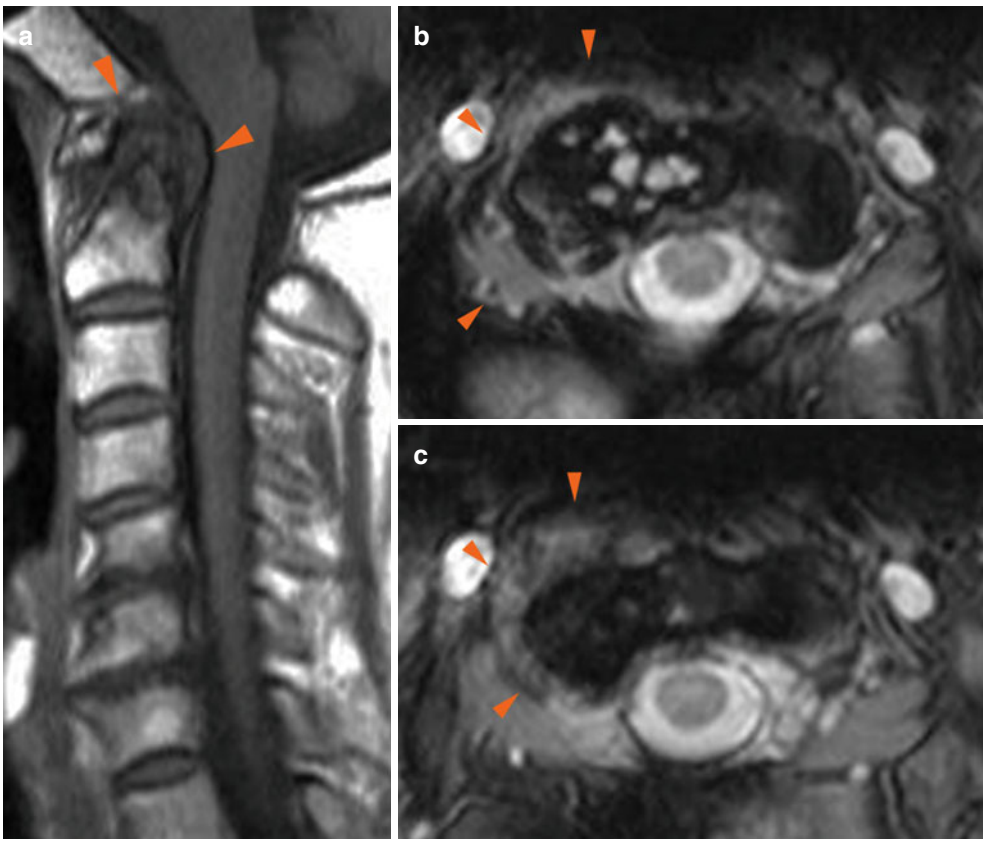


**Fig. 1** Sagittal SE T1-weighted image (**a**), axial GE T2*-weighted images (**b**, **c**), sagittal and axial SE T1-weighted images with fat saturation after the administration of contrast medium (**d**–**g**) , axial CT images (**h**, **i**). Malformation of the cranio-cervical junction, with posterior atlanto-occipital fusion (**a**). T1-weighted imaging shows pathological tissue at C1/C2 (**a**, *arrowheads*). Multiple small hyperintense areas in T2 at the level of atlanto-axial and atlanto-odontoid joints on the right side and periarticular soft tissues (**b**, **c**, *arrowheads*). The same areas enhance after administration of contrast medium (**e**–**g**, *arrowheads*). CT images show osteosclerosis, joint erosions and cysts (**h**, **i**, *arrowheads*). These findings indicate osteoarthritis in active-inflammatory phase, with periarticular soft tissue inflammation

P. D'Aprile, *MRI of Degenerative Disease of the Spine: A Case-Based Atlas*,
DOI 10.1007/978-3-319-09447-2_24, © Springer International Publishing Switzerland 2015

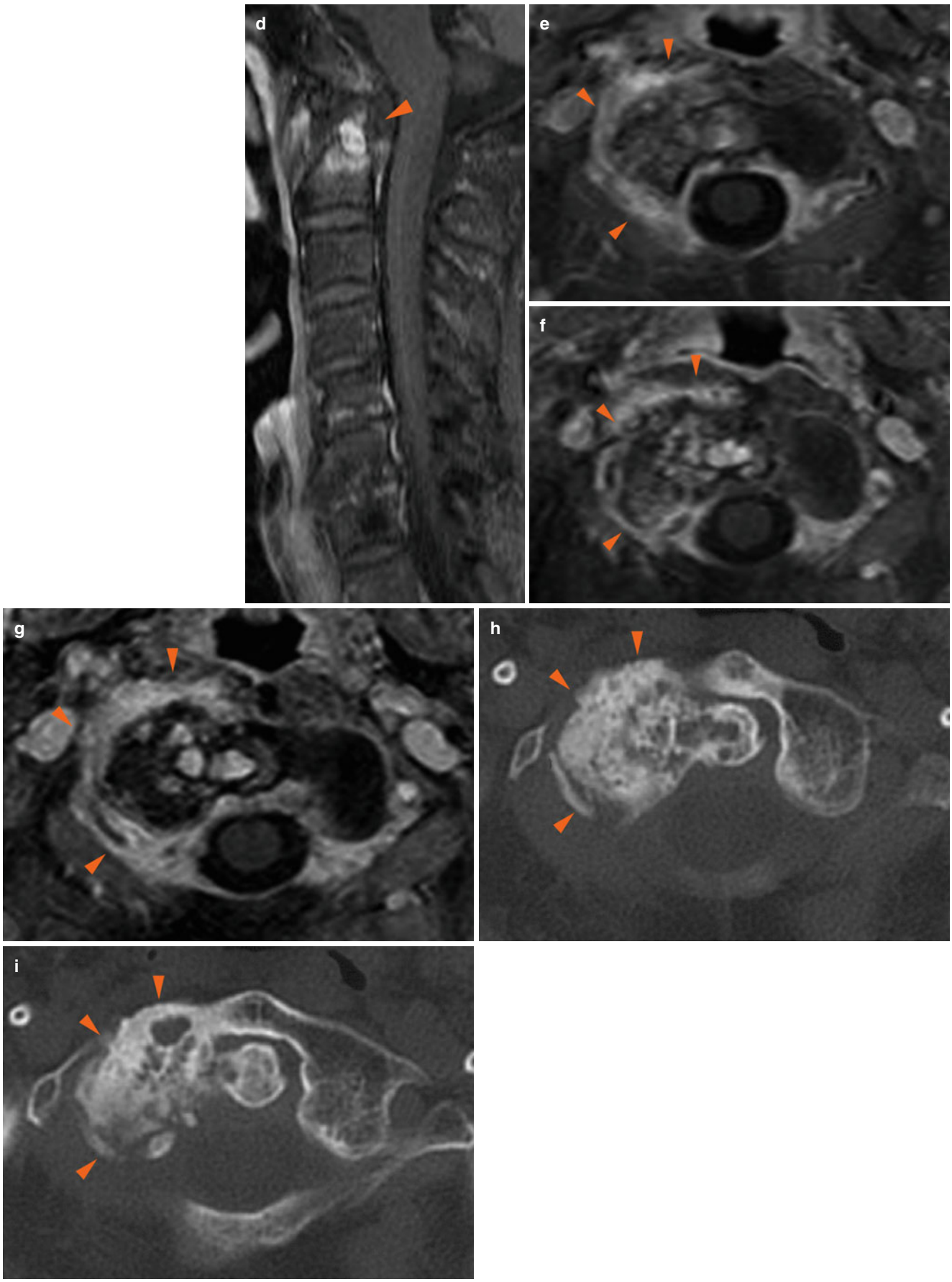


**Fig. 1** (continued)

# Spine Instability

## Facet Joint Osteoarthritis

Case **23**

- A 57-year-old man
- Low back pain exacerbated by flexion
- Point tenderness corresponding to L4/L5 right facet joint

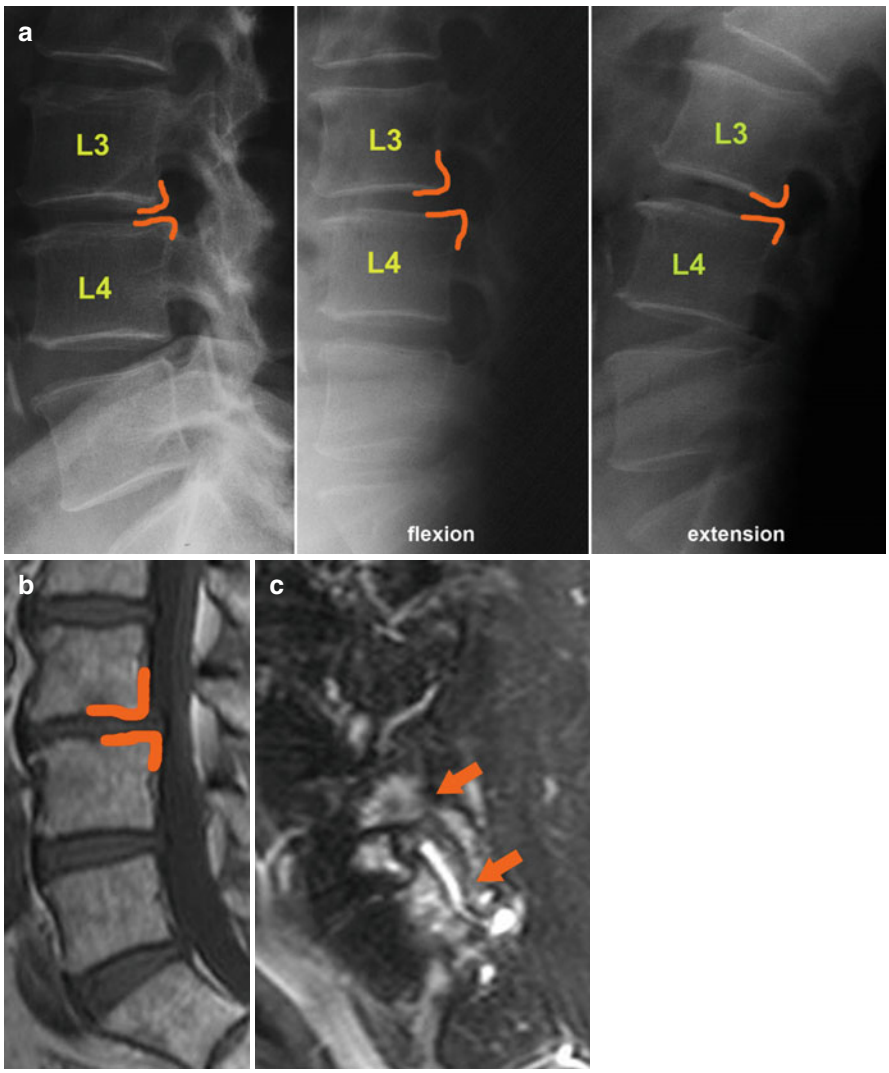


**Fig. 1** Dynamic lumbar radiography (**a**), sagittal SE T1-weighted image (**b**), sagittal and axial SE T2-weighted images with fat saturation (**c**–**f**), axial SE T1-weighted images with fat saturation following the administration of contrast medium (**g**–**i**). Anterolisthesis of L3 over L4, more marked in flexion (**a**). MRI confirms the spinal instability L3/L4 (**b**). The same patient presented right facet joint osteoarthritis at L5/S1, with bone oedema and intra-articular effusion (**c**–**f**, *arrow*). The administration of contrast medium shows enhancement of the same apophyseal processes, confirming the aseptic inflammation of the bone marrow, i.e. osteitis in the context of osteoarthritis (**g**, *arrow*). Note also synovial hypertrophy of the same joint, well delineated after contrast medium administration (**h**, **i**, *arrow*)

P. D'Aprile, *MRI of Degenerative Disease of the Spine: A Case-Based Atlas*,
DOI 10.1007/978-3-319-09447-2_25, © Springer International Publishing Switzerland 2015

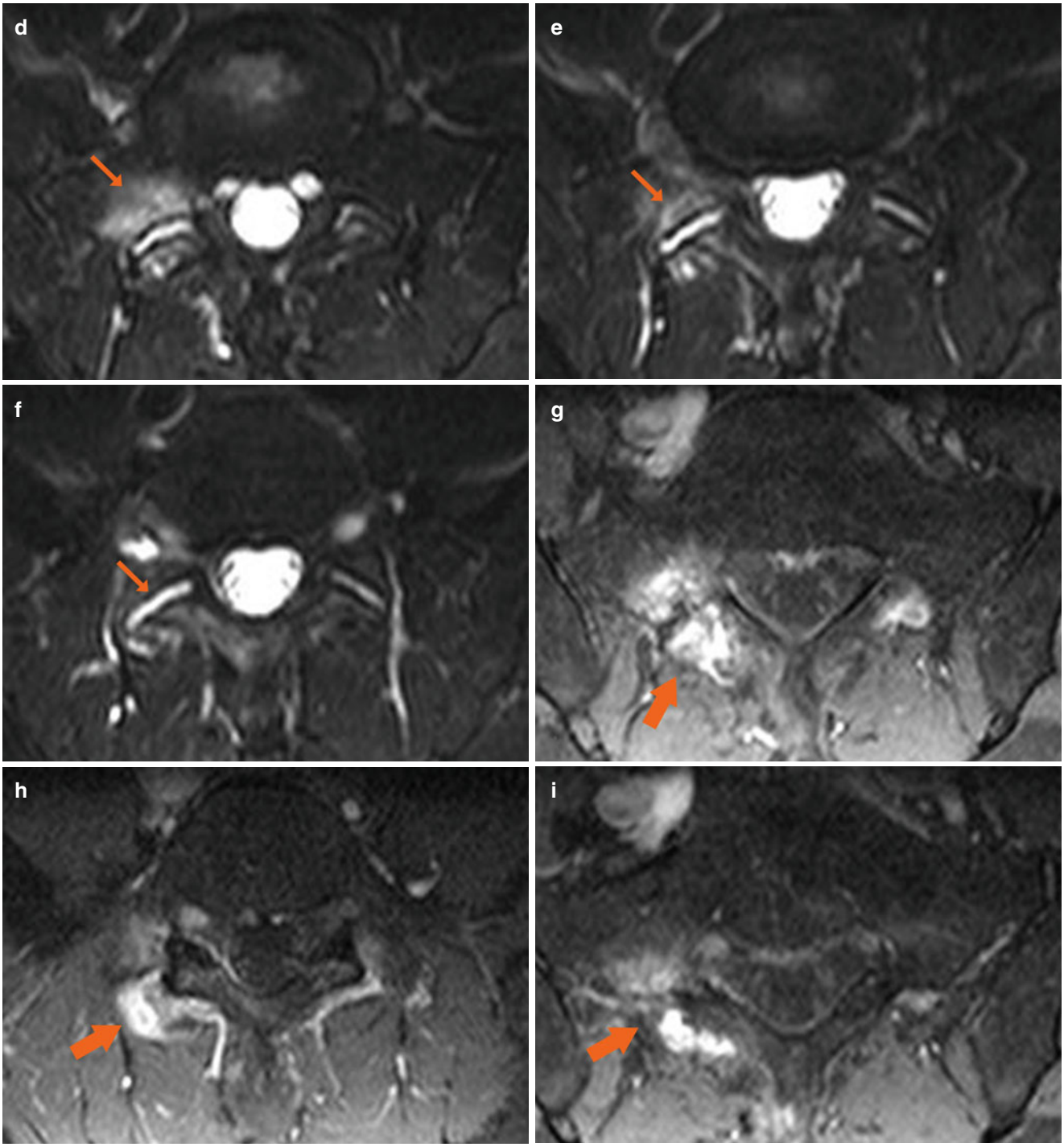


**Fig. 1** (continued)

# Synovitis

## Inflammation of Ligaments

Case **24**

- A 61-year-old man
- Persistent low back pain
- Tenderness on palpation of the lumbar interspinous spaces

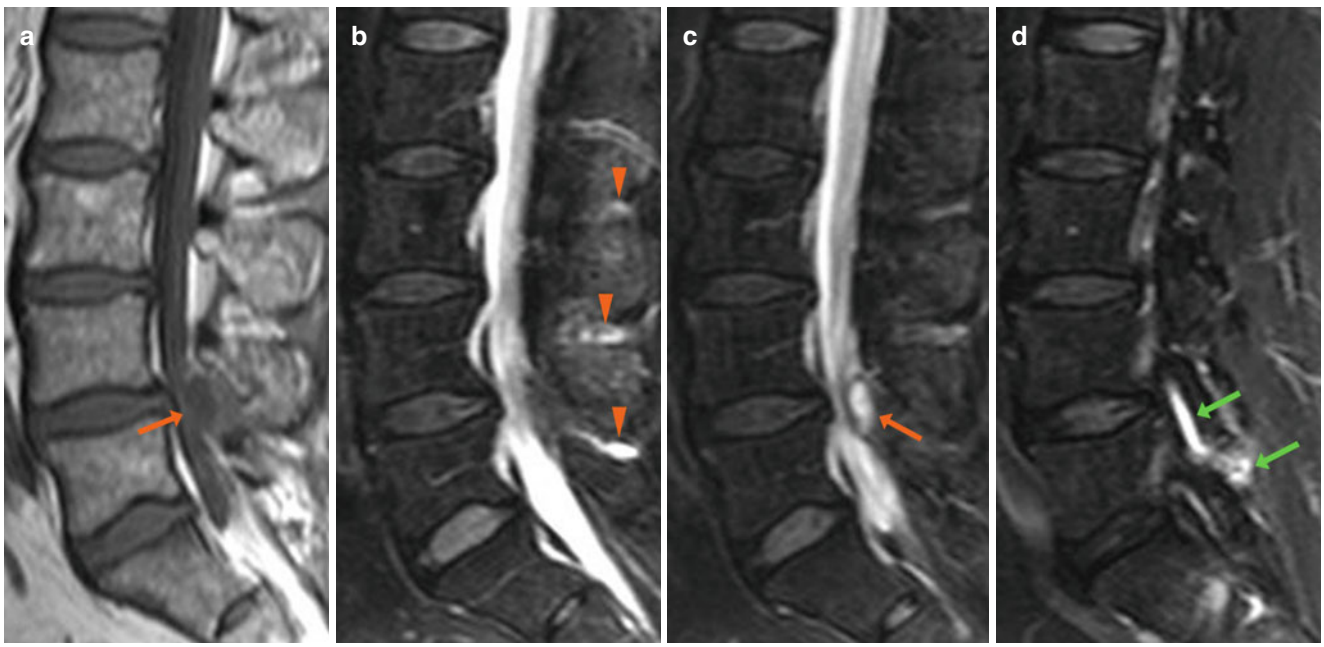


**Fig. 1** Sagittal SE T1-weighted image (**a**), sagittal TSE T2-weighted images with fat saturation (**b**–**d**), sagittal and axial SE T1-weighted images with fat saturation following administration of contrast medium (**e**–**g**, **i**, **j**), axial CISS T2-weighted image (**h**). Mild anterolisthesis of L4 over L5 (**a**–**g**). Hypertrophy and contrast enhancement of the L4/L5 left flavum ligament, due to inflammation with cystic component (**a**, **c**, **e**, **f**, **l**, *red arrow*). T2 hyperintensity and contrast enhancement of the interspinous ligament from L2 to L5, indicating ligamentous degenerative-inflammatory changes (**b**, **e**, **i**, *arrowhead*). The L4/L5 left facet joint space is widened (**d**, **h**, *green arrows*) and enhances after contrast medium administration (**g**, **l**, *green arrows*), indicating synovitis; note also synovial hypertrophy postero-inferiorly to the same joint (**g**)

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DOI 10.1007/978-3-319-09447-2_26, © Springer International Publishing Switzerland 2015

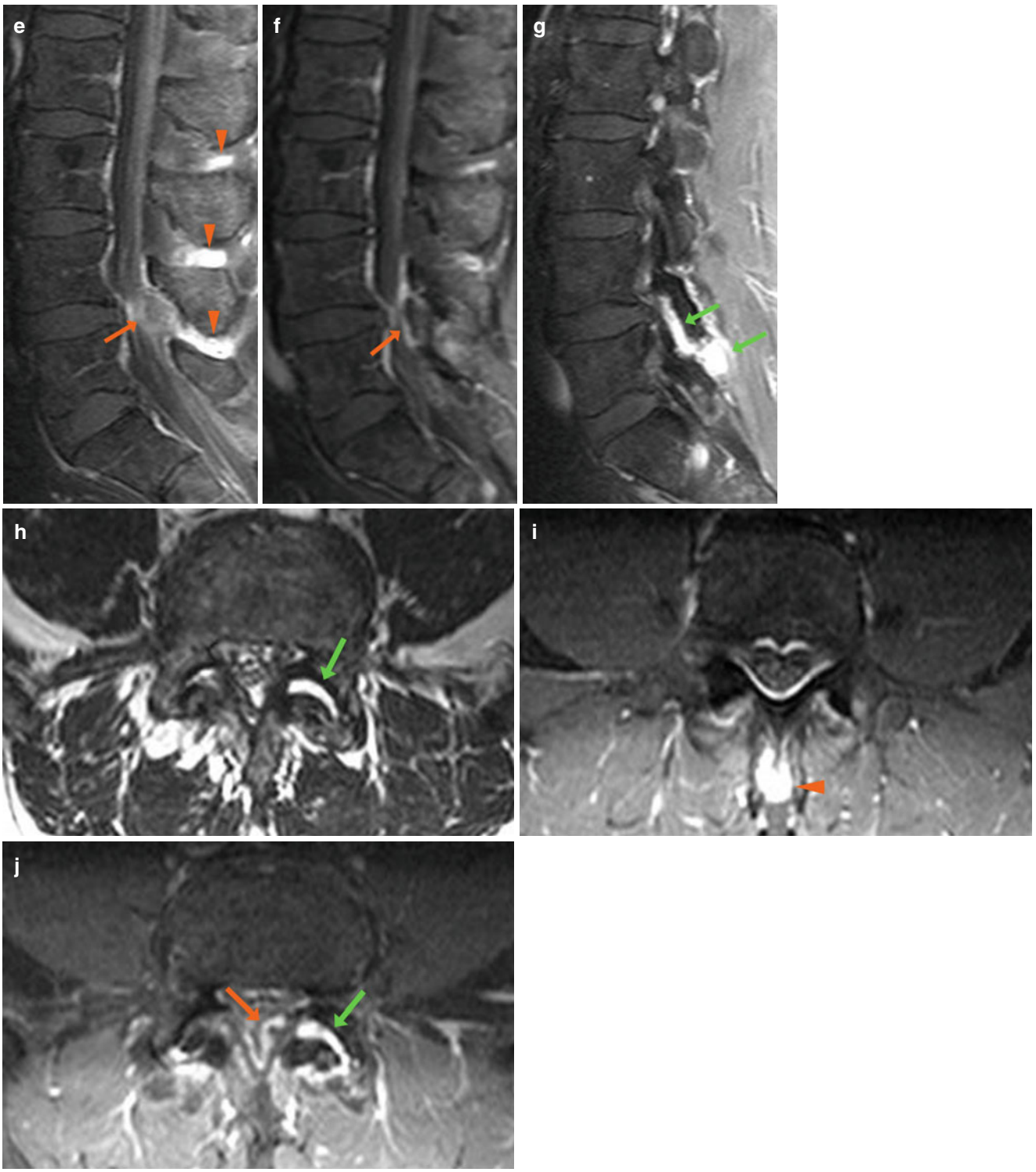


**Fig. 1** (continued)

# Synovial Cyst

Case 25

- A 59-year-old male
- Neck pain with radiculopathy on the left side

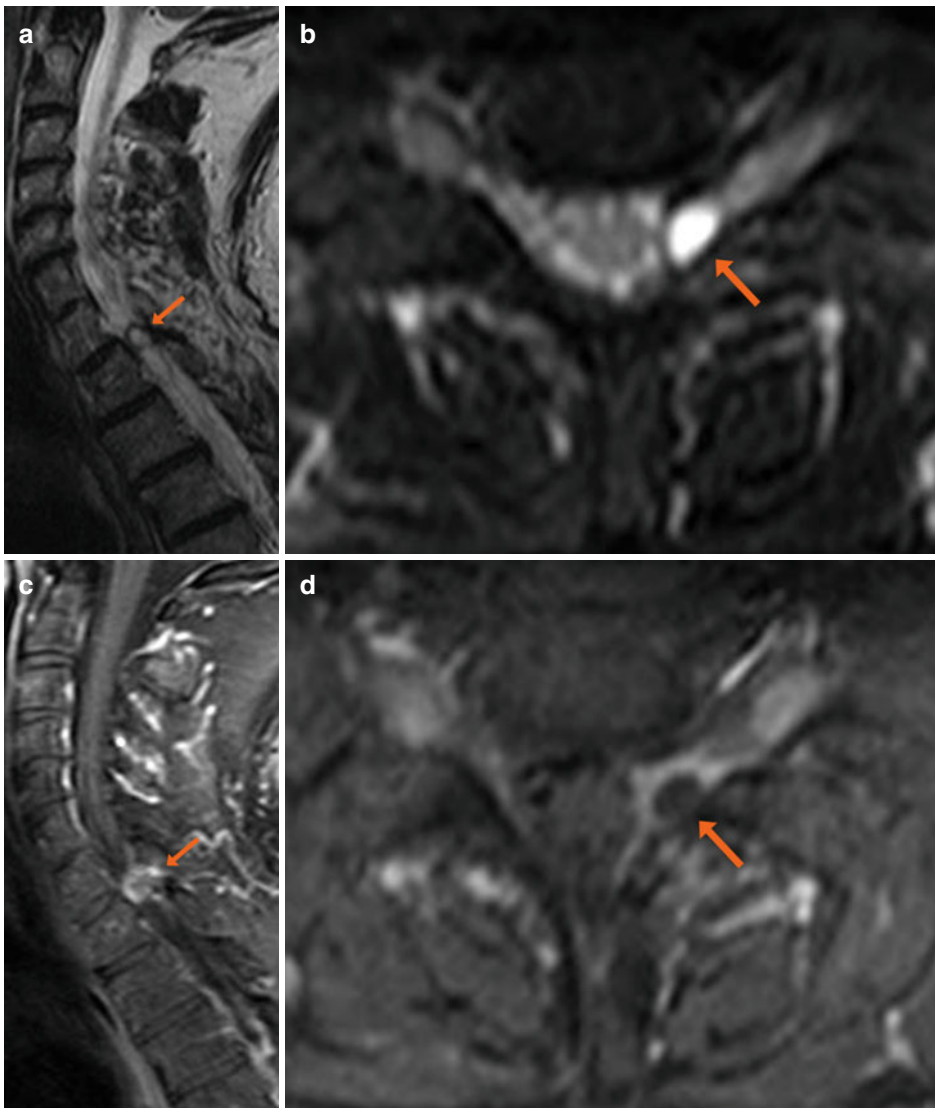


**Fig. 1** Sagittal TSE T2-weighted image (**a**), axial TSE T2-weighted image with fat saturation (**b**) , sagittal and axial SE T1-weighted images with fat saturation following administration of contrast medium (**c**, **d**). Presence of synovial cyst in the left lateral recess of the spinal canal at C7/D1 (**a**–**d**, *arrow*). The cyst is hyperintense on T2-weighted images (**a**, **b**) and hypointense in T1, with peripheral enhancement after contrast medium administration (**c**, **d**)

P. D'Aprile, *MRI of Degenerative Disease of the Spine: A Case-Based Atlas*,
DOI 10.1007/978-3-319-09447-2_27, © Springer International Publishing Switzerland 2015

# Synovial Cyst

# Facet Joint Osteoarthritis

Case 26

- A 74-year-old woman
- Patient with persistent neck pain, more marked on the right side

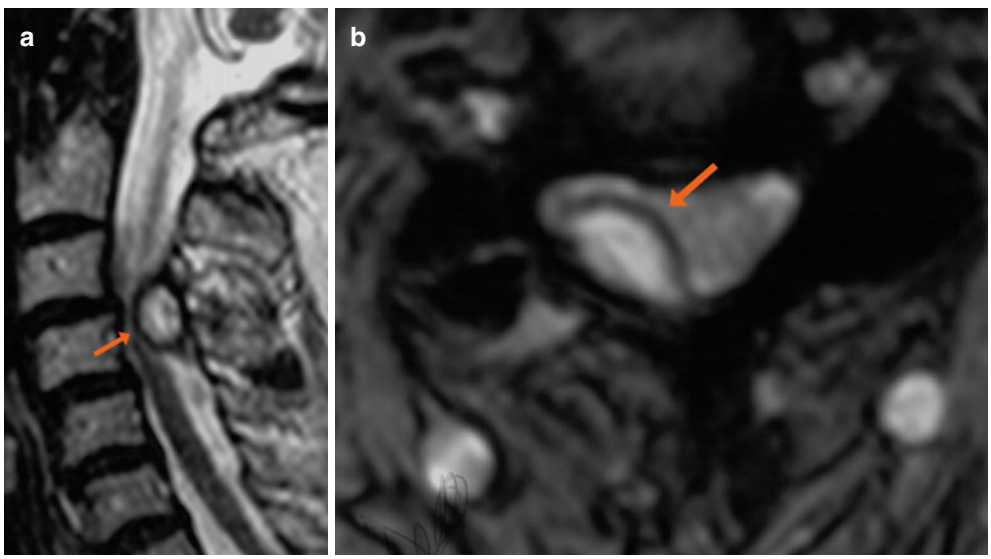


**Fig. 1** Sagittal TSE T2-weighted image (**a**), axial GE T2*-weighted image (**b**), axial SE T1-weighted image with fat saturation after administration of contrast medium (**c**), axial CT scan (**d**). Anterolisthesis of C3 over C4 and C4 over C5. Cystic lesion in the right part of the vertebral canal (*arrow*), hyperintense in T2 (**a**, **b**) and hypointense in T1, with peripheral contrast enhancement (**c**). The cyst compresses and displaces the spinal cord. This is a typical intracanal synovial cyst. Note also a right facet joint osteoarthritis, with contrast enhancement of the apophyses and periapophyseal soft tissues, indicating active inflammation (**c**, *thin arrows*). CT scan shows osteosclerosis of the apophyses and erosions of the articular surfaces (**d**, *thin arrows*). Most probably the synovial cyst is an epiphenomenon of the adjacent osteoarthritis and vertebral instability

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DOI 10.1007/978-3-319-09447-2_28, © Springer International Publishing Switzerland 2015

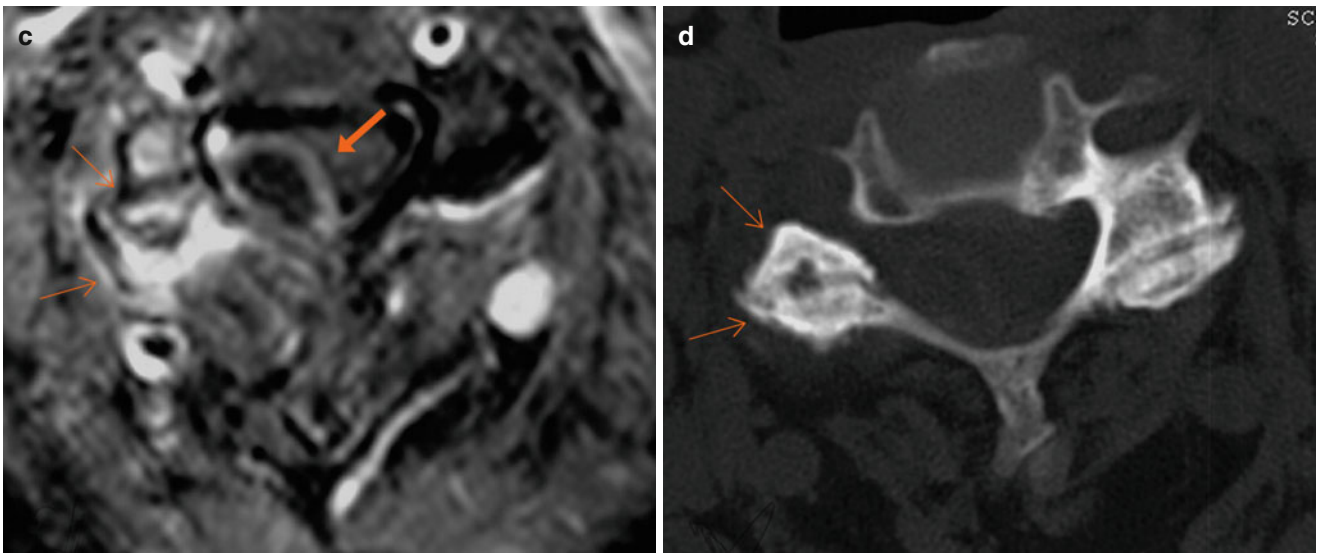


**Fig. 1** (continued)

# Synovial Cyst

Case 27

- A 64-year-old male
- Chronic neck pain

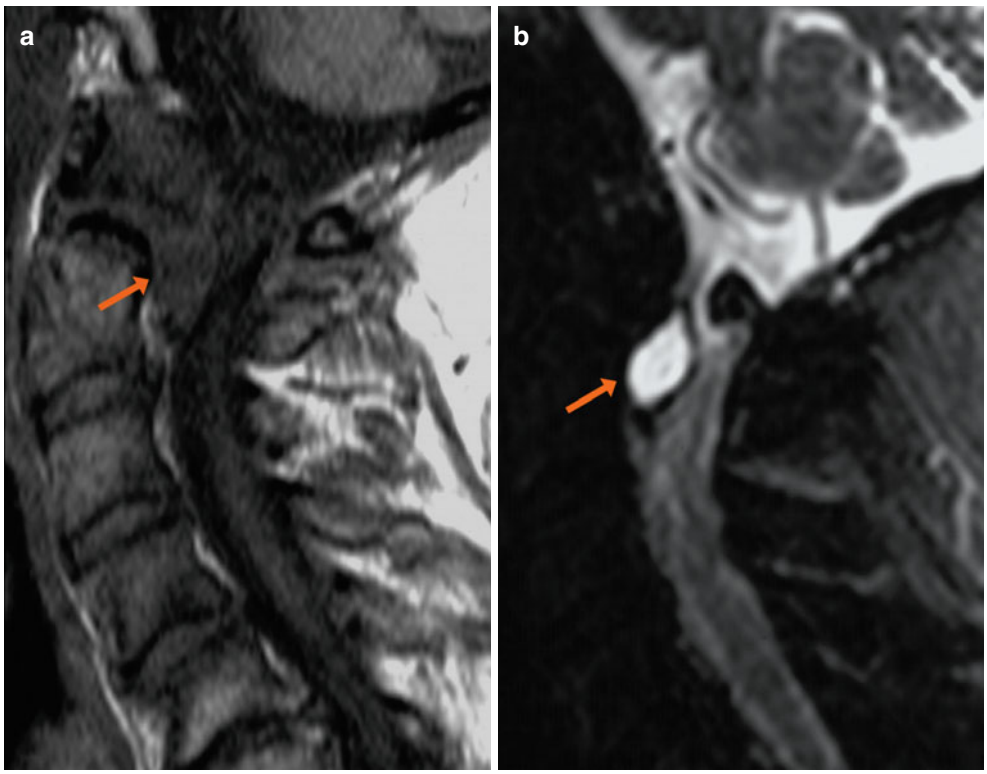


**Fig. 1** Sagittal SE T1-weighted image (**a**), sagittal and axial TSE T2-weighted images with fat saturation (**b**, **c**) , sagittal and axial SE T1-weighted images with fat saturation after administration of contrast medium (**d**, **e**). Extradural cystic lesion in the right part of the vertebral canal (*arrow*), hypointense in T1 (**a**) and hyperintense in T2 (**b**, **c**), with peripheral contrast enhancement (**d**, **e**). Diagnosis: intracanal synovial cyst

P. D'Aprile, *MRI of Degenerative Disease of the Spine: A Case-Based Atlas*,
DOI 10.1007/978-3-319-09447-2_29, © Springer International Publishing Switzerland 2015

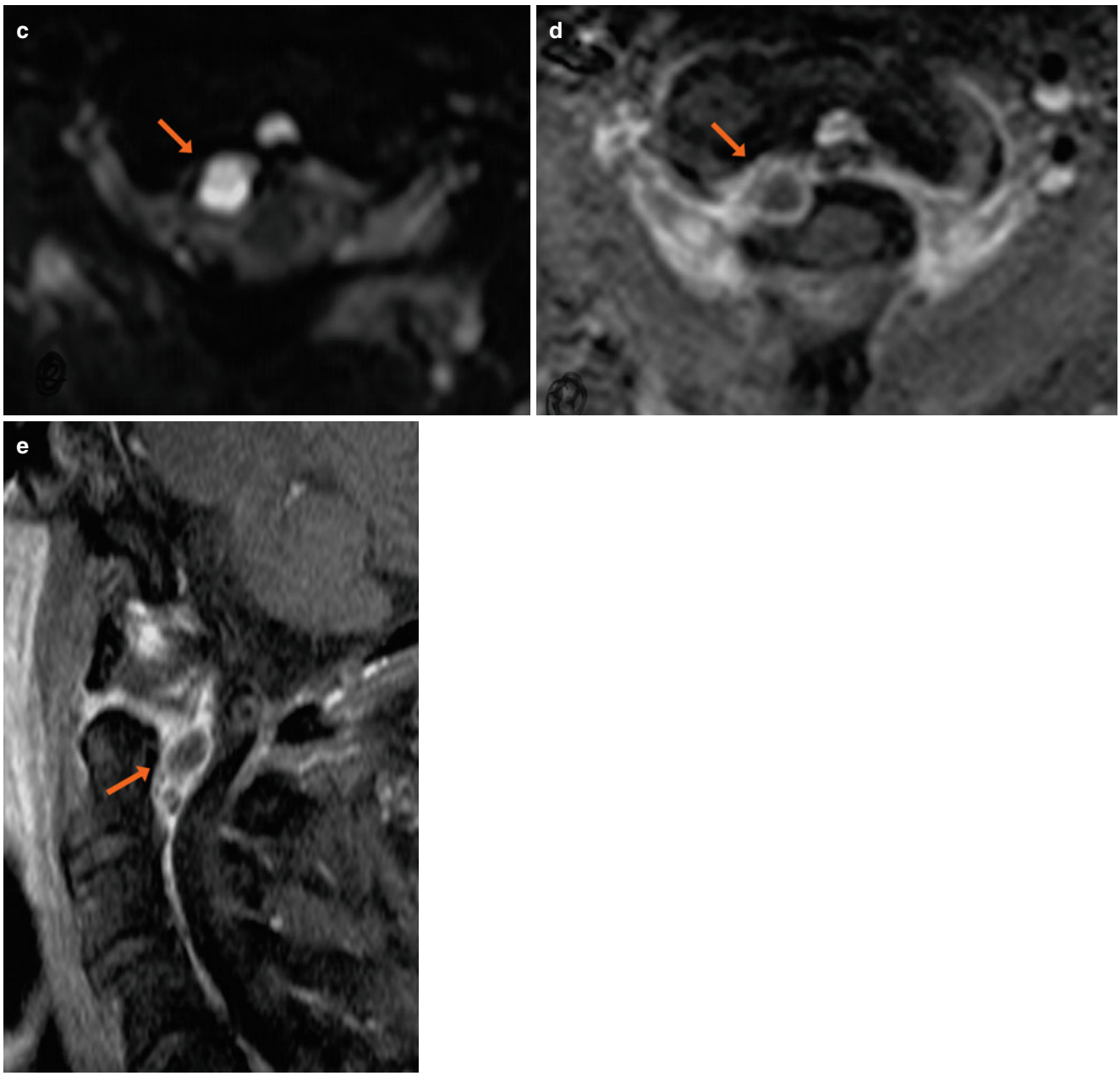


**Fig. 1** (continued)

# Synovial Cyst

Case 28

- A 43-year-old female
- Low back pain with radiculopathy on the right side

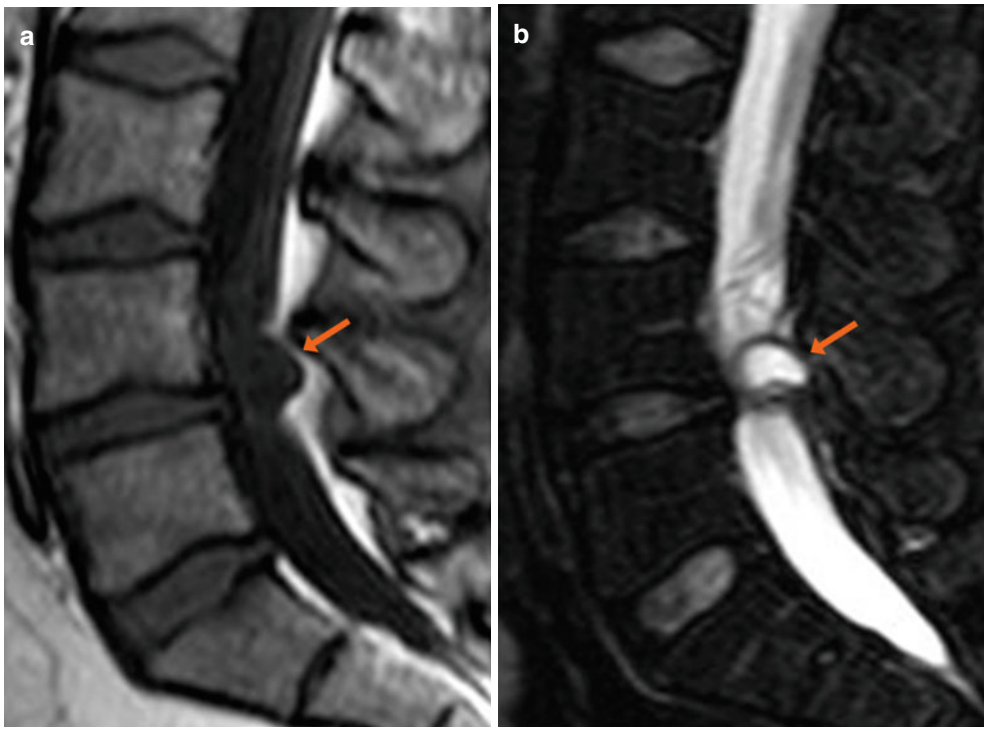


**Fig. 1** Sagittal SE T1-weighted image (**a**), sagittal and axial TSE T2-weighted images with fat saturation (**b**, **c**) , sagittal and axial SE T1-weighted images with fat saturation following administration of contrast medium (**d**, **e**). Endocanal synovial cyst at L4/L5 on the right side (**a**–**e**, *arrow*). The cyst displaces the thecal sac. Cystic content resembles CSF in all sequences (**a**–**c**). Cyst capsule enhances following contrast administration (**d**, **e**)

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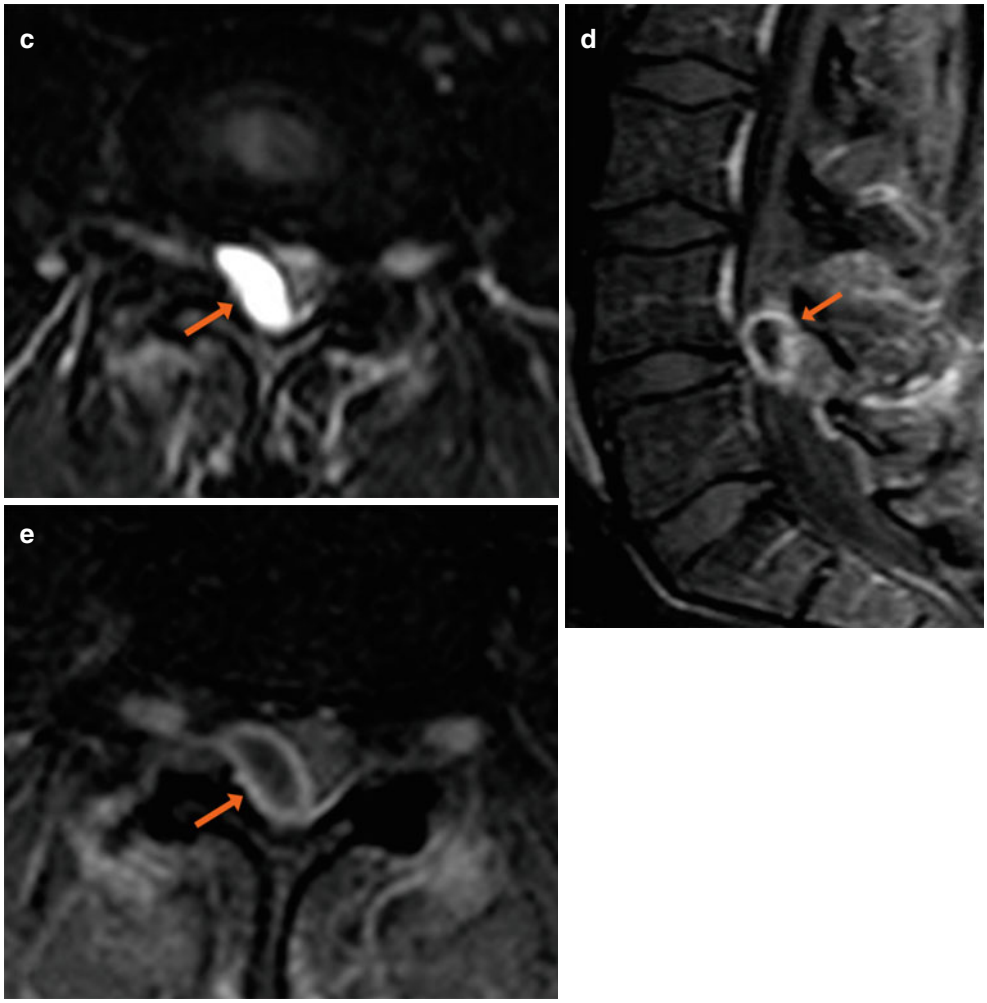
**Fig. 1** (continued)



# Synovial Cyst

Case 29

- A 45-year-old male
- Left L5 radiculopathy

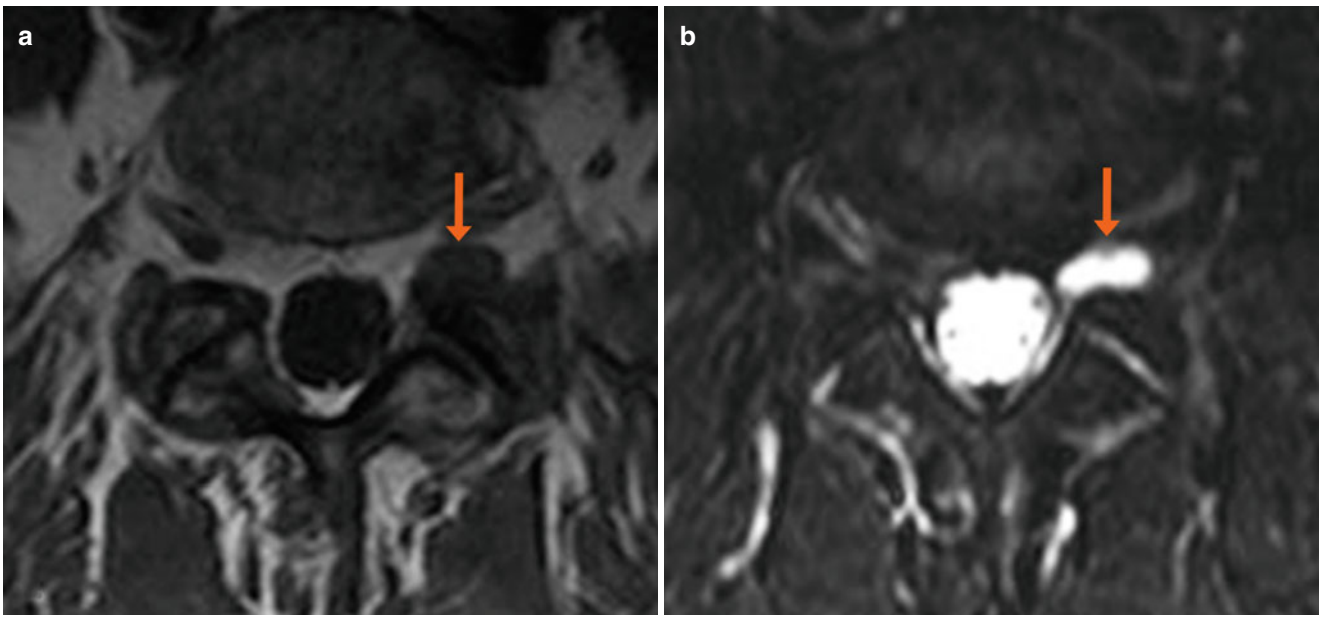



**Fig. 1** Axial SE T1-weighted image (**a**), axial TSE T2-weighted image with fat saturation (**b**) , sagittal and axial SE T1-weighted images with fat saturation following administration of contrast medium (**c**–**e**). Intraforaminal cystic lesion at L5/S1 on the left side (**a**–**c**, **e**, *arrow*). Cystic content resembles CSF. The wall of the cyst shows a thin and regular contrast enhancement (**c**, **e**). The L5 root is clearly separated and located anteriorly to the cyst (**d**, *arrow*). Diagnosis: intraforaminal synovial cyst. The major differential diagnosis of synovial cyst is a free disc fragment and cystic nerve root tumour. Signal characteristics and position of the lesion allow the proper diagnosis

P. D'Aprile, *MRI of Degenerative Disease of the Spine: A Case-Based Atlas*,
DOI 10.1007/978-3-319-09447-2_31, © Springer International Publishing Switzerland 2015

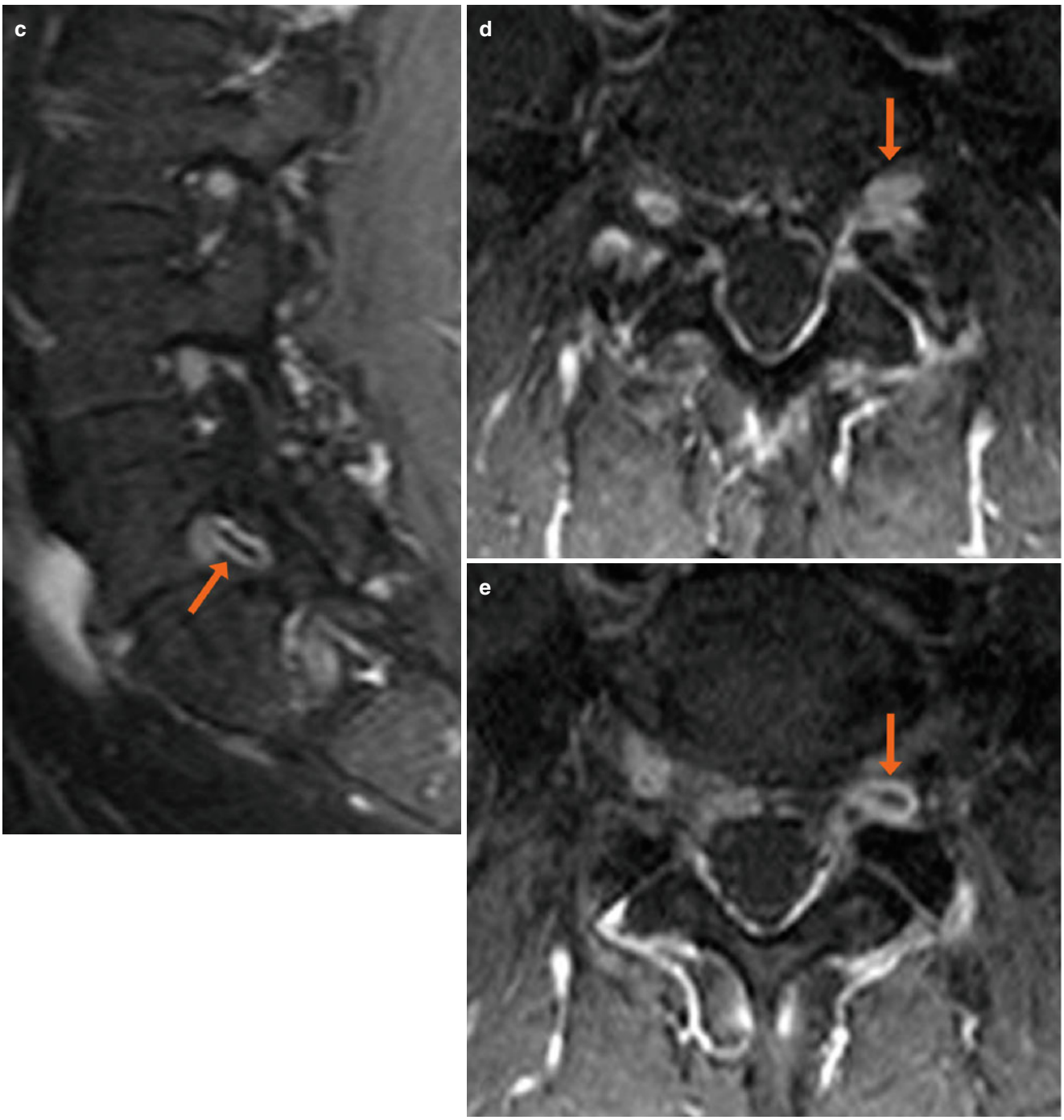



**Fig. 1** (continued)

# Facet Joint Osteoarthritis

# Synovial Cyst

Case 30

- A 54-year-old man
- Chronic low back pain, more marked in flexion and extension

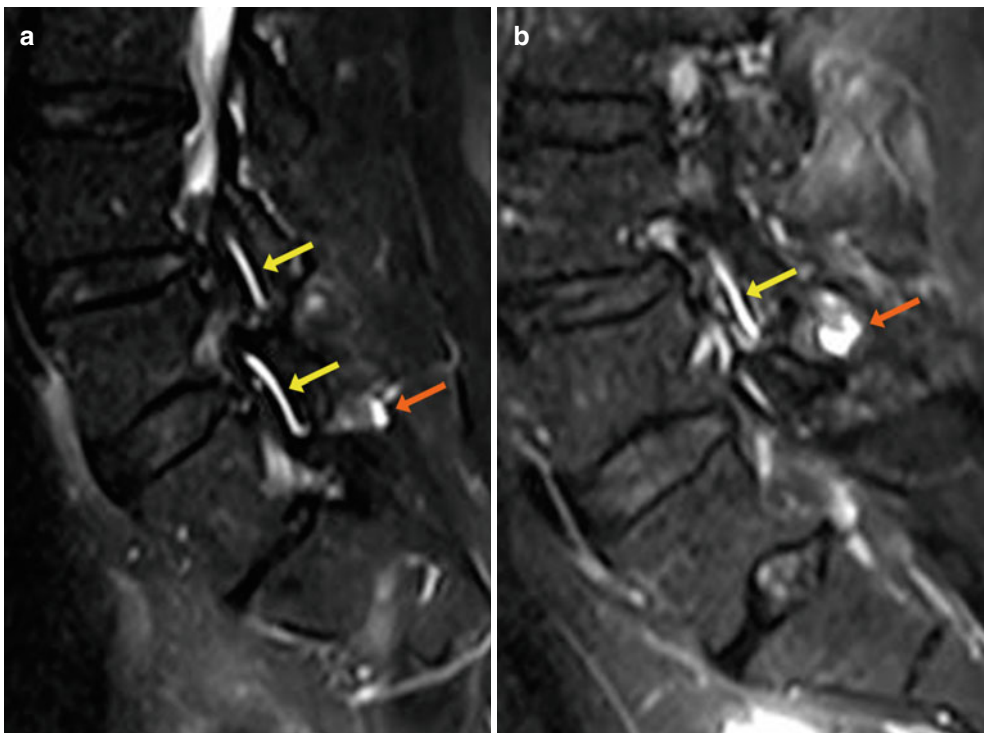



**Fig. 1** Sagittal TSE T2-weighted images with fat saturation (**a**, **b**), sagittal and axial SE T1-weighted images with fat saturation following administration of contrast medium (**c**–**f**). Facet joint arthritis (*yellow arrow*, **a**–**d**). Extracanal synovial cyst, adjacent to degenerated facet joint (*orange arrow*, **a**–**f**). The cyst is more delineated on the left side after contrast medium administration (**d**, **f**)

P. D'Aprile, *MRI of Degenerative Disease of the Spine: A Case-Based Atlas*,
DOI 10.1007/978-3-319-09447-2_32, © Springer International Publishing Switzerland 2015

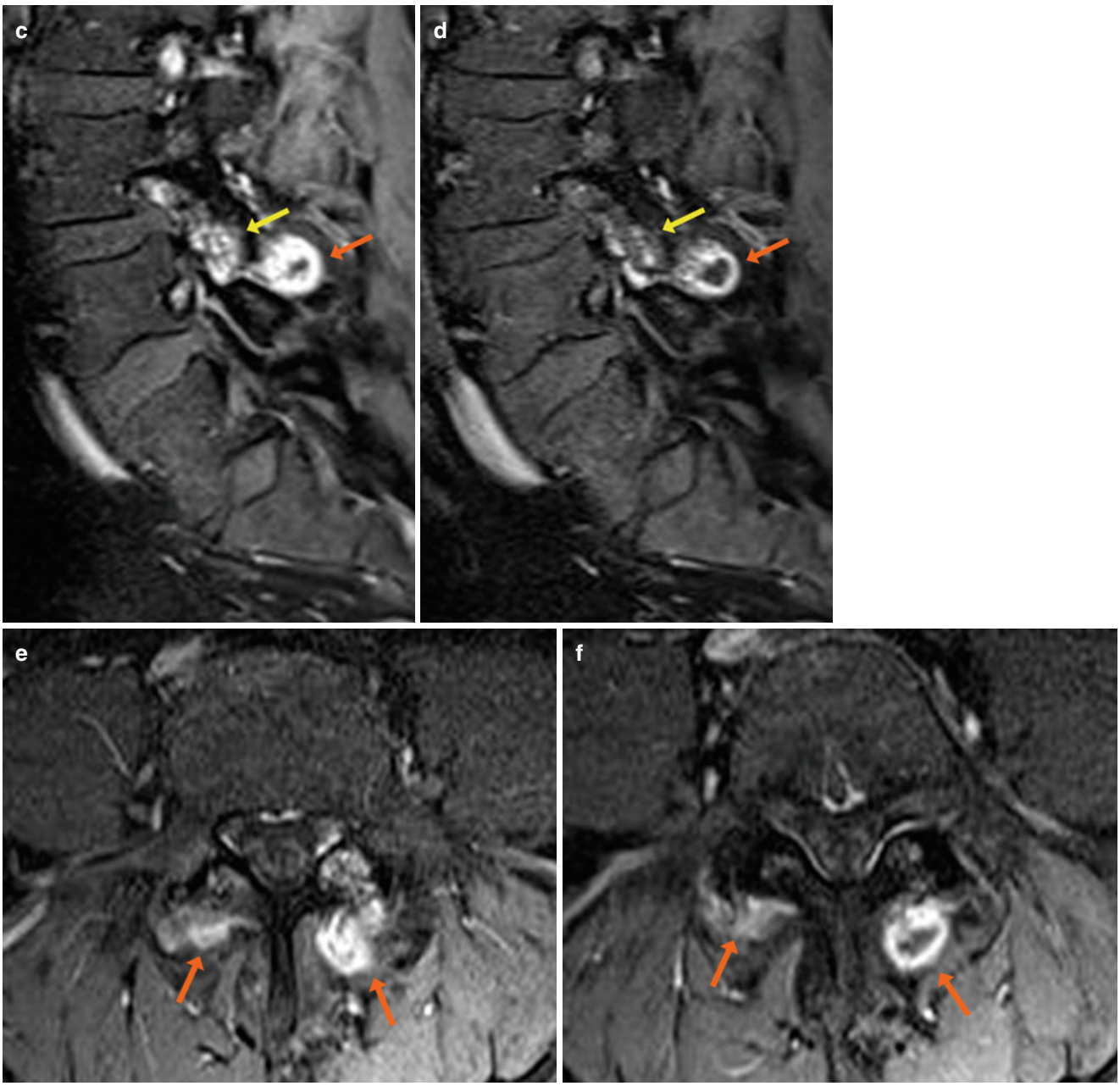



**Fig. 1** (continued)

# Facet Joint Osteoarthritis

# Synovial Cyst

Case 31

- A 46-year-old female
- Low back pain
- Pain exacerbated by flexion and extension

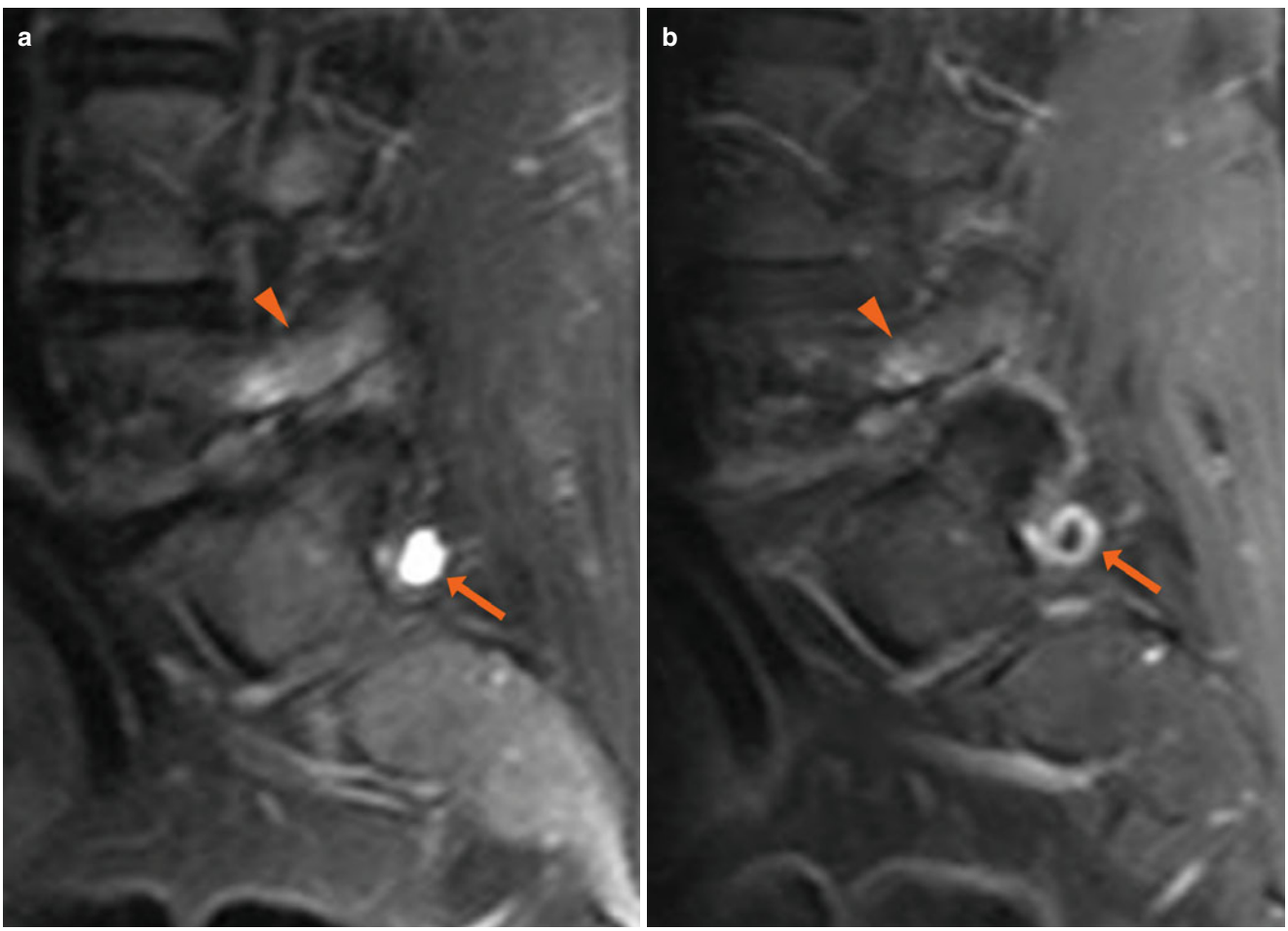



**Fig. 1** Sagittal and axial T2-weighted images with fat saturation (**a**, **c**), sagittal and axial SE T1-weighted images with fat saturation after contrast medium administration (**b**, **d**). Facet joint osteoarthritis with oedema-osteitis that extends to the left pedicle of L5 (**a**, **b**, *arrowheads*). Extracanal synovial cysts and synovial "hypertrophy" adjacent to the facet joints bilaterally (**a**–**d**, *arrows*). Synovial cysts typically show peripheral enhancement after contrast medium administration (**b**, **d**)

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DOI 10.1007/978-3-319-09447-2_33, © Springer International Publishing Switzerland 2015

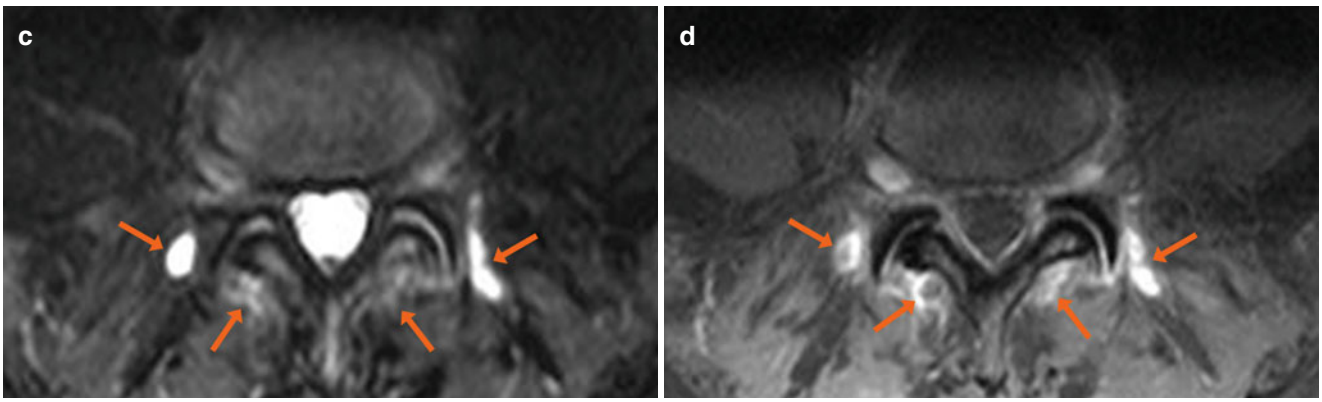


**Fig. 1** (continued)

# Spondylolysis

Case 32

- A 18-year-old male
- Acute-subacute low back pain in a young athlete

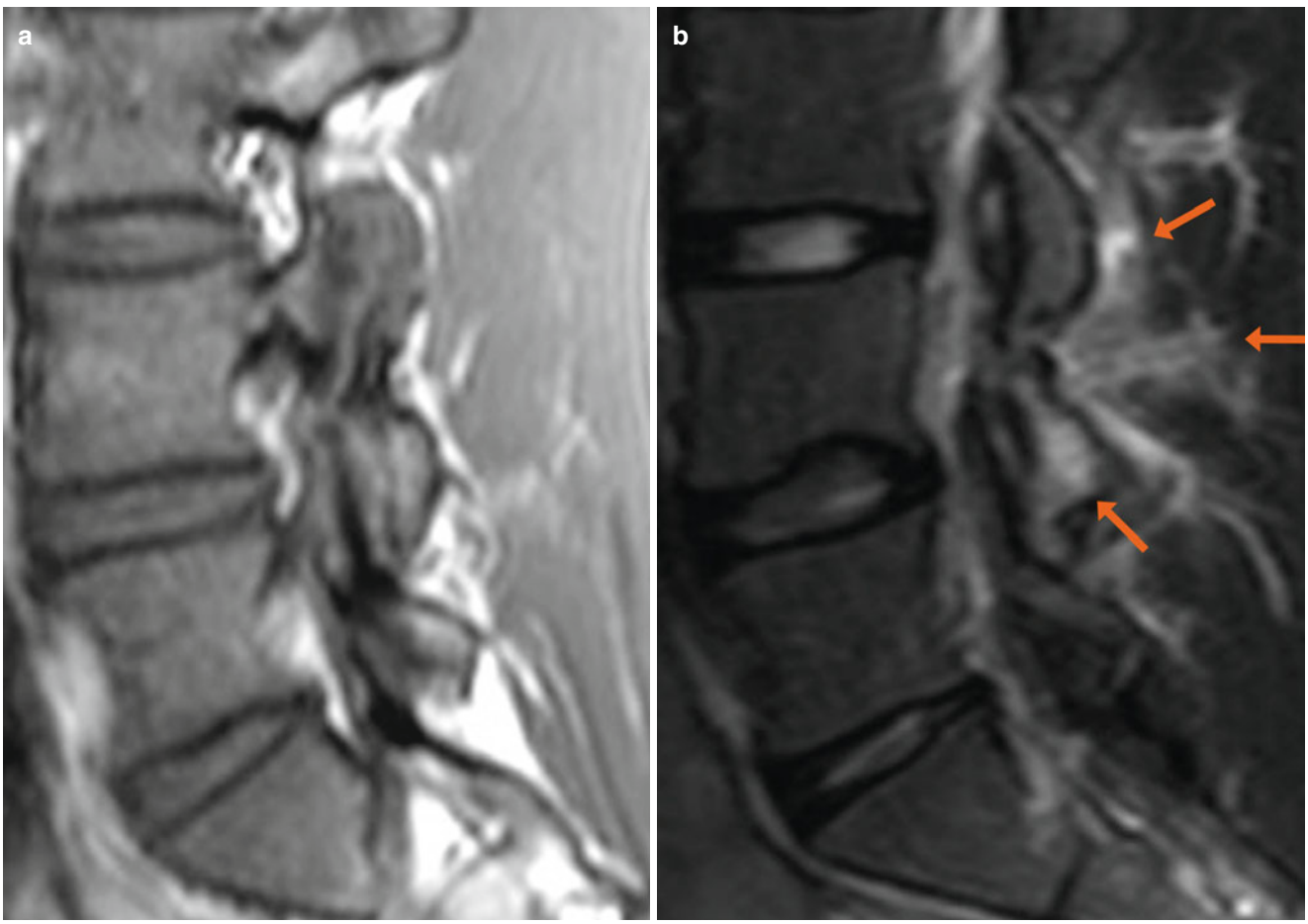


**Fig. 1** Sagittal SE T1-weighted image (**a**), sagittal and axial T2-weighted images with fat saturation (**b**–**d**), axial SE T1-weighted images with fat saturation after administration of contrast medium (**e**–**g**). T1-weighted image shows no abnormalities (**a**). T2-weighted image shows hyperintensity of the periapophyseal soft tissue, bilaterally, indicating oedema (**b**–**d**, *arrows*). The administration of contrast medium shows bilateral spondylolysis of L4, with enhancement of the pars fracture (**e**–**g**) and periapophyseal enhancement indicating inflammation of the soft tissue (**e**–**g**, *arrows*)

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DOI 10.1007/978-3-319-09447-2_34, © Springer International Publishing Switzerland 2015

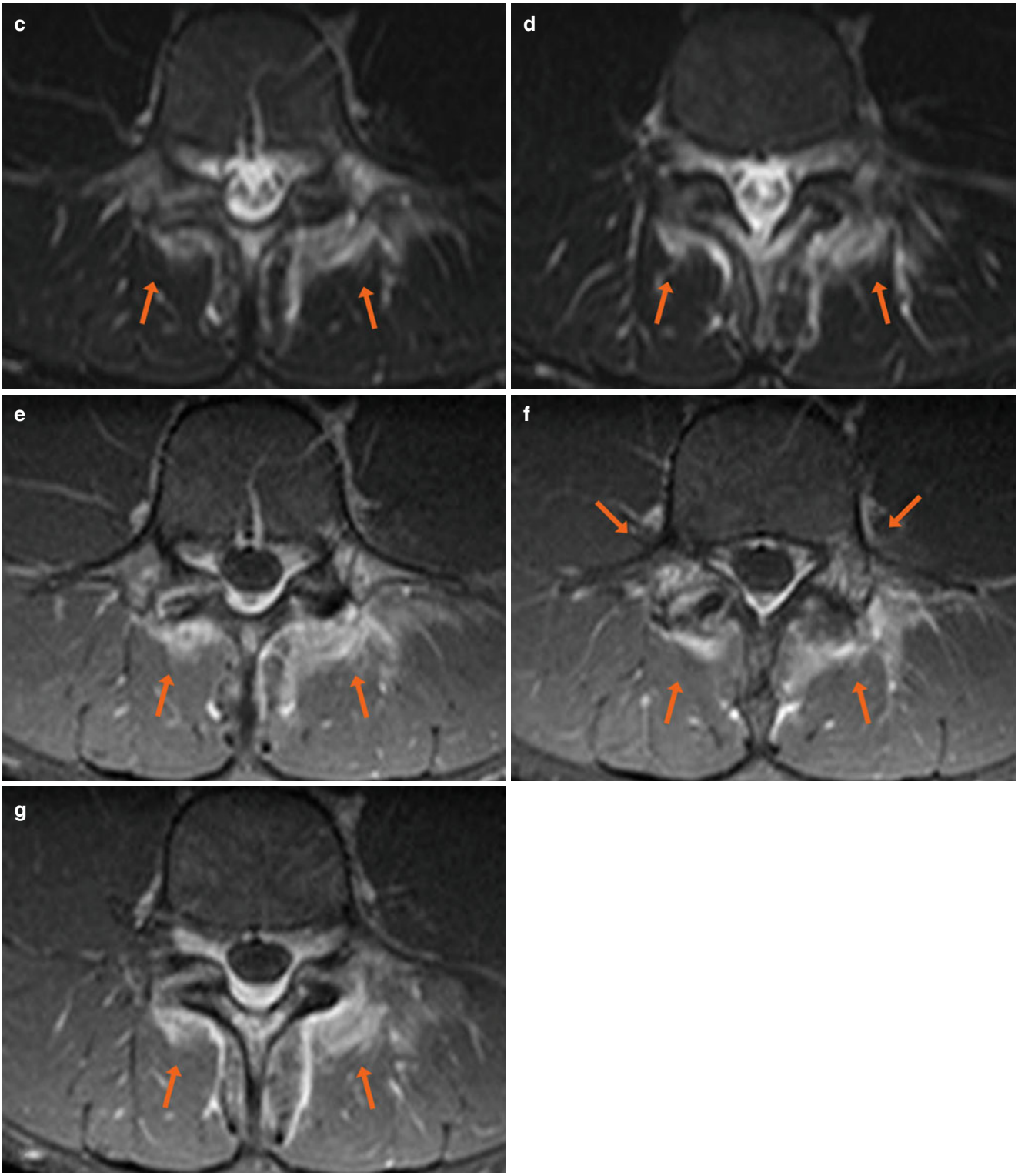


**Fig. 1** (continued)

# Spondylolysis

Case 33

- A 42-year-old male
- Persistent low back pain, more marked in flexion and extension

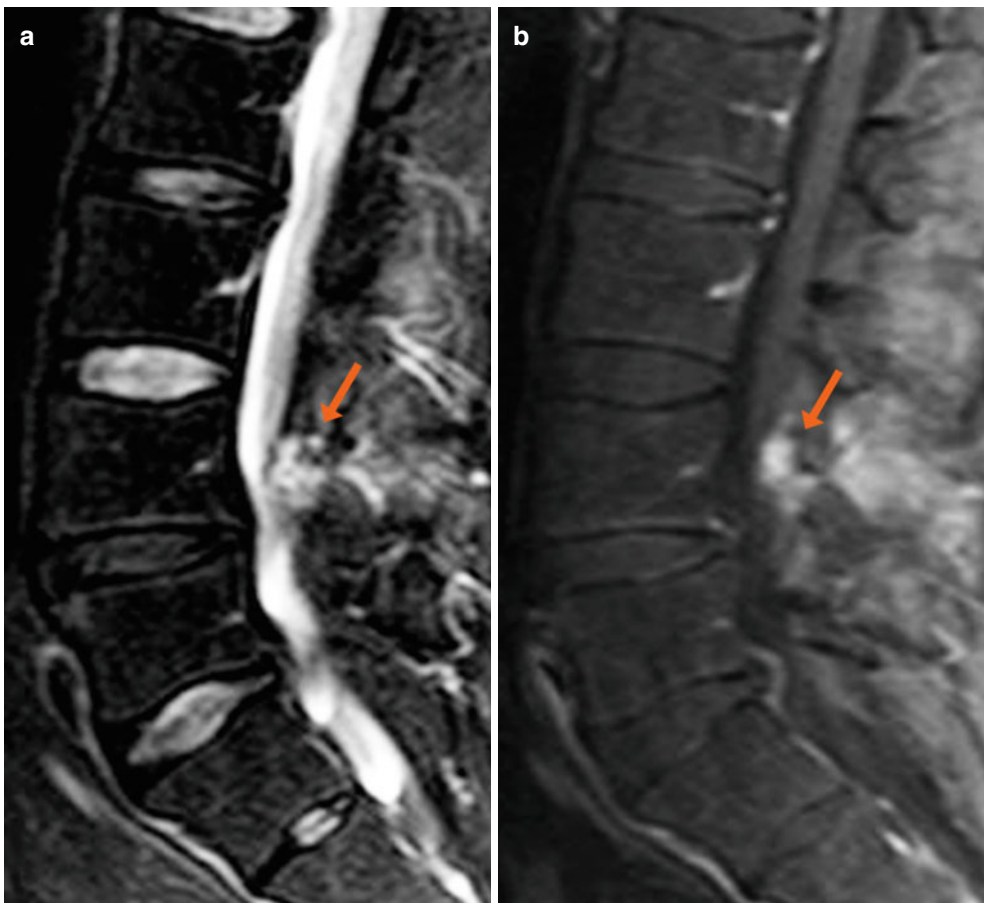


**Fig. 1** Sagittal and axial T2-weighted images with fat saturation (**a**, **c**), sagittal and axial T1-weighted images with fat saturation following contrast medium administration (**b**, **d**, **e**), axial CT scan (**f**). Hyperintense areas in T2 (**a**, **c**, *arrows*), with contrast enhancement (**b**, **d**, **e**, *arrows*), at the level of the pars interarticularis of L4, bilaterally. These findings are referable to spondylolysis with reactive inflammation of the adjacent soft tissues. Axial CT scan confirm bilateral spondylolysis of L4 (**f**, *arrows*)

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DOI 10.1007/978-3-319-09447-2_35, © Springer International Publishing Switzerland 2015

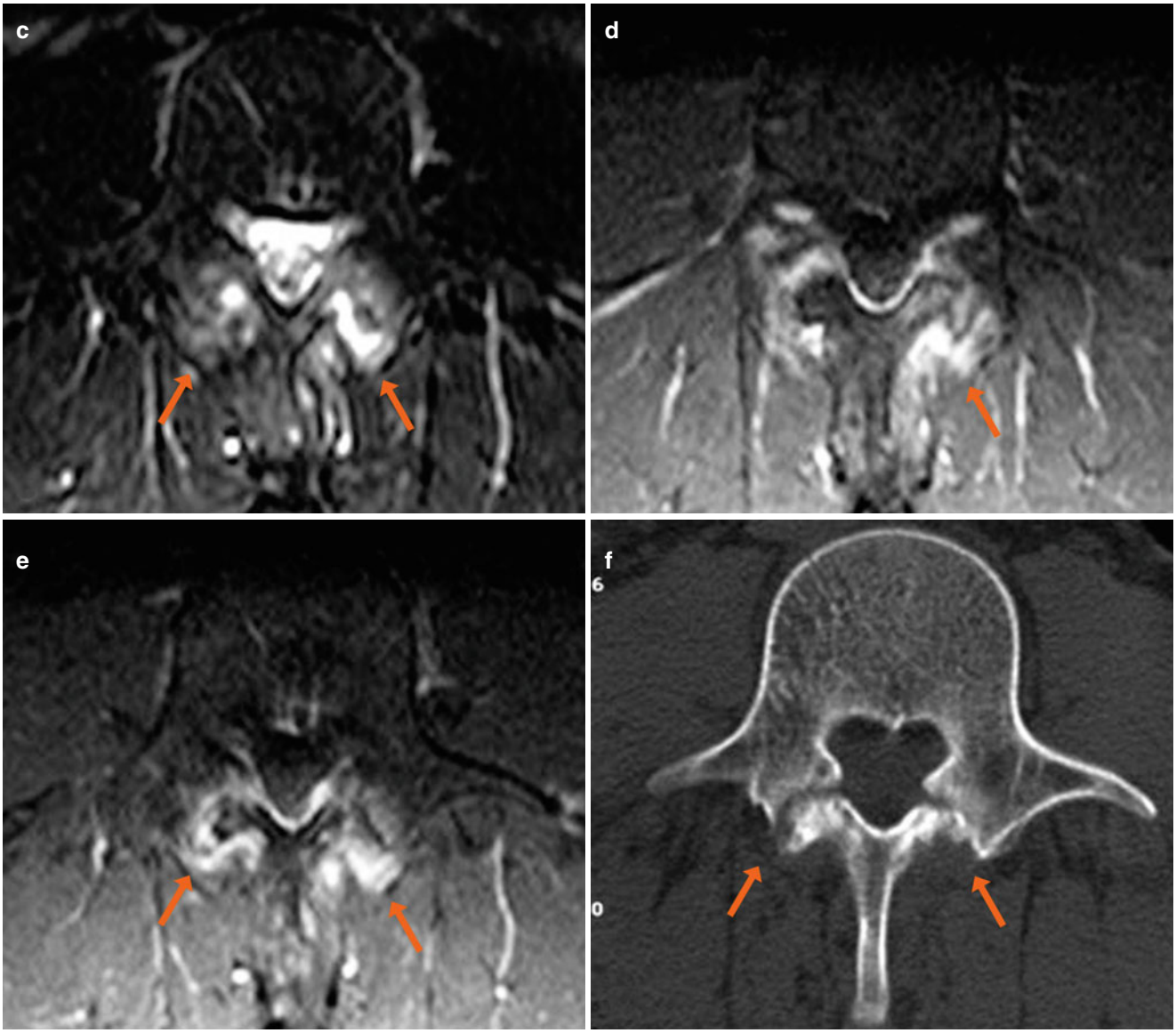


**Fig. 1** (continued)

# Spondylolysis

Case 34

- A 41-year-old male
- Persistent right low back pain

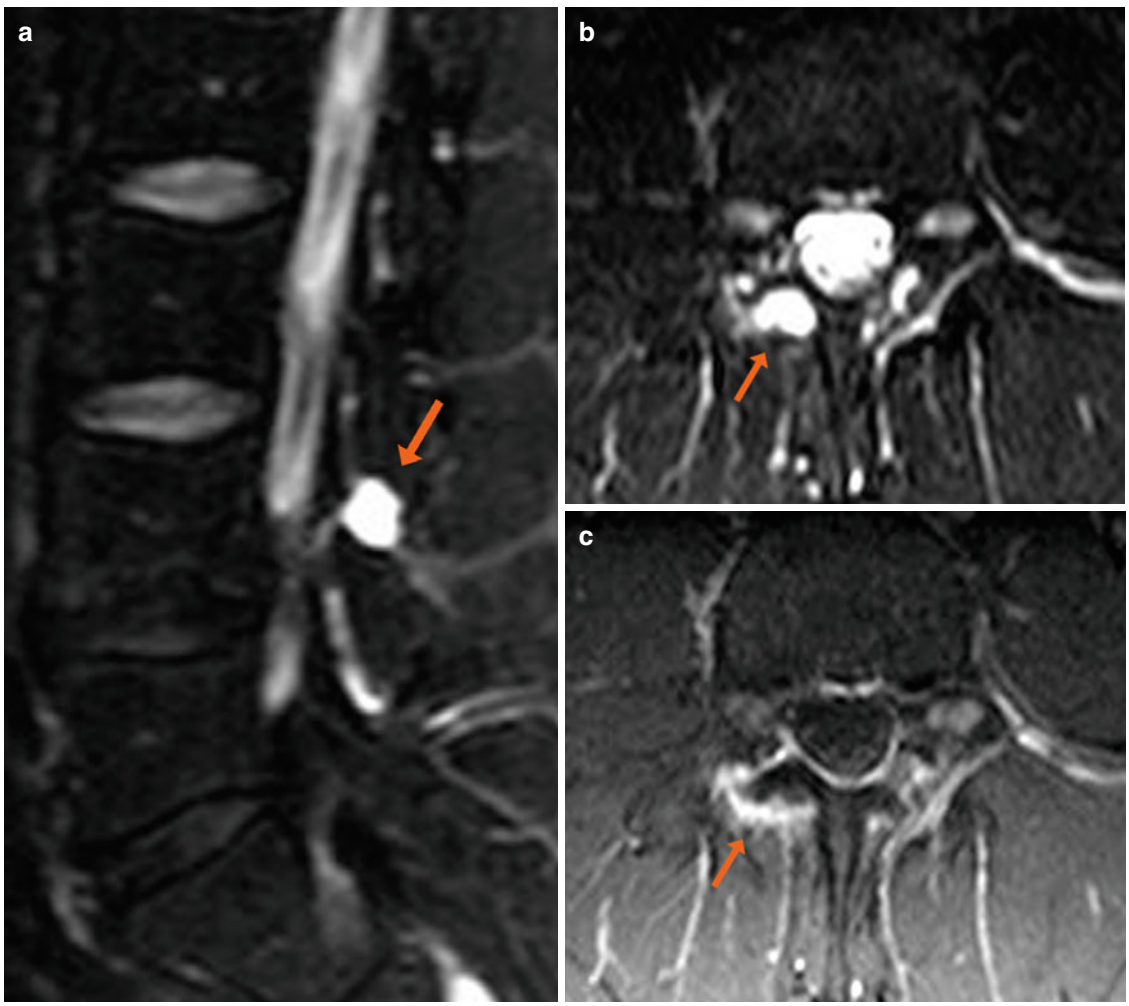


**Fig. 1** Sagittal and axial TSE T2-weighted images with fat saturation (**a**, **b**), axial contrast-enhanced SE T1-weighted image with fat saturation (**c**), axial CT scan (**d**). Right spondylolysis of L4, with neocyst formation (**a**, **b**, *arrow*). Contrast enhancement of the pars and adjacent soft tissue, due to reactive inflammation (**c**, *arrow*). CT scan confirms the fracture of the pars interarticularis (**d**, *arrow*)

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DOI 10.1007/978-3-319-09447-2_36, © Springer International Publishing Switzerland 2015

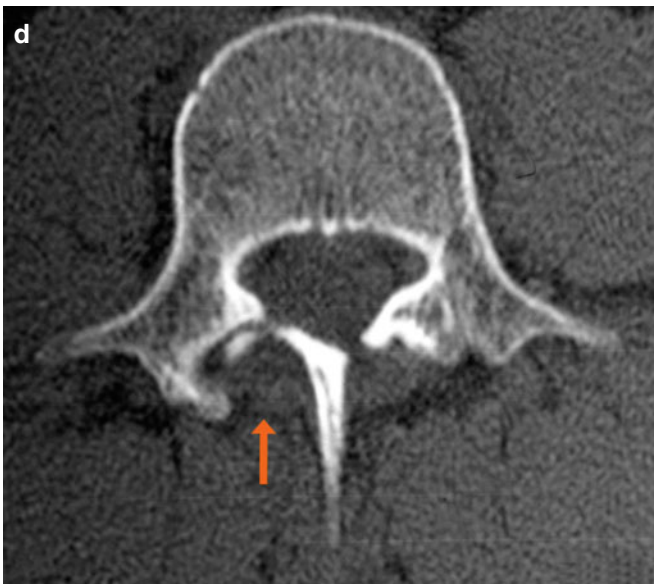


**Fig. 1** (continued)

# Spondylolysis

# Muscular Inflammation

Case 35

- An 18-year-old male
- Persistent right low back pain
- Tenderness at examination of posterior perispinal muscles at L3-L4-L5 on the right side

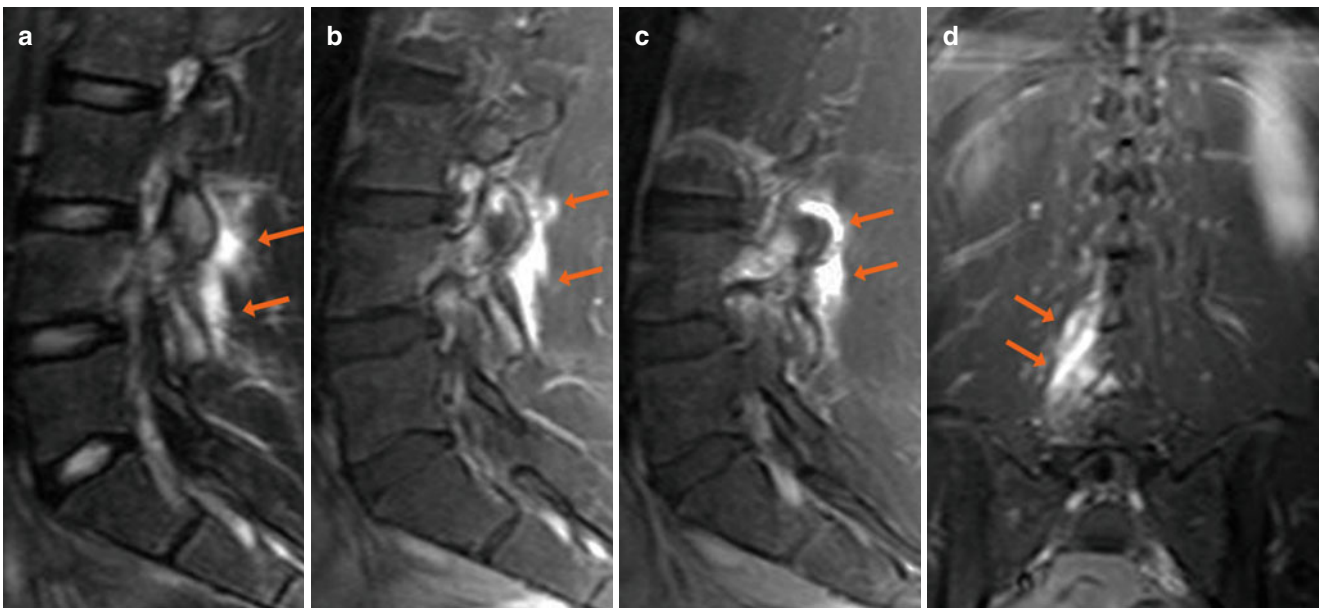


**Fig. 1** Sagittal and axial SE T2-weighted images with fat saturation (**a**, **e**–**h**), sagittal and axial SE T1-weighted images with fat saturation after the administration of contrast medium (**b**–**d**, **i**–**k**), axial CT scan (**l**). T2 hyperintensity and contrast enhancement of the right posterior arch of L4 and adjacent periapophyseal soft tissues, indicating oedema-inflammation (**a**–**c**, *arrow*). The images on the axial plane allow a better spatial resolution of the lesion (**e**–**k**, *arrows*). Edema of the perispinal muscles is well defined in coronal image (**d**, *arrows*). CT image shows right spondylolysis of L4, which is the cause of the above-mentioned oedematous lesions (**l**, *arrow*)

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DOI 10.1007/978-3-319-09447-2_37, © Springer International Publishing Switzerland 2015

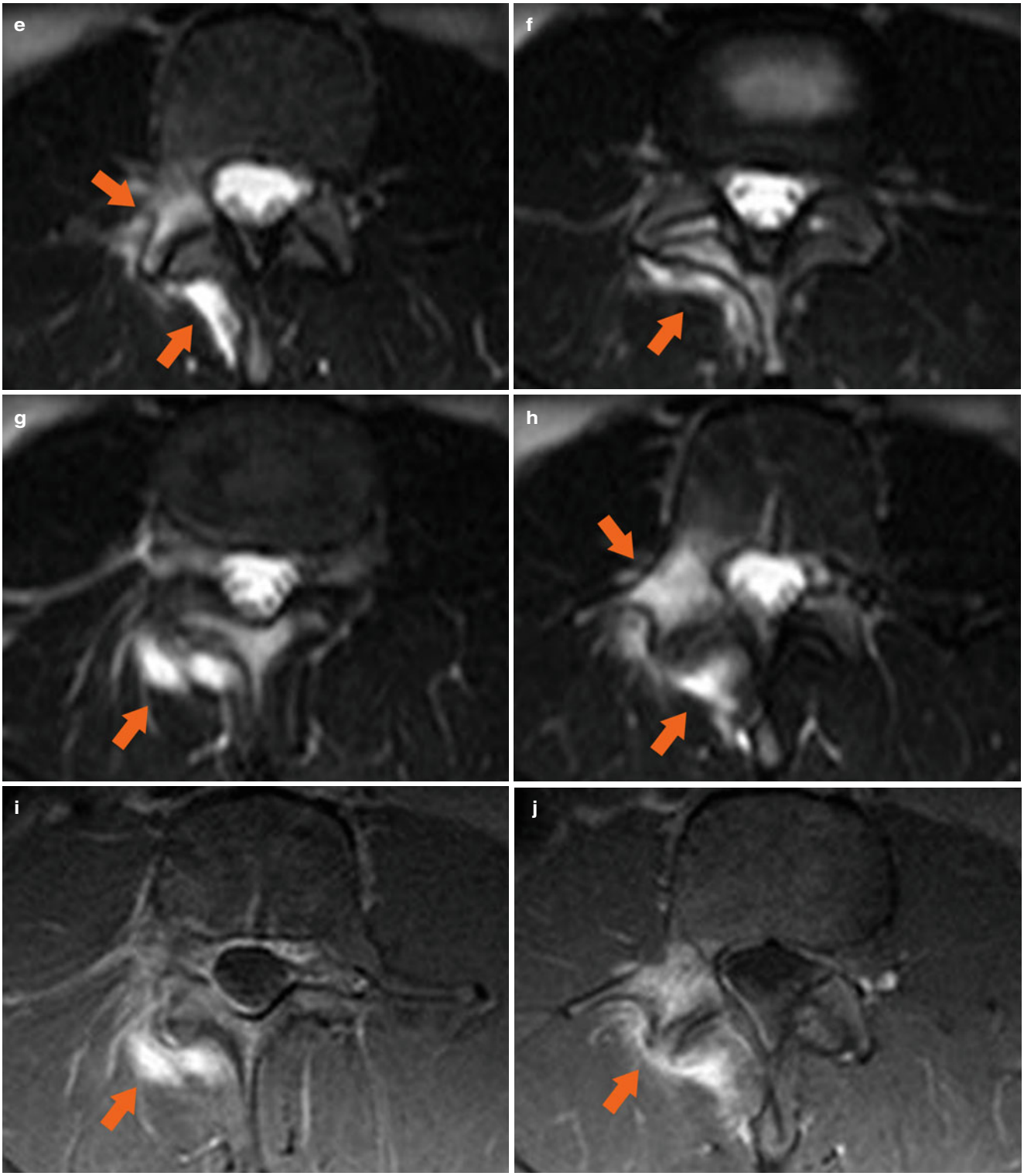


**Fig. 1** (continued)

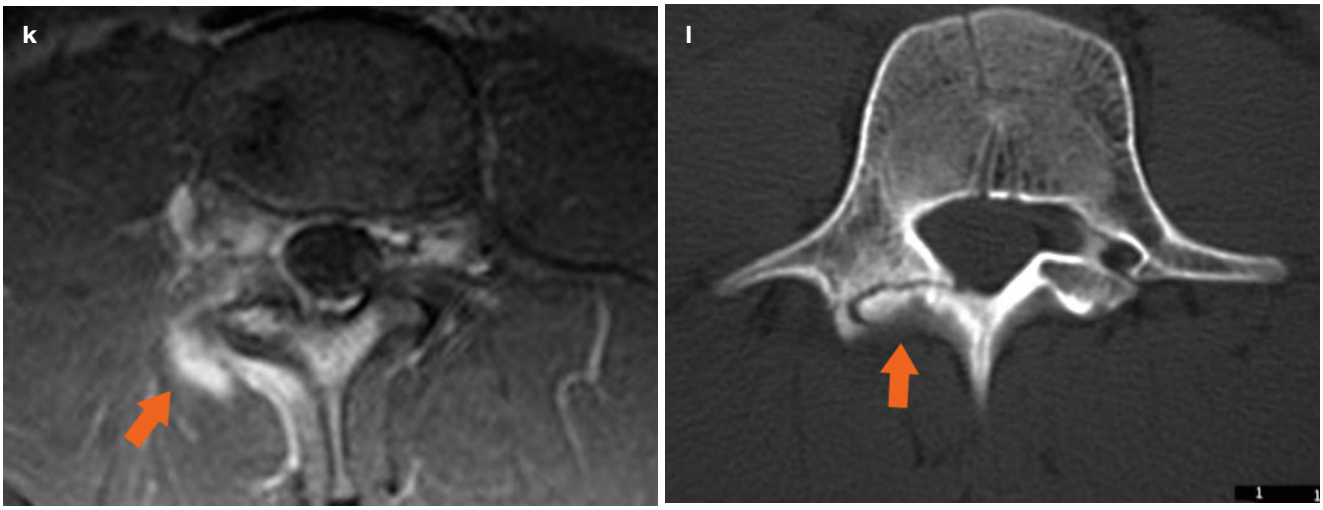


**Fig. 1** (continued)

# Spondylolysis

Case 36

- An 18-year-old male
- Patient with persistent low back pain after sports activity

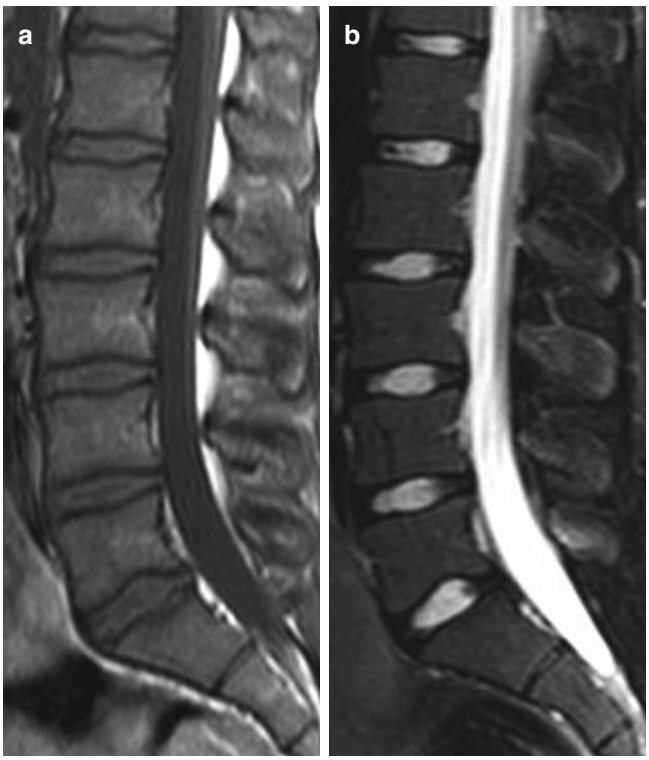


**Fig. 1** Sagittal T1-weighted image (**a**), sagittal and axial TSE T2-weighted images with fat saturation (**b**, **c**), axial SE T1-weighted images with fat saturation following administration of contrast medium (**d**, **e**), axial CT images (**f**, **g**). The images on the sagittal plane do not show significant changes. Oedematous pattern of the left pedicle at L5 and adjacent soft tissues (**c**, *arrow*). Fractures of the right pars interarticularis and left lamina; the lines of fractures are well defined after contrast medium administration (**d**, **e**, *thin arrows*). Contrast enhancement of the adjacent soft tissue, due to reactive inflammation (**d**, **e**, *large arrows*). CT examination confirmed the lines of fracture (**f**, **g**, *arrows*)

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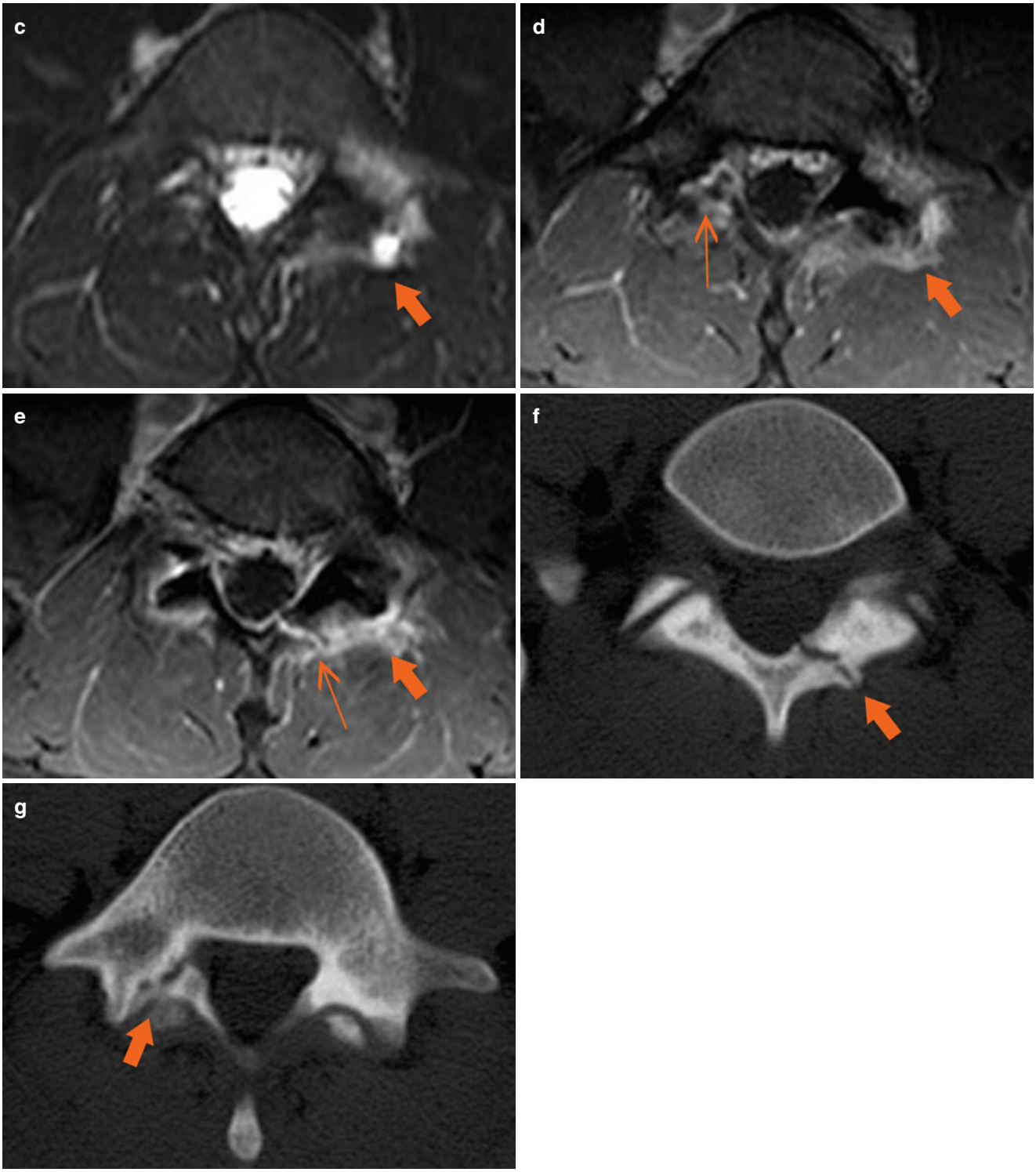


**Fig. 1** (continued)

# Spondylolysis

## Degenerative-Inflammatory Changes of Ligaments

Case **37**

- A 44-year-old male
- Patient with low back pain, more marked in flexion and extension, and focal tenderness on palpation of the interspinous space

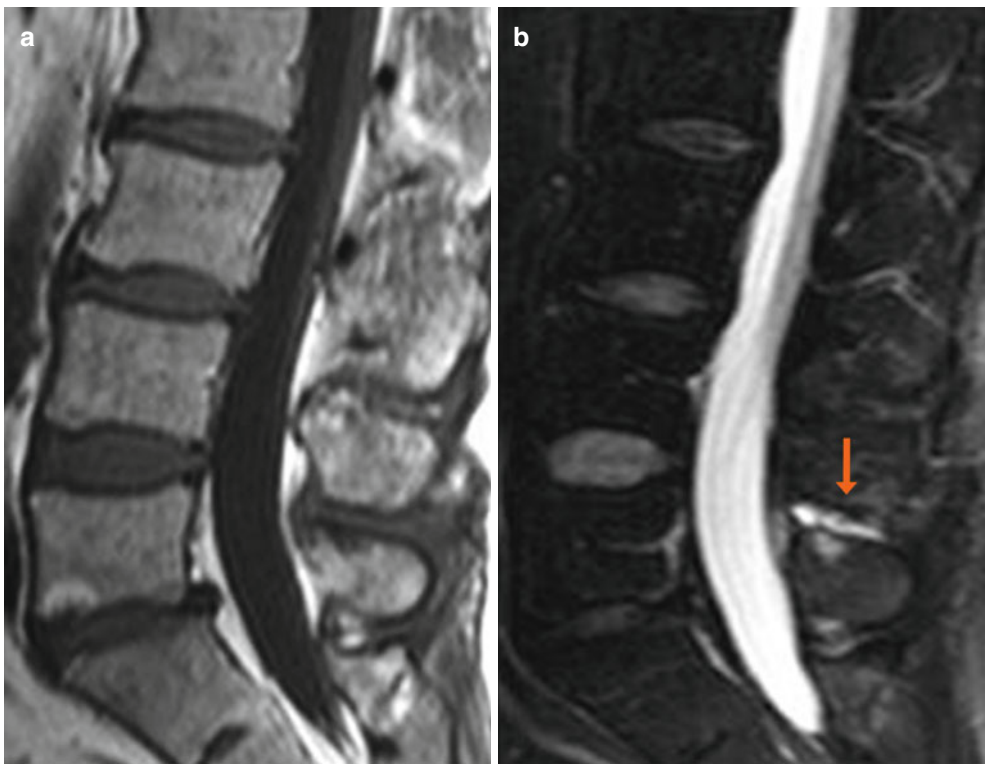


**Fig. 1** Sagittal T1-weighted image (**a**), sagittal and axial TSE T2-weighted images with fat saturation (**b**, **c**), sagittal and axial SE T1-weighted images with fat saturation following administration of contrast medium (**d**, **e**), axial CT image (**f**). Mild anterolisthesis of L5 over S1. The interspinous ligament at L4/L5 is hyperintense on T2-weighted imaging (**b**, **c**, *posterior arrow*) and enhances after contrast medium administration (**d**, **e**, *posterior arrow*); these findings indicate degenerative-inflammatory changes of the ligament. The images on the axial plane show bilateral spondylolysis of L5. The pars defect is well seen as hyperintense both in T2-weighted images with fat saturation and contrast enhanced T1-weighted images with fat saturation (**c**, **e**, *lateral arrows*). CT image confirms the lines of fractures of the pars interarticularis (**f**, *arrows*)

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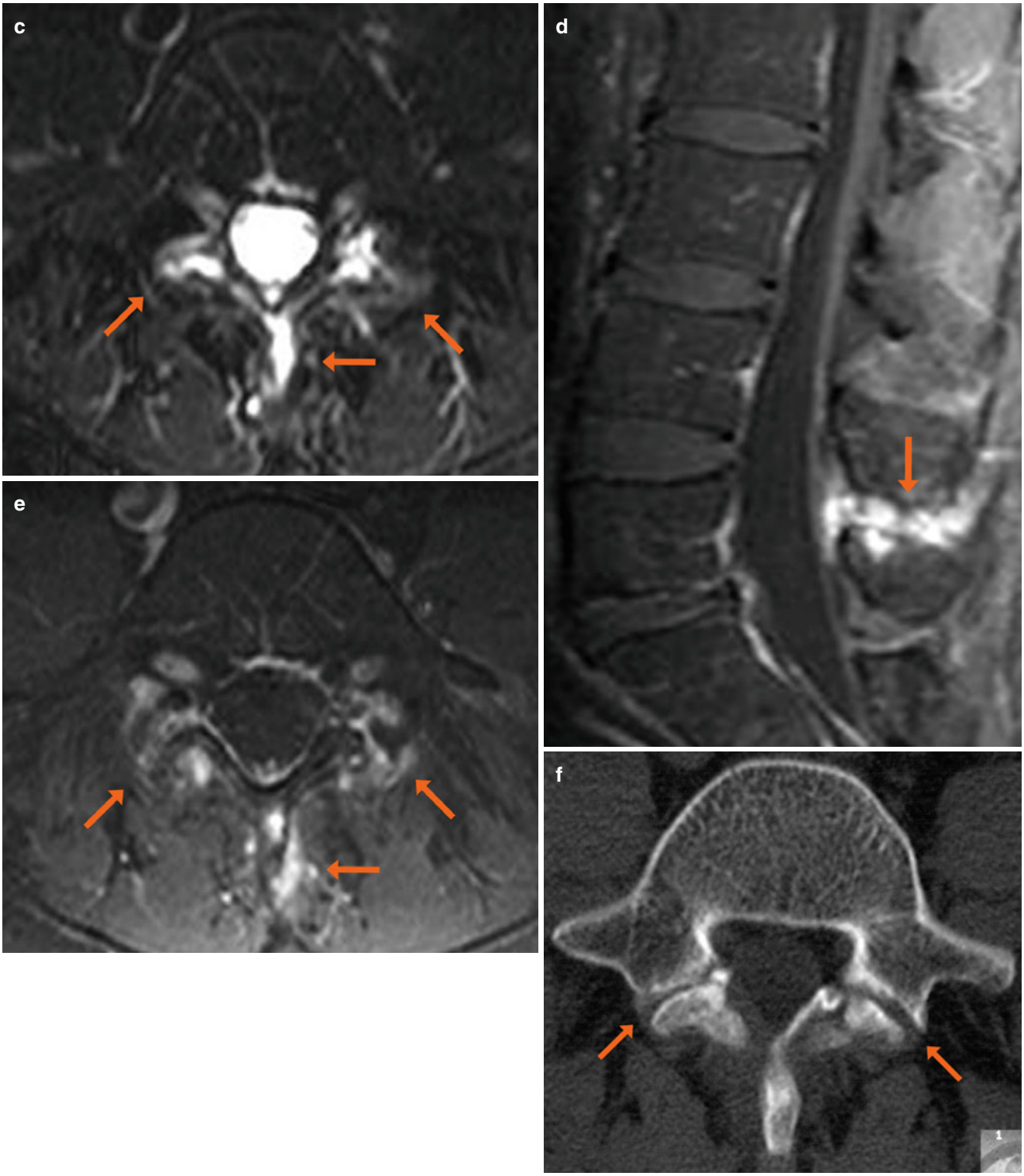


**Fig. 1** (continued)

# Spondylolysis

Case 38

- A 19-year-old male
- Patient with persistent low back pain arising after sports activity

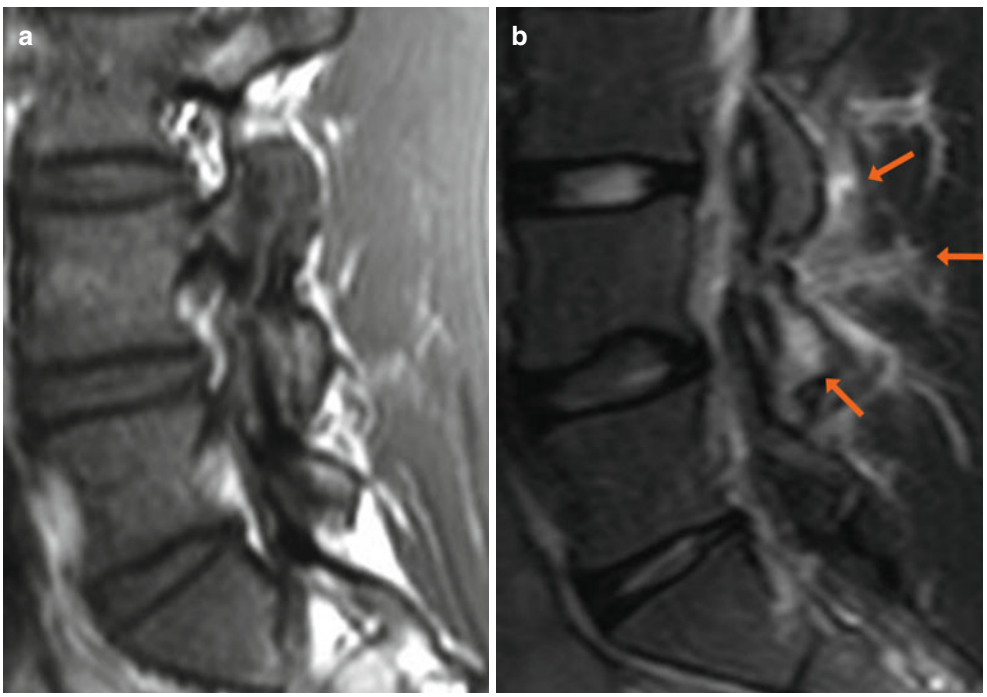


**Fig. 1** Sagittal T1-weighted image (**a**), sagittal and axial TSE T2-weighted images with fat saturation (**b**–**d**), axial SE T1-weighted images with fat saturation following administration of contrast medium (**e**–**g**). Oedema of the posterior vertebral arch of L4 and periapophyseal soft tissues (**b**–**d**, *arrows*). The administration of contrast medium reveals bilateral spondylolysis of L4 (**e**–**g**, *arrowhead*) and reactive inflammation of the adjacent soft tissues (**e**–**g**, *arrows*). Note also peridural contrast enhancement at the same level (**e**–**g**)

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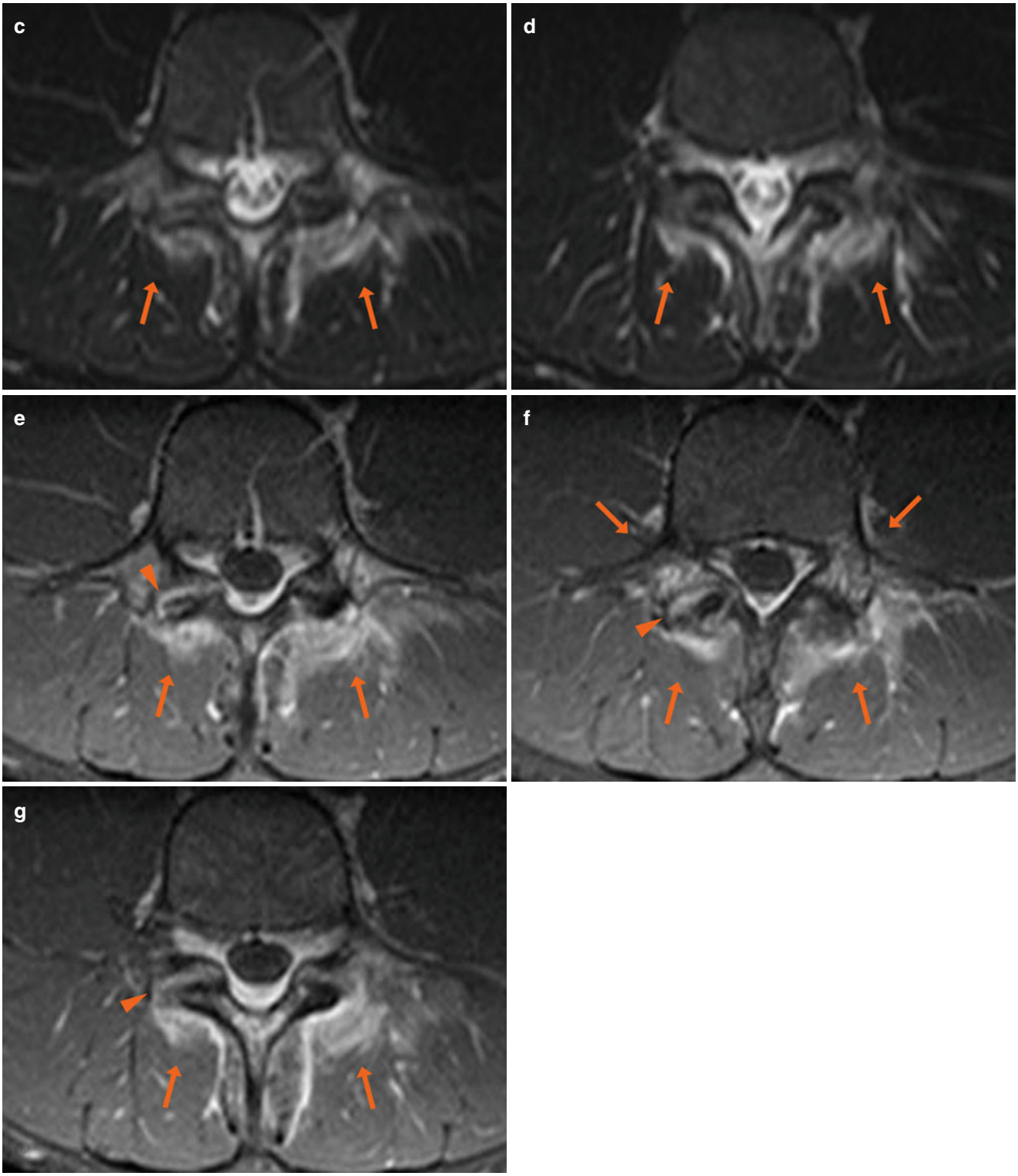


**Fig. 1** (continued)

# Degeneration-Inflammation of Ligaments

Case 39

- A 47-year-old patient
- Chronic low back pain, more marked in flexion and extension

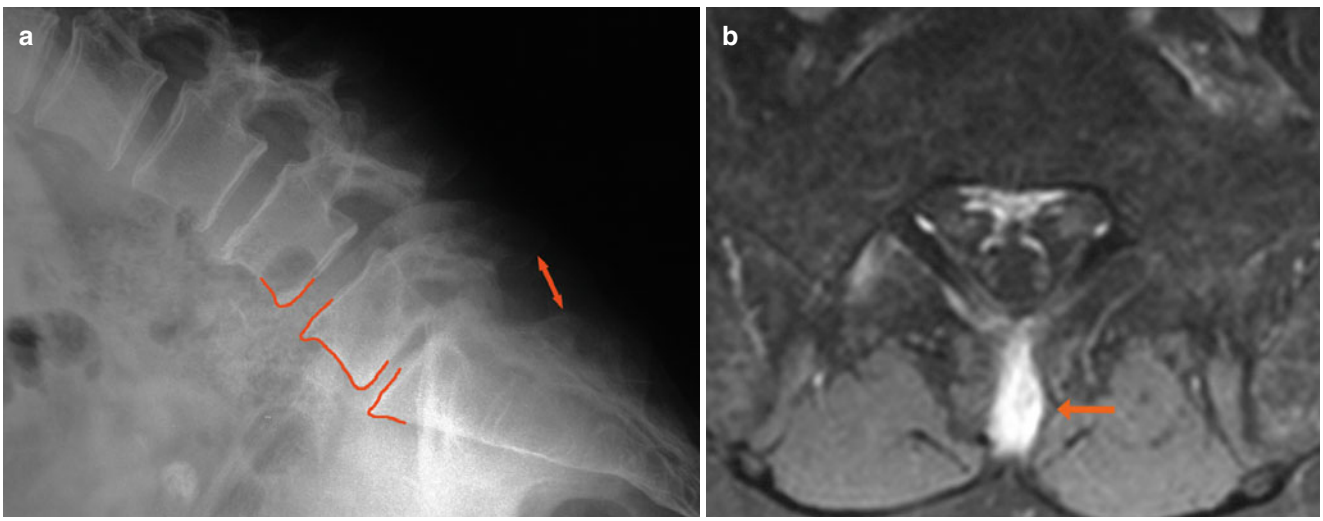


**Fig. 1** Lateral radiography in hyperflexion (**a**), sagittal and axial SE T1-weighted images with fat saturation following administration of contrast medium (**b**, **c**). The radiography shows mild retrolisthesis of L5 over S1 and widening of the corresponding interspinous space (**a**). MRI confirms the spinal instability and shows marked contrast enhancement of the interspinous ligament at L5/S1 (**b**, **c**, *arrows*), due to rupture-degeneration and inflammation of the same ligament. (**d**) Schematic figure (Modified from Jinkins)

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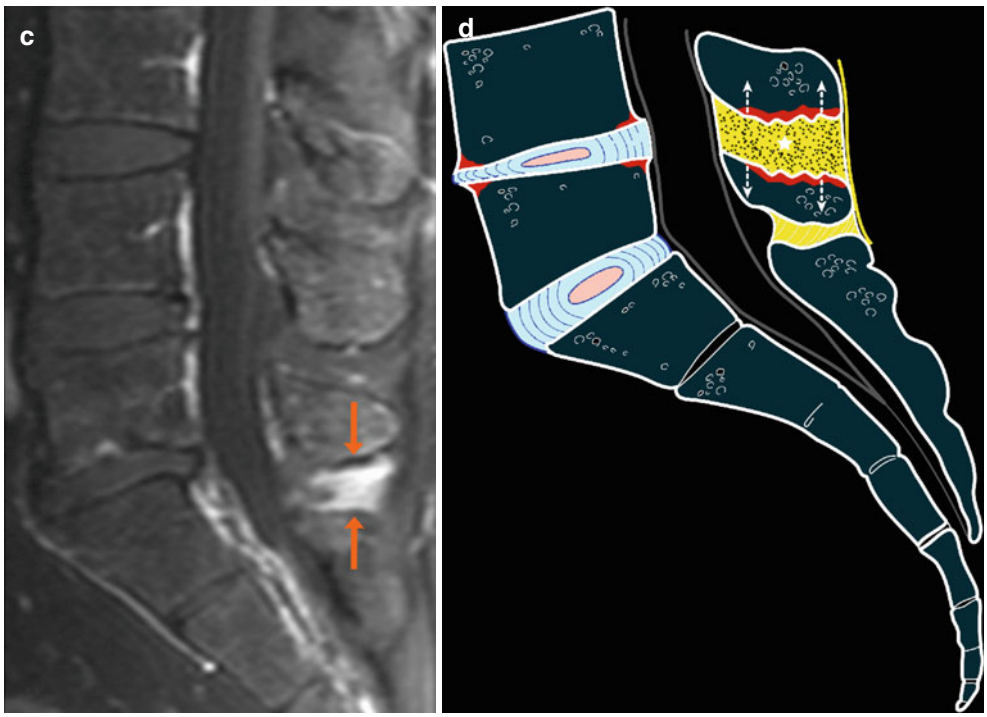
**Fig. 1** (continued)



# Degeneration-Inflammation of Ligaments

Case 40

- A 68-year-old woman
- Low back pain
- Tenderness on palpation of the lumbar interspinous spaces

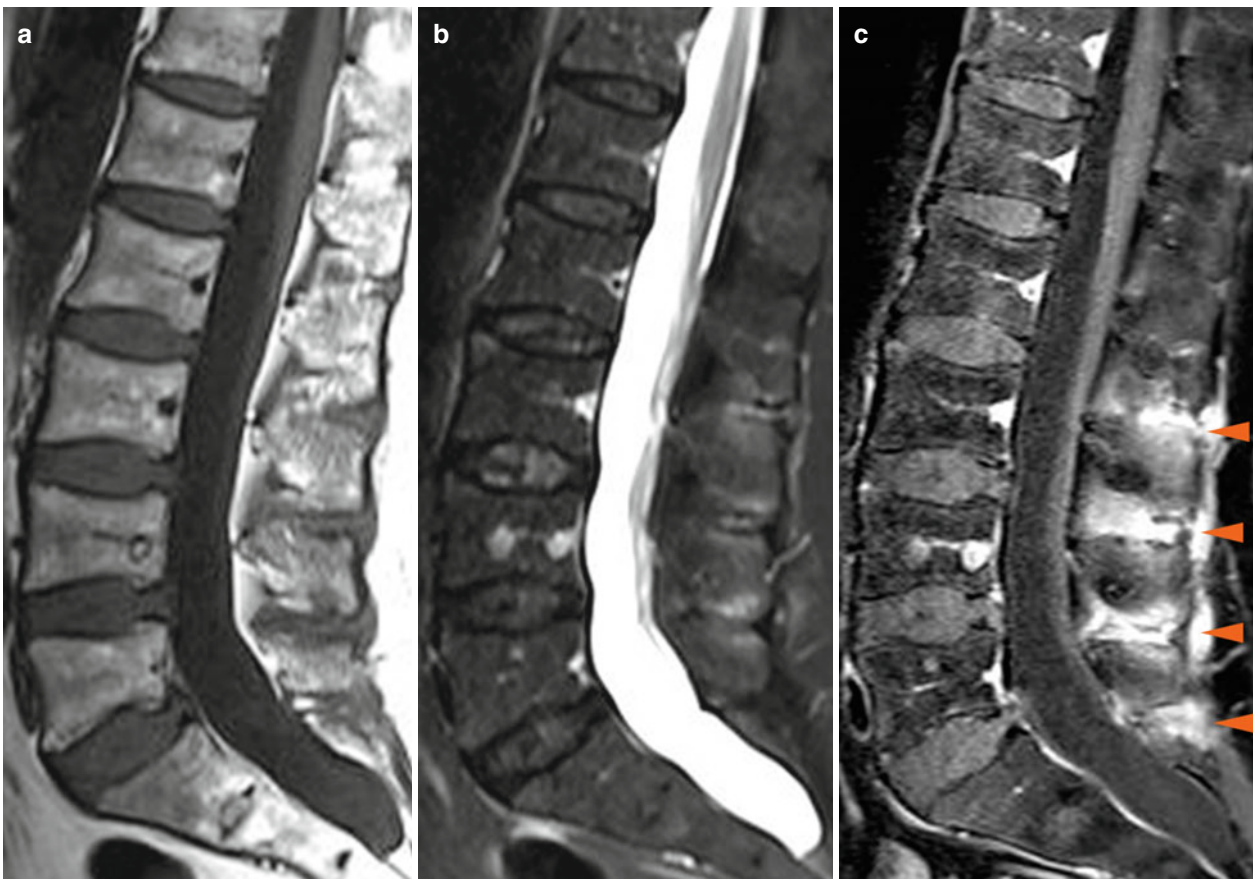



**Fig. 1** Sagittal SE T1-weighted image (**a**), sagittal TSE T2-weighted image with fat saturation (**b**), sagittal and axial T1-weighted images with fat saturation following administration of contrast medium (**c**–**f**). Mild hyperintensity in T2 of the interspinous ligaments from L2 to S1 (**b**). Contrast medium administration reveals marked enhancement of the interspinous and supraspinous ligaments from L2 to S1 (**c**–**e**, *arrowheads*). It is also evident that there is contrast enhancement of the ligamenta flava at L4/L5 and L5/S1 (**d**, **f**, *arrows*). These findings indicate degenerative-inflammatory ligamentous changes. Note that such ligamentous changes are clearly detectable after the administration of contrast medium coupled with fat-suppressed T1-weighted imaging

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DOI 10.1007/978-3-319-09447-2_42, © Springer International Publishing Switzerland 2015

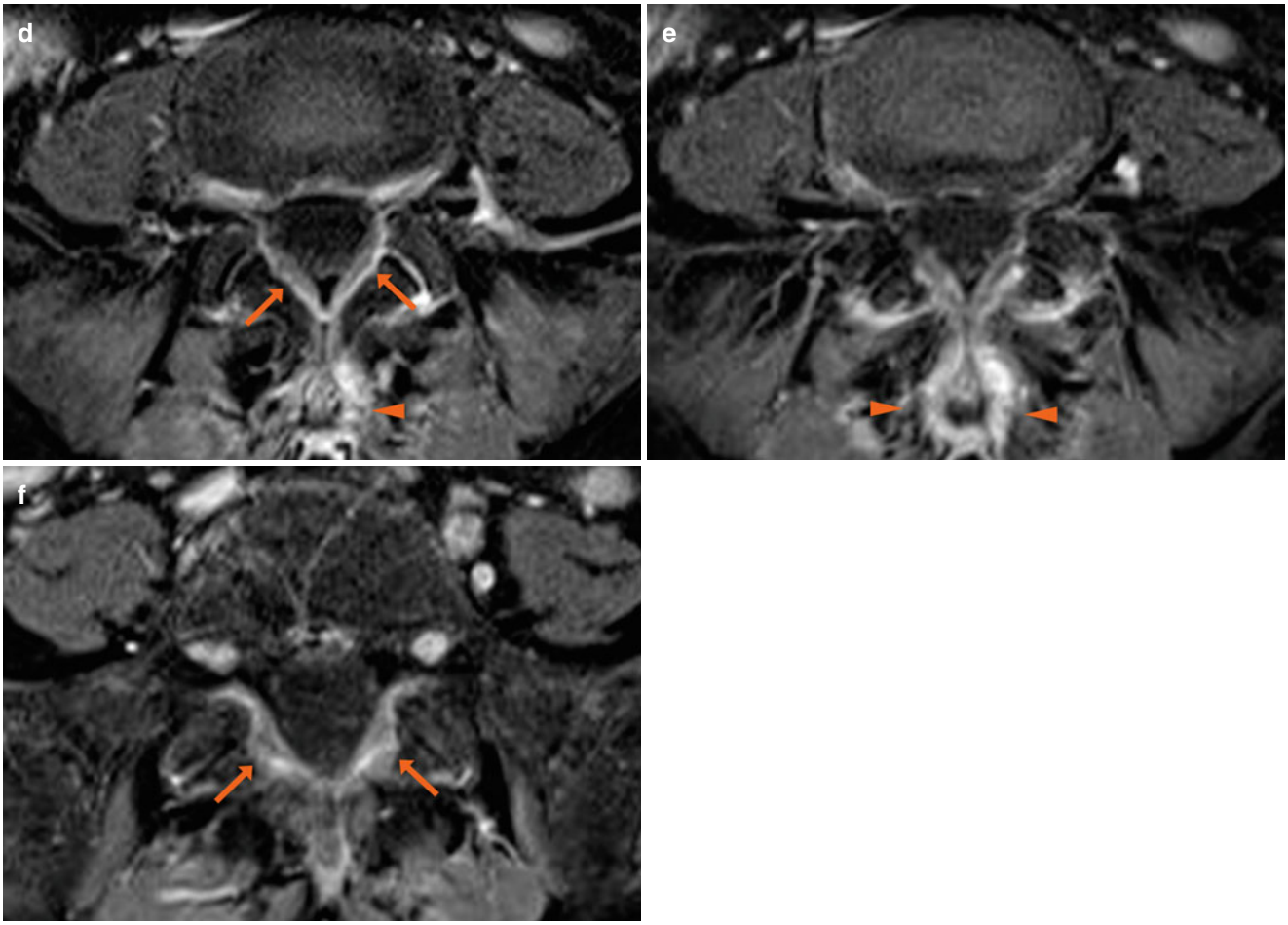


**Fig. 1** (continued)

# Degeneration-Inflammation of Ligaments

Case 41

- A 74-year-old male
- Patient with persistent low back pain
- Limited lumbar motion
- Exacerbation of pain by extension

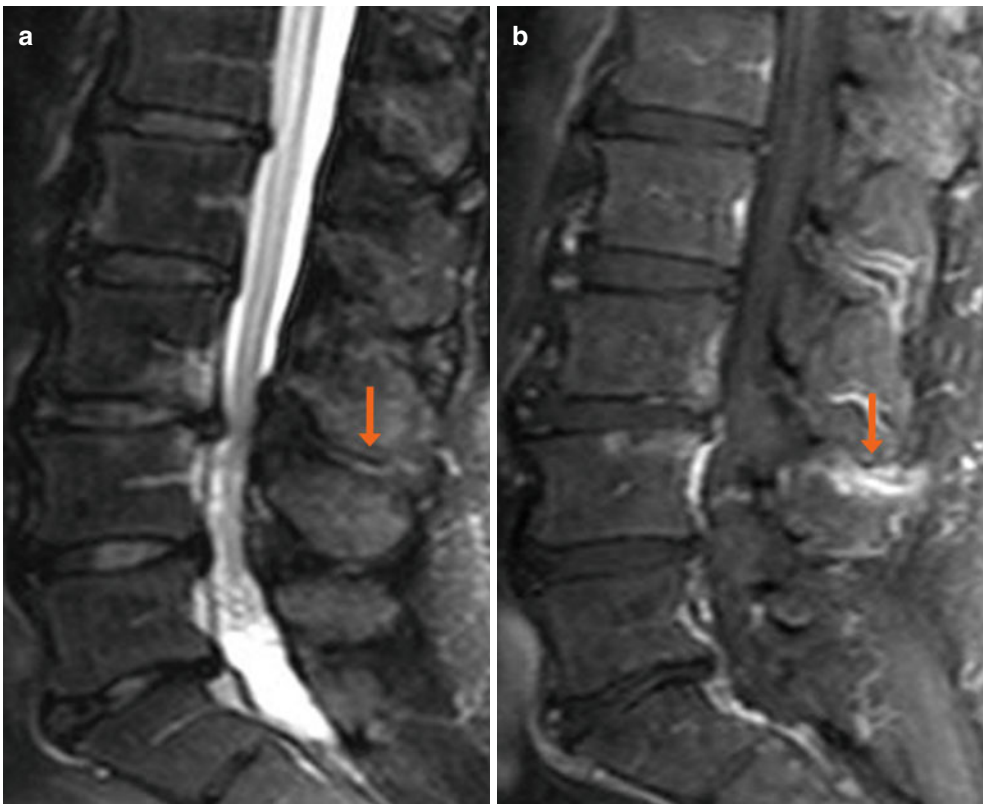


**Fig. 1** Sagittal TSE T2-weighted image with fat saturation (**a**), sagittal and axial T1-weighted images with fat saturation following the administration of contrast medium (**b**, **c**). Mild retrolisthesis of L3 over L4, with posterior osteochondritis and bulging disc at the same level L3/L4 (**a**, **b**). Reduction in height of the L3/L4 interspinous space (**a**, *arrow*). Marked contrast enhancement of the L3/L4 interspinous ligament (**b**, **c**, *arrow*), indicating degenerative-inflammatory ligamentous changes; these changes are presumably due to collision of the spinous processes and injury of the interposed ligament

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DOI 10.1007/978-3-319-09447-2_43, © Springer International Publishing Switzerland 2015

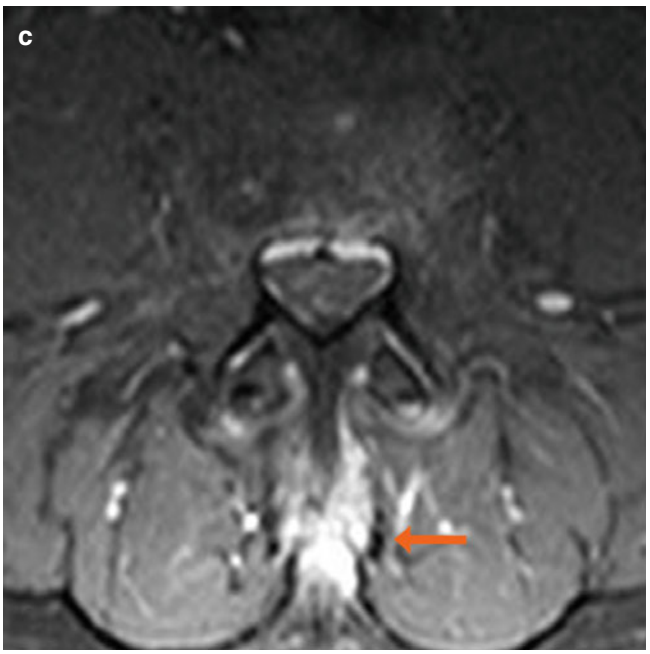


**Fig. 1** (continued)

# Degeneration-Inflammation of Ligaments

Case 42

- A 73-year-old male
- Patient with chronic low back pain
- Limited lumbar motion
- Exacerbation of pain by extension/flexion

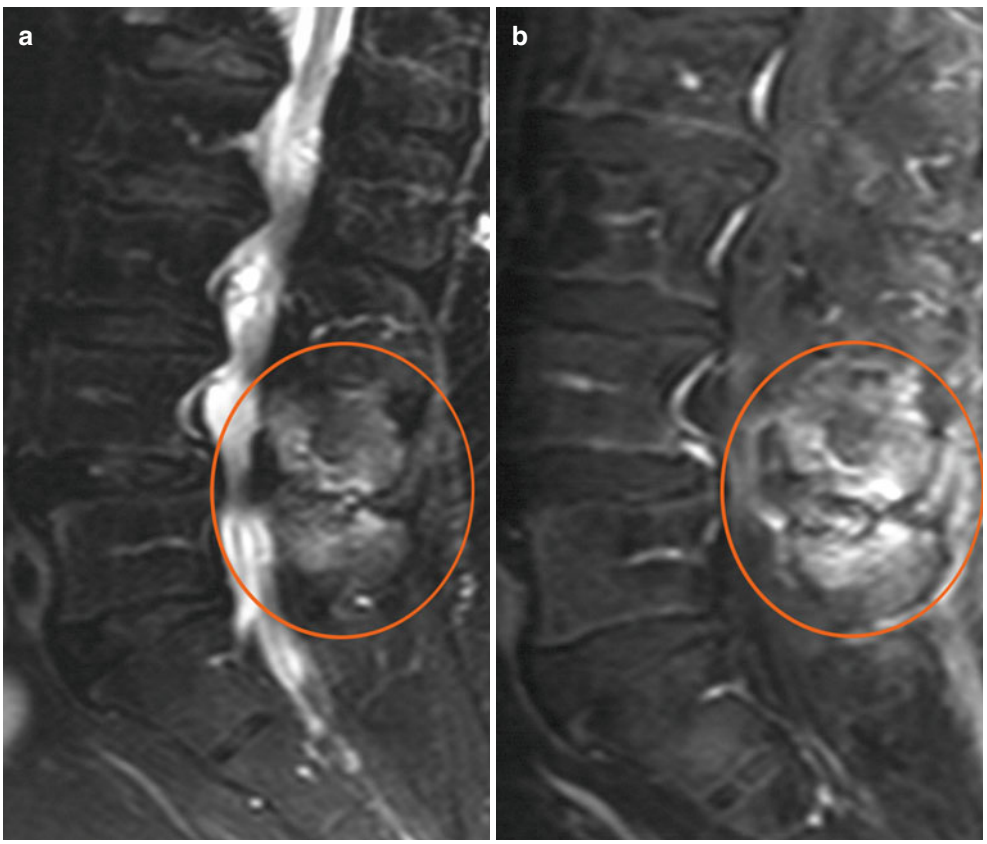


**Fig. 1** Sagittal TSE T2-weighted image with fat saturation (**a**), sagittal and axial T1-weighted images with fat saturation following administration of contrast medium (**b**, **c**). Mild anterolisthesis of L4 over L5. High signal in T2 and contrast enhancement of the opposing L4/L5 spinous processes, due to bony collision and consequent stress-related reactive-degenerative changes of the bone marrow (**a**, **b**, *circle*). The L4/L5 interspinous space is collapsed, and the corresponding interspinous ligament is degenerated. The axial image on L4/L5 shows also contrast enhancement of the perispinal muscles (e.g. interspinalis muscle) indicating degenerative-inflammatory muscular changes and contrast enhancement of the facet joints, indicating bilateral osteoarthritis (**c**, *circle*)

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DOI 10.1007/978-3-319-09447-2_44, © Springer International Publishing Switzerland 2015

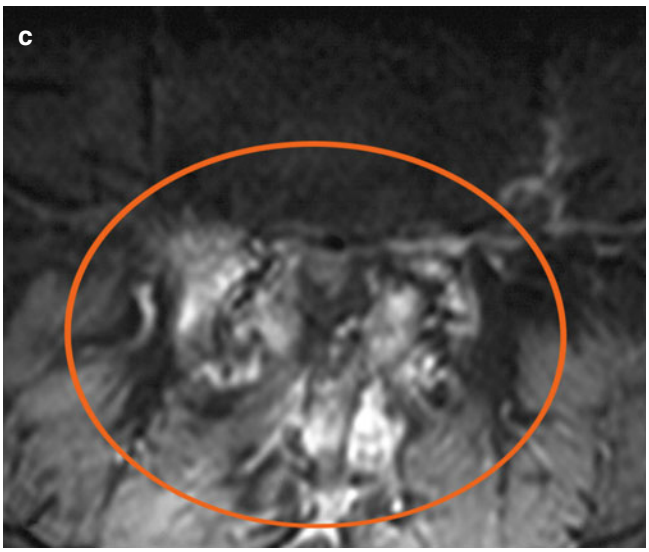


**Fig. 1** (continued)

# Degeneration-Inflammation of Ligaments

Case 43

## Baastrup's Phenomenon

- A 52-year-old patient
- Persistent low back pain
- Limited lumbar motion
- Exacerbation of pain by hyperextension
- Tenderness on palpation of the interspinous spaces

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DOI 10.1007/978-3-319-09447-2_45, © Springer International Publishing Switzerland 2015

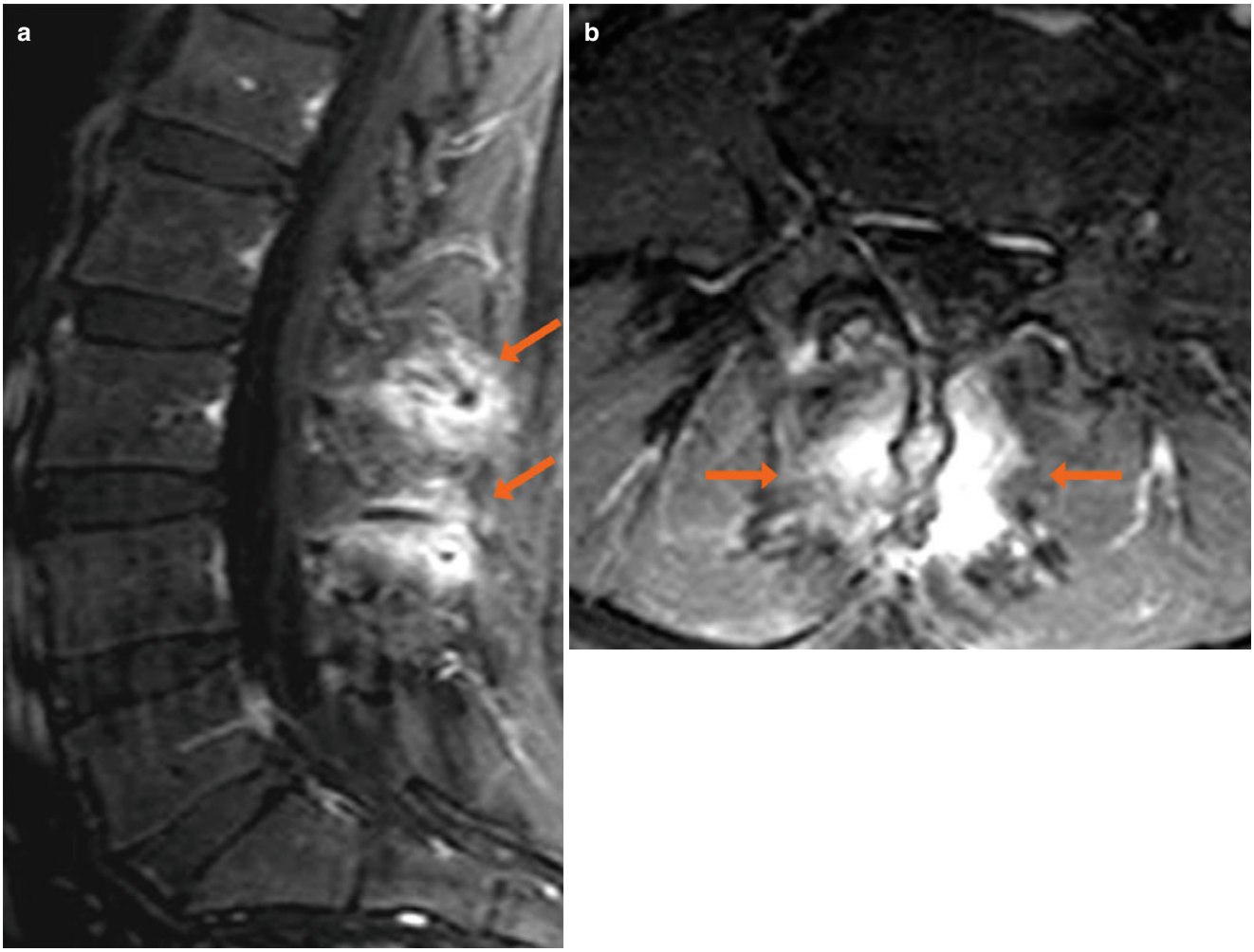


**Fig. 1** Sagittal and axial SE T1-weighted images with fat saturation following the administration of contrast medium (**a**, **b**). Lumbar hyperlordosis is present, associated with collision of the spinous processes at L2-L3-L4 (i.e. Baastrup's phenomenon) (**a**). Contrast enhancement of the same opposing spinous processes, corresponding interspinous ligaments and interspinalis muscles (**a**, **b**, *arrows*). Bone marrow changes of the spinous processes represent stress-related bony reactive-degenerative phenomena. Similarly, ligamentous and muscular changes represent stress-related degenerative-inflammatory phenomena

# Degeneration-Inflammation of Ligaments

# Case 44

## Baastrup's Phenomenon

- A 50-year-old female
- Patient with persistent low back pain
- Limited lumbar motion

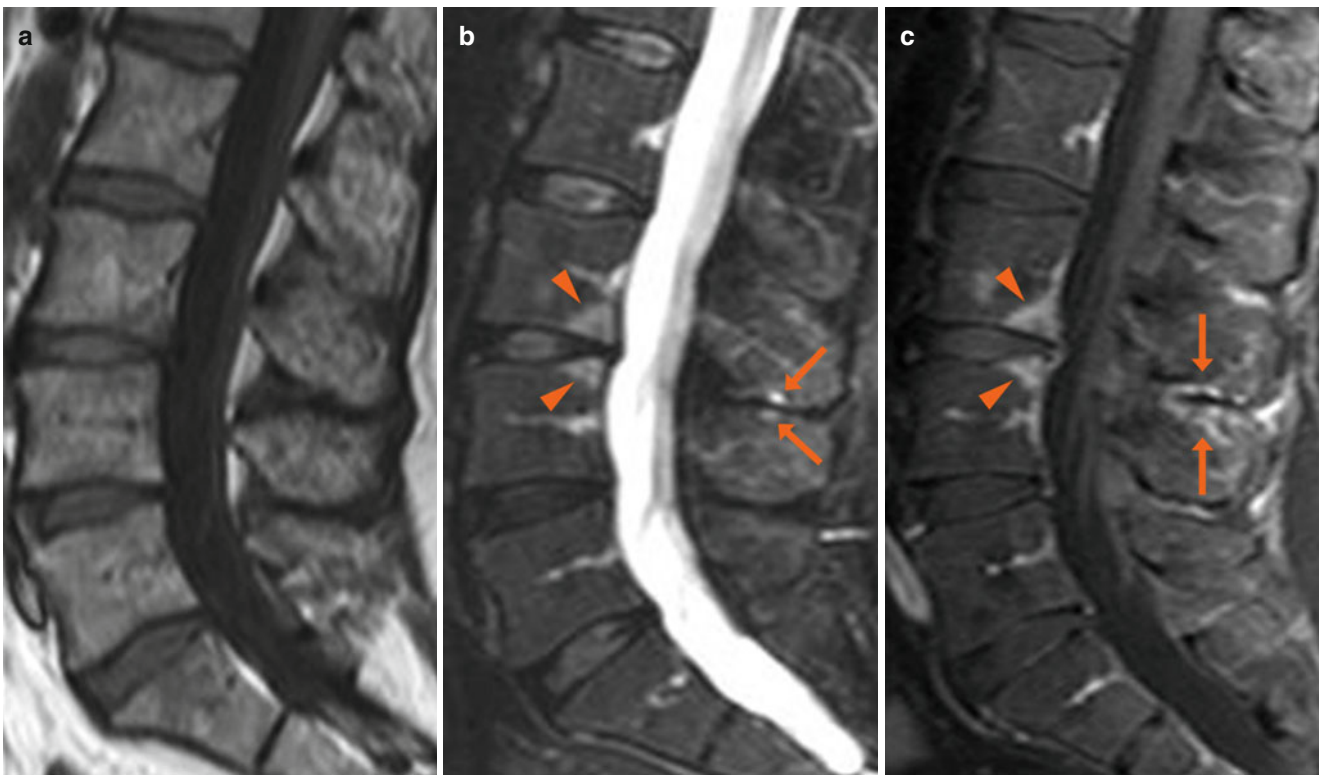


**Fig. 1** Sagittal SE T1-weighted image (**a**), sagittal TSE T2-weighted image with fat saturation (**b**), sagittal and axial SE T1-weighted images with fat saturation after contrast medium administration (**c**, **d**). Lumbosacral hyperlordosis with collision of the spinous processes at L3/L4. Note small hyperintense areas in T2 (**b**, *arrows*) and contrast enhancement (**c**, *arrows*) in the same opposing spinous processes at L3/L4, due to stress-related bony reactive-degenerative phenomena. Contrast enhancement of the interspinalis muscles at the same level (**d**, *arrows*), due to degenerative-inflammatory phenomena. This patient presented also osteochondrosic changes at L3/L4, in active-inflammatory phase (**b**, **c**, *arrowheads*)

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DOI 10.1007/978-3-319-09447-2_46, © Springer International Publishing Switzerland 2015

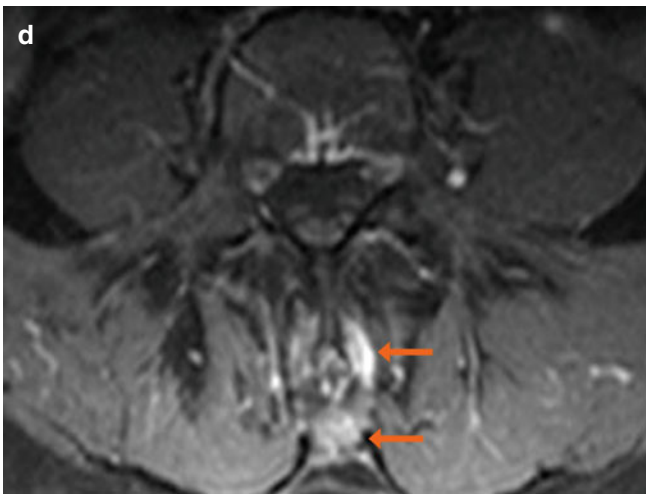


**Fig. 1** (continued)

# Osteochondritis
# Osteoarthritis
# Muscular Inflammation

Case **45**

- A 58-year-old man
- Persistent low back pain
- Focal tenderness on the right side

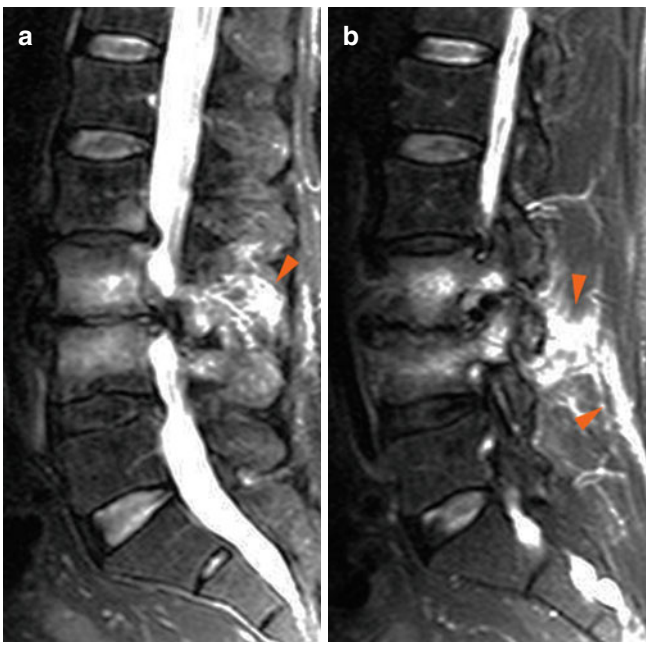


**Fig. 1** Sagittal and axial TSE T2-weighted images with fat saturation (**a**–**e**), sagittal, coronal and axial SE T1-weighted images with fat saturation after the administration of contrast medium (**f**–**m**). Osteochondritis at L3/L4, in active-inflammatory phase, with peridiscal inflammation involving the adjacent psoas muscle (**a**–**m**, *thin arrow*). Right facet joint osteoarthritis al the same level L3/L4 (**c**–**e**, **j**–**m**). Degeneration-inflammation on the right multifidus muscle that shows hyperintensity in T2 and contrast enhancement (**a**–**m**, *arrowheads*). Left-convex scoliosis (**h**). All the above-mentioned changes, i.e. osteochondritis, osteoarthritis and muscular changes, are presumably due to scoliosis with consequent biomechanical deterioration of the spine and perispinal muscles

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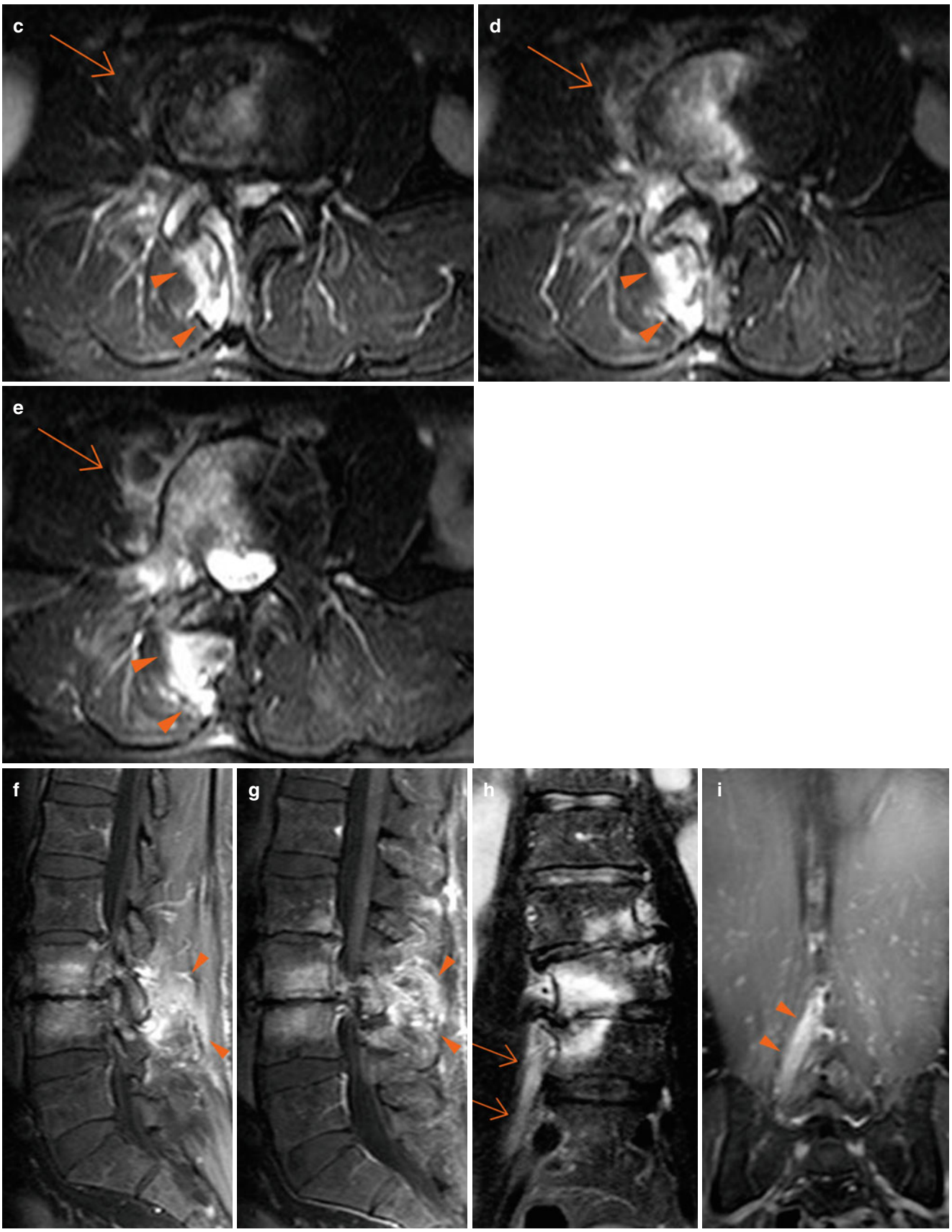


**Fig. 1** (continued)

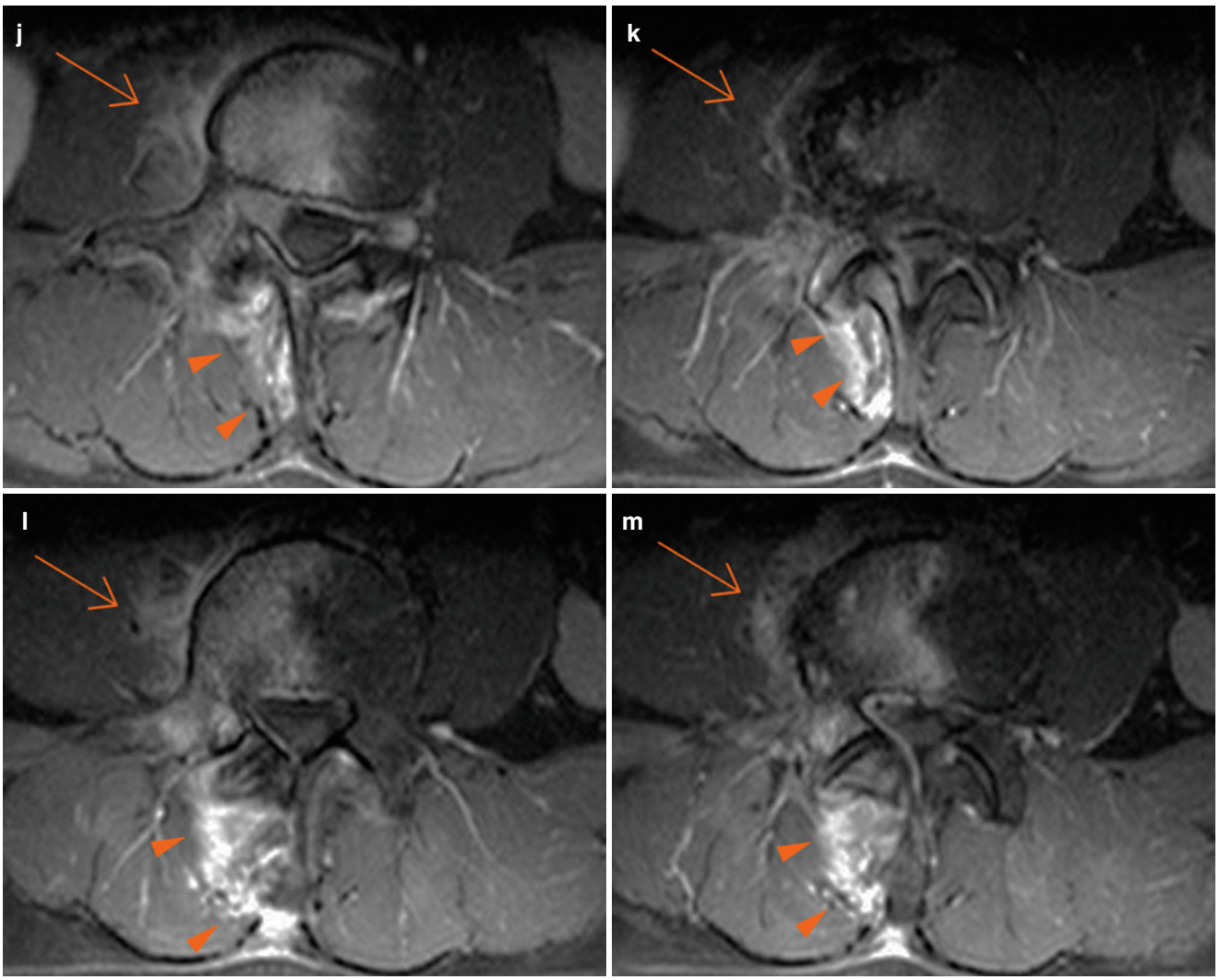


**Fig. 1** (continued)

# Degeneration-Inflammation of Ligaments

# Ligamentous Cyst

# Facet Joint Osteoarthritis

# Synovitis

Case 46

- A 68-year-old woman
- Low back pain
- Weakness in the legs
- Focal tenderness on palpation of the interspinous space at L4/L5

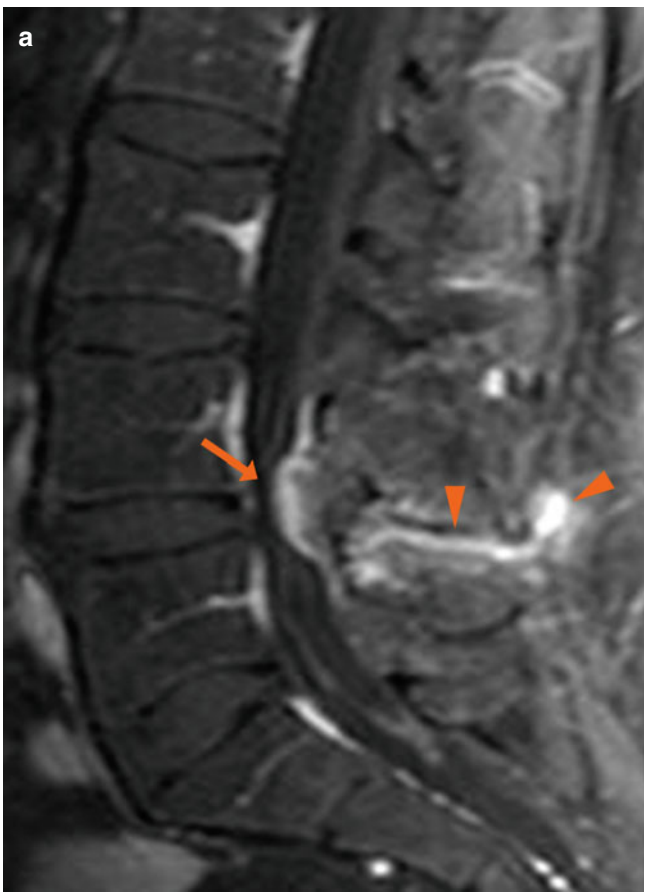


**Fig. 1** Sagittal and axial SE T1-weighted images with fat saturation following the administration of contrast medium (**a**–**c**). Mild anterolisthesis of L4 over L5. Contrast enhancement of the interspinous and supraspinous ligaments at L4/L5, indicating ligamentous degeneration-inflammation (**a**–**c**, *arrowheads*). Note the anterior neo-/pseudocyst with peripheral contrast enhancement, in the epidural space of the posterior aspect of the central spinal canal at L4/L5 in the midline (**a**, **b**, *arrow*). The cyst arises from the collision of the corresponding spinous processes and progressive degeneration of the interspinous ligament, with pseudocyst formation. The same cyst contributes to the stenosis of the central spinal canal at the same level. Bilateral osteoarthritis with synovitis-synovial hypertrophy at L4/L5 (**b**, **c**, *double arrows*). (**d**) Schematic figure (Modified from Jinkins)

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DOI 10.1007/978-3-319-09447-2_48, © Springer International Publishing Switzerland 2015

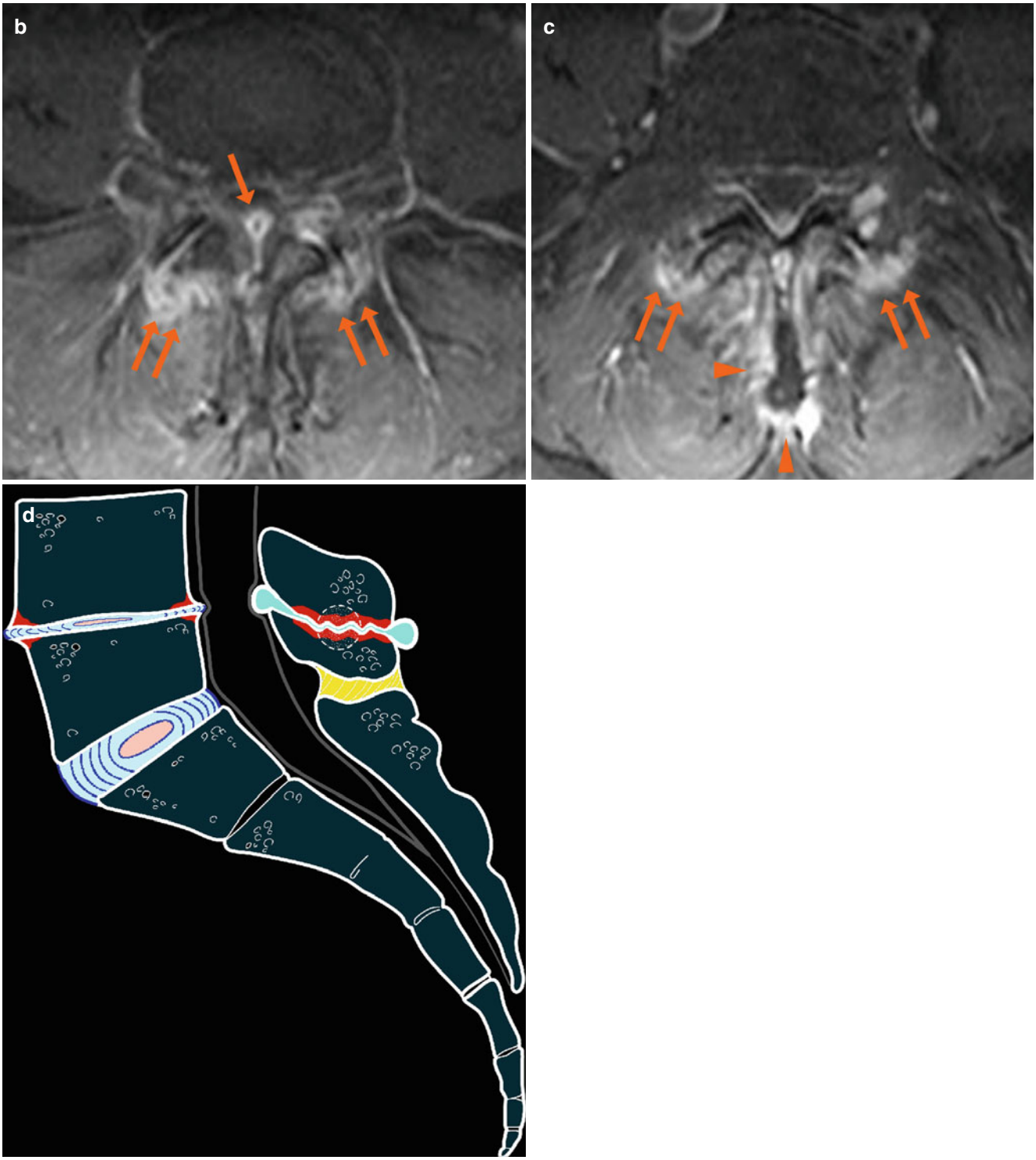


**Fig. 1** (continued)

# Degeneration-Inflammation of Ligaments and Paravertebral Muscles

Case 47

- A 66-year-old male
- Chronic low back pain
- Pain on palpation of the lumbar interspinous spaces and posterior paravertebral muscles on the left side

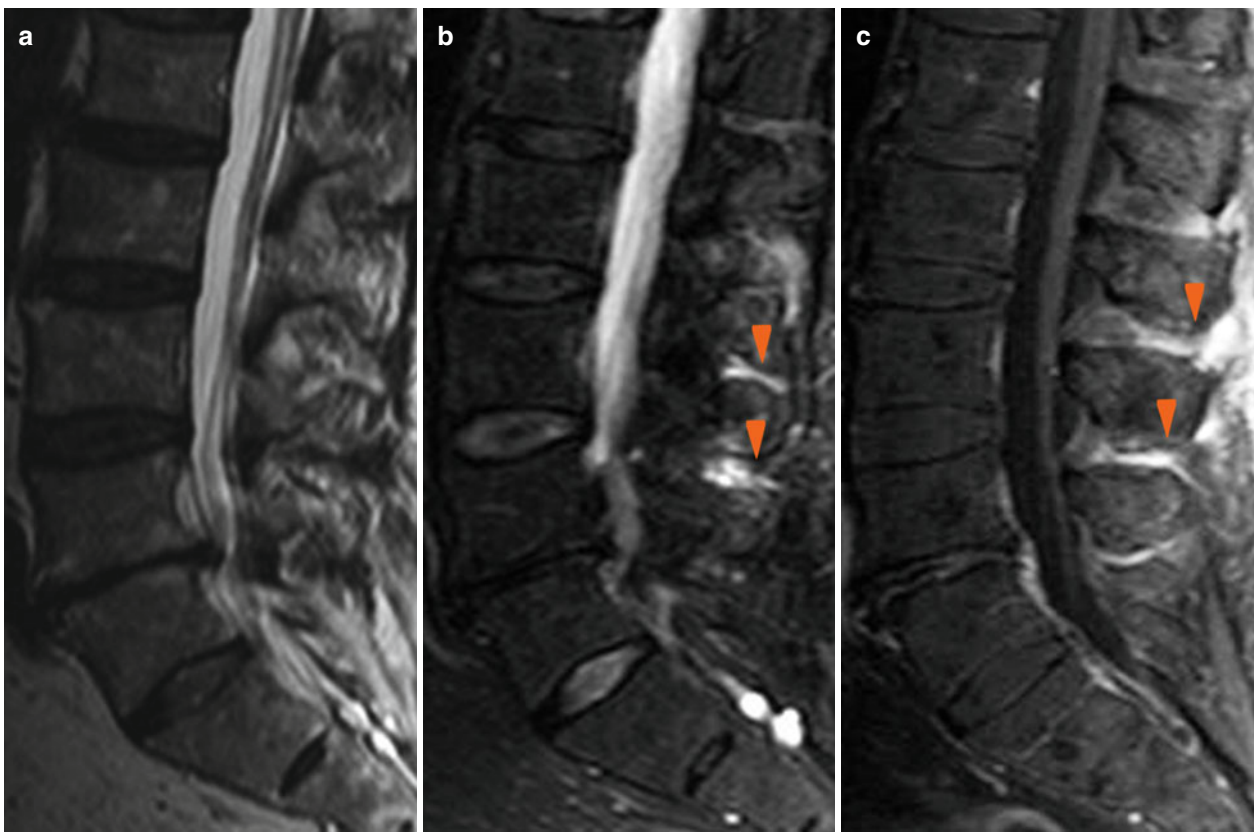


**Fig. 1** Sagittal SE T2-weighted image (**a**), sagittal and axial TSE T2-weighted images with fat saturation (**b**, **d**, **e**), sagittal and axial SE T1-weighted images with fat saturation following administration of contrast medium (**c**, **f**, **g**). Osteochondrosis at L4-L5. T2 hyperintensity and contrast enhancement of the interspinous and/or supraspinous ligaments from L1 to L5, indicating degeneration-inflammation of the same ligaments (**b**, **c**, *arrowhead*). The same patient presented T2 hyperintensity and contrast enhancement of the interspinalis muscles (**e**–**g**, *arrowhead*) and left multifidus muscle (**d**–**g**, *arrow*), referable to degeneration-inflammation of the same muscles

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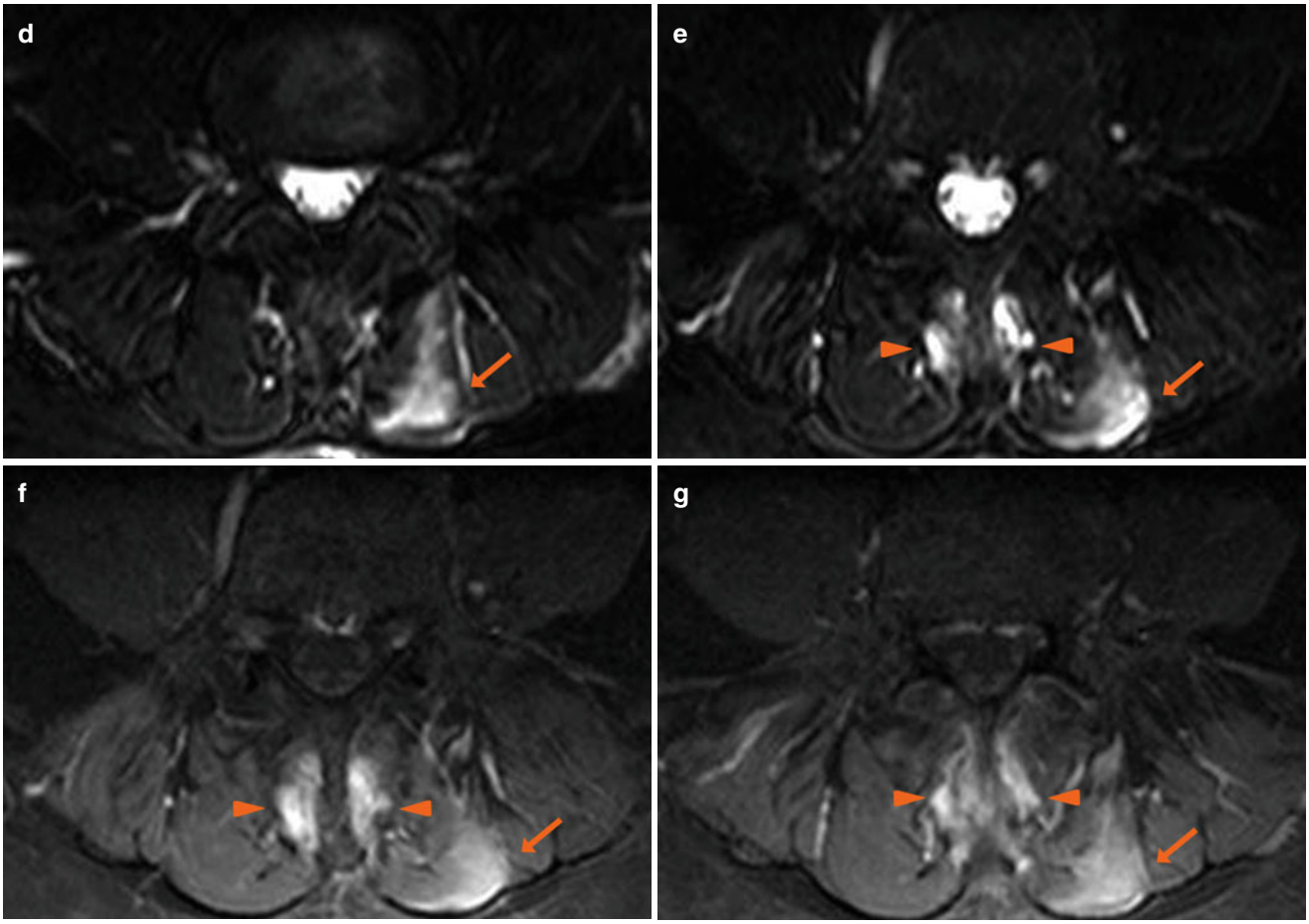


**Fig. 1** (continued)

# Muscular Inflammation

Case 48

- A 31-year-old woman
- Low back pain
- Focal tenderness on palpation of the lumbar perispinal muscles

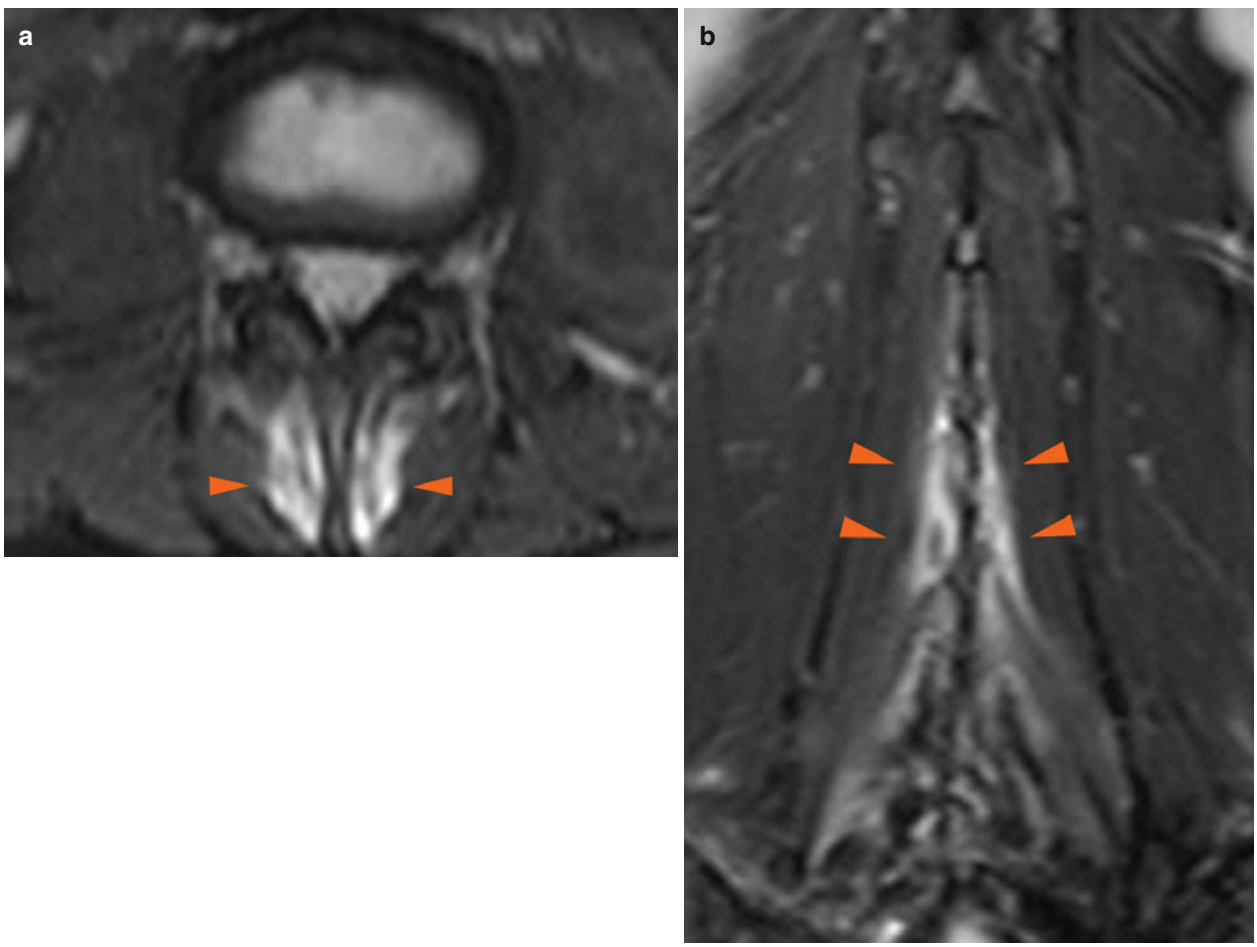


**Fig. 1** Axial and coronal TSE T2-weighted images with fat saturation image (**a**, **b**). T2 hyperintensity of the interspinalis muscles, due to oedema (**a**, **b**, *arrowheads*). This patient had a trauma 10 days before MR examination

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DOI 10.1007/978-3-319-09447-2_50, © Springer International Publishing Switzerland 2015

- A 66-year-old man
- Right-sided low back pain
- Focal tenderness

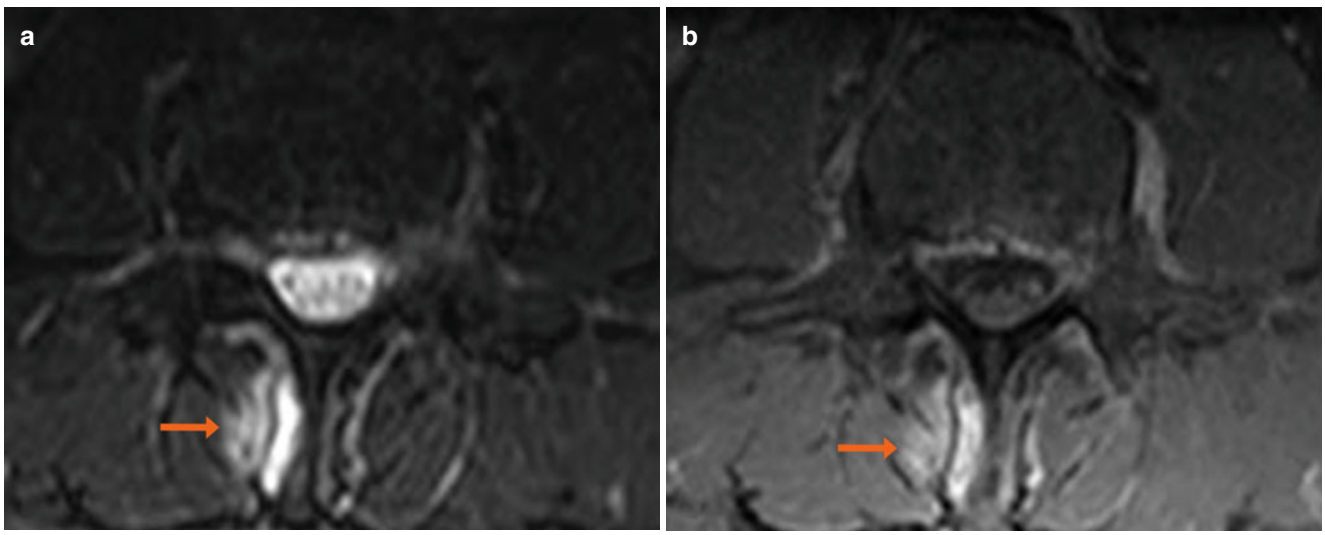


**Fig. 1** Axial TSE T2-weighted image with fat saturation (**a**), axial and coronal SE T1-weighted images with fat saturation after contrast medium administration (**b**, **c**). T2 hyperintensity of the perispinal muscles (e.g. interspinalis muscle and multifidus muscle) on the right side (**a**, *arrow*). Contrast enhancement of the same muscles (**b**, **c**, *arrows*). These finding indicate degenerative-oedematous muscular changes

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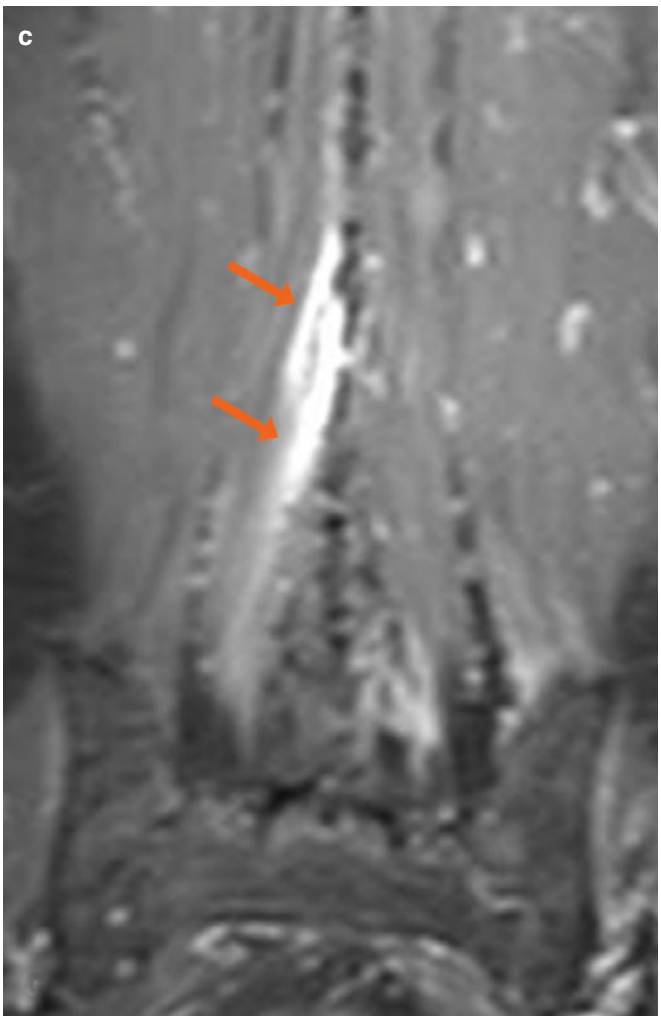


**Fig. 1** (continued)

- A 27-year-old male
- Right back pain after physical exercise

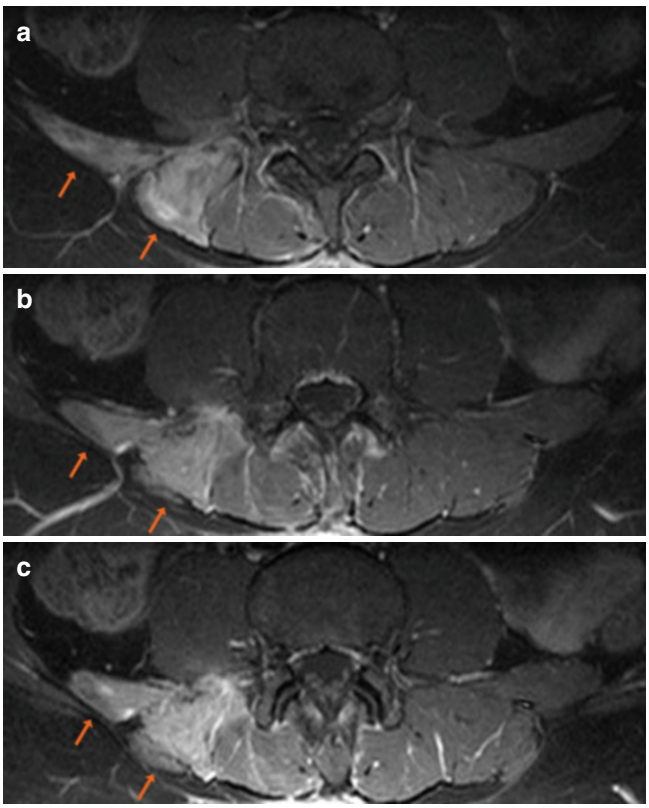


**Fig. 1** Axial SE T1-weighted images with fat saturation following administration of contrast medium (**a**–**c**), coronal TSE T2-weighted with fat saturation (**d**). Signal changes of the posterior paravertebral muscles on the right side. In particular the iliocostalis and quadratus lumborum muscles show contrast enhancement (**a**-**c**, *arrows*) and high signal in T2 (**d**, *arrow*). These findings indicate inflammatory muscular changes. There was precise correlation between MRI findings and the patient's focus of pain

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DOI 10.1007/978-3-319-09447-2_52, © Springer International Publishing Switzerland 2015

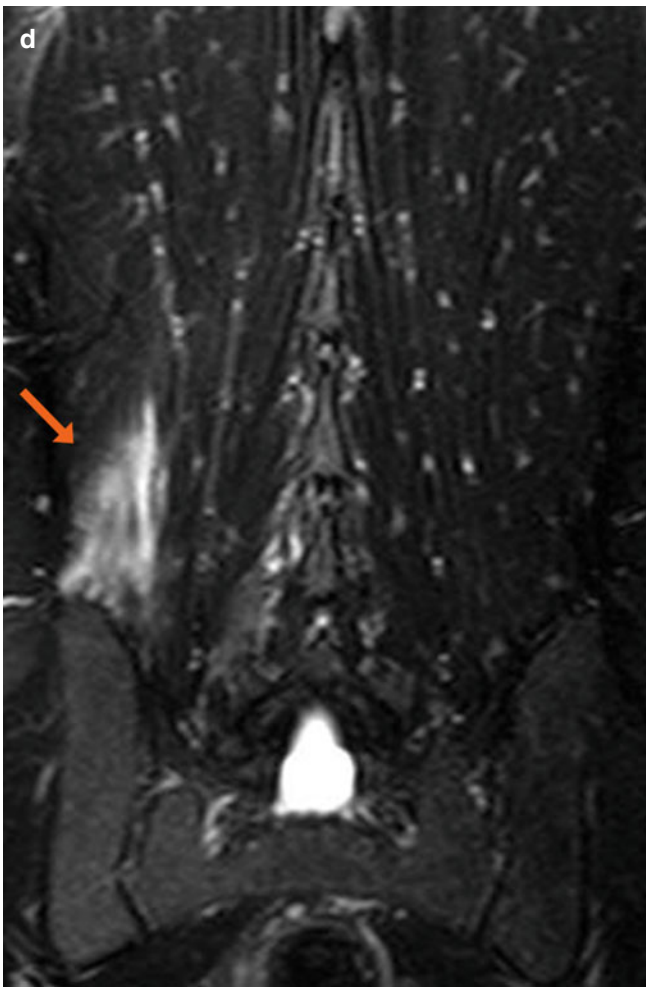


**Fig. 1** (continued)

- A 30-year-old patient
- Lumbar radiculopathy on the right side
- Limited motion
- Focal tenderness corresponding to lumbar perispinal muscles on the right side

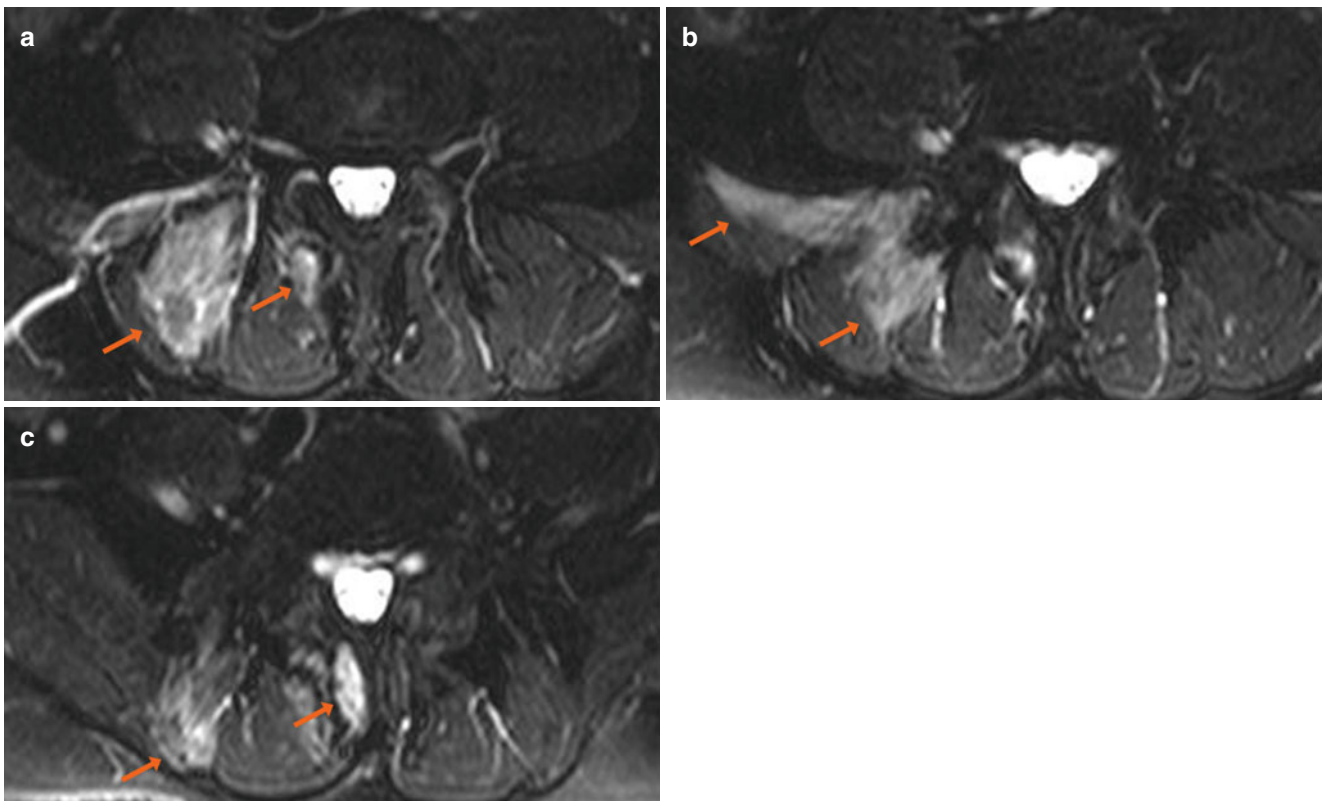


**Fig. 1** Axial TSE T2-weighted images with fat saturation (**a**–**c**), sagittal, axial and coronal SE T1-weighted images with fat saturation following administration of contrast medium (**d**–**m**). Signal changes of the lumbar perispinal muscles on the right side, e.g. interspinalis, multifidus, thoracocostalis, longissimus, quadratus lumborum (**a**–**c**, **e**–**g**, **j**, **k**, *arrows*). These muscles exhibit high signal in T2-weighted images (**a**–**c**), with marked contrast enhancement (**e**–**g**, **j**, **k**, *arrows*). The same patient presented contrast enhancement of a nerve root on the right side (**d**, **h**, **i**, *arrow*), indicating sterile radiculitis

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DOI 10.1007/978-3-319-09447-2_53, © Springer International Publishing Switzerland 2015

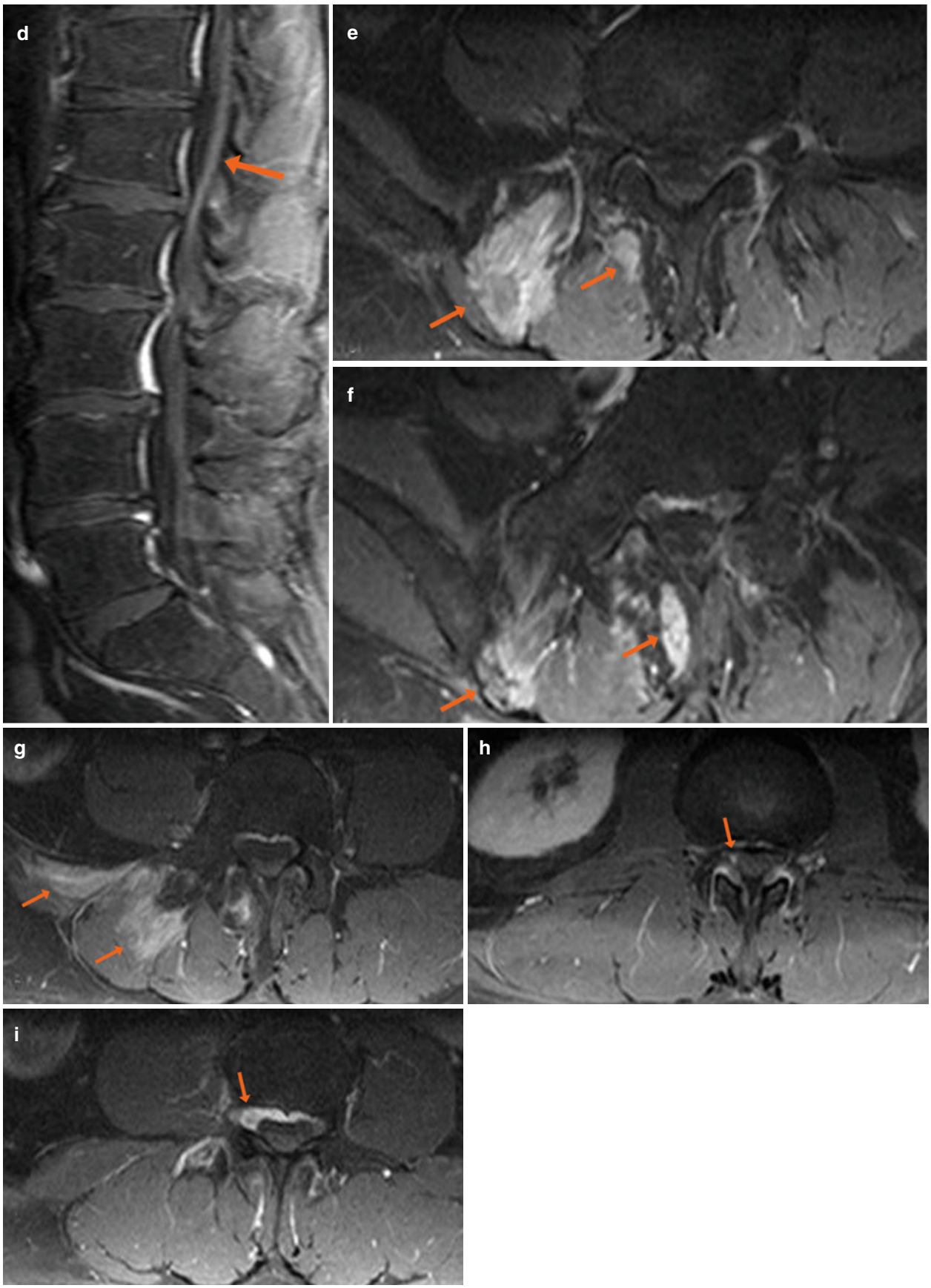


**Fig. 1** (continued)

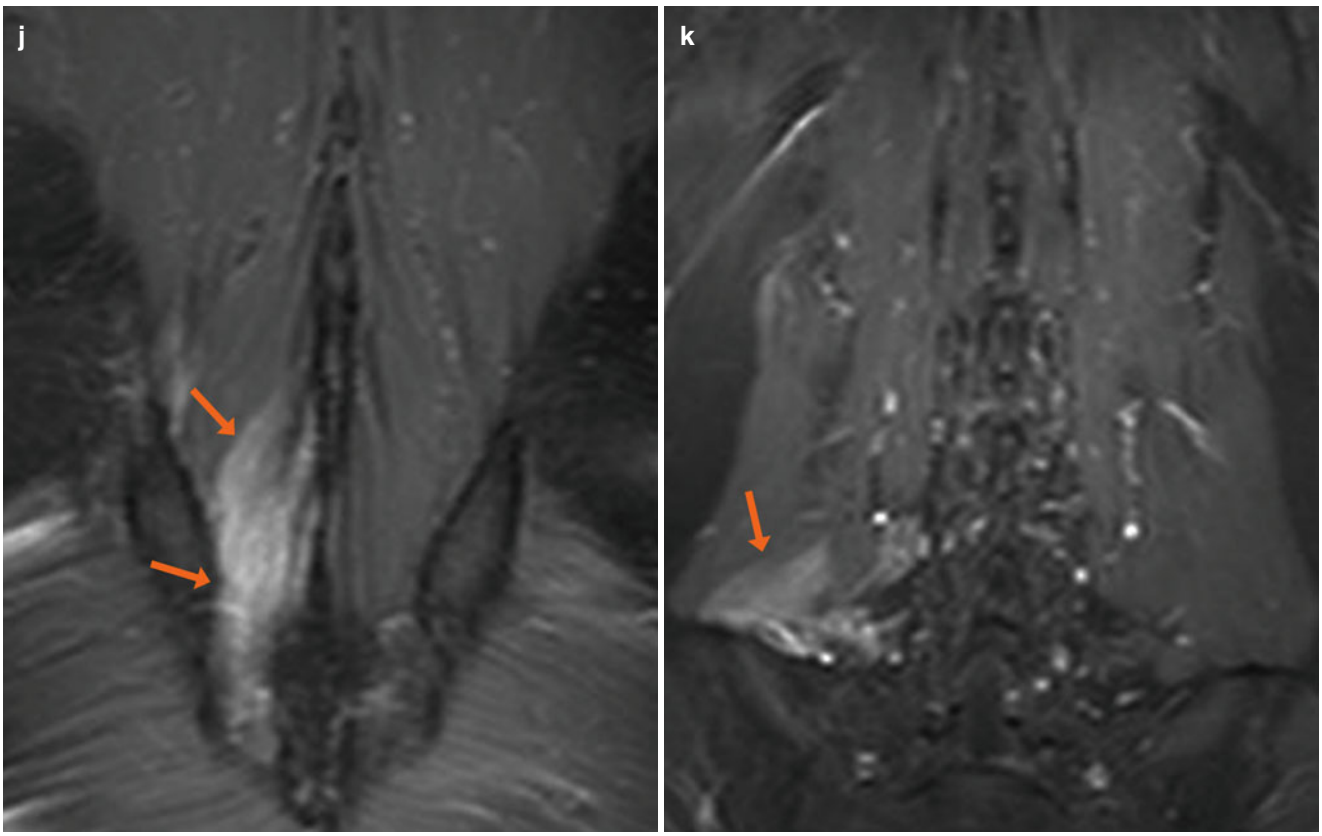


**Fig. 1** (continued)

## Unusual Case

# Herniated Disc in Unusual Location

Case **52**

- A 58-year-old woman
- Sudden onset of back pain and right anterior thigh pain following physical exercise

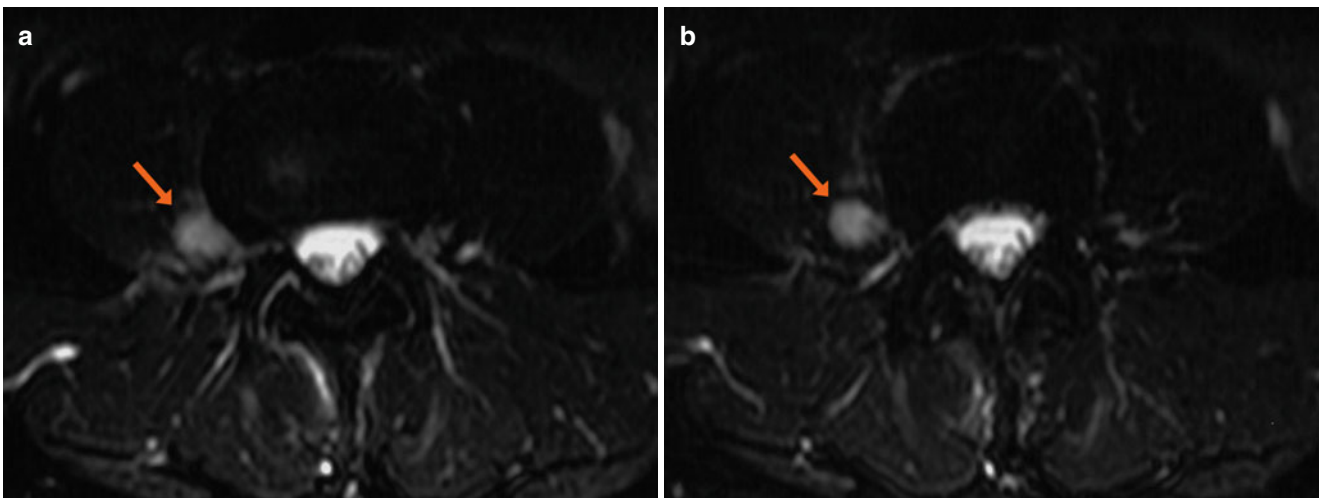


**Fig. 1** Axial TSE T2-weighted images with fat saturation (**a**, **b**), axial and coronal SE T1-weighted images with fat saturation following administration of contrast medium (**c**–**f**). Small rounded mass lateral to the right L1/L2 neural foramen and adjacent to the ipsilateral psoas. The mass shows high signal on T2-weighted images (**a**, **b**, *arrow*), probably caused by associated edema, and has an enhancing rim on contrast-enhanced T1-weighted studies (**c**–**f**, *arrow*). This is an extraforaminal disc fragment (so-called far lateral herniation), well delineated by peripheral contrast enhancement due to peridiscal inflammatory reaction

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DOI 10.1007/978-3-319-09447-2_54, © Springer International Publishing Switzerland 2015

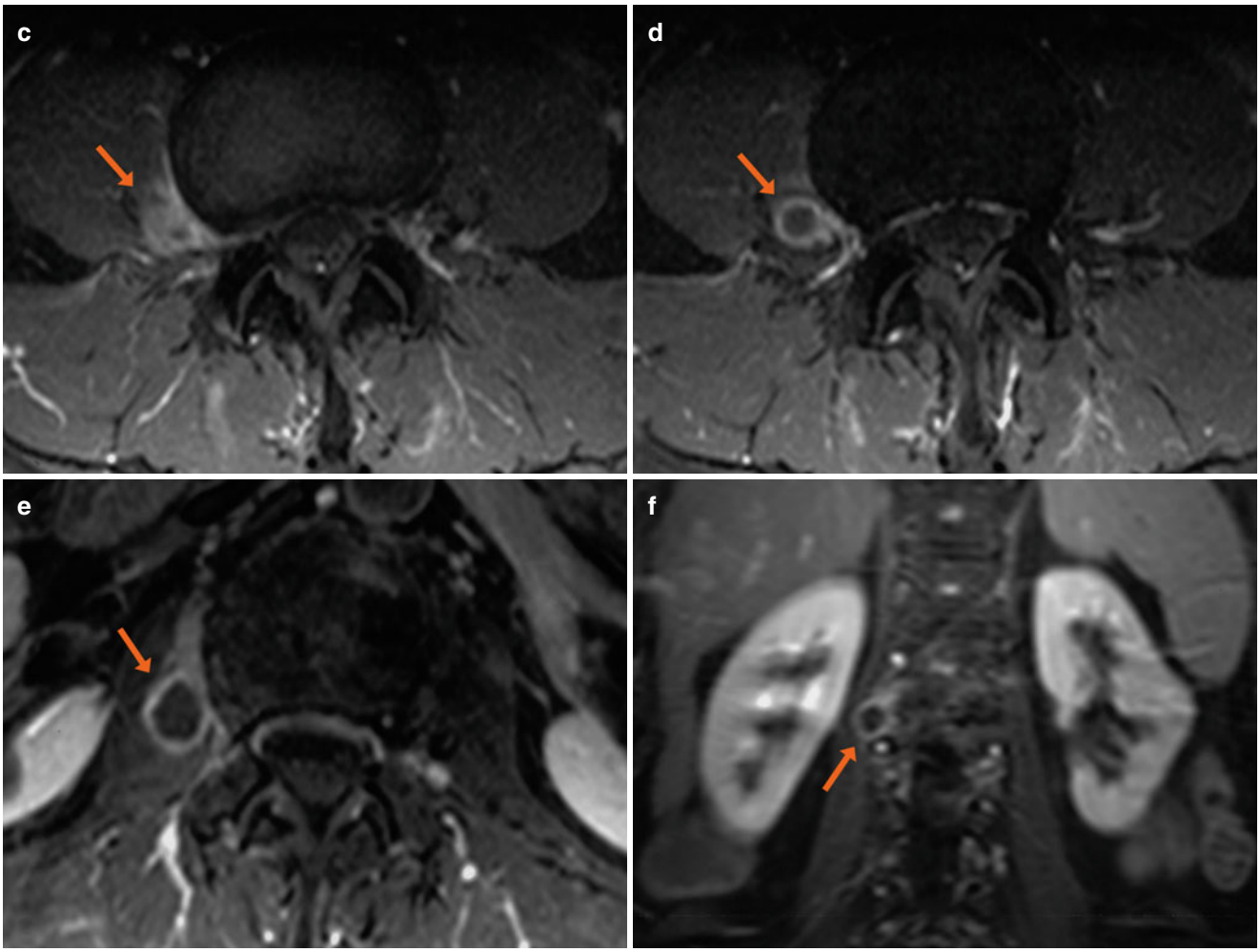


**Fig. 1** (continued)

## Unusual Case

# Herniated Disc in Unusual Location

Case **53**

- A 55-year-old man
- Persistent low back pain
- Limited lumbar motion

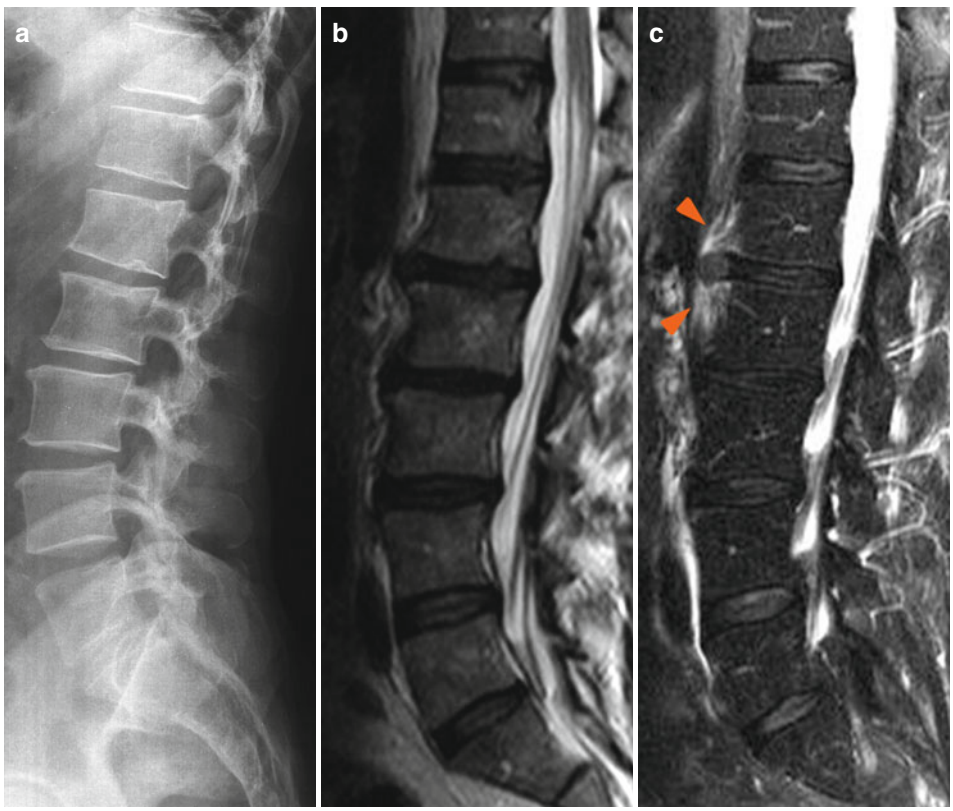


**Fig. 1** Lateral lumbosacral X-ray (**a**), sagittal TSE T2-weighted image (**b**), sagittal and axial TSE T2-weighted images with fat saturation (**c**, **e**), axial SE T1-weighted image (**d**), sagittal and axial SE T1-weighted images with fat saturation after contrast medium administration (**f**, **g**). X-ray does not show spine changes (**a**). MRI shows an anterior herniated disc at L1/L2 (**b**–**f**, *arrowheads*). Note that T2-weighted images with fat saturation (**c**) reveal peridiscal oedema that is not evident in the "standard" T2-weighted images without fat saturation (**b**). Note also peridiscal contrast enhancement indicating peridiscal inflammatory reaction (**f**, **g**, *arrowheads*)

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DOI 10.1007/978-3-319-09447-2_55, © Springer International Publishing Switzerland 2015

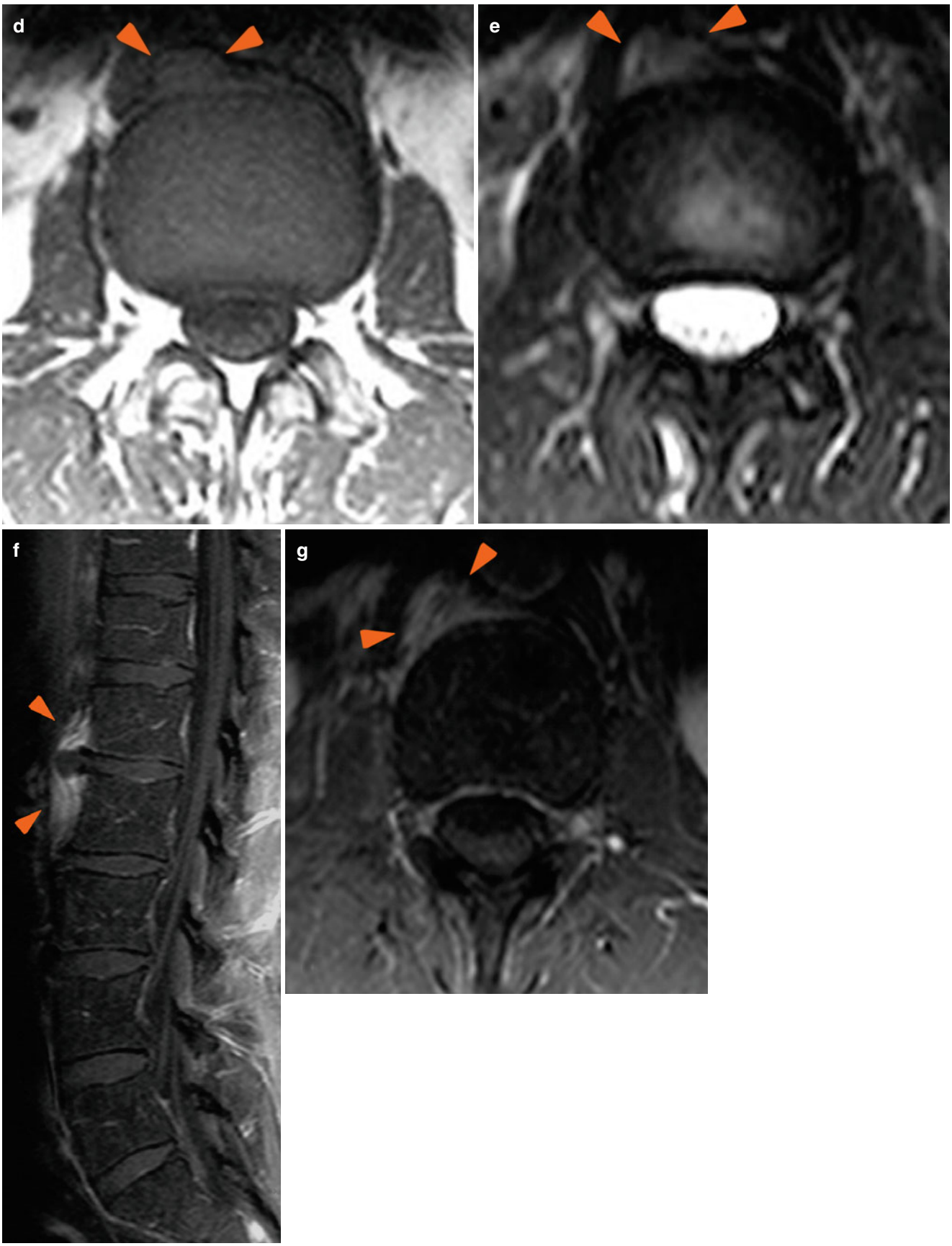


**Fig. 1** (continued)

## Unusual Case

# Giant Schmorl Hernia

Case **54**

- A 30-year-old male
- Low back pain

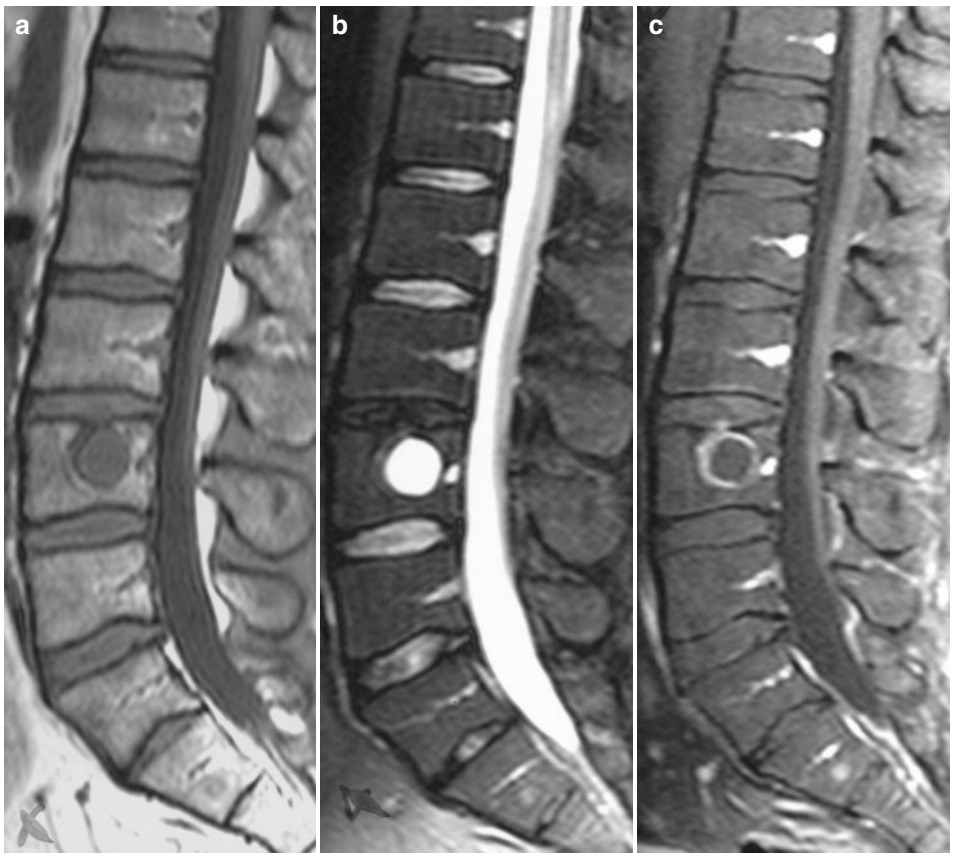


**Fig. 1** Sagittal SE T1-weighted images (**a**), sagittal TSE T2-weighted image with fat saturation (**b**, **d**), sagittal and axial SE T1-weighted images with fat saturation following administration of contrast medium (**c**, **e**), CT scan with coronal and sagittal reconstruction (MRP) (**f**–**h**). Presence of round lesion in the vertebral body of L4. The lesion shows a cystic appearance, hypointense in T1 (**a**), hyperintense in T2 (**b**, **d**), with peripheral contrast enhancement (**c**, **e**). There is a small interruption of the adjacent vertebral endplate. CT images show a thin osteosclerotic margin of the lesion and confirm the interruption of the upper adjacent vertebral endplate. Diagnosis: giant Schmorl hernia with cystic-degenerative phenomena

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DOI 10.1007/978-3-319-09447-2_56, © Springer International Publishing Switzerland 2015

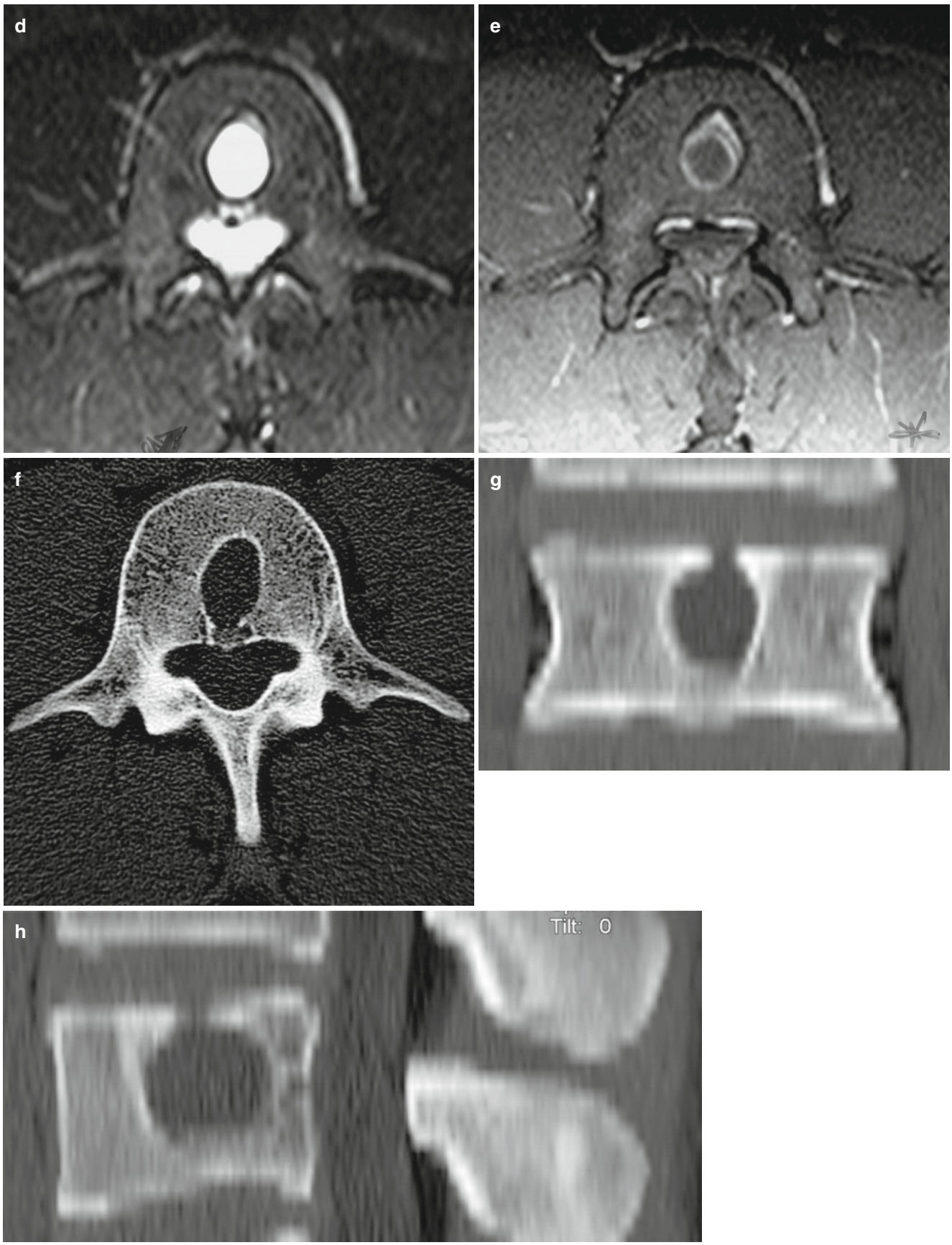


**Fig. 1** (continued)

## Unusual Case

## Ganglion Cyst

Case **55**

- An 18-year-old patient, male
- Low back pain with sciatica on the left side

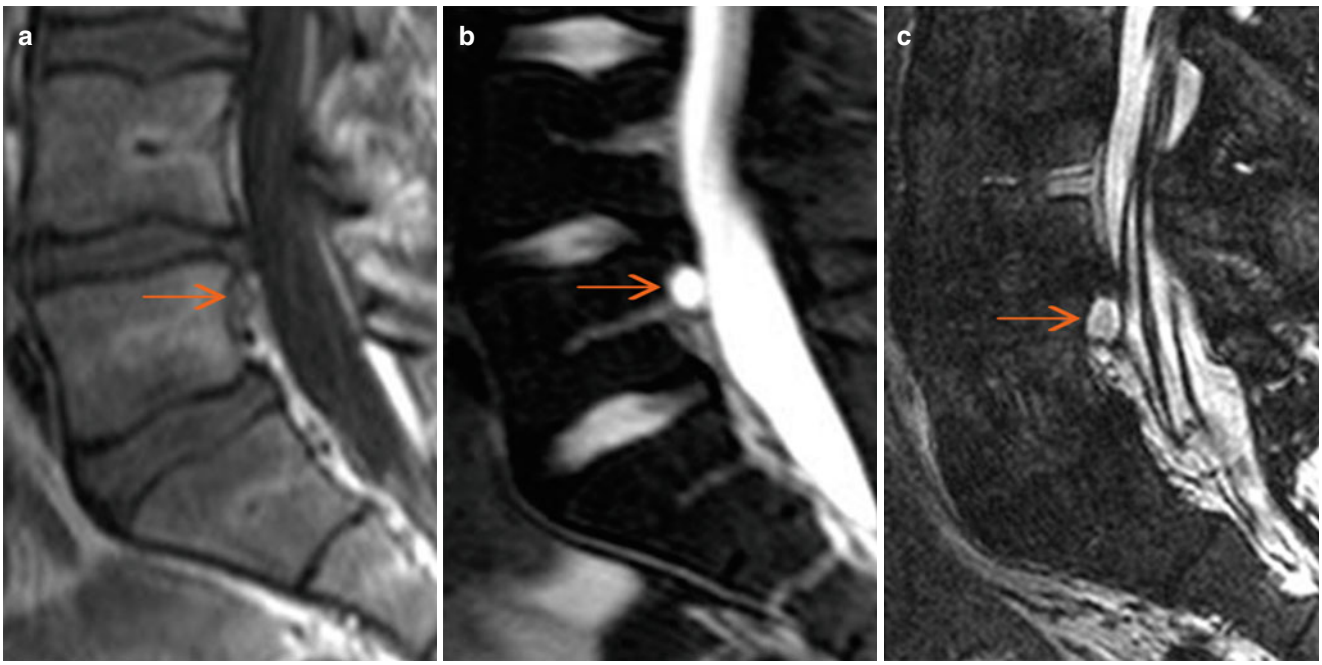


**Fig. 1** Sagittal SE T1-weighted image (**a**), sagittal TSE T2-weighted image with fat saturation (**b**), sagittal and axial CISS images (**c**, **d**), SE T1-weighted images with fat saturation following administration of contrast medium (**e**, **f**). Cystic lesion in the anterior epidural space, on the left side (**a**–**f**, *arrows*). The cyst content resembles the CSF in all pulse sequences (**a**–**d**) and shows peripheral contrast enhancement. This is a so-called ganglion cyst, arising from the posterior longitudinal ligament. Epidural cysts, either synovial or ganglion, are an unusual case of radicular compression

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DOI 10.1007/978-3-319-09447-2_57, © Springer International Publishing Switzerland 2015

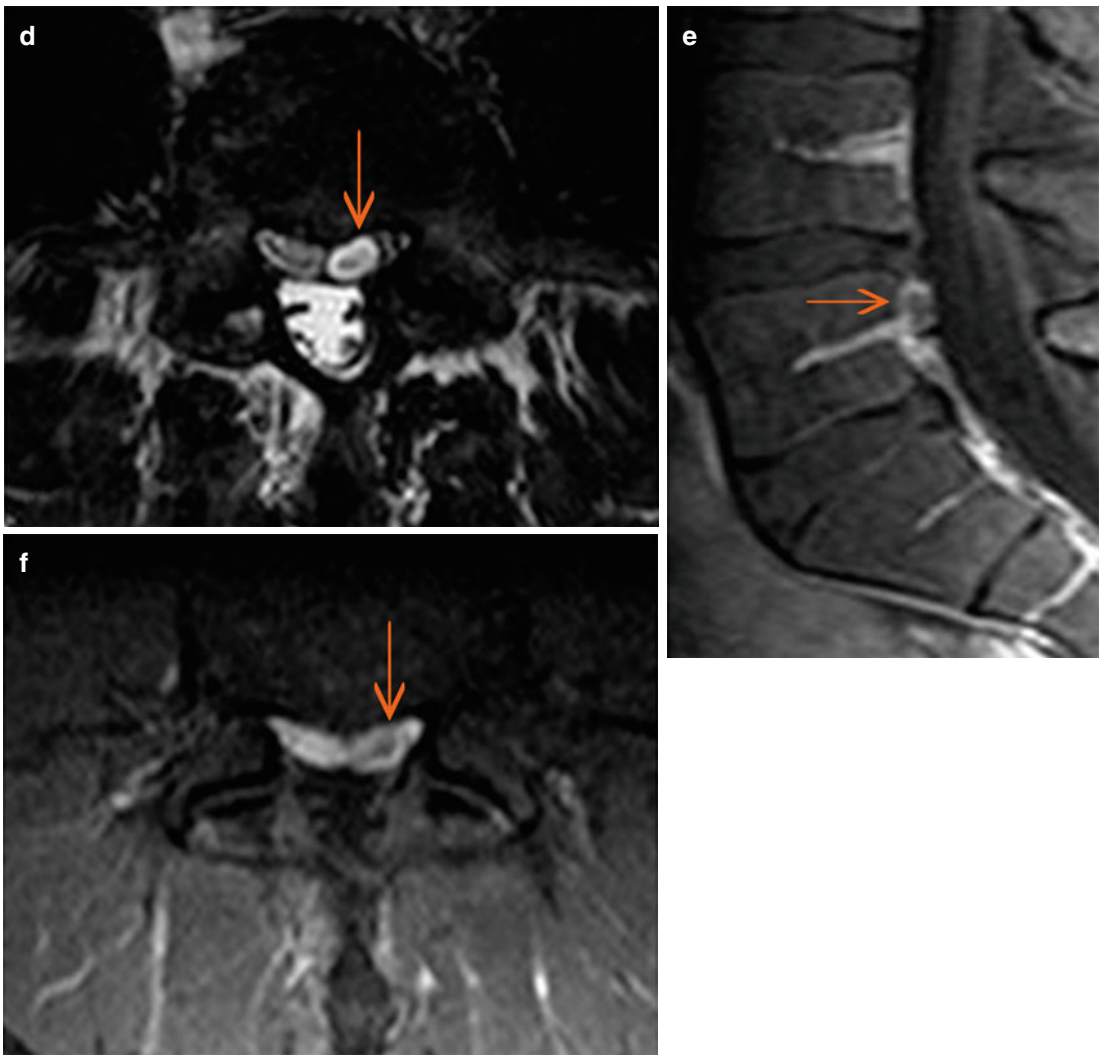


**Fig. 1** (continued)

## Unusual Case

# Periradicular Cyst

Case **56**

- A 54-year-old female
- Left sciatica

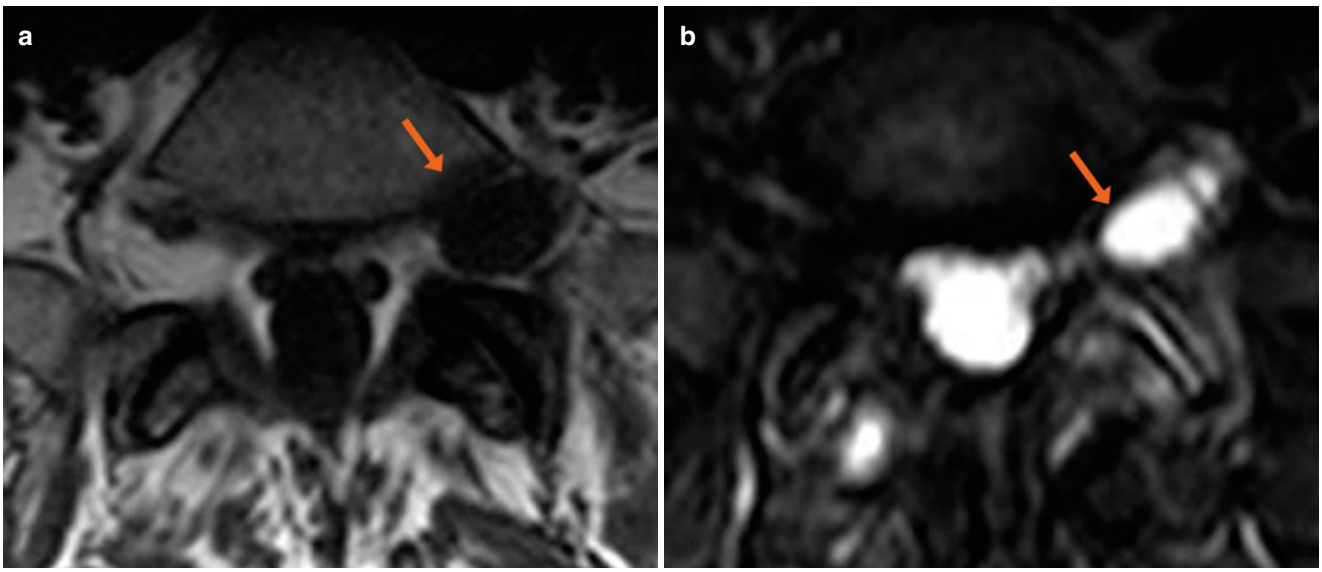


**Fig. 1** Axial SE T1-weighted image (**a**), axial TSE T2-weighted image with fat saturation (**b**), axial CISS images (**c**, **d**). Intraforaminal periradicular cyst at L5/S1 on the left side (**a**–**c**, *arrow*). The cyst compresses and displaces the adjacent nerve root (**d**, *arrow*)

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DOI 10.1007/978-3-319-09447-2_58, © Springer International Publishing Switzerland 2015

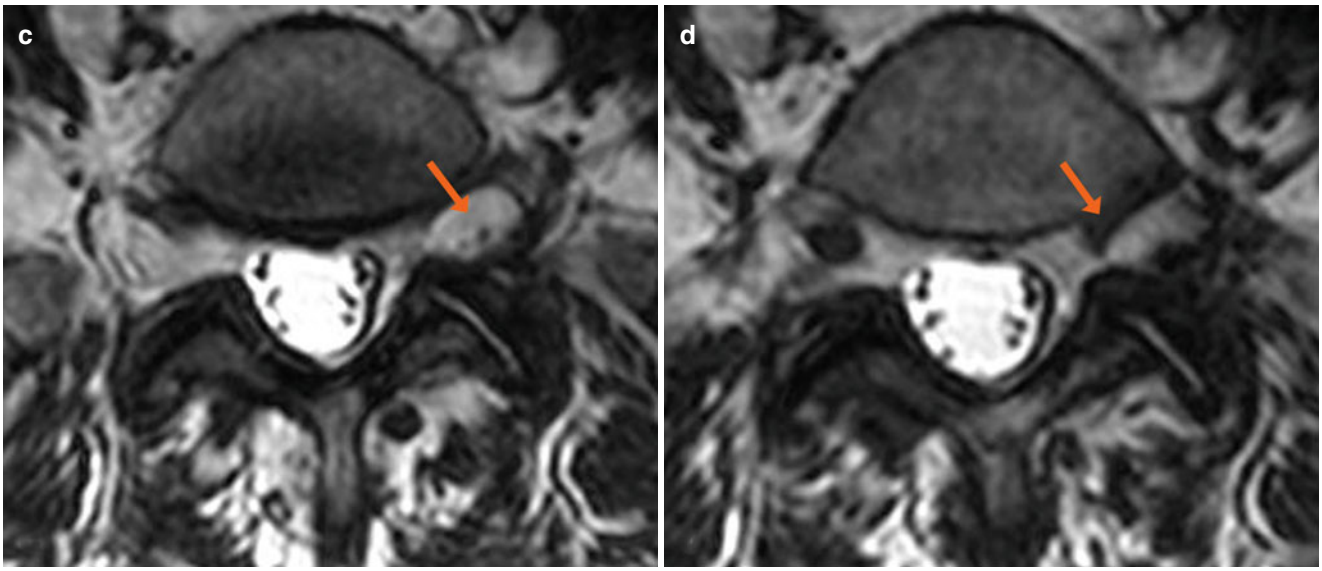


**Fig. 1** (continued)

## Unusual Case

## Periganglíar Cyst

Case **57**

- A 44-year-old woman
- Right sciatica

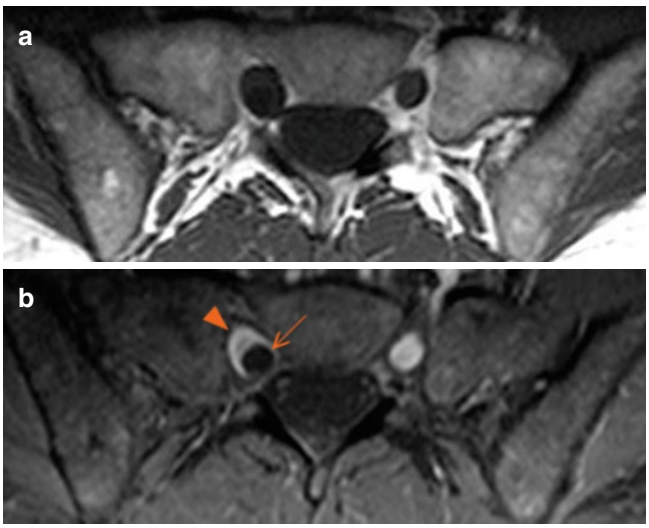


**Fig. 1** Axial SE T1-weighted image (**a**), axial SE T1-weighted image with fat saturation after contrast medium administration (**b**). Periradicular-periganglíar cyst at S1. The cyst (**b**, *arrow*) compresses and displaces the corresponding ganglion (**b**, *arrowhead*)

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DOI 10.1007/978-3-319-09447-2_59, © Springer International Publishing Switzerland 2015

## Unusual Case

# Pigmented Villonodular Synovitis

Case **58**

- A 35-year-old man
- 3 weeks history of left-sided low back pain

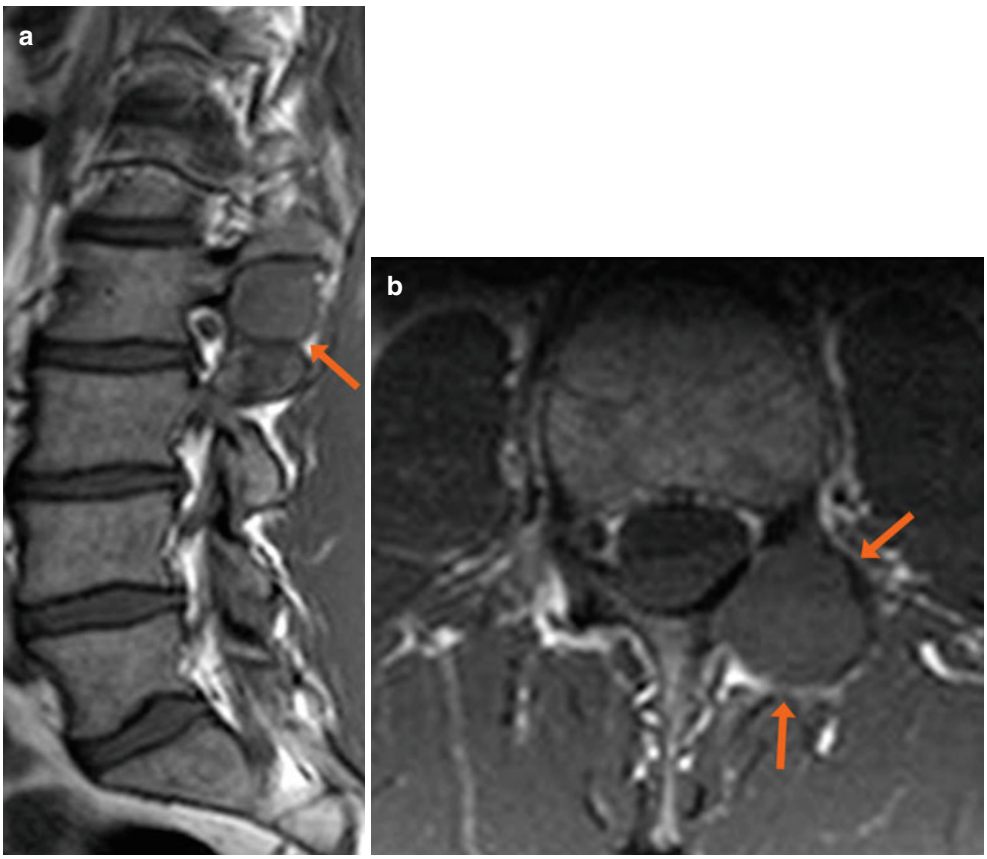


**Fig. 1** Sagittal and axial SE T1-weighted image (**a**, **b**), axial TSE T2-weighted image with fat saturation (**c**), axial GE T2*-weighted image (**d**), sagittal and axial SE T1-weighted images with fat saturation following administration of contrast medium (**e**, **f**), axial CT scan (**g**). Extradural mass arising from the left facet joint at L2/L3 (*arrow*). The lesion shows intermediate-low signal intensity in T1 (**a**, **b**) and hyperintensity in T2 (**c**, **d**), with marked contrast enhancement (**e**, **f**). CT scan shows osteolysis of the same facet joint (**g**, *arrows*). The patient underwent a biopsy of the lesion, with histological diagnosis of pigmented villonodular synovitis (PVNS). PVNS is a locally aggressive proliferative disorder affecting synovium lined joints. The anatomopathological findings of PVNS are constituted by synovial cells lined by villous fronds containing mononuclear cells, multinucleated giant cells, fibroblasts and macrophages

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DOI 10.1007/978-3-319-09447-2_60, © Springer International Publishing Switzerland 2015

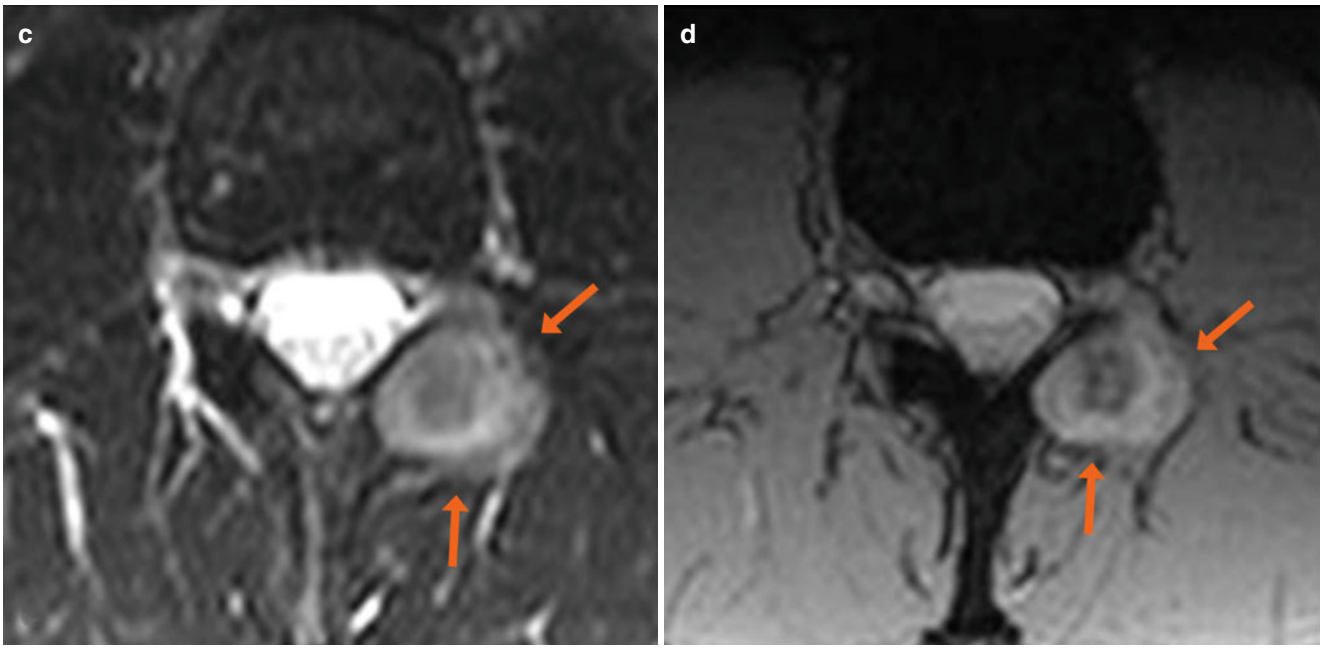
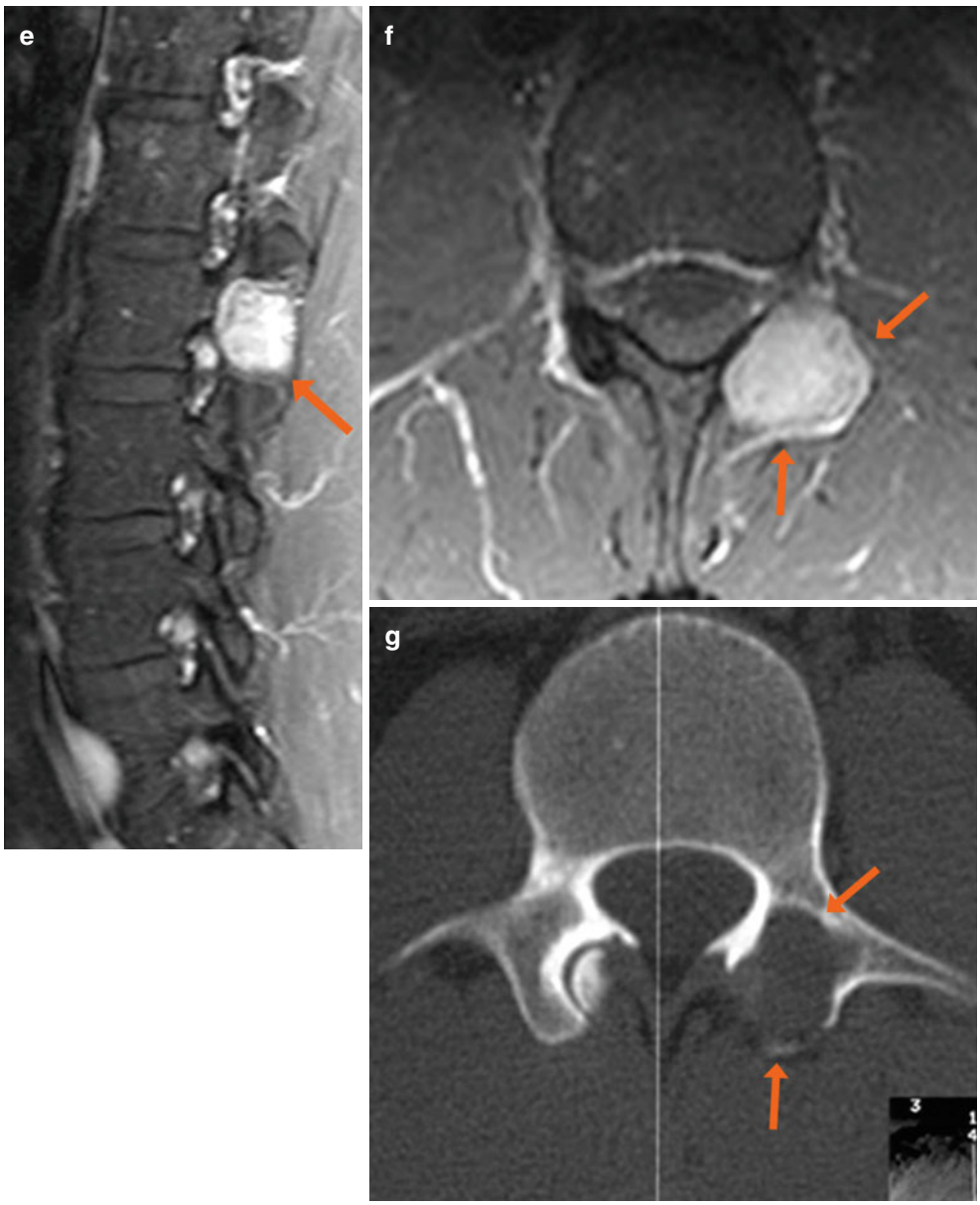



**Fig. 1** (continued)

**Fig. 1** (continued)




## Reference

1. Oreste D, Tarantino A, D'Aprile P (2009) MRI of pigmented villonodular synovitis of the lumbar spine. Euro Rad (online, case 7520). URL: http//www.eurorad.org/case.php?id=7520

# Radiculitis

Case 59

- A 43-year-old female
- Right low back pain with radiculopathy

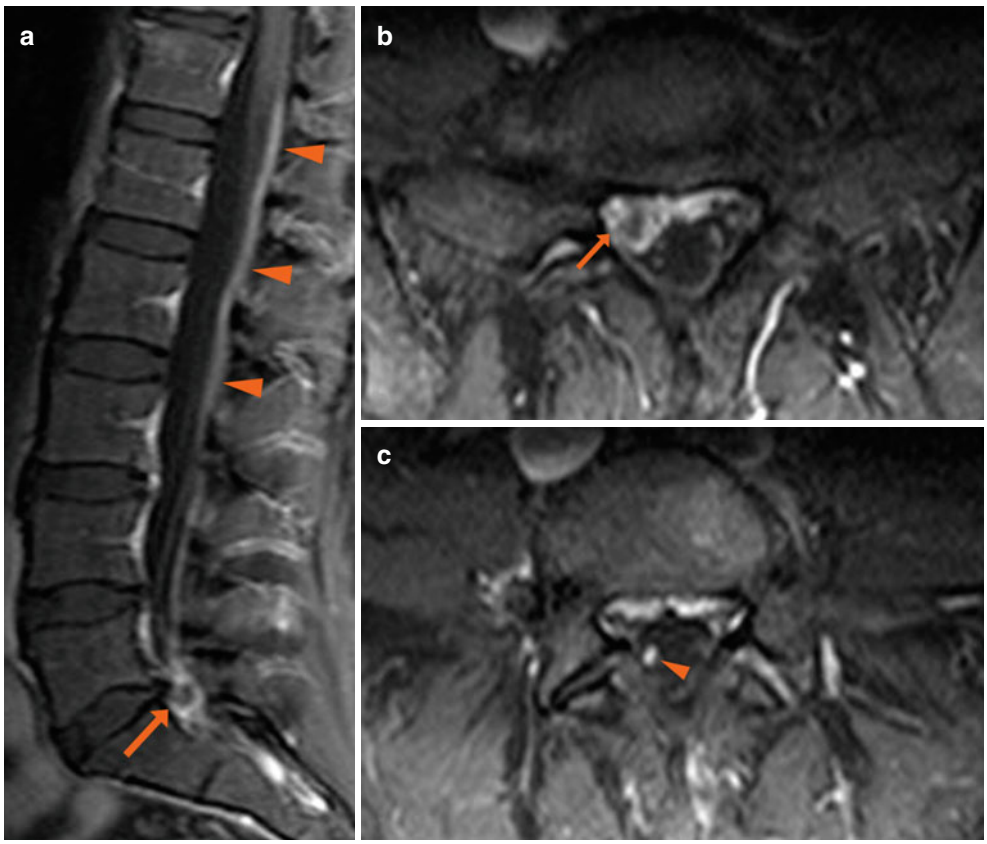



**Fig. 1** Sagittal and axial SE T1-weighted images with fat saturation after contrast medium administration (**a**–**c**). Herniated disc fragment at L5/S1, with reactive peripheral contrast enhancement (**a**, **b**, *arrow*). Diffuse intradural contrast enhancement of the compressed nerve root (**a**, **c**, *arrowhead*), referable to sterile, benign radiculitis. In some cases of lateral recess stenosis, the compressed nerve root enhances after contrast medium administration as a result of a breakdown of the blood-nerve barrier and/or intravascular enhancement of radicular veins

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# Radiculitis

Case 60

- A 65-year-old woman
- Low back pain with radiculopathy

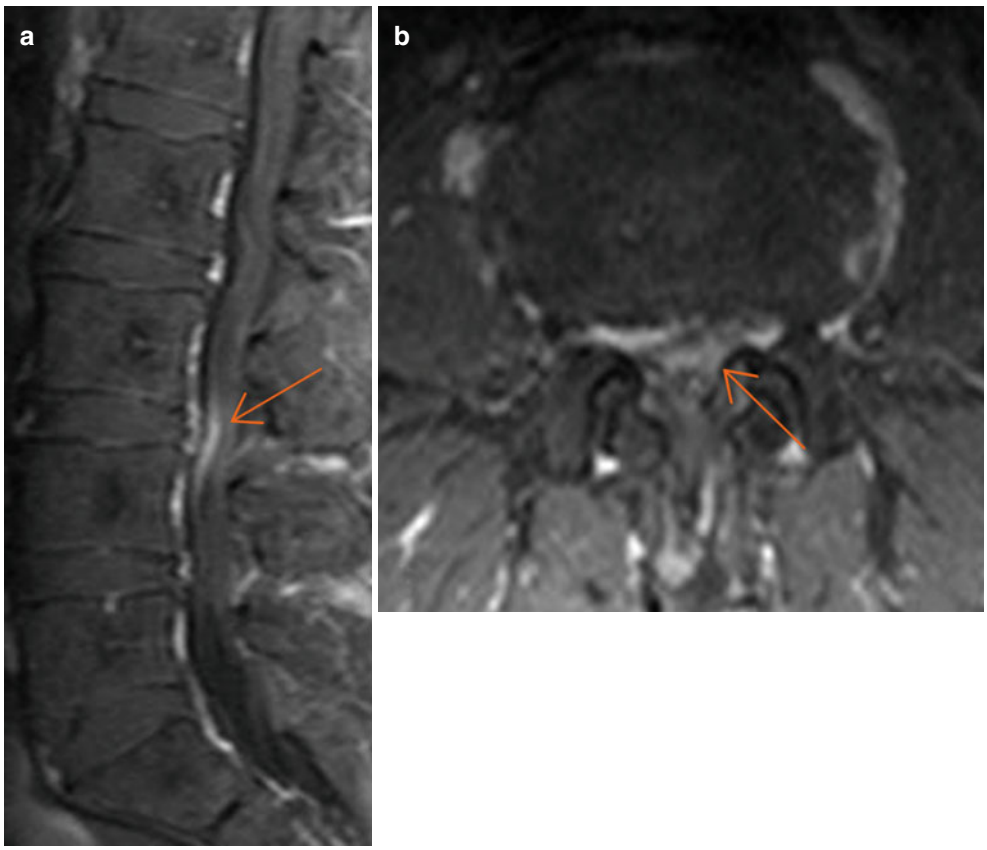


**Fig. 1** Sagittal and axial T1-weighted images with fat saturation following administration of contrast medium (**a**, **b**). Contrast enhancement of nerve roots at L3/L4 (**a**, **b**, *arrow*), in correspondence of a canal stenosis. This finding is referable to aseptic radiculitis caused by compression of nerve roots

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# Radiculitis

Case 61

- A 48-year-old male
- Low back pain with radiculopathy

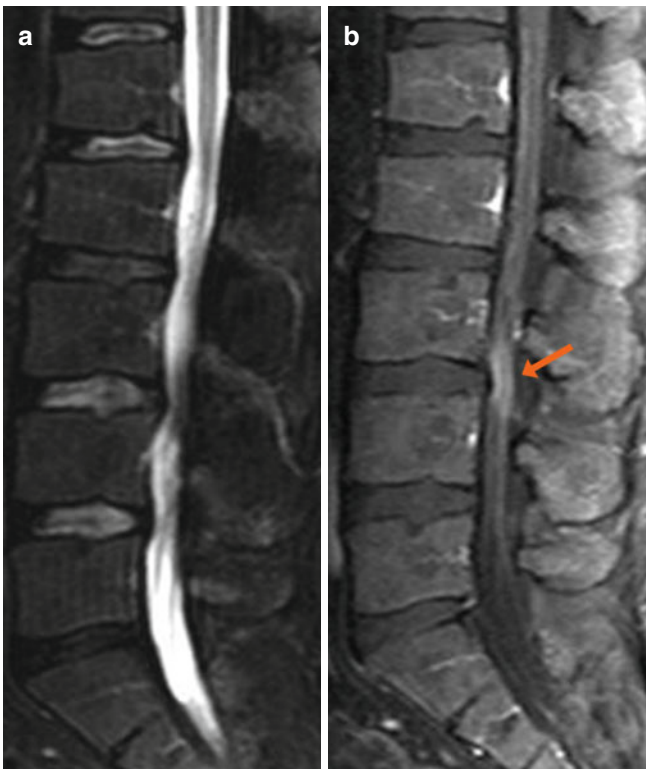


**Fig. 1** Sagittal TSE T2-weighted image with fat saturation (**a**), sagittal and axial SE T1-weighted images with fat saturation following administration of contrast medium (**b–d**). Herniated disc and canal stenosis at L3/L4. At the same level there is contrast enhancement of nerve roots (**b–d**, *arrow*). Diagnosis: radiculitis due to compression of nerve roots

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DOI 10.1007/978-3-319-09447-2_63, © Springer International Publishing Switzerland 2015

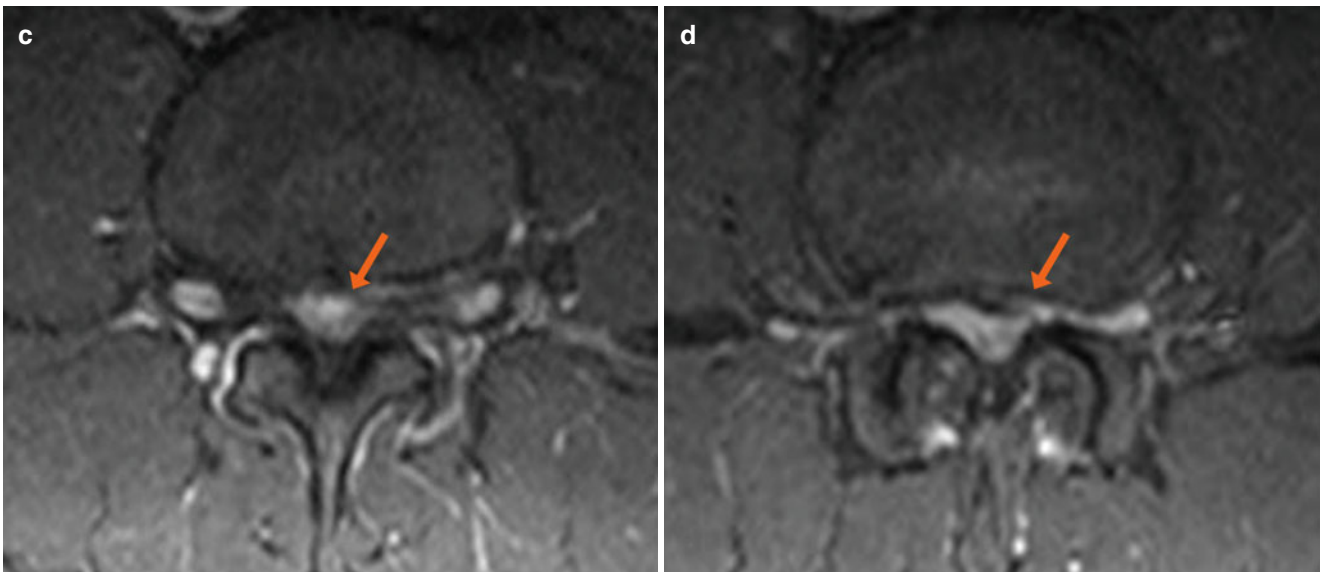


**Fig. 1** (continued)

# Radiculitis

Case 62

- A 73-year-old female
- Left low back pain with radiculopathy

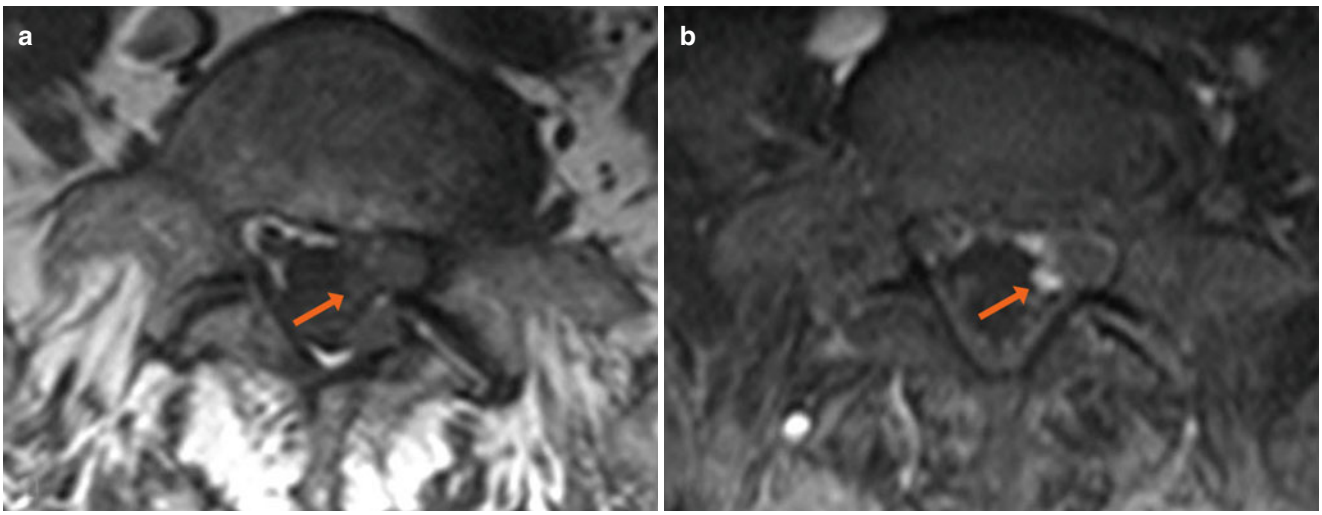


**Fig. 1** Axial SE T1-weighted image (**a**), sagittal and axial SE T1-weighted images with fat saturation following contrast medium administration (**b**–**d**). Herniated disc with inferior migratory fragment behind the L5 vertebral body on the left side (**a**, **c**, *arrows*). It coexists with contrast enhancement of the adjacent intradural nerve root (**b**, **d**, *arrows*), indicating sterile radiculitis

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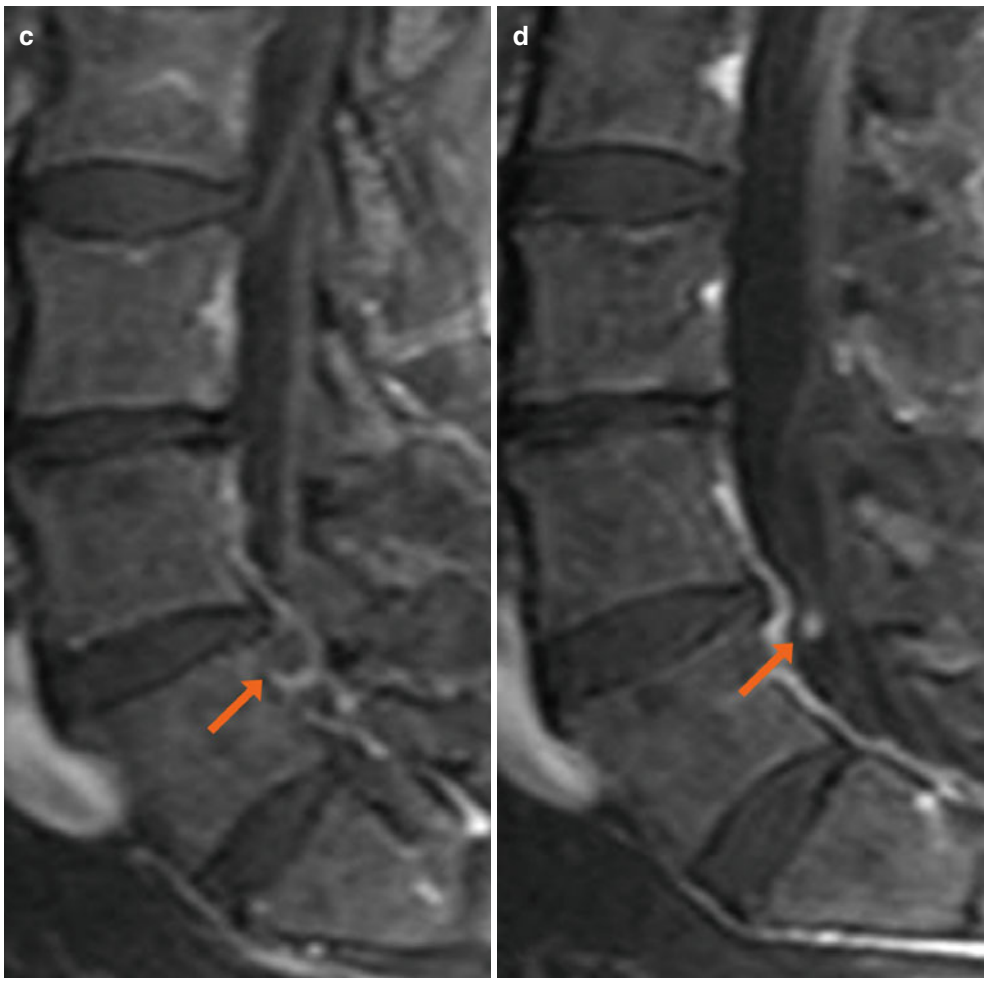
DOI 10.1007/978-3-319-09447-2_64, © Springer International Publishing Switzerland 2015

**Fig. 1** (continued)



# Radiculitis

Case 63

- A 78-year-old male
- Low back pain with bilateral radiculopathy

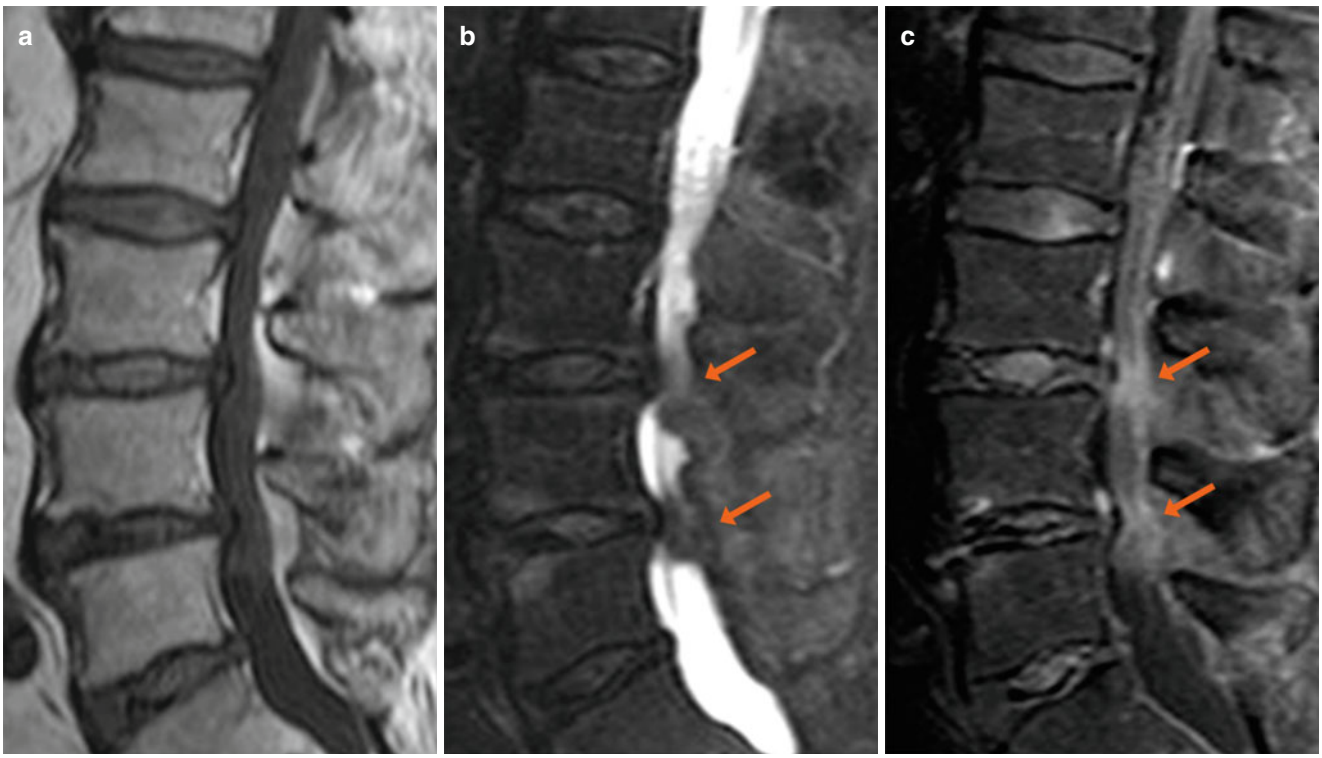



**Fig. 1** Sagittal SE T1-weighted image (**a**), sagittal TSE T2-weighted image with fat saturation (**b**), sagittal and axial SE T1-weighted images with fat saturation after administration of contrast medium (**c**–**e**). Disc herniation/protrusion and canal stenosis at L3/L4 and L4/L5 (**a**, **b**, *arrows*). Contrast enhancement of the nerve roots at the same levels (**c**–**e**, *arrow*) indicating sterile, benign radiculitis due to radicular compression

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DOI 10.1007/978-3-319-09447-2_65, © Springer International Publishing Switzerland 2015

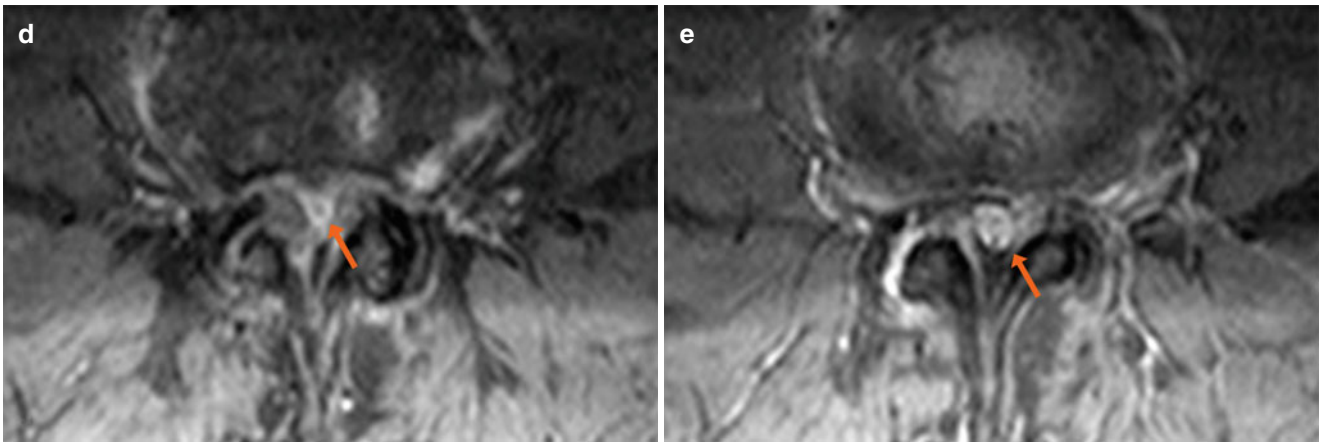


**Fig. 1** (continued)

# Neuritis

Case 64

- A 35-year-old man
- Low back pain on the right side

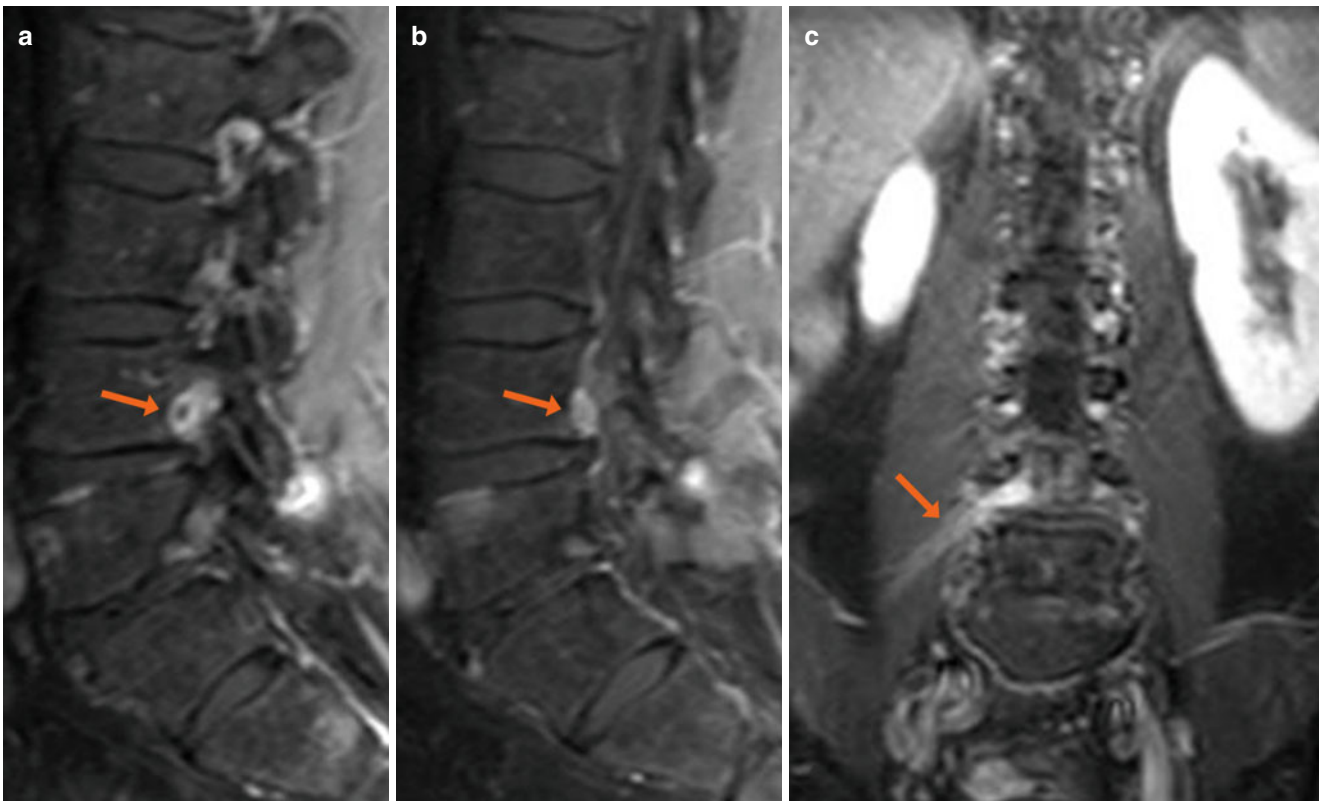


**Fig. 1** Sagittal, coronal and axial SE T1-weighted images with fat saturation following administration of contrast medium (**a**–**d**). Herniated disc with superior migratory fragment within the right L4/L5 neural foramen and behind the L4 vertebral body (**a**, **b**, *arrow*). The same disc fragment shows peripheral enhancement after contrast medium administration, due to peridiscal reactive inflammation. It coexists with contrast enhancement of the corresponding nerve passing through the fibers of the psoas muscle (**c**, *arrow*), indicating sterile neuritis. Note also contrast enhancement of the corresponding posterior perispinal muscle, e.g. thoracocostalis muscle (**d**, *arrow*)

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DOI 10.1007/978-3-319-09447-2_66, © Springer International Publishing Switzerland 2015

**Fig. 1** (continued)