Review Articles/บทฟื้นฟูวิชาการ

Imaging of Infectious Spondylitis

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Abstract

Infectious spondylitis is a common disease and increasing in incidence, which may be a primary spinal infection or secondary to a systemic infection. Because many spinal infections are life-threatening, early diagnosis leads to improved outcomes. Nowadays, imaging plays an important role in the diagnosis and follow up of these potentially life-threatening conditions. MR is the imaging modality of choice for *patients* with suspected spinal infection. Common causes of infectious spondylitis that were focus on this review article are pyogenic and tuberculous spondylitis. Pyogenic spondylodiscitis should be considered, when there is increased T2- weighted signal and/or enhancement in the disc and subchondral regions of adjacent vertebral bodies. Spinal epidural and subdural abscesses generally accompany pyogenic spondylodiscitis. MRI is critical in diagnosing both spinal epidural and subdural abscesses, which need to be treated with emergent surgery. Tuberculous spondylitis shows characteristic imaging findings including disc space sparing, subligamentous spread, and large paraspinal abscesses. Fungal spondylitis is a rare disease found in immunocompromised patients and has a nonspecific imaging appearance. Therefore, the diagnosis of infectious spondylitis should be made of radiographic abnormality with clinical correlation.

Keywords: Spinal infection, Spondylitis, Spondylodiscitis

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Introduction

Infectious spondylitis is a common disease, which may be a primary disease starting locally as a spinal infection or secondary to a systemic infection. Because many spinal infections are life-threatening, early diagnosis leads to improved outcomes. Therefore, imaging is increasingly used and plays an important role in the diagnosis, management, and follow up. The focus of this review article is on pyogenic spondylitis and tuberculous spondylitis, which are common spinal infections in the routine clinical practices. Nowadays, magnetic resonance imaging (MRI) is the diagnostic method of choice for spinal infection due to its high sensitivity and specificity. Here, we also emphasize the MRI findings in each different type of spinal infection including clues in differential diagnosis which allow the radiologist to distinguish them from other diseases.

Pyogenic spondylodiscitis

Pyogenic spondylodiscitis is caused by bacterial infection of the bony spinal column, the intervertebral discs, and/or extradural/intradural space. The most common cause of pyogenic spondylodiscitis is hematogenous spread of infection from a remote site. Urinary tract infection is the most common source. The infection typically spreads via an arterial route, although the paravertebral venous plexus. Other etiologies include direct inoculation in a surgical setting, discography, and therapeutic spinal injections as well as contiguous spread from adjacent infected sites[1,2,3]. Staphylococcus aureus is the most common causative organism, found in approximately one-third of all patients[4,5]. Peak incidences are in the sixth and seventh decades of life. Risk factors include age older than 50 years, diabetes, underlying chronic disease such as renal failure and cirrhosis, AIDS and other immunocompromised hosts, and chronic steroid use and intravenous drug use. Diabetic patients, particularly in their 50s and 60s are frequently affected, and overall spinal infections are more common in men. Patients with pyogenic spondylodiscitis typically present with fever, back pain, and associated muscle spasm with tenderness to palpation. Limited range of motion and/or rapidly progressive neurologic deficits can also be present. However, patients may present with subtle, insidious, and nonspecific symptoms such as malaise and weight loss. The time course ranges from acute to chronic. Laboratory evaluation is not reliable, although elevated erythrocyte sedimentation rate (ESR) and elevated C-reactive protein (CRP) are often present. Leukocytosis is less common[6–8].

The lumbar spine is most frequently affected (about 45% of cases), followed by thoracic spine (35% of cases), while the cervical spine is the least common site (about 15% of cases)[1-5,7]. The intervertebral discs in the adults are nearly avascular, due to involution of the rich network of intraosseous anastomoses and the profuse capillary network at the vertebral margins of the discs during adolescence. Only the cancellous bone adjacent to the cartilagenous endplate is highly vascularized in adults and is prone to blood borne organisms. In most patients the infection is limited to the disc and adjacent vertebral bodies. Multiple levels and/or skip lesions are also common[1-5]. The most widely accepted pathophysiologic hypothesis is that infected emboli cause infarction and subsequent infection in the metaphysis of vertebral bodies. Infection typically begins in the anterior aspect of the vertebral bodies and spreads early to the adjacent discs[1-7]. The remainder of vertebral bodies and opposite vertebral endplates are also involved late in the

course of the disease. Spread of infection into the paraspinal and/or epidural spaces is also common. Infection may extend deeply into the subdural or subarachnoid spaces causing subdural abscess or arachnoiditis. In contrast to adults, the primary site of infection in children is the disc due to its rich vascular supply around that allows the infection to spread directly to the disc. Thus, isolated discitis can occur in children[1-7,18].

Imaging Evaluation

In current routine clinical practice, plain radiographs are usually the first imaging modality in patients with back pain. However, plain radiographs are not sensitive for spondylodiscitis, particularly in the early phase of the disease. Radiographic findings are usually subtle and may not appear until late in the process. Therefore, the hallmark radiographic findings of spondylodiscitis (Fig. 1) such as disc space narrowing, endplate irregularity/destruction, and associated paraspinal soft tissue bulging typically lag behind the clinical evolution, and may not be present until several days or weeks after initial infection occurred. In addition, plain radiographs cannot demonstrate the intraspinal complications of spondylodiscitis, especially epidural abscess, arachnoiditis or any other lesion that results in mass effect on the spinal cord/nerve roots[4]. In the chronic stage of spondylodiscitis, findings such as endplate sclerosis and bony ankylosis can be seen. Regarding CT scan, it is more sensitive than plain film but also has limited evaluation of soft tissue changes as well as any abnormality involving the spinal cord and nerve roots.

Currently, MR is the imaging of choice for spine infection due to high sensitivity and specificity to detect spinal infections early in their course. The sensitivity of MRI for spondylodiscitis is comparable to that of radionuclide studies, but its specificity is much higher. Even in the early process of spondylitis, MRI shows a sensitivity of 96%, a specificity of 92% and accuracy of 92%[9,10]. Regarding the MRI protocol, sagittal MR images of the entire spinal axis are recommended to identify the extent of infection and possible skip lesions. In the presence of a spinal infection, there is increased T2 signal intensity in the vertebral marrow due to edema, whereas T1-weighted signal intensity is decreased due to replacement of marrow fat by edema. Gadolinium enhancement is easier seen with fat suppressed T1-weighted images and helps to delineate intraosseous infection (Fig.1 and 2)[11,12]. Therefore, fat-suppressed T2- weighted and fat-suppressed postgadolinium T1-weighted sequences are useful and should be performed in every case of spinal infection[7-8]. Pathologically, the infection typically begins in the anterior metaphyseal region of the vertebral body and then spreads to involve the disc space and adjacent vertebral body. Therefore, bone marrow signal changes typically begin at the vertebral endplates adjacent to the infected disc and appears as T1 hypointensity and T2 hyperintensity with contrast enhancement in the subchondral bone marrow. Unfortunately, this change is nonspecific and can also be seen in some degenerative endplate changes and in many other noninfectious conditions. Endplate erosion and vertebral body destruction are commonly evident. Disc involvement typically manifests as increased T2 signal which is the most reliable sign of discitis[9]. Other MRI findings of discitis include loss of the normal intradiscal band of decreased T2 signal that represents internuclear cleft, disc height loss, and disc enhancement (Fig. 1 and 2). Associated paraspinal soft tissue involvement usually presents as either solid appearing enhancing soft tissues typical of

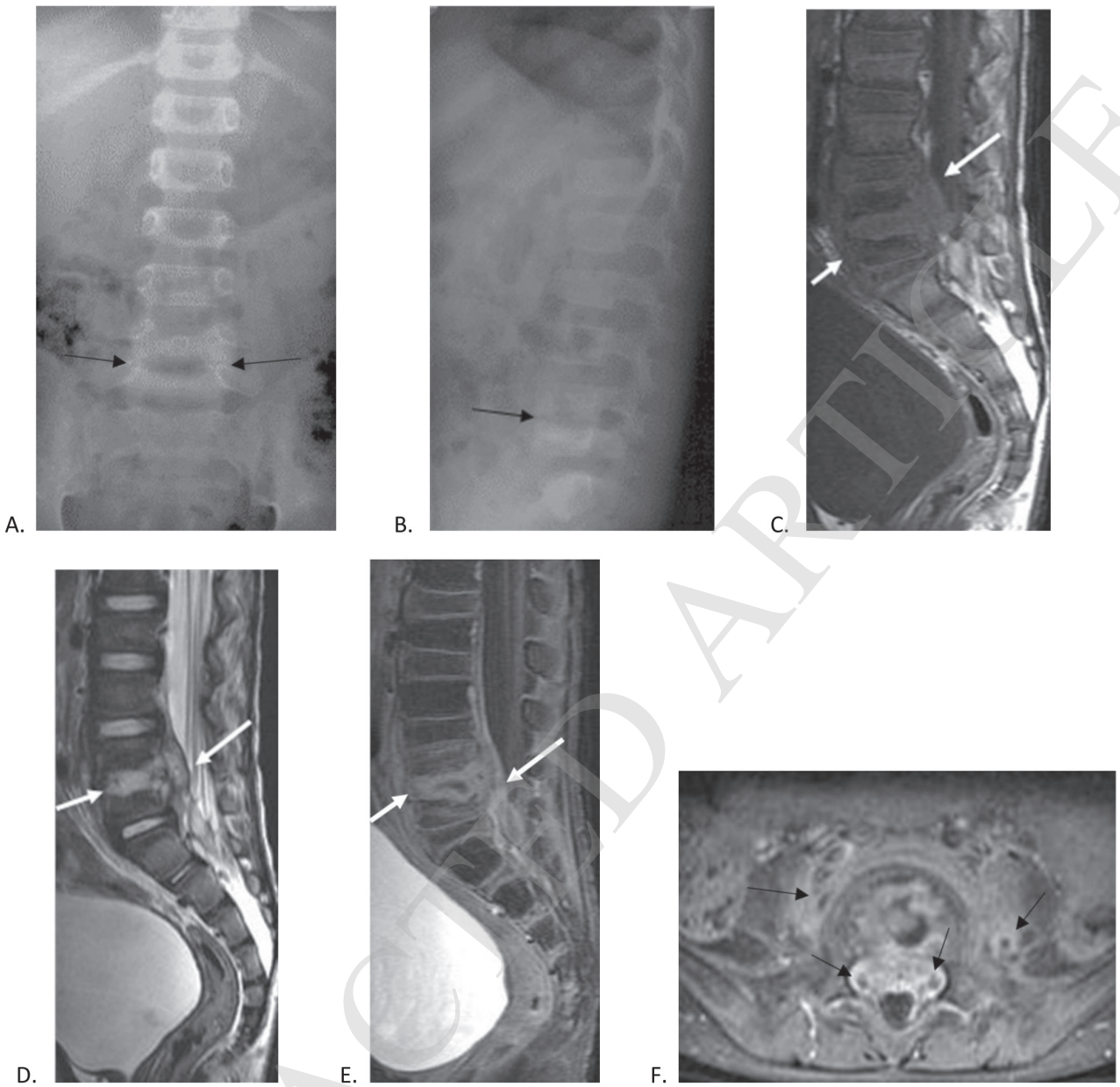

Figure 1 *Pyogenic spondylodiscitis. A 5-year-old boy, known case of acute nonlymphocytic leukemia (ANLL) post bone marrow transplant presented with low back pain for 4 days without fever. Anteroposterior (A.)and lateral (B.) plain radiographs show narrowing of the L4-5 disc space with subtle irregularity of the adjacent endplates (black arrows) C.T1- weighted sagittal MRI shows diffuse T1 hypointensity of the visualized vertebral bodies with destruction of the L4 and L5 vertebral bodies (white arrows). D.T2-weighted sagittal MRI demonstrates abnormal mild T2 hyperintensity in the L4-5 disc sapce and irregular destruction of the adjacent endplates with ventral epidural and anterior paraspinal soft tissue (white arrows). E. Postgadolinium, fat-saturated T1-weighted sagittal MRI reveals diffuse marrow enhancement of the L4 and L5 vertebral bodies as well as inhomogeneous enhancement of the L4-5 disc space (white arrows) Thick enhancing ventral epidural soft tissue (white arrows) and thin anterior paraspinal enhancement extending from lower L3 to upper S1 level is seen. F.Postgadolinium, fat-saturated T1-weighted axial MRI confirms the vertebral marrow enhancement, enhancing ventral epidural and paraspinal soft tissue. In this case, there are a few small rim enhancing portions (black arrows) in the anterior epidural and paraspinal soft tissue enhancement possibly representing small abscess formation. The blood cultures were positive for streptococci, and then he was treated for pyogenic spondylodiscitis with antibiotics.*

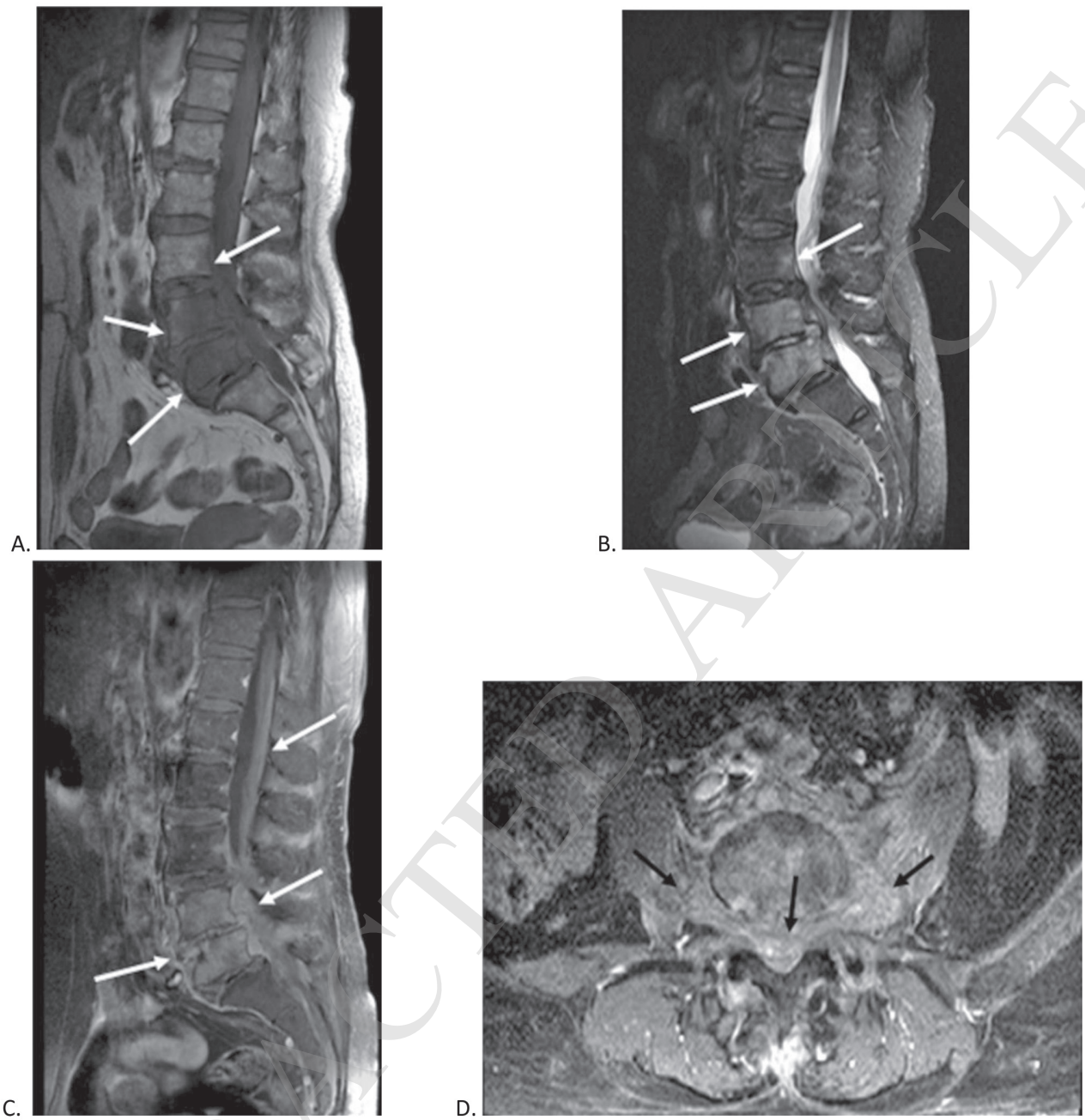

Figure 2 *Pyogenic spondylodiscitis. A 73-year-old woman with back pain and mild weakness of both legs for 1 month. A.T1- weighted sagittal MRI shows diffuse T1 hypointensity (white arrows) in the L4 and L5 vertebral bodies as well as some in the posterior aspect of L3 vertebral body. B.T2-weighted, fat-saturated sagittal MRI demonstrates abnormal T2 hyperintensity in the narrowed L4-5 disc sapce (white arrows) and T2 hyperintensity in the L4 and L5 vertebral bodies as well as some in the posterior aspect of L3 vertebral body (white arrows). C. Postgadolinium, fat-saturated T1-weighted sagittal MRI reveals marrow enhancement corresponding to the abnormal vertebral marrow signal. Evidence of disc enhancement, enhancing ventral epidural soft tissue, and thin anterior paraspinal enhancement extending from lower L3 to upper S1 level (white arrows) is seen. Enhancing cauda equina nerve roots are also seen, indicating radiculitis/arachnoiditis. Note a small nonenhancing portion in the anterior paravertebral region at the upper L5 level, representing small abscess formation. Postgadolinium, fat-saturated T1-weighted axial MRI confirms the vertebral marrow enhancement, enhancing ventral epidural and paraspinal soft tissue (black arrows). The blood cultures were positive for S. aureus, and then she was treated for pyogenic spondylodiscitis with antibiotics.*

phlegmon or T1 hypointense/T2 hyperintense fluid collections with peripheral contrast enhancement, representing frank abscess (Fig 1 and 2). The most important findings that have been suggested for the diagnosis of pyogenic spondylodiscitis include early disc space involvement manifested as T2 hyperintensity, disc space enhancement, vertebral marrow T1 hypointensity and the presence of paraspinal or epidural inflammation. In contrast, findings with relatively low sensitivity for the diagnosis of pyogenic spondylodiscitis include T1 hypointensity in the disc and disc space height loss[13,14]. Therefore, if T2 hyperintensity and/or enhancement are present in both disc and adjacent vertebral bodies, infectious spondylodiscitis should be considered.

Pyogenic spondylodiscitis may have atypical imaging features, including lack of endplate erosive changes and signal intensity abnormalities in early course of the disease, involvement of two adjacent bodies with intervening disc sparing, only single vertebral body involvement, and involvement of one vertebral body and one disc only[15]. Occasionally, pyogenic spondylodiscitis may appear as solitary or multiple discrete, enhancing bony lesions without gross abnormalities of the discs, mimicking metastatic disease or other marrow infiltrative diseases[16].

MRI is not recommended for routine follow-up because it may lag behind the clinical picture, especially of improvement and clinical response to antibiotics[16–20]. MRI findings can even worsen despite clinical improvement. The abnormal MRI findings, particularly of the affected bone and disc as well as the paraspinal soft tissues, may persist despite antibiotic therapy for 4 to 8 weeks[18] and even months after a clinical cure has been achieved[17,20,21]. The only radiographic sign correlating with healing process at MRI follow-up is reappearance of normal marrow T1 hyperintensity that is thought to be normal fatty replacement[15,16].

MRI is also useful in the setting of postoperative spinal infection. Postoperative discitis and/or osteomyelitis are relatively uncommon complications after lumbar spine surgery. MRI is less reliable in this setting, because the postoperative spine may demonstrate disc or endplate signal changes/enhancement which are part of the process. MRI cannot reliably differentiate between pathology and postoperative changes until at least 6 months after surgery. One finding that may suggest postdiscectomy changes rather than infection is the presence of two parallel thin bands of enhancement in the disc space which is different to the amorphous/patchy enhancement commonly seen throughout the disc with infection. If paravertebral enhancement is also present, it supports the diagnosis of infection, whereas the absence of T2 hyperintensity/enhancing endplate changes or disc space enhancement makes infection unlikely[22].

Regarding nuclear medicine imaging, despite relatively high sensitivity, it is used only in selected patients, due to its limited spatial resolution, low availability outside of industrialized countries, long examination time, and low specificity of positive findings. Indications for nuclear scanning include patients in whom MRI is contraindicated, equivocal MRI and CT findings, suspected multifocal infectious disease, or high clinical suspicion with negative MRI examination.

Treatment

Parenteral antibiotic therapy is the mainstay of clinical management for at least 6 weeks[6,23]. Surgery is performed in patients who have complications of spondylodiscitis, such as epidural abscess, cord or nerve root compression, and chronically spinal

deformity, or spinal instability due to collapse of vertebral bodies or discs. As mentioned previously, the clinical and imaging evolutions may be discordant. The most important predictive feature that indicates healing of spondylodiscitis is clinical improvement. Laboratory markers, such as improvement of leukocytosis and ESR can also be helpful. Persistently elevated or increased ESR and CRP at week 4 of antibiotic therapy suggest treatment failure[5].

SPINAL EPIDURAL AND SUBDURAL ABSCESSES

Spinal epidural abscesses commonly occur due to direct extension from spondylodiscitis, spontaneously or related to hematogenous spread or iatrogenic inoculation from an invasive spinal procedure. Predisposing factors include immunosuppressive conditions and underlying chronic diseases, especially diabetes. As in pyogenic spondylodiscitis, Staphylococcus aureus is the most common pathogen, recovered in approximately 70% of patients. The clinical presentation of patients with spinal epidural abscess is variable. Back pain is the most common symptom and is severe. Fever and neurologic deficits are also common as well as other symptoms such as tenderness and limited range of motion. Leukocytosis and elevations of ESR and CRP are common. Spinal epidural abscesses are most common in the thoracic spine, but can even involve entire length of the spine. The abscesses can be located either in the anterior or posterior epidural space. Rapid expansion of the abscess can cause permanent neurological deficits resulted from direct cord or nerve root compression. The neurologic deficits can be caused by thrombosis or thrombophlebitis[24].

Imaging Evaluation

MRI of the entire spinal axis is recommended when a spinal epidural abscess is clinically suspected. MRI typically reveals a T1 hypointense/T2 hyperintense lesion in the epidural space. On postgadolinium T1-weighted imaging, this lesion typically enhances either homogeneously or heterogeneously when it is a phlegmon or peripherally with central non enhancing fluid signal intensity in a mature abscess (Fig. 3)[13, 21]. Patients with an epidural abscess always have meningeal thickening and enhancement. A ventral epidural abscess usually has a bilobed configuration on axial MRI images because of the firmly anchored midline raphe and the fixed lateral membranes giving rise to the so-called “curtain” sign[25].

Engorgement of the epidural venous plexus and/or basivertebral veins may be present. Thecal sac, spinal cord and/or cauda equina nerve root compression are common. Diffusion weighted imaging may show restricted diffusion within the epidural abscess[26].

Subdural abscess of the spine is extremely rare. In it, the pus collection is located in the space between the dura and arachnoid, which shows as a ring enhancing fluid signal collection on axial postgadolinium T1 weighted MRI images. The most common location is the lumbar spine. Risk factors and clinical features are similar to epidural abscess. Staphylococcus aureus is again the most common causative organism. Emergent surgical drainage is the definite treatment with subsequent antibiotic therapy[36–38].

Treatment

The treatment of choice is emergent surgical decompression and abscess drainage, followed by antibiotic therapy. Antibiotic therapy may be delayed in neurologically stable patients until emergent

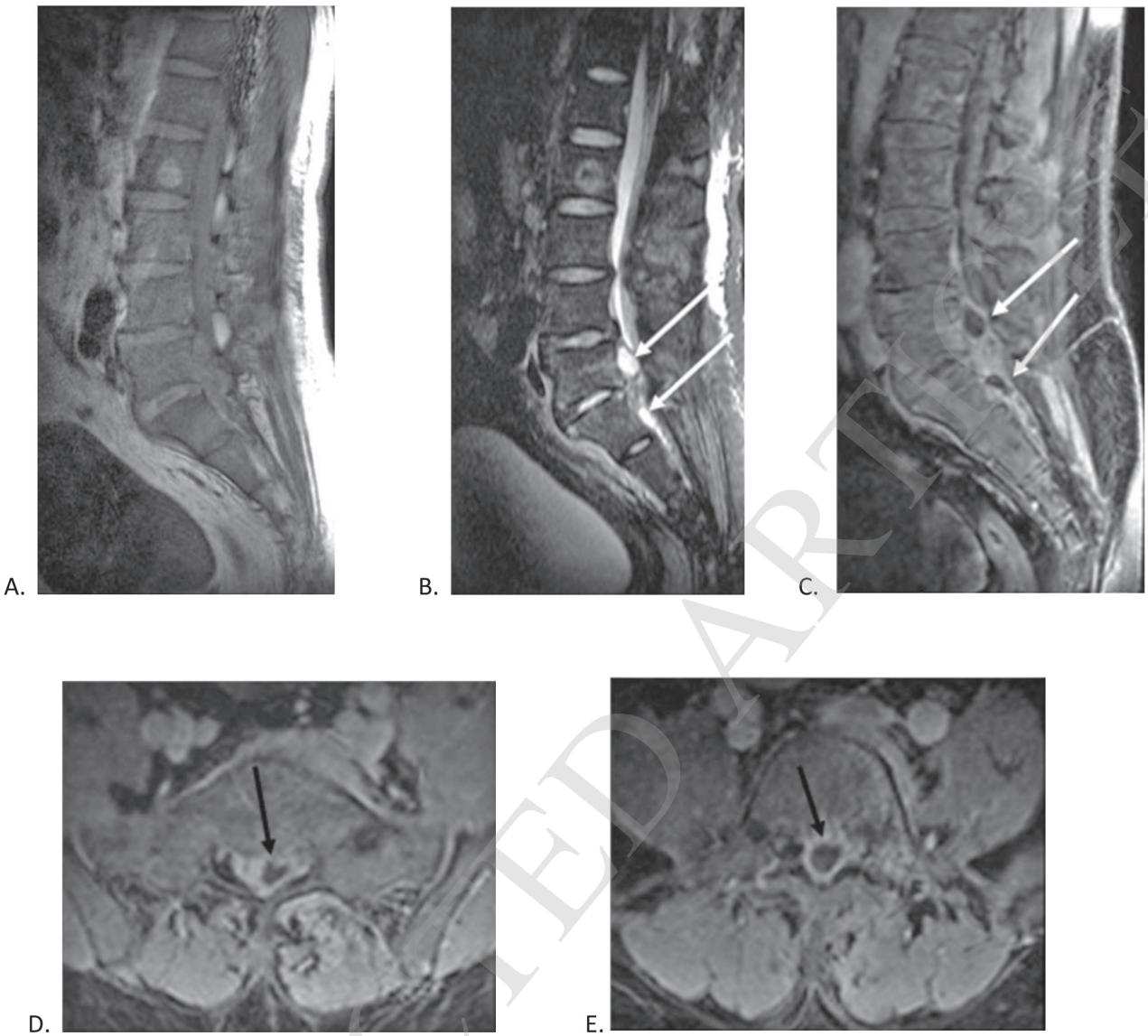

Figure 3 *Pyogenic spondylitis with epidural abscesses. A 53-year-old male presented with fever and back pain for 3 weeks. A.T1- weighted sagittal MRI B.T2-weighted, fat-saturated sagittal MRI and C. Postgadolinium, fat-saturated T1-weighted sagittal MRI demonstrate abnormal T2 hyperintensity with some enhancement in the L5-S1 disc space as well as T2 hyperintensity with marrow enhancement in the L5-S1 endplates indicating spondylodiscitis. There are enhancing anterior epidural and anterior paraspinal soft tissues extending from the upper L4 level down to the end of thecal sac. Note a few nonenhancing T2 hyperintense fluid collections (white arrows) within enhancing anterior epidural soft tissue, representing epidural abscesses. D.and E.Postgadolinium, fat-saturated T1-weighted axial MRI also demonstrate two anterior epidural abscesses (black arrows) at the L5 and S1 levels compressing the thecal sac. An enhancing left-sided anterior paraspinal soft tissue phelgmon is noted. The epidural abscesses were surgically evacuated, and S. aureus was cultured.*

surgery has been performed. A phlegmon may require a widespread decompressive approach with laminectomy, whereas an abscess may be treated by limited laminotomies and irrigation[24].

GRANULOMATOUS SPINAL INFECTION

Tuberculous Spondylitis

Tuberculous spondylitis (Pott disease) is the most common nonpyogenic infection of the spine, and also the most common overall cause of spinal infection in the developing world. Tuberculous infection is more insidious than pyogenic infection of the spine. The incidence of tuberculosis remains high in the endemic regions, even in developed countries due to increasing immigration from endemic areas, the development of drug-resistant strains, and the human immunodeficiency virus (HIV) pandemic[30]. Tuberculous spondylitis is more common in HIV-positive patients than normal hosts[31]. The clinical presentations of tuberculous spondylitis include back pain, low grade fever, and nonspecific symptoms such as malaise or weight loss and in the chronic stage, kyphotic deformity[32]. The source of tuberculosis is commonly pulmonary by hematogenous dissemination, typically from a clinically quiescent primary focus[33]. However, chest radiographs commonly do not demonstrate evidence of pulmonary tuberculosis in patients with tuberculous spondylitis[34].

Tuberculous spondylitis most commonly involves the thoracic spine, particularly the thoracolumbar junction. The tuberculous bacilli spread to the vertebral body hematogenously and lodge in the anterior subchondral regions of the vertebral body. Extension to other vertebral bodies occurs beneath the anterior and posterior longitudinal ligaments and is commonly present at the time of diagnosis. When the infection spreads posteriorly, it can cause an epidural abscess, intradural abscesses, tuberculous arachnoiditis, and spinal cord myelitis[1, 30-34].

Imaging evaluation

Similar as with other spinal infections, MR is the imaging modality of choice for evaluating tuberculous spondylitis. The classic imaging appearance of tuberculous spondylitis is similar to that of pyogenic spondylodiscitis, including involvement of one or more adjacent vertebral levels preferentially at the vertebral endplates, involvement of the disc space, and adjacent paraspinal soft tissue inflammatory changes[32]. However, disc space involvement is often not present early in the disease and is delayed when compared with pyogenic spondylodiscitis. There are many atypical features of tuberculous spondylitis such as isolated single vertebral lesions or multilevel disease (contiguous or noncontiguous) without disc involvement mimicking metastases[32, 35, 37]. Sparing of the disc space has been ascribed to the fact that Mycobacterium lacks proteolytic enzymes. The infection commonly spreads in a subligamentous fashion across one or more levels beneath the anterior or posterior longitudinal ligaments (Fig. 4). Spreading of the infection into the epidural space may also occur but is less common than extension into the anterior paravertebral regions. The subligamentous spread may be much more extensive than the degree of vertebral involvement and can lead to skip lesions of involved bones/discs characterized by intervening normal vertebral levels. Regarding tuberculous abscess, they more commonly contain calcifications, are larger and have smoother and thinner walls when compared with pyogenic abscesses (Fig.5). The posterior elements are more commonly involved than in pyogenic disease and isolated posterior element involvement has been reported in 15% of cases.

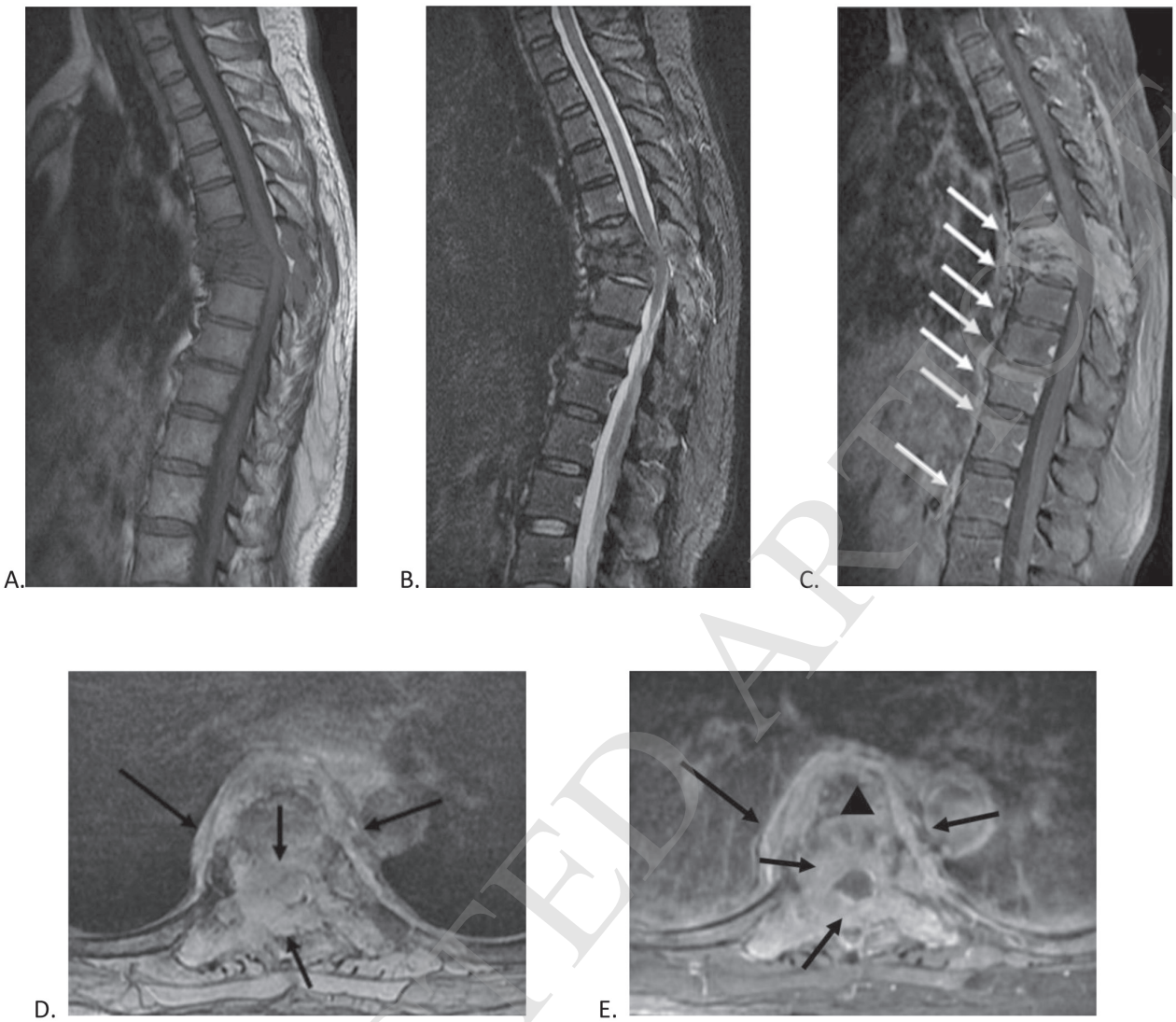

Figure 4 *Tuberculous spondylodiscitis. A 47-year-old woman with back pain and weakness of both legs for 2 months A.T1- weighted sagittal MRI shows collapsed T6 and T7 vertebrae with diffuse T1 hypointensity involving both vertebral bodies and posterior element. Kyphotic change or gibbus deformity at this level is seen. B.T2-weighted, fat-saturated sagittal MRI demonstrates abnormal T2 hyperintensity in the T6 and T7 vertebral bodies in both vertebral bodies and posterior elements. Severe narrowing of the spinal canal with cord compression is seen. Marked narrowing of the T6-7 disc space without definite T2 hyperintensity is noted. C. Postgadolinium, fat-saturated T1-weighted sagittal MRI reveals diffuse boe marrow enhancement corresponding to the abnormal vertebral marrow T1 signal. There is subligamentous spread of infection beneath the anterior longitudinal ligament down to the T12 level. (white arrows) Evidence of disc enhancement, enhancing ventral and dorsal epidural soft tissues and thin anterior paraspinal contrast enhancement is seen. D. T2-weighted, fat-saturated axial MRI and E.Postgadolinium, fat-saturated T1-weighted axial MRI confirms the vertebral marrow enhancement (black triangle) and enhancing paraspinal and epidural soft tissues (black arrows) encirle the thoracic cord resulting in cord compression. In this case, the epidural and paraspinal enhancement represents inflammatory phlegmon and/or venous engorgement without discrete abscess formation. The patient underwent T6-7 laminectomy to decompress the spinal cord and remove the epidural soft tissue. Tissue culture was positive for M. tuberculosis.*

Gibbus deformity is a classic radiographic findings of tuberculous spondylitis (Fig. 4 and 5) due to the preferential involvement of the anterior vertebral column causing collapse of the destroyed vertebral bodies. Similar to other spinal infections, radiographic findings of tuberculous spondylitis tend to lag behind the clinical setting[35, 36].

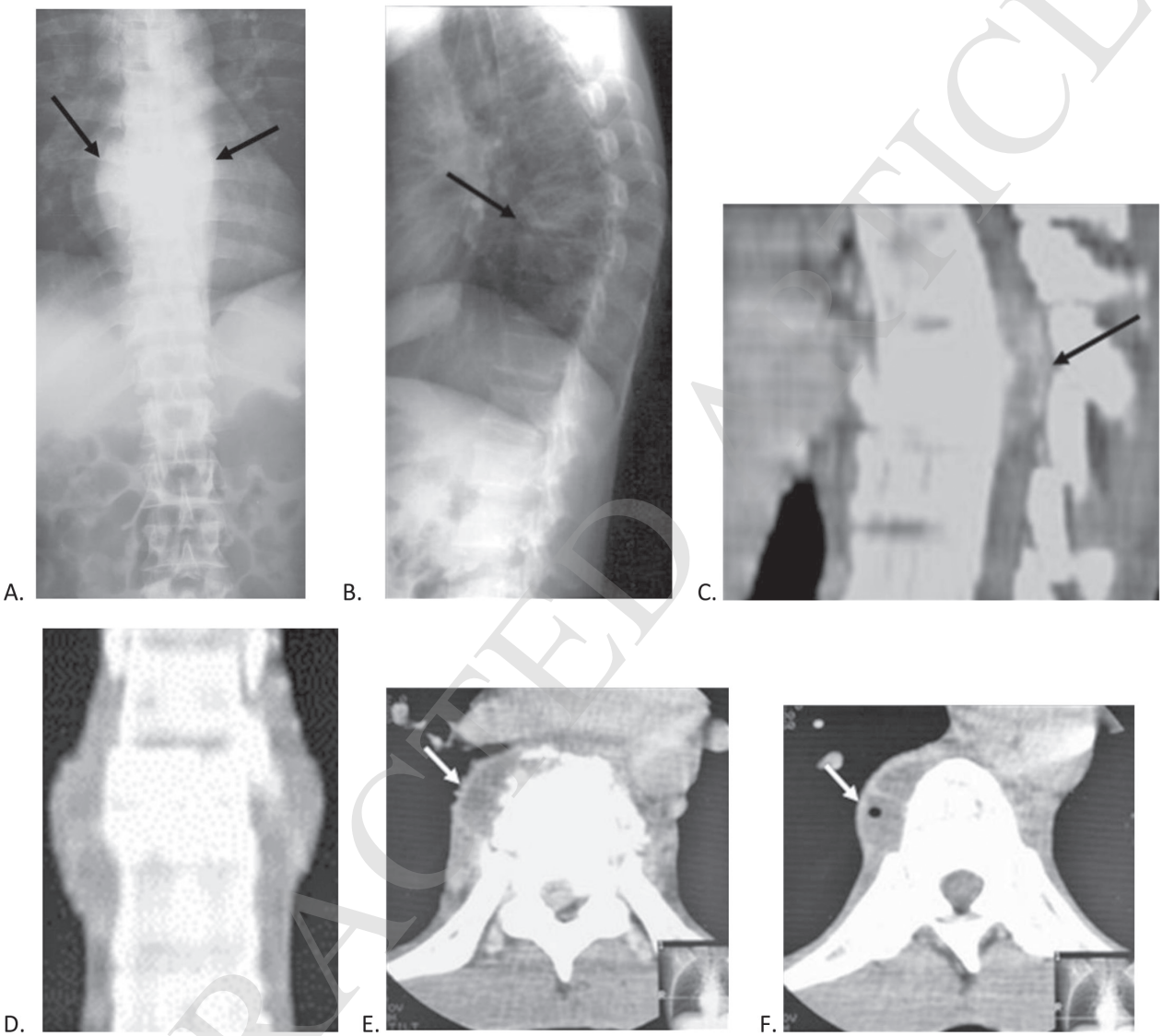

Figure 5 *Tuberculous spondylitis. A 40-year-old woman presented with chronic kyphosis and paraplegia for 2 months. Anteroposterior (A.) and lateral (B.) views of plain radiographs show mild kyphotic deformity of the upper thoracic spine, a mildly collapsed T6 vertebra, narrowing of the T6-7 disc space with indistinct endplates, and bilateral paravertebral soft tissue lesions (black arrows). C. Postcontrast sagittal CT demonstrates the mildly collapsed T6 vertebra, involvement of the T6-7 disc space with large anterior epidural soft tissue (black arrows) compressing the thoracic cord. D. Postcontrast coronal CT confirms enhancing bilateral paravertebral soft tissues. E. and F. Postcontrast axial CT at T6-7 level demonstrates a large thin smooth rim enhancing lesion (white arrows) at the right-sided paravertebral region with internal small air bubble representing a tuberculous abscess. The patient underwent T6-7 laminectomy to decompress the spinal cord and evacuate the epidural soft tissue. The paravertebral abscess was surgically drained. Pus culture was positive for M. tuberculosis.*

Treatment

Antituberculous drugs are the main treatment and the duration of treatment is generally 6 to 12 months to obtain a cure. Surgery may be performed in patients with neurological complications or medical therapy failure or occasionally for prevention of new or worsening deformity[30].

Fungal Spondylitis

Fungal spondylodiscitis occurs in immunocompromised patients. Many fungal organisms can cause spine infection, most of them are Aspergillus and Candida species. The radiographic findings of fungal spondylodiscitis are usually nonspecific and cannot be distinguished from those of pyogenic and tuberculous spondylodiscitis. One MRI finding that has been recently reported in fungal spondylitis is a lack of T2 hyperintensity in the disc spaces, in contrast to pyogenic spondylodiscitis where the disc is always T2 bright[38]. The MRI findings of aspergillus spondylitis are similar to those of tuberculous spondylitis, including involvement of multiple vertebral levels with skip lesions or subligamentous spreading, and serrated appearance of the vertebral endplates and subchondral T2 hypointensity (Fig.6). Therefore, Aspergillus spondylitis should be suspected in immunocompromised patients when either typical or atypical radiographic findings of spondylitis are present[39].

SUMMARY

Infectious spondylitis is increasing in incidence due to immigration and immunosuppression. Nowadays, imaging plays an important role in the diagnosis and follow up of these potentially life-threatening conditions. MR is the imaging modality of choice for patients with suspected spinal infection. If spinal infection is suspected, MRI protocols should include fat-suppressed T2- and fat-suppressed contrast enhanced T1-weighted sequences to better detect and delineate extent of the spinal infection. Pyogenic spondylodiscitis should be considered, when there is increased T2- weighted signal and/or enhancement in the disc and subchondral regions of adjacent vertebral bodies. Spinal epidural and subdural abscesses generally accompany pyogenic spondylodiscitis. MRI is critical in diagnosing both spinal epidural and subdural abscesses, which need to be treated with emergent surgery. Tuberculous spondylitis is the most common spinal infection worldwide and its characteristic imaging findings include disc space sparing, subligamentous spread, and large paraspinal abscesses. Fungal spondylitis is a rare disease found in immunocompromised patients and has a nonspecific imaging appearance. Therefore, the diagnosis of infectious spondylitis should be made of radiographic abnormality with clinical correlation.

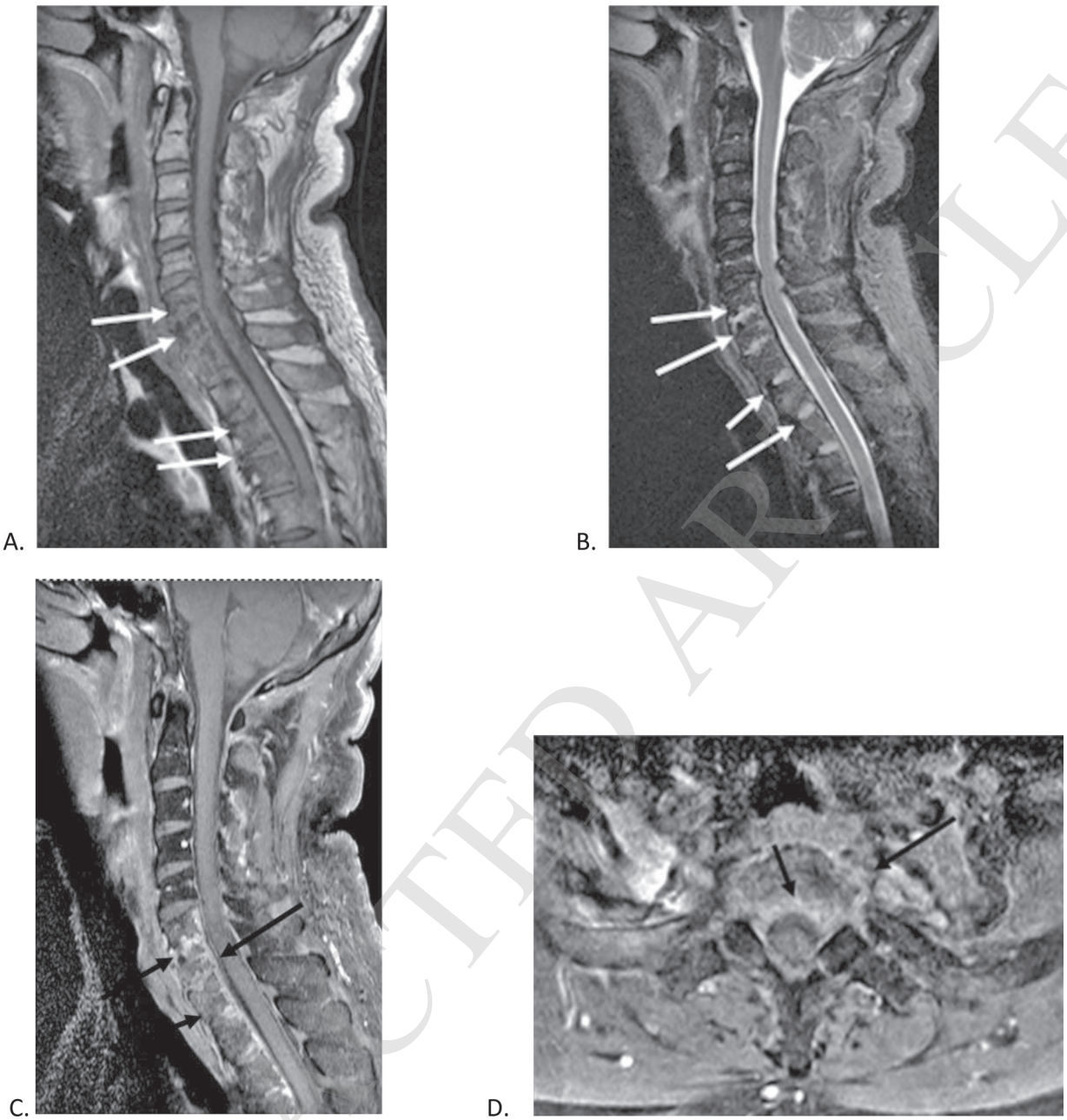

Figure 6 *Aspergillus spondylitis. A 60-year-old male, with diabetes presented with neck pain and weakness of the upper extremities of one month duration. A.T1- weighted sagittal MRI and B.T2-weighted, fat-saturated sagittal MRI show diffuse T1 hypointensity/T2 hyperintensity extending from C6 to the T4 vertebral bodies. There are endplate irregularities with subchondral low signal intensity (white arrows) adjacent to the discs and some hypointense foci in the anterior aspect of discs (white arrows). Abnormal T2 hyperintensity in the C6-7 down to T3-4 disc sapces is also seen. C. Postgadolinium, fat-saturated T1-weighted sagittal MRI reveals diffuse bone marrow enhancement (black arrows) at the C6 to T4 vertebrae corresponding to the abnormal vertebral bone marrow signal. Evidence of disc enhancement and thin enhancing ventral epidural soft tissue and anterior paraspinal enhancement (black arrows) is seen. D.Postgadolinium, fat-saturated T1-weighted axial MRI confirms the vertebral marrow enhancement, enhancing ventral epidural, and thin anterior paraspinal soft tissue. His blood culture was positive for Aspergillus fumigatus. He was treated for fungal spondylitis with antifungal therapy.*

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Review Articles/บทฟื้นฟูวิชาการ

ภาพวินิจฉัยทางรังสีของภาวะการติดเชื้อในกระดูกสันหลัง

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บทคัดย่อ

ภาวะการติดเชื้อในกระดูกสันหลังเป็นโรคที่พบได้บ่อยและมีอุบัติการณ์สูงขึ้นเรื่อยๆ โดยสาเหตุเป็นทั้งได้จากการติดเชื้อแรกเริ่มที่กระดูกสันหลังหรือเป็นภาวะแทรกซ้อนจากการติดเชื้อในกระแสเลือด เนื่องจากภาวะนี้มีความอันตรายถึงแก่ชีวิตได้โดยเฉพาะถ้าได้รับการวินิจฉัยและรักษาล่าช้า ดังนั้นในปัจจุบันการตรวจด้วยภาพทางรังสีโดยเฉพาะการตรวจด้วยเครื่องเอกซเรย์คลื่นแม่เหล็กไฟฟ้าจึงมีบทบาทสำคัญในการช่วยวินิจฉัยภาวะนี้ให้รวดเร็วและแม่นยำมากขึ้น โดยสาเหตุที่พบบ่อยของการติดเชื้อมาจากการติดเชื้อแบคทีเรียและวัณโรคซึ่งจะพบความผิดปกติของภาพรังสีที่ค่อนข้างแตกต่างกัน ในภาวะติดเชื้อในกระดูกสันหลังที่เกิดจากเชื้อแบคทีเรียนั้นส่วนใหญ่จะพบว่ามีความผิดปกติทางรังสีที่หมอนรองกระดูกสันหลังและขอบกระดูกสันหลังที่อยู่ติดกันตั้งแต่ระยะเริ่มแรกจากการตรวจด้วยเครื่องเอกซเรย์คลื่นแม่เหล็กไฟฟ้า นอกจากนี้ยังสามารถให้การวินิจฉัยการเกิดฝีหนองใต้เยื่อหุ้มกระดูกสันหลังซึ่งเป็นภาวะแทรกซ้อนที่พบร่วมได้บ่อยและต้องได้รับการรักษาด้วยการผ่าตัดระบายหนองออกโดยเร็ว ในขณะที่การติดเชื้อในกระดูกสันหลังที่เกิดจากเชื้อวัณโรคนั้นส่วนใหญ่จะพบว่ามีความผิดปกติทางรังสีที่เป็นลักษณะเฉพาะได้แก่ ไม่พบความผิดปกติทางรังสีที่หมอนรองกระดูกสันหลังในระยะเริ่มแรก, มีการติดเชื้อลุกลามไปตามชั้นใต้เยื่อหุ้มกระดูกสันหลัง และมักพบฝีหนองขนาดใหญ่ที่บริเวณด้านข้างกระดูกสันหลังที่ติดเชื้อ ส่วนการติดเชื้อในกระดูกสันหลังที่เกิดจากเชื้อรานั้นเป็นโรคที่พบไม่บ่อยมักเจอในผู้ป่วยที่มีภาวะภูมิต้านทานต่ำ โดยจะพบความผิดปกติของภาพรังสีที่มีลักษณะไม่จำเพาะทำให้ยากต่อการวินิจฉัย ดังนั้นในการวินิจฉัยภาวะติดเชื้อในกระดูกสันหลังทุกรายจำเป็นต้องอาศัยความผิดปกติของภาพวินิจฉัยทางรังสีร่วมกับข้อมูลทางคลินิกของผู้ป่วยมาประกอบกัน

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