Imaging of Hematogeneous Pyogenic Spondylodiscitis

Sumer N. Shikhare and Wilfred C. G. Peh

Contents

Abstract

Pyogenic spondylodiscitis (PSD) is an infection of the intervertebral disk and adjacent vertebrae, which may also involve the paravertebral soft tissues. The incidence of PSD is increasing because of the growing number of elderly people and immunocompromised patients. Hematogeneous spread is by far the commonest route of spinal infection, due to the rich arterial supply of the vertebral body. PSD warrants early diagnosis and prompt treatment to prevent morbidity and mortality. Diagnosis is often challenging and requires a high index of clinical suspicion, blood and tissue cultures, appropriate imaging, and/or imaging-guided biopsy to enable an early diagnosis. This chapter aims to provide an overview of PSD, particularly its pathophysiology and imaging features. The roles of different imaging modalities such as radiography, computed tomography,

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magnetic resonance imaging (MRI), and nuclear scintigraphy are discussed, with a focus on MRI as the imaging modality of choice.

Abbreviations

CT	Computed tomography
FS	Fat-suppressed
MRI	Magnetic resonance imaging
STIR	Short-tau inversion recovery
T1-W	T1-weighted
T2-W	T2-weighted

1 Introduction

Pyogenic spondylodiscitis (PSD) is an infection of the spine which affects primarily the intervertebral disk and the adjacent vertebra. The incidence of PSD has been on the rise over the recent few years, due to the increasing life expectancy of older patients and several other factors. The indolent nature of the disease usually contributes to a delay in timely diagnosis and treatment, thus affecting clinical outcome. The diagnosis cannot be established solely on the basis of clinical assessment and almost always requires imaging. Characteristic imaging findings, together with image-guided microbiological isolation of the causative agent, are the main tools for appropriate diagnosis. This chapter discusses various aspects of PSD such as its epidemiology, pathogenesis, and clinical features, with emphasis on the role of various imaging modalities in confirming its diagnosis.

2 Epidemiology

PSD is relatively rare and comprises only 2–7% of all cases of musculoskeletal infections (Tyrrell et al. 1999; Stäbler and Reiser 2001; Cheung and Luk 2012). The reported incidence in developed countries ranges from 4 to 24 per million per year (Lazzeri et al. 2019). The incidence of PSD has been rising in the recent few years, due to improved life expectancy of older patients with chronic debilitating diseases and increased prevalence of chronic liver or renal disease, diabetes mellitus, long-term steroid use, intravenous drug abuse, and human immunodeficiency virus infection. The increase in number of spinal surgeries and improved diagnostic sensitivity, particularly related to the more widespread use of magnetic resonance imaging (MRI), has also contributed to these rising numbers (Kehrer et al. 2014; Nickerson and Sinha 2016; Lazzeri et al. 2019).

PSD can affect any age group but is most frequently seen between the fifth to seventh decades, with a male preponderance (Cheung and Luk 2012; Lazzeri et al. 2019). The most frequent site of vertebral infection is the lumbar spine (50%), followed by the thoracic (35%) and cervical spine (15%) (Cheung and Luk 2012). In approximately 95% of cases, PSD involves the vertebral body, with or without the intervening disk. Involvement of the posterior elements is seen in only 5% of cases (Cheung and Luk 2012). The common organisms causing PSD are *Staphylococcus aureus* including methicillin-resistant *Staphylococcus aureus* (MRSA) and streptococci, accounting for more than 50% of the cases (Cheung and Luk 2012; Boody et al. 2018). Other organisms causing PSD are *Escherichia coli*, *Klebsiella* spp., *Proteus* spp., *Enterobacter* spp., *Salmonella*, and *Pseudomonas aeruginosa* (Fantoni et al. 2012). Moreover, 1 in 10 cases of PSD is polymicrobial, highlighting the importance of blood cultures (Boody et al. 2018).

3 Pathogenesis

PSD commonly results from hematogeneous spread of bacteria from the skin, respiratory tract, gastrointestinal tract, genitourinary tract, or oral cavity (Govender 2005; Skaf et al. 2010). The arterial mode of spread is more common than the venous route (Figs. 1 and 2). This is explained by a rich arterial supply of the vertebral bodies derived from the vertebral arteries, the aorta, or the iliac arteries, depending on vertebral location. In septicemia, microemboli are carried in the arterial system and get impacted in the vertebral end arterioles (Ratcliffe 1985; Leone et al. 2012). PSD typically involves two adjacent vertebral bodies and the intervening disk and is explained by the

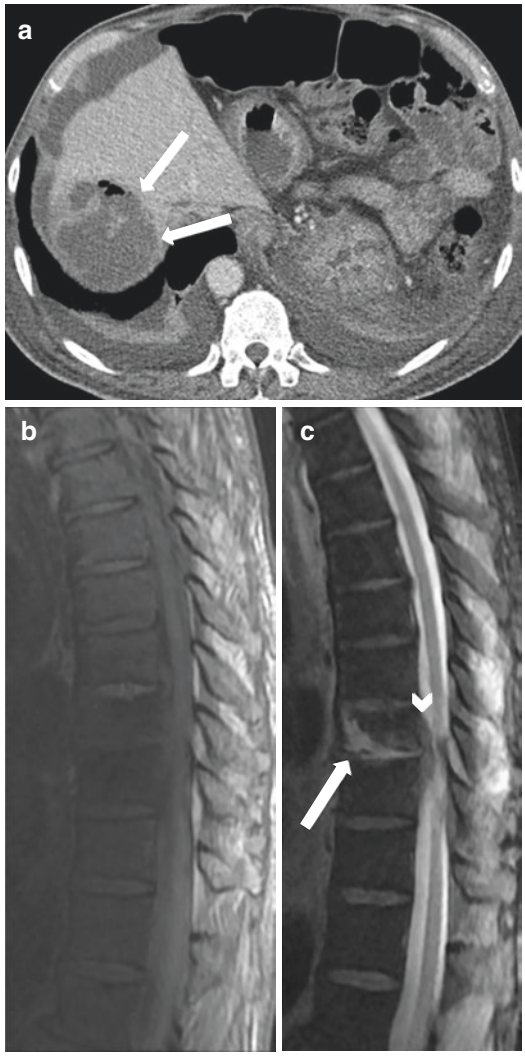

Fig. 1 A 69-year-old man with pyogenic spondylodiscitis due to hematogeneous spread of infection from liver abscess secondary to *Klebsiella pneumoniae*. (**a**) Axial contrast-enhanced CT image of the abdomen shows a large necrotic liver abscess with air locules within (arrows). Two months later, the patient presented with fever and back pain. Sagittal (**b**) T1-W and (**c**) STIR MR images show changes of pyogenic spondylodiscitis involving T8 and T9 vertebral bodies as well as the T8/T9 disk in the form of T8 and T9 vertebral body end-plate irregularity (arrow) and diffuse T1-hypointense signal in the T8 and T9 vertebral bodies with corresponding subtle T2-hyperintense signal. The T8/T9 disk also demonstrates marked T2-hyperintense signal intensity similar to that of fluid. There is an epidural component with resultant T8/T9 anterior epidural space indentation (arrowhead)

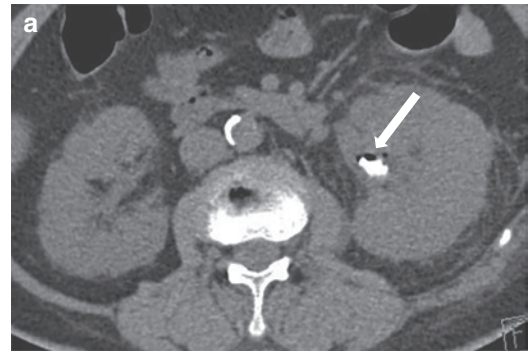

Fig. 2 A 73-year-old woman with pyogenic spondylodiscitis due to hematogeneous spread of infection from left emphysematous pyelonephritis due to *Escherichia coli*. (**a**) Axial unenhanced CT image of the abdomen shows air foci within the left renal pelvis (arrow) with a renal calculus. The left kidney is swollen with perinephric fat stranding. One month later, the patient developed fever and low back pain. Sagittal (**b**) T1-W, (**c**) FS T2-W, and (**d**) contrast-enhanced FS T1-W MR images show typical changes of pyogenic spondylodiscitis involving the L4-L5 vertebral bodies. The intervening disk demonstrates increased signal intensity on STIR images (arrow). There is T1-hypointensity, T2-hyperintensity, and marrow enhancement involving L4-L5 vertebral bodies (star). There is an enhancing epidural component with resultant L4/L5 anterior thecal sac indentation (curved arrow)

arterial supply of the axial skeleton. The same segmental spinal artery bifurcates and supplies the lower portion of the upper vertebra, the upper portion of the adjacent lower vertebra, and the intervening disk (Cheung and Luk 2012). The arterioles enter the vertebral body through central nutrient foramina, undergo ramification, and are most abundant at the vertebral body end plates, particularly in the anterior subchondral region where the infective process usually commences (Wiley and Trueta 1959; Ratcliffe 1985; Leone et al. 2012).

The intervertebral disk is avascular in adults; hence it is usually not directly infected by hematogeneous spread of infection. In elderly patients however, disk degeneration may be followed by ingrowth of vascularized granulation tissue. This secondary vascularization of the disk material may be the reason why primary discitis is possible in older patients (Yeom et al. 2016). As infection worsens, it causes bone infarction, followed by vertebral body and end-plate destruction, and direct extension of infection through the end plates into the intervertebral disk (Fantoni et al.

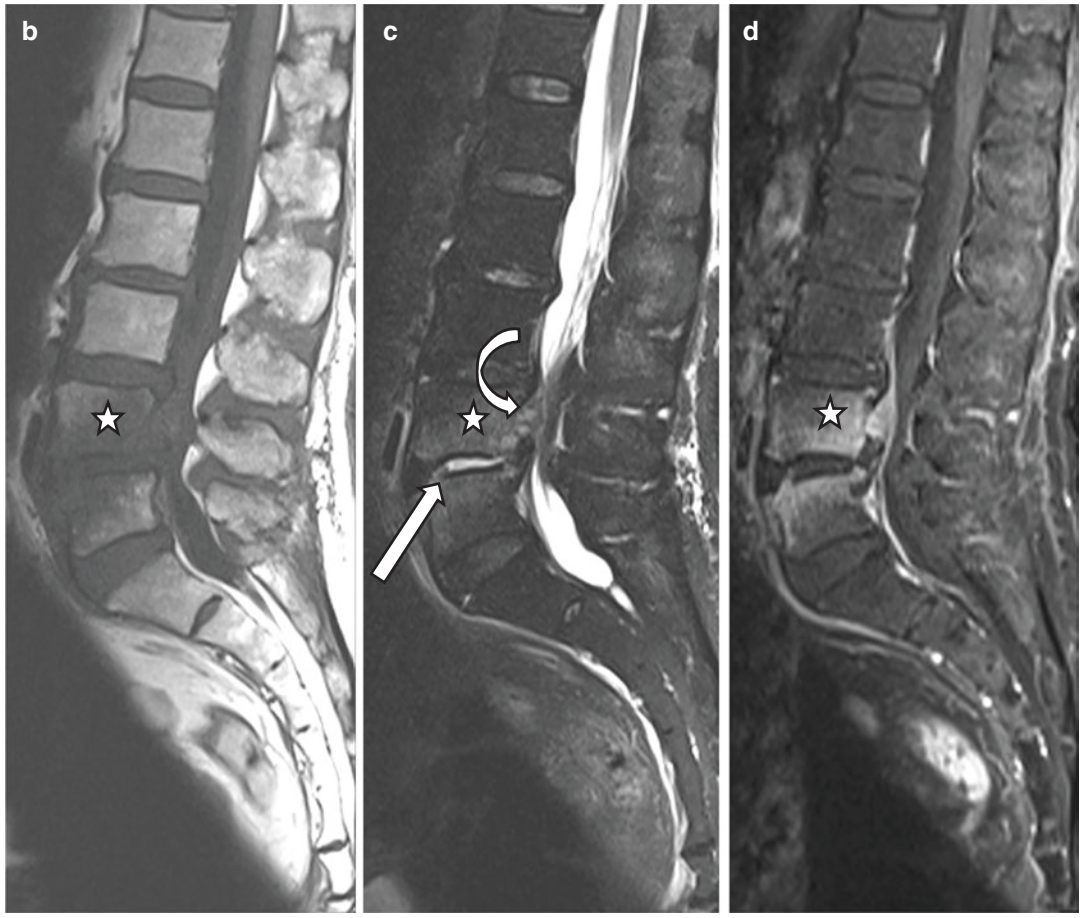

Fig. 2 (continued)

2012). If untreated, the infection may progress and spread into the paravertebral soft tissue and the spinal canal.

Due to their poor blood supply, the posterior elements of the vertebrae such as facet joints are rarely involved by the hematogeneous spread of infection (Babinchak et al. 1997; Yeom et al. 2016). Facet joint infection is an uncommon condition which may result from hematogeneous spread of infection from the skin and respiratory and urinary systems (Narváez et al. 2006; Diehn 2012; Yeom et al. 2016). It may also result from direct inoculation during an interventional procedure, such as therapeutic facet joint injection. When present, facet joint infection is most commonly found in the lumbar spine and usually affects the facet joint unilaterally. Occasionally, when bilateral facet joints are involved, the suggested route of transmission is through the retro-ligamentous space of Okada (Narváez et al. 2006; Diehn 2012; Yeom et al. 2016).

The venous spread of infection, such as via epidural venous plexus within the central canal and Batson paravertebral plexus, is less common. These are a series of valveless veins along the length of the spinal canal. The venous route of spread is of particular importance in instances of sepsis originating in the urinary bladder, bowel, and female pelvic organs. As the Batson plexus is a valveless system, increasing intra-abdominal pressure allows retrograde hematogeneous spread from the pelvis and abdominal organs to the vertebral column, particularly the lumbar spine (Tyrrell et al. 1999; Govender 2005; Cheung and Luk 2012; Tali et al. 2015). In the cervical spine, the infection may spread via the prevertebral pharyngeal venous plexus

in patients with infections in the head and neck region (Wiley and Trueta 1959; Cheung and Luk 2012).

4 Clinical Features

PSD is often an indolent disease with nonspecific symptoms and signs. Hence, the time from onset of symptoms to diagnosis is often long, ranging from 2 to 12 weeks, and on occasion, even 3 months or more (Cheung and Luk 2012). The commonest presenting symptom is unremitting back or neck pain which is present in more than 90% of patients (An and Seldomridge 2006; Cottle and Riordan 2008; Skaf et al. 2010). Fever is less common and occurs in 60–70% of patients (Gasbarrini et al. 2005; An and Seldomridge 2006; Skaf et al. 2010). A nonspecific back pain with no significant fever may be due to a wide list of differential diagnoses, the most common being degenerative spine disease. This symptom does not necessarily prompt spinal imaging, with possible resultant delayed diagnosis, unless there is a high index of suspicion.

Other constitutional symptoms include nausea, vomiting, malaise, anorexia, and weight loss (Cottle and Riordan 2008). Patients with PSD of the cervical spine may present with difficulty in swallowing, secondary to a retropharyngeal abscess (Cheung and Luk 2012). In later stages of PSD, patients may present with limb weakness, numbness, sphincter loss, and paralysis, which may be related to neurological compromise caused by spinal cord or cauda equina compression (An and Seldomridge 2006; Leone et al. 2012). Physical examination may reveal localized paravertebral muscle tenderness and spasm, with a limited range of motion (Cottle and Riordan 2008).

5 Laboratory Investigations

The white blood cell count (WBC) can be normal and may be elevated in less than 50% of cases (Hadjipavlou et al. 2000; Cottle and Riordan 2008). C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR) levels are elevated in more than 90% of patients with spinal infection (Lam and Webb 2004; An and Seldomridge 2006; Cottle and Riordan 2008). Although not pathognomonic, elevated CRP and ESR levels serve as good screening and surveillance tests in the diagnosis and treatment of spinal infections. In suspected cases of PSD, blood cultures, urinalysis, and urine for culture should be obtained. These may be helpful in guiding the choice of antimicrobial therapy (Cheung and Luk 2012). If blood cultures are negative, an attempt should be made to obtain direct cultures using computed tomography (CT)-guided needle biopsy or a surgical biopsy (Skaf et al. 2010). Unless the patient is critically ill, antibiotic therapy should not be started until the appropriate cultures have been performed.

6 Imaging

6.1 Radiography

Radiographs are usually the first imaging study employed in patients with suspected spinal infection. However, radiographs have a very low sensitivity and specificity. Infection usually results in loss of normal bone matrix, causing reduced bone density and osteolysis. In general, osteolysis must compromise 30–40% of bone mineral content to produce noticeable changes on radiographs. These radiographic changes may take at least 2 weeks after acute onset of infection (Gillams et al. 1996; Tali et al. 2015). Thus, a negative study does not rule out an underlying infection. The earliest radiographic sign is ill-defined and irregular borders of the anterior corner of the vertebral body end plate occurring at 2–8 weeks after infection onset. Vertebral body end-plate destruction is considered the most reliable radiographic sign (Tali et al. 2015). This is followed by reduced disk space, vertebral osteolysis, and loss of height of the affected vertebral bodies (Fig. 3a).

As infection progresses, further osteolysis results in vertebral body collapse and wedging, causing a gibbus deformity (Leone et al. 2012). The posterior elements of the vertebral body are uncommonly involved. Paraspinal extension may

be seen radiographically as an abnormal psoas shadow or widening of the mediastinum or retropharyngeal space (An and Seldomridge 2006; Cottle and Riordan 2008). In the later stages of spinal infection, increased bone density due to sclerosis, obliteration of the disk space, ankylosis of the vertebral bodies, and kyphotic or scoliotic deformities may be seen (Leone et al. 2012) (Fig. 3b).

6.2 Computed Tomography

CT is not the modality of choice for diagnosing PSD. However, due to its superior anatomical resolution, CT is more sensitive than spine radiographs in the early detection of loss of disk height, vertebral body end-plate destruction, vertebral osteolysis, and paravertebral soft tissue involvement (Fig. 4). CT can also be useful in identifying other causes of back pain such as degenerative spine disease, fracture, or metastatic disease. Intravenous contrast agent is usually not administered, unless MRI is contraindicated. Compared to radiographs, CT enables better evaluation of paraspinal abscess, seen as a rim-enhancing fluid collection (Raghavan et al. 2018). CT may also be used in the evaluation of facet joint infection and may demonstrate bony destruction at or soft tissue changes around the infected facet joint (Diehn 2012) (Fig. 5a).

Another major application of CT is in guiding percutaneous needle biopsy and abscess drainage. Currently, the modality of choice for

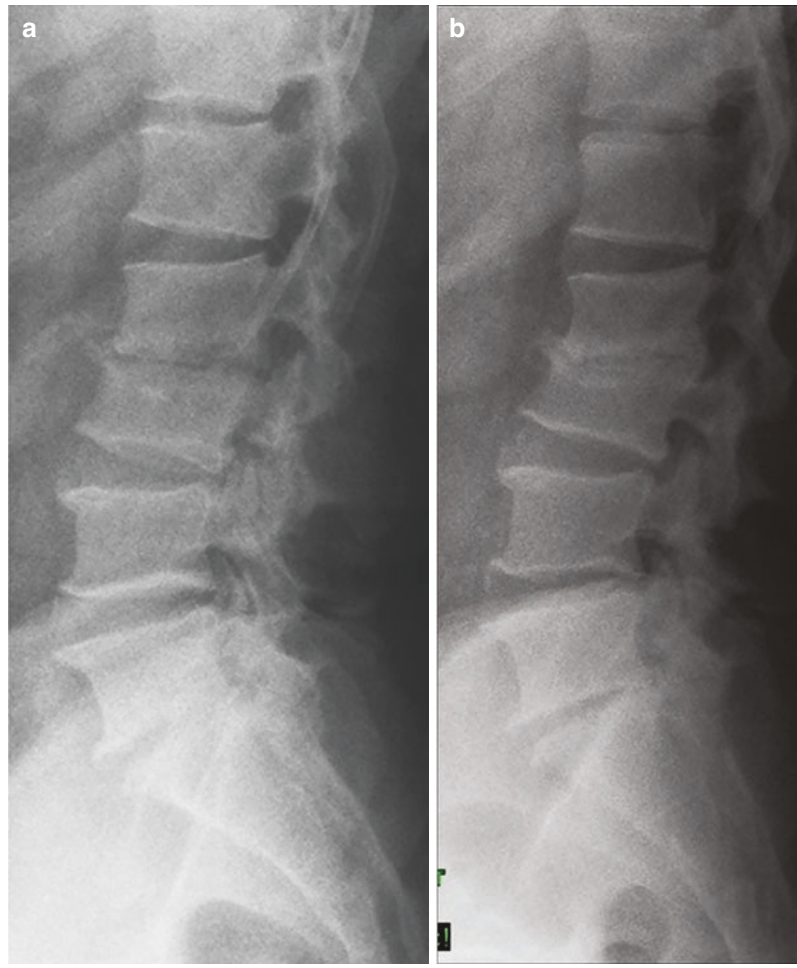

Fig. 3 A 73-year-old man presenting with fever and lower back pain. (**a**) Lateral radiograph of the lumbar spine shows a reduction of the L2/3 disk space with ill-defined irregularities of the adjacent vertebral body end plates and mild loss in height of both L2 and L3 vertebral bodies, indicating early spondylodiscitis. (**b**) Lateral radiograph of the lumbar spine taken 9 months later, following completion of antibiotic treatment, shows chronic changes in the form of obliteration of the L2/L3 disk space with L2 and L3 vertebral body end-plate sclerosis, vertebral body collapse, and mild kyphotic wedging. Causative organism on blood cultures was *Staphylococcus aureus*

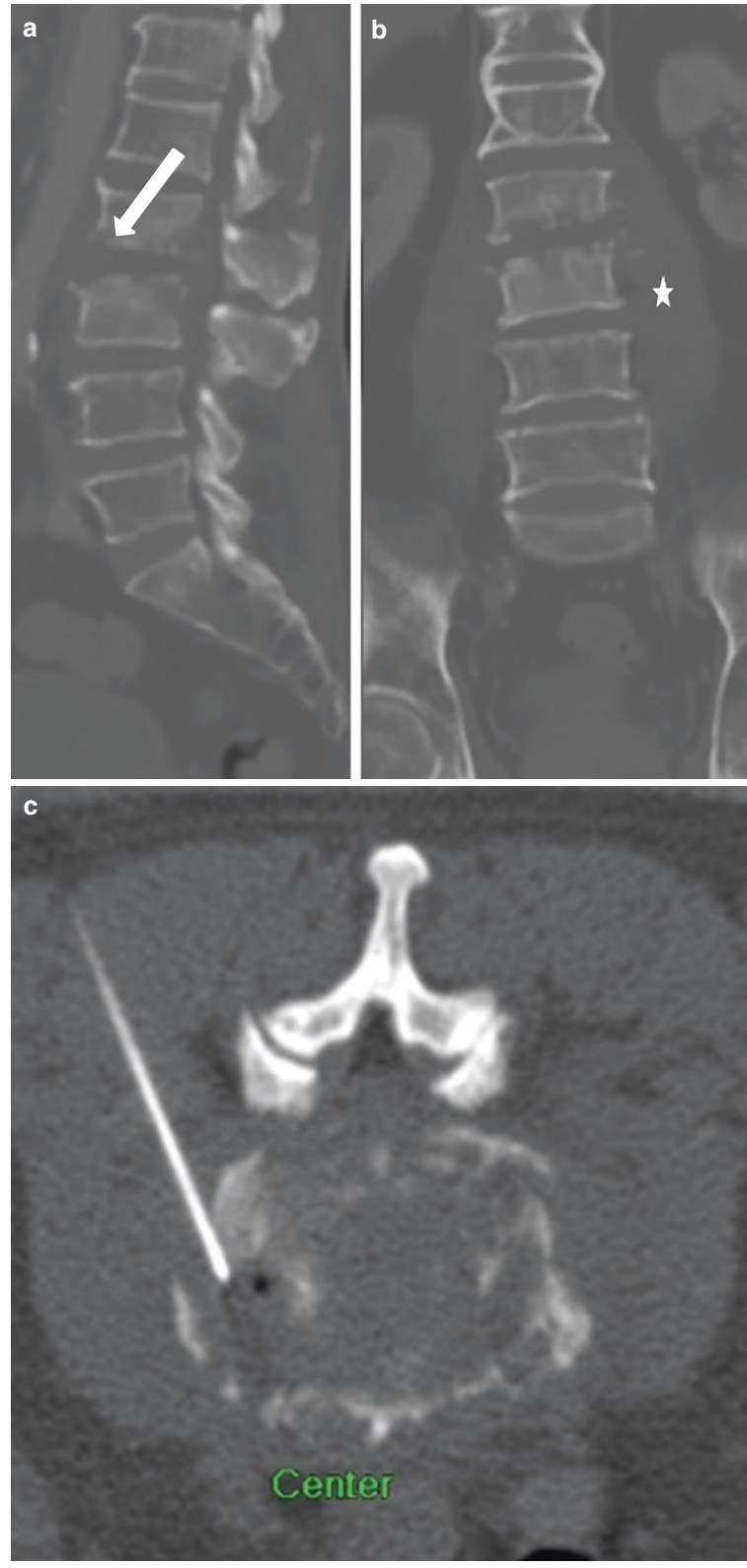

Fig. 4 An 80-year-old woman presenting with constitutional symptoms and back pain of insidious onset. (**a**) Sagittal and (**b**) coronal CT images of the lumbar spine show vertebral body end-plate destruction (arrow) and reduced disk space at L2 and L3 vertebral levels with associated paravertebral soft tissue swelling (star). (**c**) CT-guided percutaneous biopsy was performed and *Escherichia coli* was isolated

performing percutaneous biopsies of the spine is CT fluoroscopy, due to its higher accuracy and repeatability in the event of an inconclusive result (Fig. 4c). Other advantages are patient comfort, ability to be performed as a day procedure, and significantly lower post-procedure complications and patient morbidity compared to open biopsy (Gogna et al. 2008; Srinivasan and Peh 2011). CT-guided percutaneous needle biopsy has better a diagnostic yield than blood cultures, ranging from 70 to 100%; while open biopsies are diagnostic in more than 80% of patients (An et al. 2006; Skaf et al. 2010).

6.3 Magnetic Resonance Imaging

MRI is the modality of choice for the diagnosis and assessment of PSD due to its high sensitivity (96%), specificity (94%), and accuracy (94%). Other advantages are high-contrast resolution, high sensitivity to detect soft tissue and bone marrow abnormalities, multiplanar imaging capability, and lack of ionizing radiation (Varma et al. 2001; Lazzeri et al. 2019; Shikhare and Peh 2019). The major advantage of MRI is its capability to diagnose PSD at an early stage, when other imaging modalities are usually normal (Cheung and Luk 2012; Tali et al. 2015). The protocol should include fat-suppressed (FS) T2-weighted (T2-W) or short-tau inversion recovery (STIR) sequences which are fluid-sensitive sequences that are highly sensitive in demonstrating marrow edema before destructive changes appear; T1-weighted (T1-W) imaging for morphological assessment; and contrast-enhanced FS T1-W sequences to complete a full diagnostic assessment and to differentiate between vascularized and necrotic inflammatory components (Yeom et al. 2016; Lazzeri et al. 2019).

Hematogeneous PSD in adults usually begins near the anterior subchondral region of the vertebral body and subsequently spreads to involve the intervertebral disk and adjacent vertebral body. The infection causes marrow edema which is seen as an area of hypointense signal on T1-W images and hyperintense signal on FS T2-W or STIR images, with corresponding contrast enhancement (Tins and Cassar-Pullicino 2004; Tali et al. 2015; Kawakyu-O'Connor et al. 2016; Lazzeri et al. 2019) (Figs. 2, 6, and 7). When destruction of the vertebral body end plates occurs, there is loss of definition or absence of the normal hypointense rim of cortical bone on T1-W images (Go et al. 2012; Yeom et al. 2016) (Figs. 2 and 7). The infected intervertebral disk may have abnormal hyperintense fluid signal on T2-W images (Figs. 1, 2, 7, and 8). Another reliable finding of spondylodiscitis is the loss of the disk intranuclear cleft on T2-W images (Tali

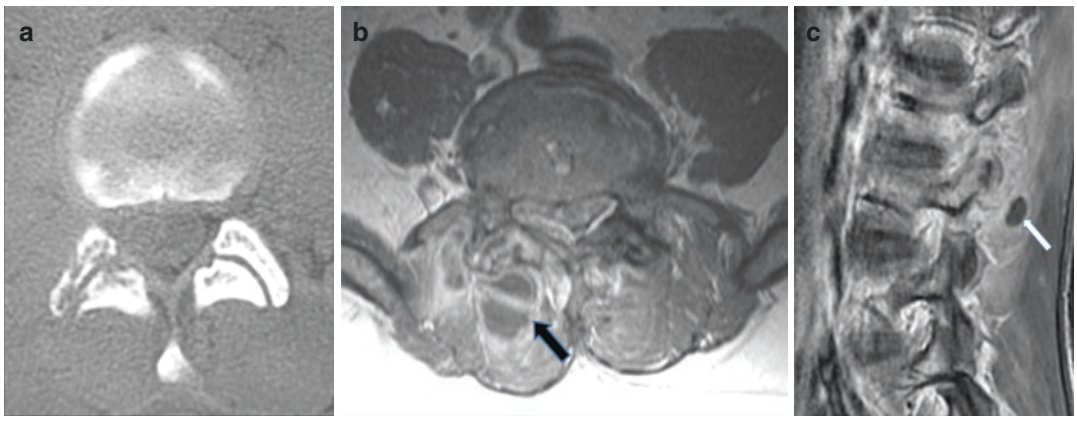

Fig. 5 A 42-year-old man with fever and back pain. (**a**) Axial CT image of the lumbar spine shows destructive subchondral osteolysis of the right facet joint. (**b**) Axial and (**c**) sagittal contrast-enhanced T1-W images confirm subchondral osteolysis involving the right facet joint with intra- and periarticular abscesses (arrows) and extension to the right posterior epidural space. [Case courtesy of Professor Fethi Ladeb, Tunis, Tunisia]

2004) (Figs. 2 and 8). On contrast-enhanced images, the disk may demonstrate homogeneous enhancement, patchy non-confluent areas of enhancement, or thick or thin areas of peripheral enhancement (Hong et al. 2009) (Figs. 2, 6, and 7). The involvement of disk helps differentiate infection from neoplasm, as the disk is usually not affected in neoplasm (Shikhare and Peh 2019). The early changes of PSD may involve only one vertebral body, one vertebral body and one disk, or two vertebral bodies without intervening disk involvement (Varma et al. 2001).

Dagirmanjian et al. (1999) showed that T1-hypointense signal in the vertebral body and disk with associated end-plate changes is a more reliable and consistent finding of PSD than

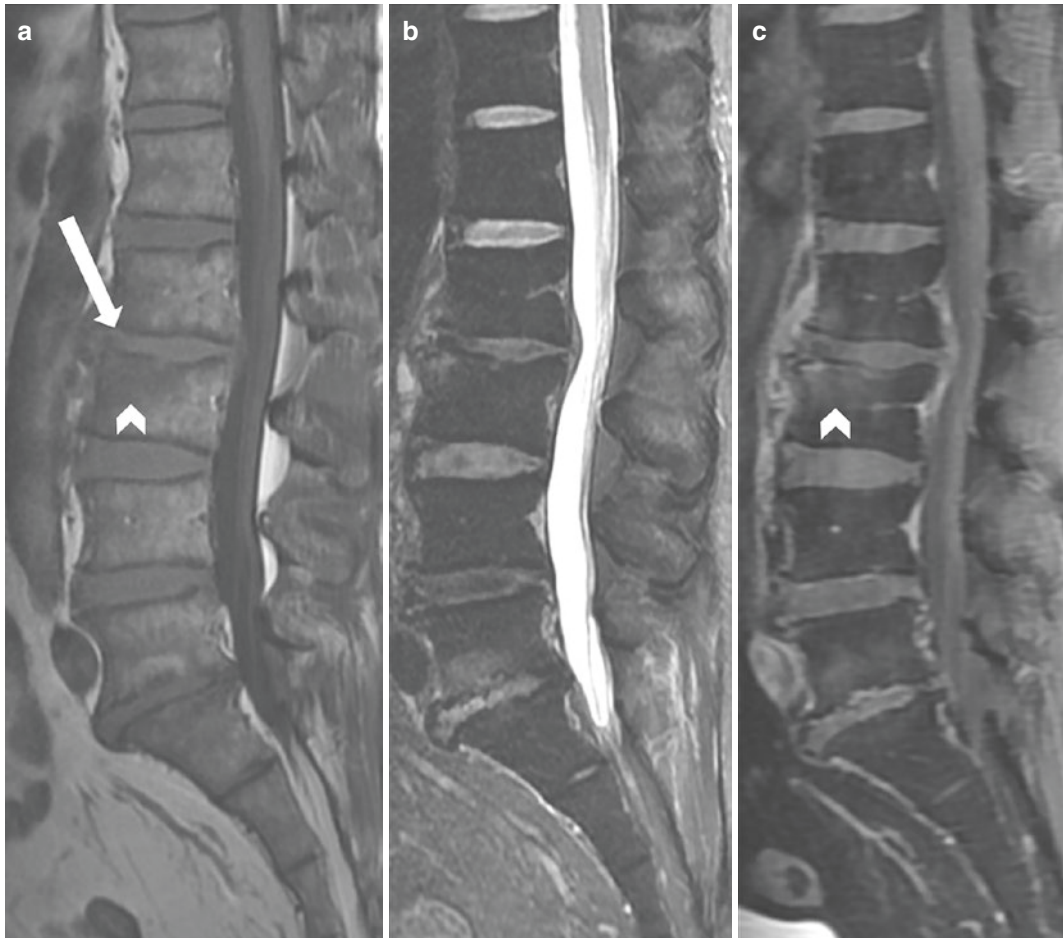

Fig. 6 A 73-year-old woman presenting with constitutional symptoms and back pain of insidious onset. (**a**) Sagittal T1-W, (**b**) FS T2-W, and (**c**) contrast-enhanced FS T1-W images show early changes of pyogenic spondylodiscitis (PSD). There is L2 and L3 vertebral body end-plate irregularity (arrow) and T1-hypointense signal in anterior subchondral regions of L2-L3 vertebral bodies, with corresponding subtle T2-hyperintense signal and contrast enhancement (arrowhead). This case demonstrates a hypointense signal on T1-W images as a more reliable and consistent finding of PS than T2-hyperintense signal. There are Modic-type II vertebral body end-plate changes at L5-S1 level. One month later, (**d**) sagittal T1-W, (**e**) FS T2-W, and (**f**) contrast-enhanced FS T1-W MR images show the progression of PSD involving L2-L3 vertebral bodies, with extensive T1-hypointensity (star), T2-hyperintensity, and marrow enhancement (star). The L2/L3 disk also demonstrates marked T2-hyperintense signal intensity similar to that of fluid (arrow), with patchy enhancement. There is an enhancing epidural component with resultant L2/L3 anterior thecal sac indentation (curved arrow). Causative organism on blood cultures was methicillin-resistant *Staphylococcus aureus*

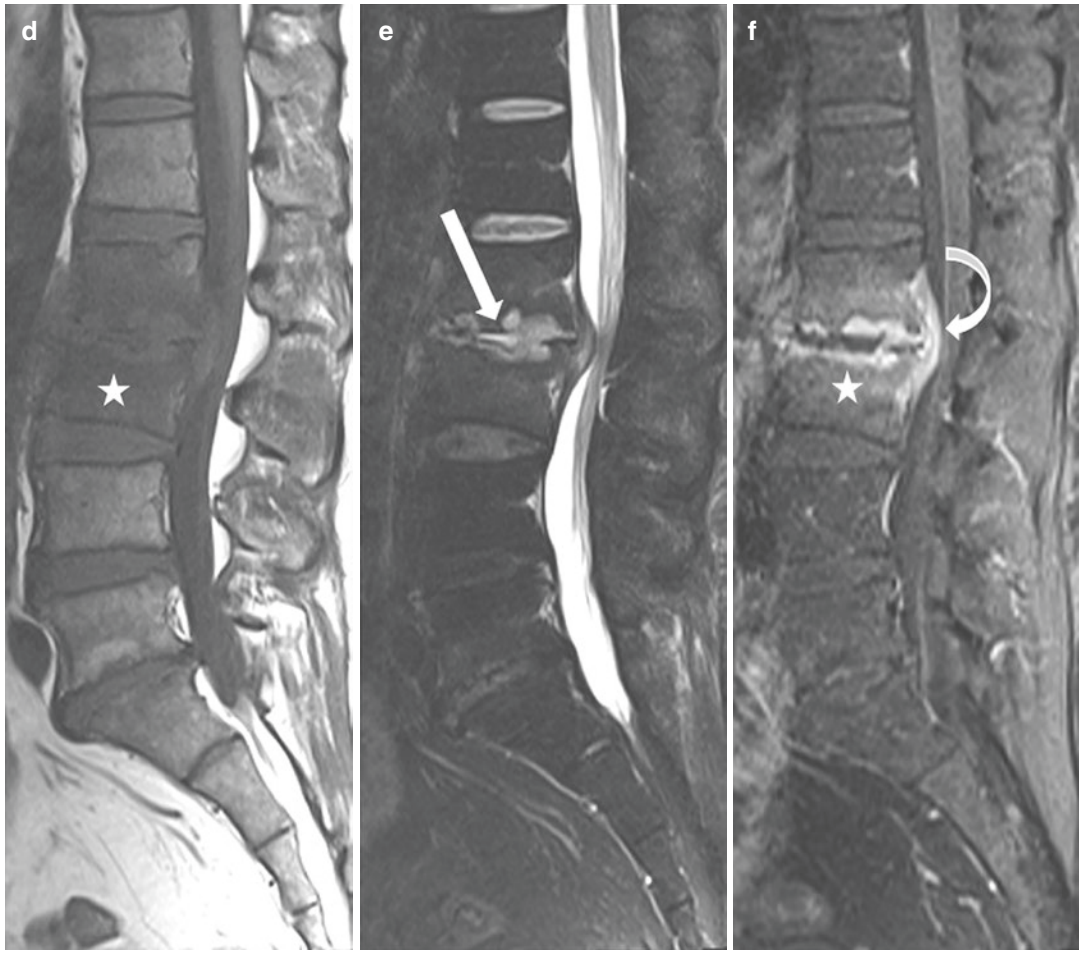

Fig. 6 (continued)

hyperintense signal seen on T2-W images (Fig. 6a, b). Abnormal T2-hyperintense signal may not be that consistent, due to the presence of sclerosis in the vertebral bodies which may cause T2-hypointense signal (Dagirmanjian et al. 1999; Varma et al. 2001). Contrast enhancement of the vertebral body marrow and adjacent intervertebral disk, however, remains a consistent diagnostic finding (Dagirmanjian et al. 1999) (Figs. 2, 6, and 7). Age-related changes in the MRI signal intensity caused by the marrow composition should be considered. Younger patients have predominant red marrow which is relatively hypointense on T1-W images. This may mask marrow edema-related hypointense signal, which is more apparent in elderly patients due to hyperintense signal from predominant fatty marrow (Dagirmanjian et al. 1996). Radiologists should be aware of these diagnostic pitfalls.

Contrast-enhanced FS T1-W sequences are also useful in assessing paravertebral space involvement (Leone et al. 2012; Shikhare and Peh 2019). In early spondylodiscitis, enhancement of paravertebral soft tissue may be the only MRI finding in the absence of vertebral body end-plate changes (DeSanto and Ross 2011; Yeom et al. 2016). Paravertebral soft tissue may be involved in the form of either a phlegmon or an abscess. Phlegmons are seen as poorly defined areas of signal hyperintensity on T2-W images with diffuse contrast enhancement, in contrast to abscesses which demonstrate hyperintense signal intensity on T2-W images with thick irregular rim-like enhancement on FS T1-W images (An

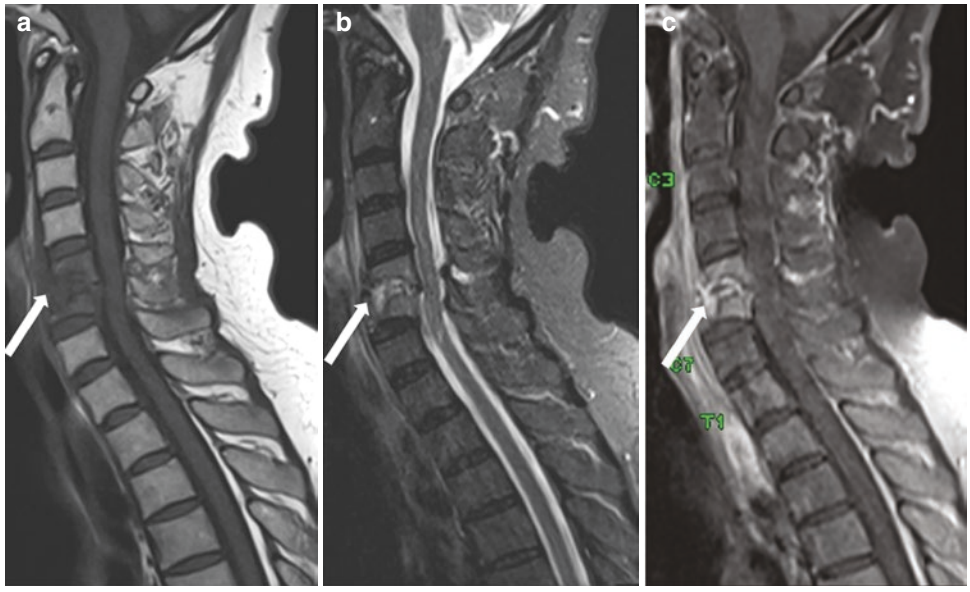

Fig. 7 A 52-year-old man presenting with fever and neck pain. Sagittal (**a**) T1-W, (**b**) STIR, and (**c**) contrast-enhanced FS T1-W MR images show vertebral body endplate destruction with T1-hypointensity, T2-hyperintensity, and marked enhancement involving C5 and C6 vertebral bodies. The intervening C5/C6 disk also shows marked T2-hyperintensity similar to that of fluid, with heterogeneous contrast enhancement (arrow). Causative organism on blood cultures was *Staphylococcus aureus*

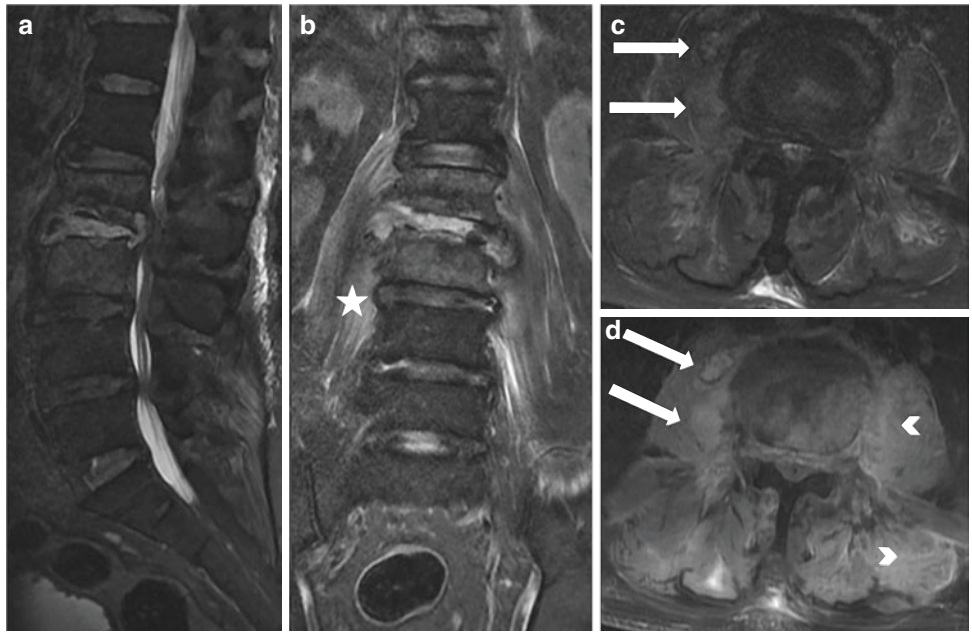

Fig. 8 An 80-year-old woman presenting with fever and severe low back pain. (**a**) Sagittal, (**b**) coronal, and (**c**) axial FS T2-W MR images show changes of pyogenic spondylodiscitis involving the L2 and L3 vertebral bodies with T2 hyperintense signal within both the psoas muscles (star). The L2/L3 disk also shows marked T2-hyperintensity similar to that of fluid. (**d**) Axial contrast-enhanced FS T1-W image shows corresponding pre- and paravertebral soft tissue enhancement with phlegmons (arrowheads) and rim-enhancing abscesses (arrows) in the psoas muscles, as well as the erector spinae and multifidus muscles. CT-guided percutaneous biopsy was performed, and *Escherichia coli* was isolated

et al. 2006) (Fig. 8). In facet joint infection, MRI may demonstrate destruction of the subchondral bone surface of the facet joint, fluid-filled facet joint with surrounding edema, and facet capsular enhancement (Diehn 2012). Associated periarticular abscesses and epidural extension are also well shown (Fig. 5b, c).

Imaging features of PSD may mimic tuberculous (TB) spondylodiscitis in approximately 75% of cases (Shikhare et al. 2011). MRI features which favor TB spondylodiscitis and help differentiate it from PSD include relative preservation of the intervertebral disk, multiple vertebral or entire vertebral body involvement, skip lesions with subligamentous spread of infection involving three or more vertebral levels (Fig. 9), well-defined abnormal signal in the paraspinal region, thin and smooth-walled abscess, presence of paraspinal or intraspinal abscess, and thoracic spine involvement (Smith et al. 1989; Jung et al. 2004; Harada et al. 2008).

MRI may also be used as a tool in assessing the therapeutic response and to help guide clinical decision-making (Hong et al. 2009). However, this can sometimes be challenging as MRI findings can lag behind the clinical picture by 4–8 weeks, or even months, after the initiation of antibiotic therapy. Hence, MRI is not a

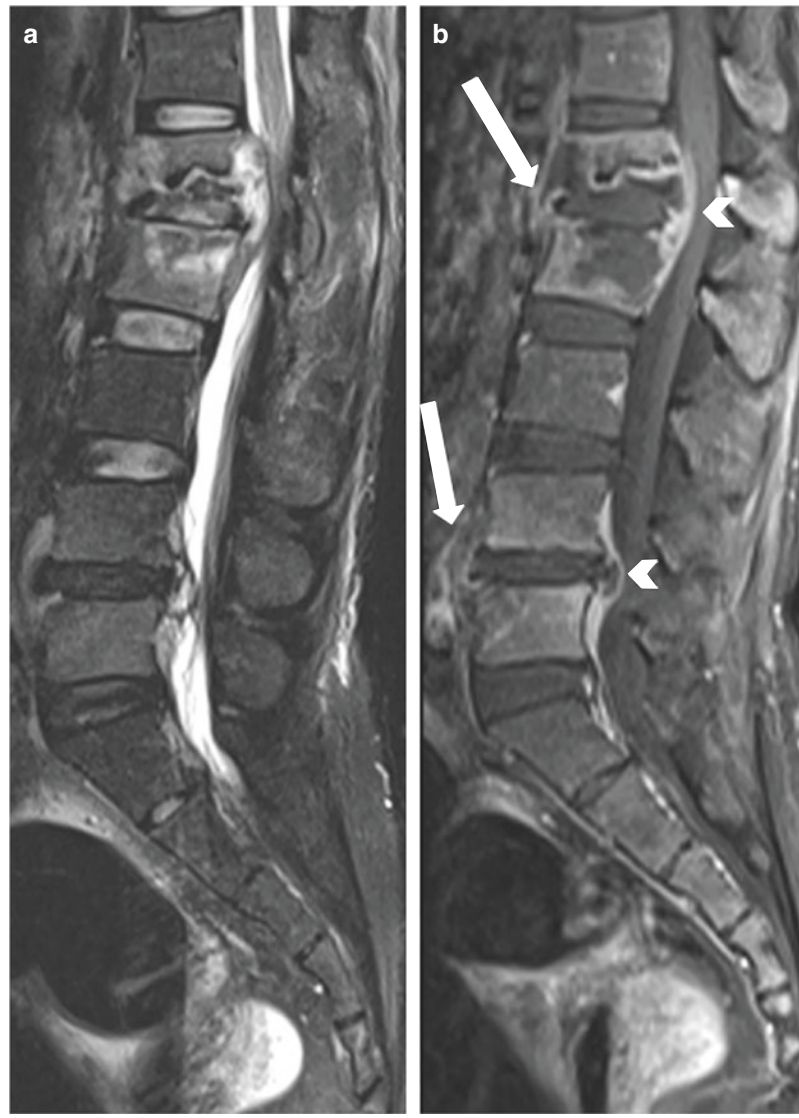

Fig. 9 Tuberculous spondylodiscitis in a 68-year-old man presenting with evening fever, chills, weight loss, and severe back pain. Sagittal (**a**) T2-W and (**b**) contrast-enhanced FS T1-W MR images show involvement of multiple vertebral bodies (L1-L2 and L4-L5). There are skip lesions with a subligamentous spread of infection (arrow) and epidural extension at both levels (arrowhead). Both L1/L2 and L4/L5 disks are relatively preserved

fully reliable technique in evaluating treatment response. The early marker of healing at follow-up MRI is the reduction in contrast enhancement, which may be seen few weeks to months after the initiation of treatment. Other signs of healing include fatty marrow restoration, seen as reinstitution of hyperintense signal in the bone marrow on T1-W images and corresponding decrease of hyperintense marrow signal on STIR or T2-W images (Fig. 10), as well as resolution of paravertebral soft tissue changes. The disk space, however, may appear persistently narrowed on follow-up imaging (Tins and Cassar-Pullicino 2004; Leone et al. 2012; Tali et al. 2015) (Fig. 10).

6.4 Nuclear Medicine Imaging

Radionuclide studies are more sensitive than radiographs in detecting early infection. Radiopharmaceuticals commonly used in the assessment of spinal infection include technetium-99m-labeled methylene diphosphonate (99mTc-MDP), gallium-67 citrate, and fluorine-18 [18F] fluoro-deoxyglucose positron

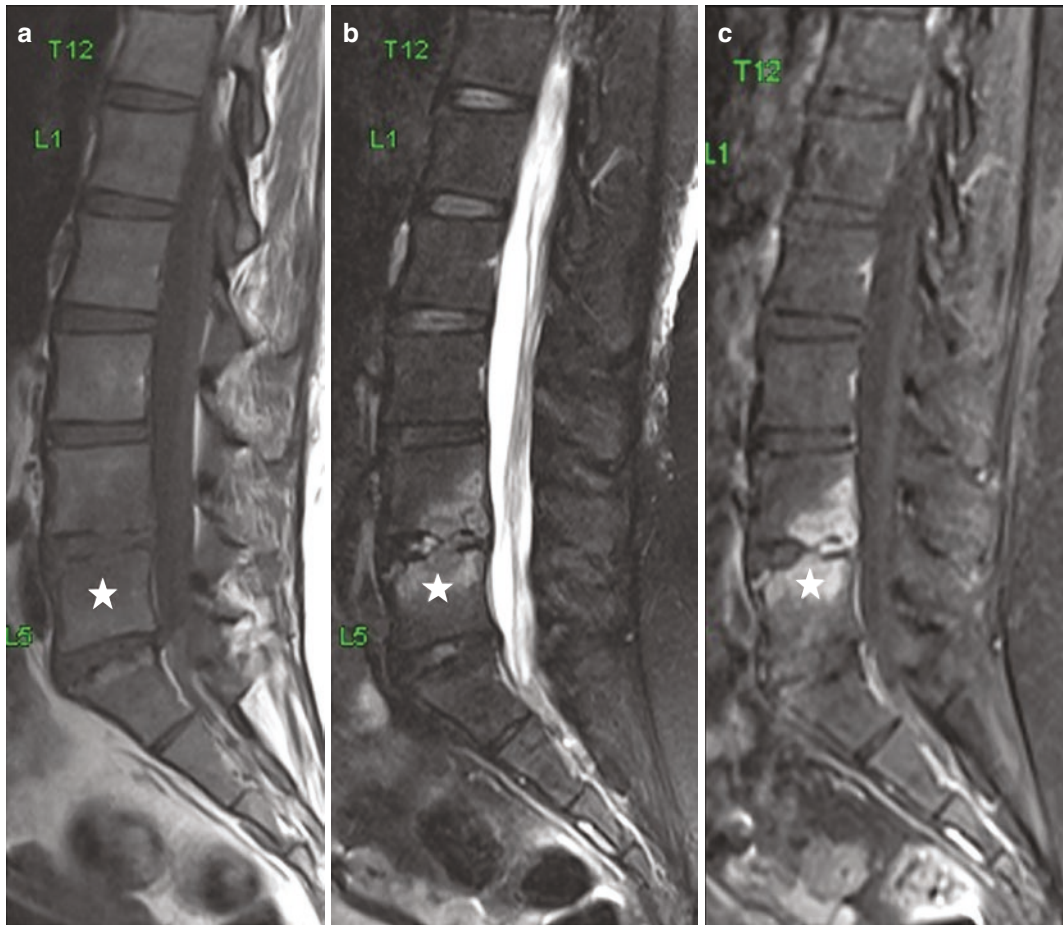

Fig. 10 A 72-year-old woman presenting with fever and back pain. Sagittal (**a**) T1-W, (**b**) FS T2-W, and (**c**) contrast-enhanced FS T1-W MR images show typical appearances of pyogenic spondylodiscitis at L4-L5 level (star) secondary to *Staphylococcus aureus*. The patient was treated with antibiotics. Ten months later, corresponding sagittal (**d**) T1-W, (**e**) FS T2-W, and (**f**) contrast-enhanced FS T1-W MR images show features of healing in the L4 and L5 vertebral bodies with fatty marrow restoration, seen as reinstitution of normal fatty signal in the bone marrow on T1-W (star) and T2-W images. There is no significant enhancement. The previously seen abnormal T2-hyperintense L4/L5 disk signal has resolved but the disk remains slightly narrowed (arrowhead)

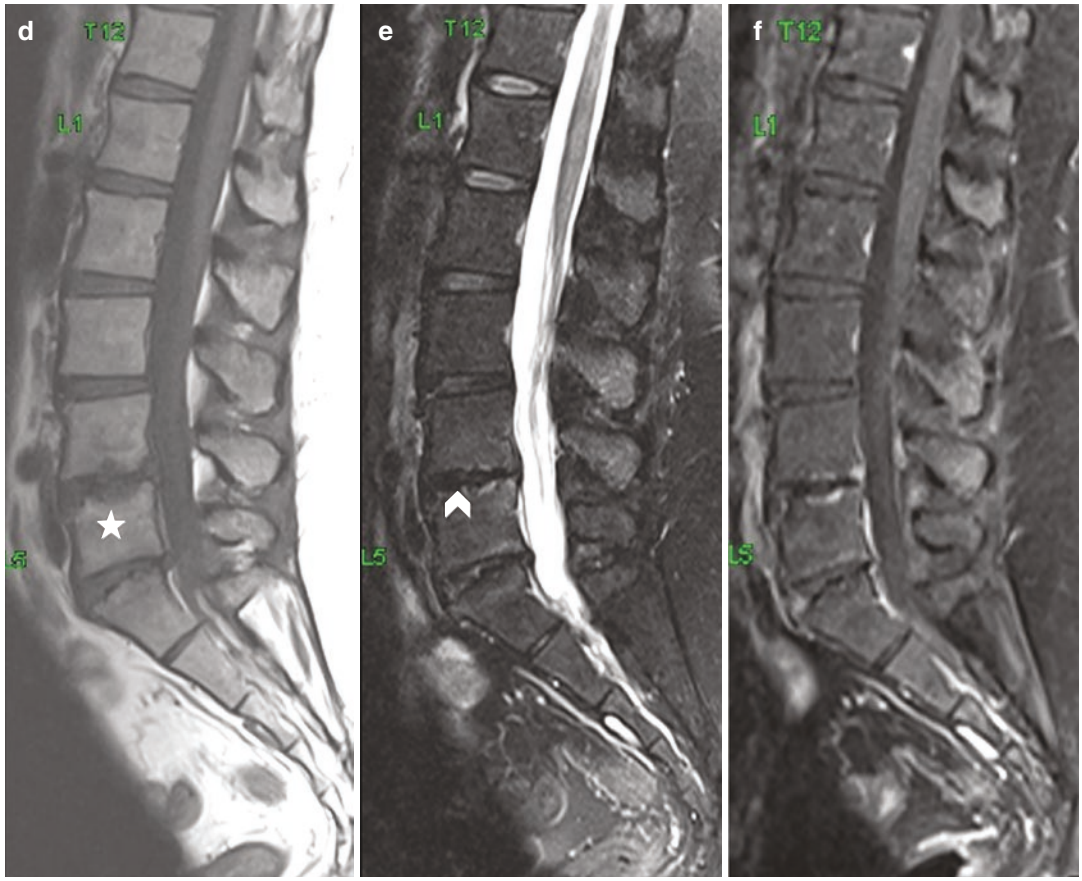

Fig. 10 (continued)

emission tomography (FDG-PET) (Lazzeri et al. 2019). Three-phase 99mTc-MDP scan has a sensitivity of 87–98% and a specificity of 91–100%, whereas the sensitivity and specificity of gallium-67 citrate bone scans is 89% and 85%, respectively, for diagnosing spinal infection (Govender 2005; Cheung and Luk 2012). Radionuclide studies lack specificity, as they may show increased activity in other conditions such as degenerative disk disease, osteoporotic fractures, and neoplasms. Additionally, bone scintiscans provide poor anatomical detail and may show persistent increased activity, even after the vertebral infection has healed (Tali et al. 2015; Shikhare and Peh 2019).

The diagnostic accuracy can be increased by using combination of technetium and gallium radionuclides, with sensitivity rates of up to 94% (Tali et al. 2015). Gallium scan is a better tool for follow-up treatment response compared to technetium, as they provide a more accurate degree of the infectious activity (Go et al. 2012; Tali et al. 2015). FDG-PET has a reported sensitivity of 97% and specificity of 88% for the diagnosis of PSD (Prodromou et al. 2014). FDG-PET combined with CT provides better anatomical details (Lazzeri et al. 2019). The advantages of FDG-PET/CT include its high negative predictive value, short acquisition time, and superior image quality. Drawbacks are its low specificity (ranging from 35 to 88%) and failure to consistently differentiate infection from marked degenerative spine disease and neoplastic lesions (Rosen et al. 2006).

7 Treatment

PSD is usually treated conservatively using antimicrobial therapy and nonpharmacological treatments such as bed rest and physiotherapy

(Gouliouris et al. 2010) (Fig. 10). Patients are often started on empirical antibiotic therapy, covering *S. aureus*, gram-negative organisms, and anaerobes, while microbiological results are still pending (Gouliouris et al. 2010). Afterwards, specific antibiotic treatment is continued intravenously, once an organism has been isolated. The recommended duration of intravenous antibiotics is 6–8 weeks, aiming to reduce failure and recurrence rates (Cheung and Luk 2012). Surgical intervention is usually indicated in cases of failed response to conservative management, intractable pain, neurological complications, and chronic infection causing extensive bone destruction leading to spinal instability or severe kyphotic deformity (Gouliouris et al. 2010). Surgical management usually includes aggressive radical debridement and decompression, along with anterior fusion for spinal stabilization (Pola et al. 2012) (Fig. 11).

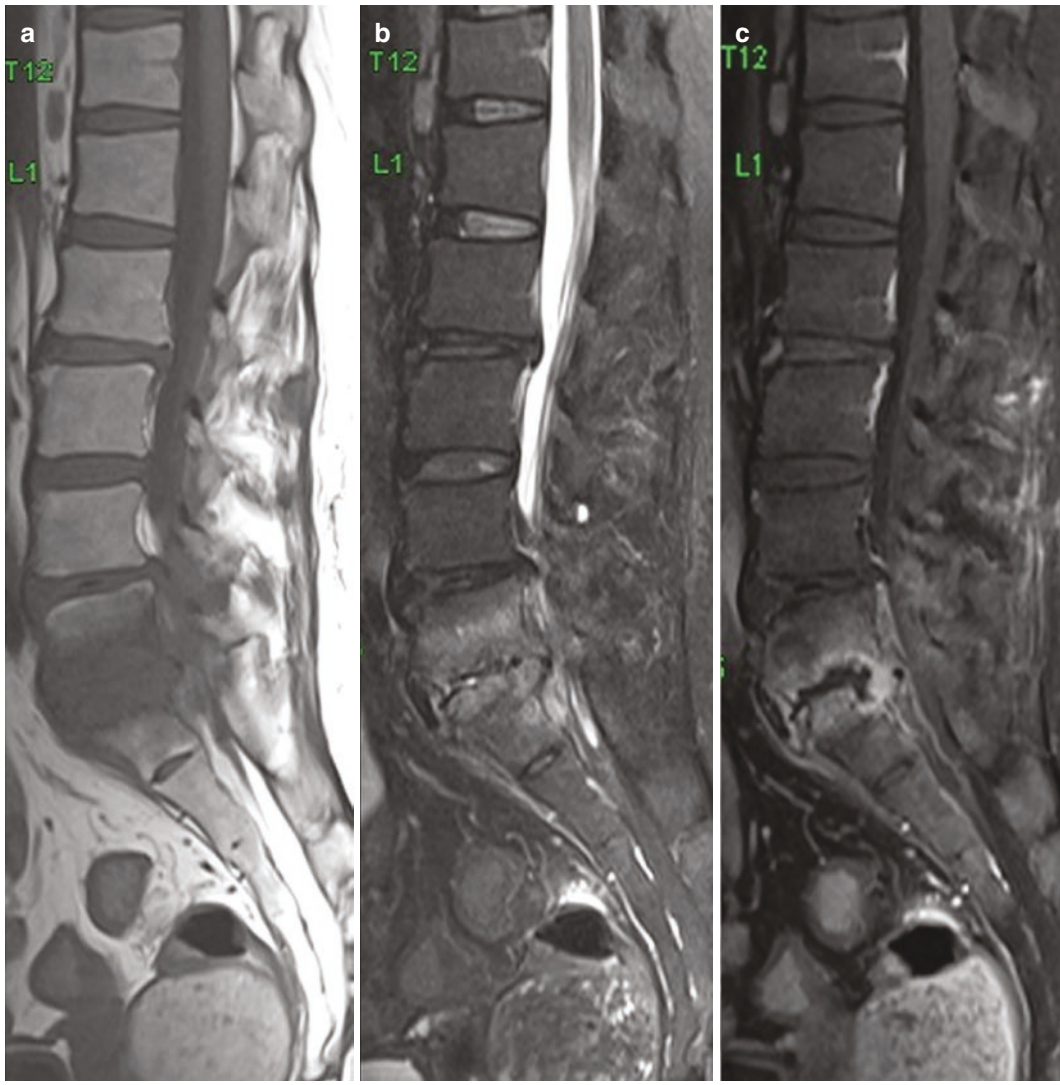

Fig. 11 A 71-year-old woman presenting with fever and back pain. Sagittal (**a**) T1-W, (**b**) FS T2-W, and (**c**) contrast-enhanced FS T1-W MR images show typical appearances of pyogenic spondylodiscitis at L5-S1 level secondary to methicillin-resistant *Staphylococcus aureus*. Patient failed conservative management and developed neurological complications, hence underwent surgery (laminectomy at L4-L5 level). Three years later, sagittal (**d**) T1-W and (**e**) STIR MR images show features of healing and bony ankylosis at L5-S1 level

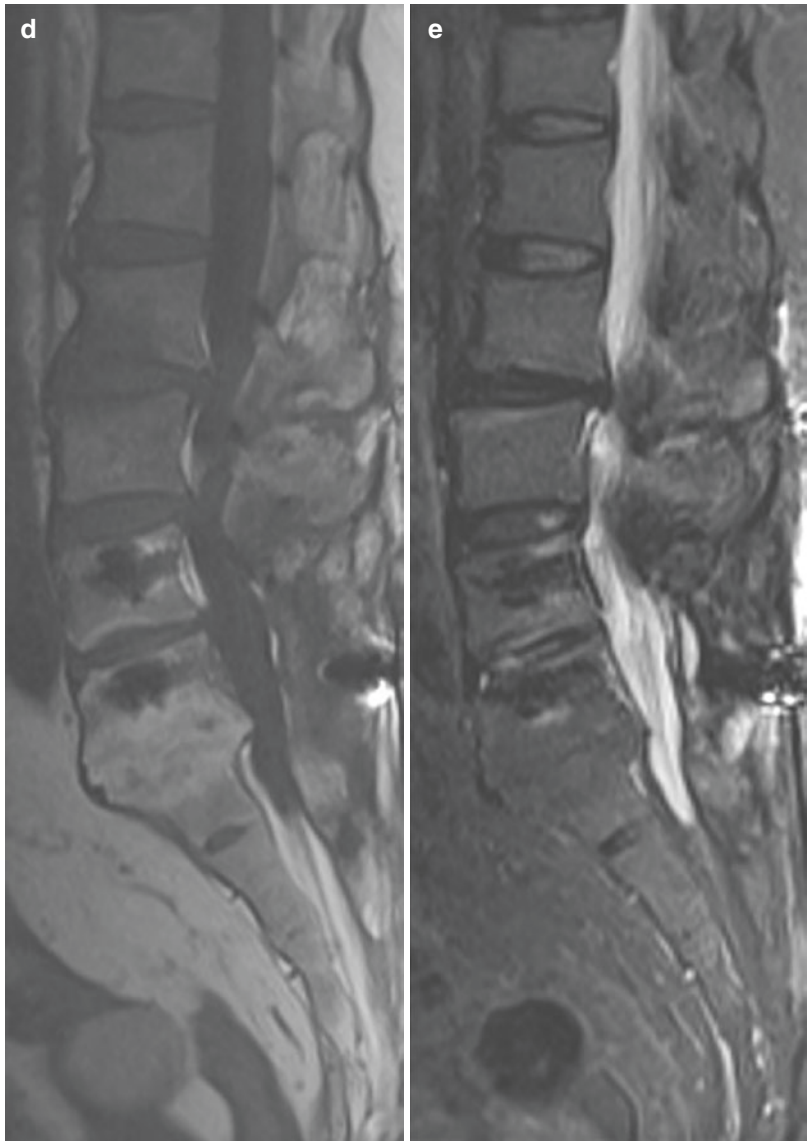

Fig. 11 (continued)

8 Conclusion

The incidence of PSD has been on the rise over the recent few years. Early diagnosis requires a high degree of clinical suspicion and knowledge of early imaging features, with the aim of preventing morbidity and mortality. The use of multiple imaging modalities has led to a greater sensitivity and specificity in the diagnosis of PSD. Radiologists thus play a crucial role and should be aware of varied imaging manifestations of PSD, particularly the MRI features. Currently, MRI is the modality of choice for diagnosing PSD at an early stage and to differentiate PSD from other conditions such as neoplasms. MRI is also useful for defining the extent of disease process and complications.

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