Spinal Infections and Parasitic Infestation

Mohammod Raziul Haque, Esma Azam Zico and Forhad H. Chowdhury

Abstract: Spinal infection is not uncommon. It can affect both the bone and soft tissue of the spine and it may be spontaneous or iatrogenic following surgery or spinal tap. It usually presents with pain and fever, with or without neurological deficit/spinal deformity, and its causes can be pyogenic, tubercular, viral, fungal, or, very rarely, parasitic. Spinal infection can be treated both conservatively and/or with surgery. Conservative management includes antibiotic and/or antitubercular therapy; surgical intervention ranges from ultrasound-guided aspiration of pus to deformity correction by instrumentation. Neurological outcomes are excellent if there is a neuro-deficit due to tubercular infection; the worst outcomes are following pyogenic infection. This chapter will briefly discuss pyogenic, tubercular, and fungal spinal infections and spinal parasitic infestations, including their neurosurgical management.

Abbreviations

AFB	acid-fast bacilli	ATD	antitubercular therapy
CBC	complete blood count	CRP	C-Reactive Protein
ESBL	extended-spectrum Beta-lactamase	SEA	spinal epidural abscess
CES	cauda equina syndrome	CT	computed tomography
ESR	erythrocyte sediment rate	FDG	5 Fluoro-deoxy Glucose
MRSA	methicillin-resistant *Staphylococcus aureus*	TC	total count
IGRA	interferon-gamma release assay	IV	intravenous
MRI	magnetic resonance imaging	SOL	space-occupying lesion
HIV	human immunodeficiency virus	HSV	Herpes simplex virus

1. Introduction

Spinal infection means infection and inflammation of the spinal and/or paraspinal tissue. It leads to considerable acute and chronic morbidity in the patient and causes significant financial loss to the patient and the healthcare system. Due to the relatively high incidence and difficulty in diagnosis, there must be a thorough understanding of the diagnostic and management principles in order to successfully treat these patients.

2. Incidence

The prevalence of spinal infections ranges from 2% to 7%, with a mortality rate of 2%–15%. Spinal infection has a bimodal occurrence, affecting primarily juvenile patients under the age of 20 and adult patients aged 50–70 years. The frequency of spinal infections has not decreased as antibiotic therapy has advanced, particularly among the elderly, owing to a rise in patients with risk characteristics (Tayles and Buckley 2004; Frangen et al. 2006).

3. Types of Spinal Infection

3.1. According to Anatomical Site

(i) Vertebral osteomyelitis/spondylitis: Infection of the vertebral body without involving the disc is called spondylitis; it most commonly involves the lumbar segment of the spine.
(ii) Spondylodiscitis/discitis: This is an infection of the disc and bone and it can occur spontaneously or postoperatively. Symptoms include severe pain during spine movement and radiating pain in different body regions with fever and chills.
(iii) Spinal epidural abscess: This is a neurosurgical emergency. These infections can cause weakness, back pain, spinal tenderness, and bowel and bladder disturbance.
(iv) Spinal subdural empyema: This infection is rarer and usually spreads from an infection in another anatomical area.
(v) Meningitis/arachnoiditis: This is an infection and inflammation of the meninges of the spine. This infection can spread swiftly and can lead to severe life-threatening complications if not managed properly.
(vi) Spinal cord abscess: This usually presents as an intramedullary SOL. It is caused by both pyogenic and tubercular organisms.

3.2. According to Aetiology

(i) Pyogenic: Most common organisms are *Staphylococcus aureus*, *Streptococcus*, and *E coli*.
(ii) Granulomatous: Most commonly caused by M. Tubercular (Figures 1 and 2); other rare granulomatous infections are caused by M. Brucellar, syphilis, and fungi (aspergillus).
(iii) Viral: Human immunodeficiency virus (HIV), Herpes zoster, Epstein–Barr virus (EBV), Cytomegalo virus, herpes simplex virus (HSV), West Nile virus, human T-cell lymphotropic virus type 1 (HTLV-1), polio virus.
(iv) Parasitic: Parasitic infections of the spine are very rare. Infectious myelopathy is caused by Schistosoma infections, which are one of the most frequent parasites. Africa, South America, and Eastern Asia are the most common places to find these. *Echinococcus granulosus* cysts in dogs can compress the spinal cord (Frangen et al. 2006; Sobottke et al. 2008; Duarte and Vaccaro 2013).

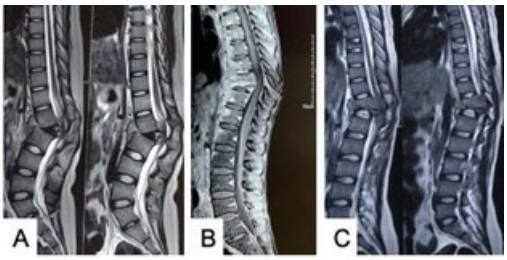


Figure 1. (**A**–**C**) Tubercular spondylodiscitis with loss of vertebral body with instability. Source: Figure by authors.

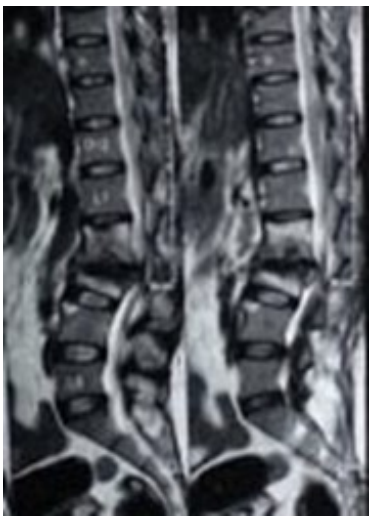


Figure 2. Lumbar tubercular spondylodiscitis. Source: Figure by authors.

4. Predisposing Factors

1. Elderly age;
2. Diabetes mellitus;
3. IV drug abuse;
4. Haemodialysis/kidney or hepatic failure;
5. Oncological history and chemotherapy;
6. Previous spinal surgery;
7. Septicaemia;
8. Infectious endocarditis;
9. Immunosuppressive conditions, including alcohol abuse, HIV infection, chronic steroid use, rheumatologic disease, and Dengue fever (Figure 3) (Sobottke et al. 2008; Duarte and Vaccaro 2013).

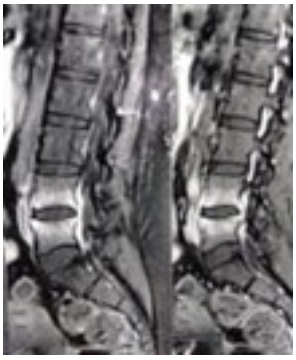


Figure 3. Spontaneous spondylodiscitis after Dengue fever. Source: Figure by authors.

4.1. Post-Procedure Spinal Infection

Spinal infection can follow any intervention, even a single spinal tap (Figure 4).

Post-procedure spinal infection can be superficial or deep and can be early or late. Classification is depending on the grade of infection and likelihood of host response. The severity of infection is divided into three groups: (1) superficial or deep infection with a single organism, (2) deep infection with multiple organisms, and (3) deep infection and myonecrosis with multiple or resistant organisms (Rasouli et al. 2012).

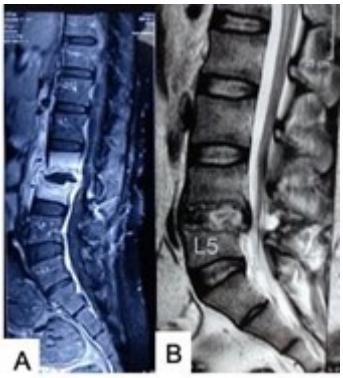


Figure 4. (**A**,**B**) Contrast-enhanced MRI showing spondylodiscitis after spinal anaesthesia at L2/3 and L4/5. Source: Figure by authors.

5. Aetiopathogenesis

Spinal infection can start from three sources; the most common source is haematogenous, then spread from a contiguous site, and, lastly, from the direct entry of an organism due to iatrogenic or penetrating trauma.

Haematogenous spread can be through the arterial or venous system. The Batson plexus is a potential route of infection. Pelvic infections can spread to the lumbar vertebrae; pulmonary infection can spread to the thoracic vertebrae and beyond. The urinary tract also a frequent source of infection. Adjacent structures, such as a retropharyngeal abscess, oesophageal perforation, or aortic implants, can transfer an infection to the spine in a contiguous manner. An iatrogenic aetiology is common, and infection can occur after epidural treatments. In the paediatric group, the spine has extensive arterial anastomosis, so arterial spread is more common and spontaneous disc infection is not rare. However, this extensive blood supply combats infection, resulting in rapid healing and little bone destruction. Discs have blood supply until the age of 15; then, they become avascular (Duarte and Vaccaro 2013; Rasouli et al. 2012; Tyrrell et al. 1999; Butler et al. 2006; Fantoni et al. 2012; Maslen et al. 1993).

6. Clinical Presentation

Spinal infection usually presents with back pain or neck pain which is constant, aggravated by even mild spinal movement, and worse at night. There may be some constitutional symptoms, like fever with chills and rigor if the infection is pyogenic or low-grade evening fever with weight loss and/or loss of appetite if the infection is caused by TB. Clinical features depend on the extent of the involvement of the spine; epidural abscesses usually present with profound neurological deficits and paraplegia/quadriplegia in low-income countries and may

present with radiculopathy. Mechanical compression and vascular impairment are prone to cause neurologic abnormalities (most prominently with epidural abscesses) (Frangen et al. 2006; Tyrrell et al. 1999; Fantoni et al. 2012; Broner et al. 1996; Gouliouris et al. 2010).

Localized spinal discomfort, muscular spasms, and a markedly reduced range of motion are all possible symptoms. Neck stiffness is the most common symptom of cervical spinal infections. They can also produce a retropharyngeal abscess, which can lead to dysphagia, dysphonia, and torticollis (Broner et al. 1996; Carragee 1997; Schimmer et al. 2002).

Irritability, refusal to sit, crawl, or walk, and bowel and bladder incontinence are all symptoms that can be seen in children. Lumbar lordosis is also visible. Fever and neurologic impairments, on the other hand, are uncommon in paediatric patients.

Infection may spread to adjacent tissue and may present with psoas abscesses. It may cause bone destruction and spinal instability, leading to spinal deformity like kyphosis or gibbus (Schimmer et al. 2002; Brown et al. 2001; Mylona et al. 2009).

7. Diagnosis

Initially, some haematological tests, like ESR, TC, DC, and CRP, are essential. A high ESR is more sensitive, and 90% of individuals with spondylodiscitis have an elevated CRP. Both can be utilized to track a patient's reaction to therapy. A 25% decrease in ESR at 1 month is a great indicator of a positive treatment response, but CRP is more precise and returns to normal sooner. If patient presents with high-grade fever, blood culture is essential before starting antibiotics (Sobottke et al. 2008; Carragee 1997; Lillie et al. 2008).

At the time of the initial assessment, plain radiography should be taken. The absence of definition and irregularities of vertebral endplates is the first evidence of spinal infection that may be noticed on plain X-rays. This normally happens 2–8 weeks after the first symptom appears. This causes endplate fragmentation and a decrease in the height of the intervertebral disc. X-rays are also useful in determining whether or not there is any global malalignment as a result of illness or bone damage (coronal or sagittal).

The gold standard for diagnosing a spinal infection is MRI. For the imaging of spinal infections, MRI has the highest specificity and sensitivity. It possesses a 96% sensitivity and a 94% specificity. Furthermore, it shows more epidural space with soft-tissue detail. Discs and vertebral bodies show a hypointense signal on T1W and a hyperintense signal on T2W, attributable to oedema in spinal infections. Gadolinium-enhanced MRI improves MRI accuracy by helping to distinguish infection from degenerative and tumour-associated pathology. Endplate alterations cause a hypointense signal on T2W imaging in degenerative disease, although there is no oedema. In comparison to normal bone marrow, tumour lesions show a relatively hypointense signal on T1W image. However, no one imaging feature can tell the difference between a tumour and an infection. Extensive bone deterioration with conservation of the intervertebral disc, heterogeneous augmentation of the body of vertebra, and the development of a paravertebral abscess are among the MRI findings in tuberculous spondylitis (Broner et al. 1996; Krogsgaard et al. 1998; Diehn 2012; Sharif 1992).

CT scan is the modality of choice to see bony details both for diagnostic and surgical planning. Also, where MR imaging is contraindicated, CT myelography can be helpful. Early abnormalities in vertebral endplates can be identified this way (Broner et al. 1996; Jevtic 2004).

Bone scan has an improved specificity over MRI, of 91%–100%, to identify spinal infection. PET scan with FDG shows increased specificity. FDG uptake is highest in sites of inflammation containing macrophages and neutrophils, enabling high-resolution imaging of acute and chronic infections. Degenerative disease, including fractures, has no FDG uptake (Gemmel et al. 2006, 2010).

Tissue diagnosis is essential to differentiate malignancy from infection and to guide antibiotic selection in adults, but empirical antibiotics can be started prior to tissue diagnosis in paediatric patients. Tissue diagnosis is important if a fungal or tubercular infection is suspected and if empirical therapy failed in that group.

Obtaining sufficient and accurate tissue for diagnosis is the key factor. CT-guided FNAC is least invasive, but it has an accuracy of 70% and has some limitations, like inadequate tissue, radiation, and complications. So, core biopsy is preferred to FNAC if the abscess cavity can be accessed easily. Core biopsy can be taken in different ways, like percutaneous fluoroscopic guidance or endoscope assistant (Ratcliffe 1985; Kornblum et al. 1998; Gasbarrini et al. 2012).

Open biopsy is sometimes essential if closed biopsy fails to detect an organism or if the infection site is inaccessible or if the patients needs surgery for a neurological deficit or for a spinal deformity.

The biopsy specimen should be delivered for Gram stain, AFB stain, fungal stain, and anaerobic bacteria, aerobic bacteria, fungal, and tubercular cultures. Gene X-pert for M. tuberculosis with rifampicin sensitivity should also be used. An interferon-gamma release assay (IGRA) from whole-blood plasma, as well as an acid-fast bacilli (AFB) smear including culture, can be employed, with a sensitivity rate of up to 88% if there is a high index of expectation for tuberculosis. Biopsy specimens should be handed over for histopathologic investigation if there is any suspicion of a tumour or fungal illness (Ratcliffe 1985; Cheng et al. 2004; Kumar et al. 2010; de Lucas et al. 2009; Rankine et al. 2004; Michel et al. 2006).

8. Management

The management of spinal infections includes both nonsurgical and surgical techniques.

8.1. Nonsurgical Management

Nonsurgical management includes antibiotics, improvement in nutritional status, immobilization with a thoracolumbar brace or cervical collar, treatment of comorbidities, and close monitoring for any neurological deterioration.

8.1.1. Indications for Nonsurgical Management

Nonsurgical management is indicated for spinal infection that does not cause any neurological deficit or spinal instability, for patients unfit for surgery, or for patients who have been completely paralyzed for more than 36 h. However, recent observations suggest that those who have been completely paralyzed due to compression of the spinal cord by a non-malignant mass for 2–3 months without any vascular compromise can benefit from surgical intervention.

8.1.2. Choice of Antibiotic

Broad-spectrum antibiotics covering *Staphylococcus aureus*, MRSA, ESBL, and *Escherichia coli* should be administered. Intravenous antibiotics should be given for 6–8 weeks, followed by oral antibiotics, depending on clinical and laboratory proof of resolution of infection. Before starting antibiotics, specimens must be collected and/or blood must be drawn for culture if fever is present (Sobottke et al. 2008; Broner et al. 1996; Ratcliffe 1985; Khanna et al. 1996; Del Curling et al. 1990; Danner and Hartman 1987).

8.1.3. Minimally Invasive Nonsurgical Treatment

This is indicated for psoas abscess or other paravertebral abscesses and for pyogenic spondylodiscitis. Here, abscesses can be drained as guided by USG/CT.

8.2. Surgical Management

8.2.1. Aims of Surgical Management

These include the decompression of the neural elements and the debridement of infective necrotic tissue.

8.2.2. Indications

(1) Neurological deficit from compression by phlegmon or any other elements resulting from a destructive consequence of infection. (2) Spinal instability. (3) Failure of conservative management.

8.2.3. Relative Indications

Epidural abscess; sepsis in the cervical and dorsal region with or without neurological deficit; uncontrollable pain.

The threshold for surgery for infections in the cervical and dorsal spine is low, as mild compression may have disastrous consequences.

8.2.4. Surgical Approach

The surgical approach to spine infections depends on the site of the lesion, extent of bony damage, and neurological deficit.

9. Follow-Up

To see the response to treatment, CRP and ESR can be assessed weekly. CRP is a good early measure of how well a treatment is working because it starts to normalize within the first week. Imaging can be performed to see fusion and radiological improvements in the infection.

10. Outcome

Mortality rates are very low, less than 5%; this is because of advancements in radio-imaging have resulted in early diagnosis and prompt treatment. Morbidity is due to neurological deficit; complete paralysis for more than 12 h due to pyogenic infection is very unlikely to improve, but complete paralysis due to Pott's disease without neurovascular compromise has very favourable outcomes (Tyrrell et al. 1999; Schimmer et al. 2002; Del Curling et al. 1990; Delafuente 1991).

11. Recurrence

In the paediatric population, relapses of spinal infections are uncommon. Adults have a recurrence risk of up to 14%, with 75% of recurrences occurring in the first year in patients with medical conditions. Recurrent infection is linked to paravertebral abscesses, recurrent bacteraemia, and chronic draining sinuses, among other things.

12. Spinal Tuberculosis/Pott's Disease

12.1. Introduction

Sir Percival Pott reported tuberculous spondylitis and its clinical manifestation of paraplegia in European patients with kyphotic deformities in 1779 (Sobottke et al. 2008). Spine tuberculosis (STB) is a particularly deadly type of skeletal tuberculosis because it can cause neurological deficits as a result of the compression of nearby nerve tissues and considerable spinal deformity. So, early detection and treatment of spinal tuberculosis is critical for precluding catastrophic consequences (Dobson 1972).

12.2. Incidence

Extrapulmonary tuberculosis (EPTB) has a low incidence of 3%, yet there has been no substantial decrease in the frequency of EPTB as there has been with pulmonary tuberculosis. Skeletal tuberculosis (STB) accounts for about 10% of EPTB cases, and spinal TB is the most prevalent site of STB, accounting for roughly 50% of skeletal EPTB cases. The most frequently impacted part of the spinal column is still the thoracolumbar junction, followed by the cervical and lumbar spine. The spinal column is implicated in less than 1% of all tuberculosis (TB) cases (Luk 1999; Pertuiset et al. 1999; Kulchavenya 2014).

12.3. Pathophysiology of Spinal TB

The mycobacterium tuberculosis complex, which comprises roughly 60 species, causes tuberculosis. Humans are only known to be affected by Mycobacterium TB (the commonest), Mycobacterium bovis, Mycobacterium africanum, and Mycobacterium microti (Jain and Dhammi 2007).

It is a fastidious, slow-growing aerobic bacillus. Infections can start in the lungs, mediastinal lymph nodes, gastrointestinal tract, mesentery, genitourinary system, or any other viscera. When aerobic circumstances are good, the bacilli tend to stay latent for long periods of time and multiply every 20–30 h. Haematogenous diffusion of the bacillus from a primary focus causes spinal infection, which is always secondary (Schirmer et al. 2010; Tuli 1993).

The paradiscal arteries separate on both sides of the disc and then reach the subchondral zone of the top and lower endplates of each disc, making the intervertebral disc an avascular anatomical structure. The fact that the vertebra has an artery supply promotes subchondral bone inclusion on both sides of the disc, known as "paradiscal," which is the most prevalent kind (Rasouli et al. 2012). "Central" involvement results in vertebral body loss; "posterior" involvement involves neural arch structures; and "nonosseous" involvement promotes abscess

creation. TB causes granulomatous inflammation, classically characterized by epithelioid cells and lymphocytic infiltration, which may unite to create the characteristic Langhans-type giant cells, resulting in caseating necrosis of the afflicted tissues and the formation of a cold abscess. Kyphosis is a deformity of the spine caused by a gradual destruction of the body of the vertebra, leading to a deformation of the spine (Rajasekaran et al. 2014; Jain 2010).

12.4. Clinical Presentation of Spinal TB

The intensity and length of the disease, as well as the location of the disease and the existence of comorbidities, all influence how spinal TB manifests (Su et al. 2010). There are two types of spinal tuberculosis: complex and simple. Patients with complex tuberculosis have abscesses, sinus formation, deformity, instability, and neurological deficits. Simple spinal tuberculosis is one in which the diagnosis is made before the onset of problems. Backache is by far the most prevalent symptom. It is mostly related to bone inflammation during the active period, and it can be radicular in character on rare occasions. The intensity of rest pain at the affected level is proportional to the extent of bone loss and instability. PTB is more commonly related to constitutional symptoms like weight loss, loss of appetite, malaise, and fever than to spinal TB (Hayes et al. 1996).

12.5. Diagnosis

The diagnosis of spinal tuberculosis is made utilizing clinical and classic MRI findings, and it is proved by either culture with sensitivity, the Gene Xpert PCR test, or histological evidence.

12.6. Management

12.6.1. Conservative

There are some controversies about the duration of ATD therapy, but the authors recommend 18 (3 + 15) months (Figure 5).

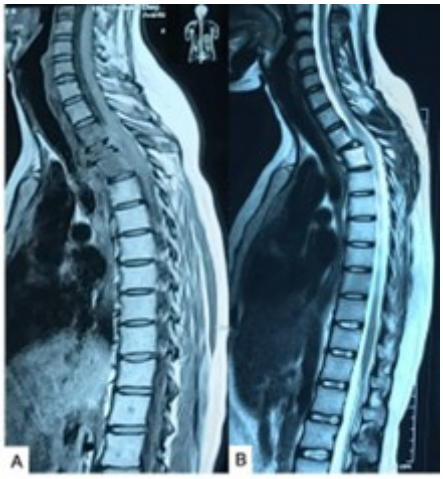


Figure 5. (**A**) MRI of spine showing D1, D2, and D3 tubercular spondylodiscitis with complete collapse of D2, epidural abscess, and cord compression. (**B**) MRI of spine 18 months after surgery and anti-TB therapy. Source: Figure by authors.

Drug Resistance (MDR, XDR)

MDR-TB (Figure 6) is resistant to both INH and rifampicin. It usually happens as a result of poor treatment; however, resistant strains can also spread. Resistance to INH and rifampicin, as well as any fluoroquinolone and at least one injectable second-line anti-TB treatment, is known as extensively drug-resistant TB (XDR-TB) (Jain 2010; Su et al. 2010). Velayati et al. suggested the term "totally drug-resistant tuberculosis" (TDR-TB) in 2009 to describe TB strains that demonstrated in vitro resistance to all first- and second-line medications tested (Pawar et al. 2009).

Figure 6. Dorsal Pott's (**A**) with positive gene X-pert and rifampicin sensitivity (**B**). Source: Figure by authors.

12.6.2. Surgical Management

Indications for surgical therapy:

(1) Chemotherapy failure or recurrence;
(2) Significant weakness at presentation;
(3) Instability;
(4) Static or growing neurological impairment even after starting chemotherapy;
(5) Incapacitating pain;
(6) Deformities.

Fundamentals of surgical management:

- Optimum decompression and debridement;
- Maintenance and augmentation of stability;
- Correction or slowing of deformity progression.

Surgical objectives (Figure 7):

- Drainage of abscess;
- Infected material debridement;
- Debridement and fusion +/− stabilization (Velayati et al. 2009).

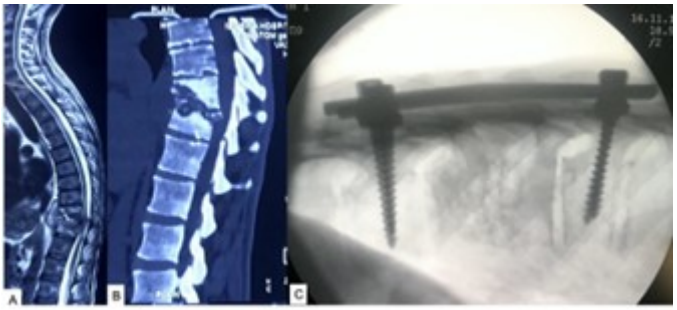


Figure 7. (**A**) MRI of spine and (**B**) CT scan of dorsal spine showing dorsal spinal tuberculosis with cord compression. (**C**) Intraoperative X-ray of after decompression, fusion, and stabilization. Source: Figure by authors.

12.7. Cold Abscess

Chemotherapy alone heals the majority of cold abscesses, with draining being necessary in rare cases, like respiratory discomfort or dysphagia caused by a big cervical paravertebral abscess or pseudo-hip flexion deformity as a result of a huge psoas abscess (Mallick et al. 2004; Oniankitan et al. 2014).

12.7.1. Surgical Approach

The surgical approach to spine infections is based on the site of lesion, the extent of bone loss, and neurological deficit. Anterior and posterior approaches, global reconstruction by posterior approach, or mixed procedures are used to accomplish debridement plus fusion with or without instrumentation.

(i) Anterior approach: usually performed in the cervical region.
(ii) Posterior approach: most commonly performed due to familiarity of route, ease of access, and the easy learning curve.

In the cervical spine, for ventral locations, an anterior approach is recommended, including the debridement of the infected disc and bone followed by normal saline wash, then by the placement of an iliac crest graft/allograft or artificial cage and stabilization with plates and screws. For posterior lesions, a posterior approach and stabilization with lateral mass screws and cervical pedicle screws should be used. In the thoracic spine if the lesion is an epidural abscess only with/without neurological deficit, laminectomy, hemi-laminotomy, or laminoplasty is recommended; if the lesion is associated with bone loss or instability, then stabilization with pedicle screws is recommended. For ventral thoracic lesions, several approaches exist. The ventral approach helps to debride infected ventral bone and tissue and should be followed by ventral or dorsal stabilization; the posterolateral approach (costo-transversectomy) involves decompression and stabilization. In the thoracolumbar junction and lumbar region, decompression and debridement by TLIF followed by stabilization is recommended. TLIF on one or two levels can be accomplished by both MISS and an open technique. Fusion can be performed with a tricortical iliac crest bone graft or with irradiated bone with bone morphogenic protein/autologous small bone chips (Al Sebai et al. 2004; Hodgson et al. 1960; Tuli 2007; Govender and Parbhoo 1999; Benli et al. 2007; Christodoulou et al. 2006; Chen et al. 2003; Lee et al. 2006; Mizuno 1967; Shang et al. 2010).

12.7.2. Outcome

Over 90% of cases of Pott's paraplegia have very good recovery. If the patient develops thromboangiitis obliterans, the outcome is unsatisfactory.

13. Spinal Epidural Abscess

13.1. Introduction

A spinal epidural abscess (SEA) is an emergency subtype of spinal infection which demands prompt diagnosis and treatment; otherwise, disastrous results can be seen.

Incidence: SEAs are a very uncommon ailment. Pyogenic SEAs occur in 0.2–1.2 instances per 10,000 hospital admissions, with the number of cases probably rising. With a male-to-female ratio of roughly 2:1, there is a masculine predominance (Khanna et al. 1996; Del Curling et al. 1990).

13.2. Aetiopathogenesis

Pyogenic and nonpyogenic organisms can cause a spinal epidural abscess. Although the causative pathogen varies by geographical region, *Staphylococcus aureus* is the commonest pathogen (70%), followed by Streptococcus species (7%). But the frequency of SEAs is rising, in part as a result of an increase in the number of elderly people, IV drug abusers, invasive spinal interventions, and HIV cases, as well as due to advancements in radiologic imaging techniques. SEAs caused by Gram-negative bacilli (more frequently seen in IV drug abusers), fungal species, mycobacterium tuberculosis, and parasitic pathogens have been documented, despite their rarity. Around 35% of persons who develop a SEA have used IV drugs in the past; 50%–60% are immunocompromised as a result of a chronic disease; and 10%–20% have had spine surgery (Michel et al. 2006; Khanna et al. 1996; Del Curling et al. 1990).

13.3. Clinical Features and Diagnosis

Systemic features include fever, malaise, and local features according to the site of infection. These are as follows:

- In cervical SEA: neck stiffness, retropharyngeal abscess, quadriparesis.
- In thoracic SEA: back pain, local tenderness, paraparesis.
- In lumber SEA: lower back pain, local tenderness, CES.

Diagnosis: CBC with ESR, CRP, and contrast MRI (Figure 8) are the recommended diagnostic tools. For the detection of organisms, CT-guided aspiration of large ventral abscesses is recommended, but for localized abscesses within the spinal canal, surgical decompression and collection of pus and abscess wall should be performed. The latter should be sent for Gram stain, AFB stain, and culture in blood agar media and Lowenstein–Jensen media; fungal stain should also be sought.

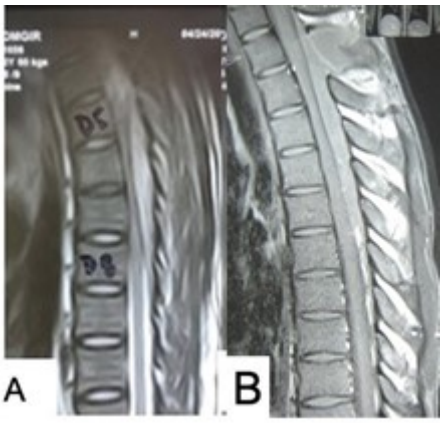


Figure 8. (**A**,**B**) Dorsal spinal epidural abscess. Source: Figure by authors.

13.4. Treatment

Early identification, CT scan-guided aspiration/drainage, and empirical antibiotic therapy followed by cultures with sensitivities are the mainstays of treatment. If the patient has acute neurological deficits, more than minor sensory disturbances, or is neurologically deteriorating, surgical decompression and debridement plus/minus spinal stabilization should be performed; postoperative complications, like arachnoiditis, may permanently worsen the patient's neurological status. As a result, the risks and advantages of a surgical procedure must be carefully weighed (Michel et al. 2006; Khanna et al. 1996; Del Curling et al. 1990).

13.5. Outcome

SEAs are fatal in 4–31% of cases (Tuli 2007). Patients with severe neurologic deficits rarely improve, even with surgical intervention within 6–12 h of the start of paralysis. Pott's disease has a 75% recurrence rate.

14. Pyogenic Spondylodiscitis

14.1. Introduction and Aetiopathogenesis

Pyogenic spondylodiscitis is a potentially fatal infection of the intervertebral disc(s) and/or neighbouring vertebrae (Skaf et al. 2010). It can happen as a result of haematogenous implantation during bacteraemia, direct dissemination from a nearby infection, or implantation during spinal surgery (Skaf et al. 2010). Spondylodiscitis is becoming more common, despite the fact that it is still a rare condition (Govender 2005). Abscess development in the epidural space and neighbouring soft tissues and muscle is common with spondylodiscitis. Localized inflammation and abscess formation can lead to spinal cord compression plus/minus vertebral column instability, resulting in long-term neurological impairment (Skaf et al. 2010).

In paediatric patients, an isolated intervertebral disc infection occurs first, followed by the involvement of the neighbouring endplates. By the age of 15, the anastomoses between the equatorial and circumferential superficial metaphyseal arteries shrink to the point of atrophy. In adults, nutrient end arteries supply the subchondral spongy bone, where a small septic embolus may lodge in the presence of bacteraemia and proceed to grow, resulting in a bone infarct and subsequent osteomyelitis. Infection spreads to neighbouring vertebral bodies by bridging anastomotic vessels from one metaphysis to the next (Skaf et al. 2010). After settling in the subchondral space, the infection usually progresses into the disc, resulting in osteomyelitis and discitis. The infection can then spread across the disc and into neighbouring endplates (Wong-Chung et al. 1999). Unlike spontaneous spondylodiscitis in adults, which begins in the body of the vertebra and then spreads to the disc space, iatrogenic or postoperative spondylodiscitis is characterized by direct disc space involvement. It is important to mention that its incidence, predisposing factors, causative organisms, and management strategies vary throughout the world.

14.2. Clinical Presentation

Patient may have symptoms long before the diagnosis. Patients commonly experience pain (up to 90%) and fever (only 52%). The commonest symptom is pain, which is usually confined to the spine, is aggravated by movement, and can radiate (Wong-Chung et al. 1999). The commonest symptoms of spondylodiscitis include paravertebral muscle soreness and spasm, as well as limitations in spine movement. In some situations, neurologic problems like spinal cord or nerve root compression, as well as meningitis, may occur (12%). The development of an epidural abscess is indicated by the evolution of spinal discomfort to radicular symptoms, followed by weakening and paralysis (Sapico and Montgomerie 1990), or by the infected level's kyphotic collapse. Because of mostly anterior cord compression, sensory impairment is uncommon, although motor and long-tract symptoms are more typical (Eismont et al. 1983).

14.3. Investigations

14.3.1. Imaging

X-ray of the spine may show reduced disc space, fracture, and deformity. CT scan of the spine shows bone involvement and deformity. MRI of the spine shows soft-tissue involvement, abscess, and cord compression (Figures 9–11).

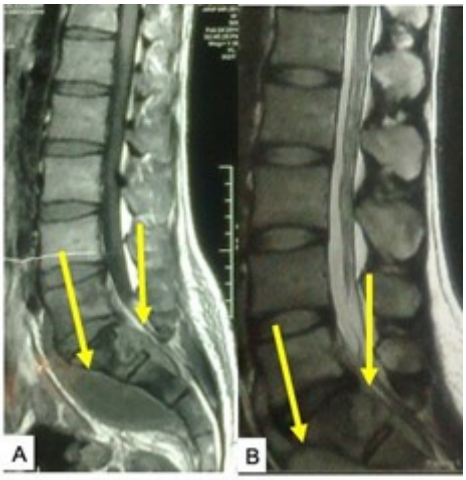


Figure 9. (**A**,**B**) MRI of lumbosacral spine showing lumbosacral spondylodiscitis (pyogenic) with epidural and presacral abscess (arrow marked). Source: Figure by authors.

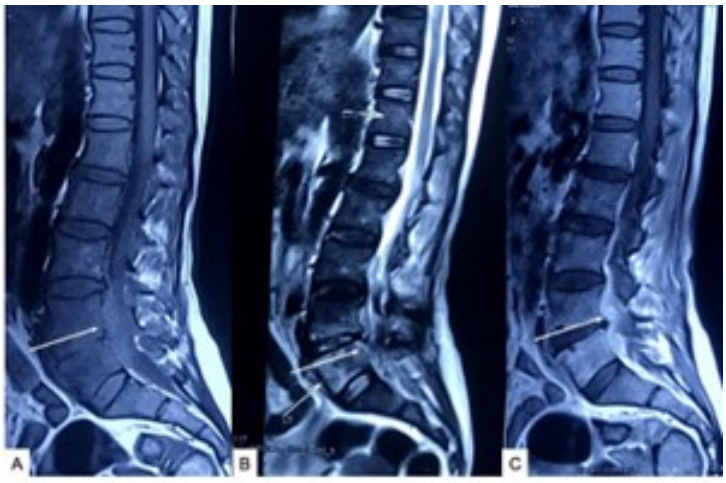


Figure 10. (**A**–**C**) MRI of lumbosacral spine showing L5 and S1 spondylodiscitis (pyogenic) with epidural abscess. Source: Figure by authors.

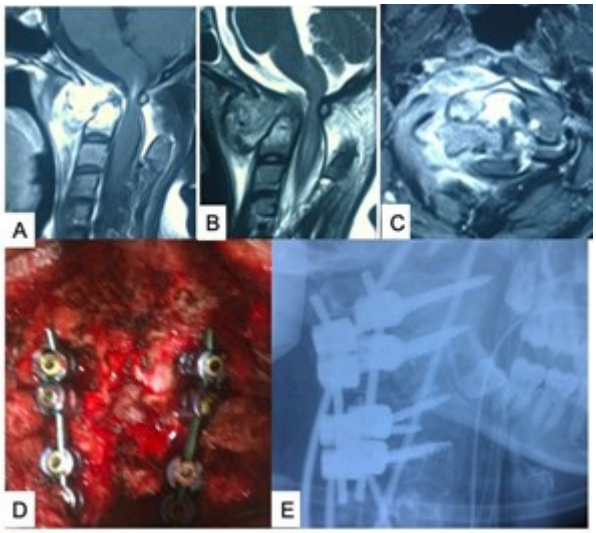


Figure 11. (**A**–**C**) MRI of craniovertebral junction (CVJ) showing pyogenic spondylodiscitis resulting in CVJ instability and spinal cord compression. (**D**) Intraoperative picture of fixation of CVJ (C1, C2, and C3). (**E**) Intraoperative X-ray of fixation. Source: Figure by authors.

14.3.2. Laboratory Investigations

The WBC could be abnormally high. In almost all situations, an increase in ESR is noted, even if it is nonspecific. A steady decline in ESR is frequently observed with appropriate medical treatment. Although nonspecific, C-reactive protein (CRP) may be a more clinically relevant marker than ESR and should be utilized to track the progression of the disease (Rath et al. 1996). Culture specimens should be taken from the blood, urine, and focused suppurative processes. Blood cultures can be positive in around 50% of patients, and they can help with antimicrobial medication selection.

14.3.3. Image-Guided Needle Biopsy

If an organism cannot be detected using less intrusive culture procedures, direct cultures from the involved vertebral body or/+ disc space are used. Percutaneous needle biopsy guided by CT or fluoroscopy (accuracy rate ranges from 70% to 100%, while open biopsies are accurate in more than 80% of cases) can be performed (An et al. 2006).

14.3.4. Open Biopsy

Open biopsy techniques have an accuracy of 93.3% (Razak et al. 2000) but are associated with morbidity (Lestini and Bell 1990).

14.3.5. PCR

Non-culture amplification-based DNA analysis (PCR) is also highly specific and sensitive; it commonly identifies the Mycobacterium.

Biopsy material should be sent for pyogenic, bacterial, tubercular, and fungal cultures with histological examination, staining (Gram and AFB), and PCR. The bacteria responsible for spontaneous pyogenic spondylo discitis arelisted in Table 1.

14.3.6. Differential Diagnosis

Inflammatory, granulomatous, neoplastic, or degenerative processes are all possible causes of pyogenic spondylodiscitis (Sapico and Montgomerie 1990). Inflammatory disorders such appendicitis, pyelonephritis, abdominal abscesses, and intestinal infarction might mimic spondylodiscitis in terms of clinical presentation.

14.3.7. Complications

Cervical spine infections can cause a retropharyngeal abscess, and dorsal spine infections can be complicated by life-threatening mediastinitis. Spondylodiscitis at any level can cause complications like epidural abscess,

meningitis, subdural abscess, absence of lordosis, vertebral collapse with consequent spinal instability, and progressive neurological deterioration. A terrible consequence is epidural abscess. The prognosis of epidural abscesses is worse than that of epidural granulation tissue. Vertebral osteomyelitis can cause long-term spinal impairment. Neurological impairments, a diagnosis delay of at least 8 weeks, and chronic debilitating disorders were all found to be predictors of a poor prognosis. Some SEA patients may still experience irreversible paralysis. Long-term rehabilitation is frequently required. The mortality rate for vertebral osteomyelitis has been published to be between 2 and 20%; it is around 5% for SEA (Kourbeti et al. 2008).

Table 1. Bacterial aetiological organisms in pyogenic spontaneous spondylodiscitis.

Gram-Positive Aerobic Cocci	**Percentages**
Staphylococcus aureus	57
Streptococcus pyogene	4.1
Coagulase-negative staphylococci	3.4
Other streptococci	2
Enterococcus spp.	0.7
Gram-Negative Aerobic Bacilli	
Escherichia coli	10.5
Proteus spp.	6.7
Pseudomonas aeruginosa	5.7
Klebsiella pneumoniae	1.8
Enterobacter spp.	1.8
Salmonella spp.	1.8
Serratia marcescens	0.5
Anaerobic Bacteria	
Propionibacterium spp.	2
Bacteroides fragilis	0.5
Peptostreptococcus spp.	0.5

Source: Authors' compilation based on data from Sapico and Montgomerie (1990).

14.3.8. Early Diagnosis

Spondylodiscitis is generally recognized at a later stage. Early detection is predicated on a significant index of suspicion, with a focus on the following factors: (1) presence of an infectious focus; (2) presence of predisposing risk factors like increased age, rheumatoid arthritis, diabetes mellitus, immunosuppression, steroid utilization, ethanol abuse, history of recent invasive diagnostic or surgical spinal procedure, and infectious endocarditis (Eismont et al. 1983; Lestini and Bell 1990); (3) localized spinal pain along with paravertebral muscle spasm, fever, restriction of movement, and presence of neurological deficit.

14.3.9. Management and Outcome

Management should attempt to alleviate pain, avoid or aid in recovery from neurologic impairments, eliminate infection, avoid relapse, and restore spinal stability.

Conservative Treatment

Conservative management principles include (a) establishing a correct microbiological diagnosis; (b) treating with proper antibiotics; (c) immobilization of the spine; and (d) monitoring for clinical and radiological sign/s of instability of the spine, infection progression, or neurological impairment.

- Spinal column immobilization.
- Specimens for microbiological tests should be obtained at admission, and blood for blood cultures should be collected three times.
- A percutaneous biopsy of the afflicted disc is required if these tests are negative. Following surgery, blood cultures should be acquired on a regular basis (Cherasse et al. 2003).
- Unless clinical circumstances dictate differently, such as in patients with neutropenia or severe sepsis, antimicrobial therapy should not be started until the organism has been isolated and identified (Grados et al. 2007). Depending on the clinician's best judgement of the likely organism/s (Ozuna and Delamarter 1996) and the patient's risk factors, the patient should be started on empiric wide-spectrum antibiotic therapy.

Direct antibiotics should be given intravenously once an organism has been detected. Treatment failure was found to be more common when parenteral antibiotic therapy was given for fewer than four weeks, according to studies (Eismont et al. 1983). Most guidelines recommend 6–12 weeks of intravenous antibiotic treatment for pyogenic spondylodiscitis. When debridement is adequately accomplished, theoretically, further intravenous antibiotic treatment duration can be shorter than that of conservative therapy alone (Li et al. 2018).

Surgical Treatment

Indications for neurosurgical management in pyogenic spontaneous spondylodiscitis:

1. Failure of conservative therapy to work.
2. Neurologic impairments that are severe or worsening.
3. Septic embolization or big paraspinal abscess causing local mass impact.
4. Substantial osseous disease affecting two nearby vertebral bodies or a single vertebral body with more than 50% loss.
5. Spinal malformation that worsens over time with or without intractable spinal pain.

Principles of neurosurgical treatment (Figure 11):

- Complete debridement and elimination of diseased tissue.
- Decompression of nerve components.
- Spinal alignment restoration.
- Adjustment of spinal instability.
- Infection usually affects just the anterior vertebral components. Intact posterior components normally preserve some degree of rigidity, preventing major subluxation. As a result, decompression laminectomy alone may destabilize the spine even more, leading to a greater neurological deficit (Eismont et al. 1983).
- In situations of dorsally located epidural abscesses, only laminectomy is recommended. A limited disc space infection can be managed with posterolateral debridement in some patients. However, for thoracic and lumbar lesions, anterior procedures are generally recommended for thorough debridement of the intervertebral disc and vertebral bodies to return to healthy bone, followed by autologous bone grafting utilizing either an anterolateral (Gasbarrini et al. 2005) or a posterolateral (Eismont et al. 1983; McGuire and Eismont 1994) route that preserves the facets, pedicles, and laminae. Several studies have recommended the utilization of autologous grafts after appropriate debridement (Schuster et al. 2000). Recent research has backed the usage of titanium mesh cages to avoid the morbidity of autografts and the slow rate of assimilation of structural allografts (Fayazi et al. 2004).
- Moreover, despite surgery, there is a substantial number of studies urging prolonged bed rest du e to a worry of reinfecting the patient with foreign implants.
- Depending on the number of segments affected, bone quality, and the existence of pre-existing kyphotic deformity, a few recent papers have advocated further posterior fixation following anterior decompression and fusion (Rea et al. 1992).
- Patients who have already had posterior fixation or titanium cages have a lower risk of postoperative problems (Hee et al. 2002).
- Recently, Rath et al. revealed that a posterolateral technique can be used to successfully perform debridement, autologous interbody bone grafting, and internal fixation, allowing for early patient mobilization (Rath et al. 1996).
- Recent research has shown that in cases of acute spinal infection, primary arthrodesis and stabilization can be accomplished (Faraj and Webb 2000).
- Spinal instrumentation, when utilized in the case of kyphotic deformity or subluxation, is a very important adjunct that can be used successfully if a careful debridement of contaminated tissue is performed along with antibiotics.

15. Postoperative and Iatrogenic Spondylodiscitis

15.1. Introduction

The only way an adult with an avascular intervertebral disc space can contract genuine discitis is through direct inoculation of the pathogen. After an ordinary lumbar discectomy, postoperative discitis has been documented to occur in between 0.7 and 2.8% of patients (Kraemer et al. 2003). When a fusion is added to the operation, the frequency rates jump from 0.9% to 6%. The rate of infection following spinal instrumentation

is, on average, 7% with a range of 1.3–12% (Gepstein and Eismont 1990). Infection can occur after laminectomy, lumbar puncture, myelogram, lumbar sympathectomy, chemonucleolysis, and discography, as well as other spinal operations.

15.2. Predisposing Factors and Aetiology

Age, uncontrolled diabetes, malnutrition, steroid medication, radiated area, pre-existing malignancy, long preoperative hospital stays, insufficient sterile practices, longer procedures, and more operating room traffic are all predisposing factors. Patients with postoperative spondylodiscitis have been shown to be younger, to have fewer underlying disorders, and to have a longer time between the beginning of symptoms and diagnosis than those with spontaneous spondylodiscitis (Dufour et al. 2005).

The most typical symptom of a postoperative infection is a brief respite from symptoms after surgery, preceded by a recurrence of back pain 2–6 weeks later, aggravated by almost any spinal motion and occasionally radiating to the hip, groin, leg, abdomen, scrotum, or perineum. Fever, increased perspiration, and chills are common constitutional complaints. In 33% of the instances, local tenderness is present (Natale et al. 1992). In most cases, the surgical site looks to be benign. Neurologic deficits are uncommon, but if they do occur, an epidural abscess or cauda equina syndrome due to recurrent disc prolapse should be suspected (Heller 1992). ESR is frequently raised despite the lack of leukocytosis, although the trend of alterations on periodic ESR testing might be very informative. Plain radiographs are initially normal in postoperative spondylodiscitis, but later (on average, after 3 months) demonstrate reduced disc space height and a blurring of the affected endplates.

The preferred test is MR imaging, which yields results that are akin to those found in spontaneous pyogenic spondylodiscitis. In postoperative spodylodiscitis, *Staphylococcus epidermidis* is the most prevalent pathogen, followed by *Staphylococcus aureus* bacteria. Gram-negative bacteria such as *Escherichia coli* and Pseudomonas aeruginosa can also be blamed (Jimenez-Mejias et al. 1999).

15.3. Treatment

Analgesics, muscle relaxants, antibiotics, immobilization in a halo vest or brace, and bed rest should be used to treat postoperative and iatrogenic spondylodiscitis. Any suspected postoperative infection should be subject to a CT-guided needle aspirate with culture. If pyogenic infection is confirmed or highly suspected, a minimum of 6 weeks of culture-specific injectable antibiotics is suggested (or until ESR drops sufficiently), starting with an anti-staphylococcal antibiotic (e.g., vancomycin $+/-$ rifampin) and a broad-spectrum anti-Gram-negative antibiotic, pending the identification of the organism, and then modifying the regimen depending on the sensitivity data. When the organism detected is MRSA, and because vancomycin monotherapy has been linked to low success rates, combined therapy or a newer anti-staphylococcal medication should be considered. Epidural abscess formation, sepsis, and increasing neurological impairments all require surgery. The surgical route is mostly determined by the severity of the condition. Patients who are diagnosed earlier in the course of the disease can be managed by re-exploration later on. An anterior approach is recommended in more extensive or chronic situations (Skaf et al. 2010).

16. Fungal Spinal Infection

Fungal infections of the spine are rather infrequent. Fungi like Coccidioides immitis as well as Blastomyces dermatitidis are only found in some parts of the world, although cryptococcus, aspergillus, and candida can be found wherever. Candida and Aspergillus are natural body commensals that cause disease in those who are susceptible (patients with uncontrolled diabetes, immunosuppressed and immunocompromised individuals, patients with HIV infection, etc.) when they enter the circulatory system through intravenous lines, prosthetic device implantation, or surgically. For other fungi, spinal infection is frequently the result of the fungus spreading through the blood or directly from a pulmonary source of infection. Vertebral compression fractures and severe deformities of the spine can occur when the vertebral bodies are involved. Psoas or paravertebral abscesses can develop if an infection spreads along the anterior longitudinal ligament. Early detection of the disease necessitates a surgeon's experience, a high index of suspicion, a thorough travel history, and a thorough physical examination, especially in patients with weakened immune systems. Treatment is mainly founded on the rapid administration of suitable medication (typically injectable at first, then long-term oral medicine) and ongoing clinical monitoring. Indications for surgery (debridement and stabilization plus spinal fusion) include resistance to medicinal therapy,

spinal instability, and neurologic impairments. The patient's premorbid status, the variety of the fungal pathogen, and the time of treatment commencement all influence the prognosis (Kim et al. 2006).

17. Parasitic Spinal Infestation

In poorer countries with insufficient sanitation, parasitic illnesses are more common. The spine can be affected by a variety of different parasite infections that impact the CNS. Patients may present with common symptoms like back pain, weakness, numbness, or autonomic incontinence, prompting the clinician to conduct appropriate spine imaging. These lesions can readily be misidentified for other commoner medically curable lesions in cases of parasite infection (Majmundar et al. 2019).

17.1. Neurocysticercosis

Taenia solium is the causal parasite of cysticercosis, the commonest parasitic infestation of the CNS. The disease is brought on by the consumption of embryonated parasite eggs. The parasite enters the bloodstream through the small intestine and travels to a range of locations, including the eyes, skeletal muscles, and neurological systems. This condition is more likely to cause intracranial involvement; spinal cysticercosis accounts for just 1.5 to 3% of all cysticercosis cases (Figure 12).

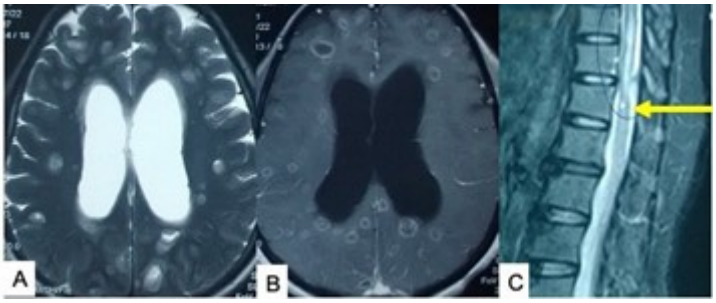


Figure 12. (**A**,**B**) MRI of the brain showing extensive neurocystocercosis. (**C**) MRI of the spine of the same patient showing neurocystocercosis in the spinal cord. Source: Figure by authors.

Spinal neurocysticercosis affecting the spinal cord is exceedingly rare, and it is present in about 1–6% of persons diagnosed with neurocysticercosis (Torabi et al. 2004). The intramedullary type is less prevalent than leptomeningeal involvement (Majmundar et al. 2019; do Amaral et al. 2015). Neurocysticercosis can affect the body of the vertebra, the epidural/subdural/subarachnoid spaces, and the spinal cord itself, similar to (intramedullary) neoplasms. Neurological deficiencies arise as a result of the cysts' mass effect and as an inflammatory response to treatment. The best imaging modality is MRI with contrast, which shows oedema, the mass effect, and enhancement, as well as cystic fluid intensity. Furthermore, the cyst and scolex can be seen using high-resolution T2W images (3D constructive interference in steady state [3D- CISS]). In asymptomatic cases, an antiparasitic drug, frequently albendazole, is administered alongside an anti-inflammatory drug, usually corticosteroids, to minimize inflammation caused by larval death. Surgery is only used in patients who have mass lesions that are causing neurological impairments. The presence of intramedullary lesions is a rare indication for surgery (Majmundar et al. 2019).

17.2. Neuroschistosomiasis

Schistosomiasis is a blood-borne infection caused by platyhelminths (flatworms) of the genus Schistosoma, which is found in Africa, Asia, and the Americas (Carod Artal 2012; Shih and Koeller 2015). Schistosoma japonicum, S. mansoni, and S. hematobium are the three primary species that can infect humans (Shih and Koeller 2015). The dispersion of eggs through venous shunts or retrograde passage of mature worms from the abdominal veins to the vertebral venous plexus are thought to be the mechanisms of CNS infection (Carod Artal 2012; Shih and Koeller 2015; do Amaral et al. 2015). When eggs are deposited into the spinal cord, the host responds with inflammation, resulting in many of the neurological issues related to advanced schistosomiasis. Inflammatory processes can result in space-occupying granulomatous masses in severe situations. Acute or subacute clinical presentation most commonly affects the lower spinal cord (Ferrari and Moreira 2011). Low back discomfort radiating down the legs, lower limb weakness and paraesthesias, deep tendon reflex abnormalities, bladder

dysfunction, sexual impotence, and constipation are all clinical characteristics. Conus medullaris syndrome, acute myelopathy, or acute/subacute lower extremity myeloradiculopathy are all possible symptoms. Due to intramedullary granuloma formation, MRI may show swelling of the spinal cord, particularly in the lower part of the spinal cord and in the conus medullaris (Ferrari and Moreira 2011). Thickened cauda equina roots with uneven contrast enhancement are another common result (Ferrari and Moreira 2011; Adeel 2015). The enzyme-linked immunosorbent assay (ELISA) is the most dependable immunological approach for diagnosis, with a sensitivity of 50% and a specificity of 95%. The sensitivity of the indirect haemagglutination assay (IHA) testing ranges from 70% to 90%, and the integration of both immunological tests has a specificity of 93% and a sensitivity of 90%. However, tissue biopsy by surgery is the most conclusive form of diagnosis. A granuloma tissue sample would reveal schistosome ova encircled by necrosis, inflammation, and demyelination Carod Carod Artal (2012).

Schistosomicidal medicines, like praziquantel, and steroids are among the treatment possibilities Carod Carod Artal (2012). However, in cases of significant spinal cord compression and tissue diagnosis, surgical granuloma excision and decompressive laminectomy may be necessary for symptomatic relief (Majmundar et al. 2019).

17.3. Toxoplasmosis

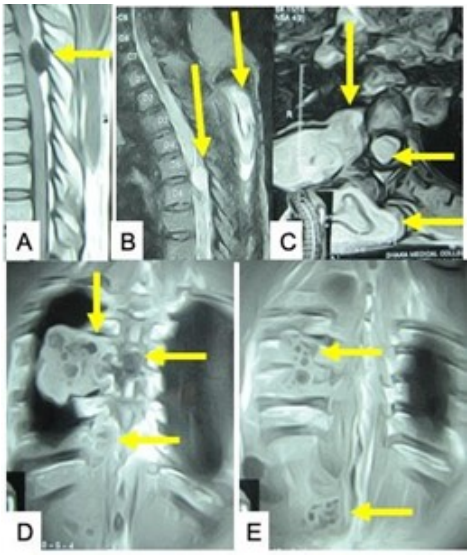
Toxoplasmosis is the commonest type of opportunistic CNS infection among AIDS patients. Toxoplasma gondii, which produces the disease, is an obligate intracellular protozoan parasite (Ashraf et al. 2013).

Spinal cord involvement is not common. Furthermore, spinal cord infection is rarely found alone and is frequently coupled with intracranial involvement. Clinical signs include limb weakness, loss of sensation, and incontinence. A typical MRI finding is localized intramedullary ring-enhancing lesions (Garcia-Gubern et al. 2010). Immunological antibody testing and CSF cytology are also very useful. A summation of pyrimethamine, sulfadiazine, and folinic acid is the preferred therapeutic therapy. Trimethoprim–sulfamethoxazole is another option for treatment. Steroids have also been successfully utilized to relieve symptoms (García-García et al. 2015). In many circumstances, surgical intervention has no clearly defined purpose (Majmundar et al. 2019).

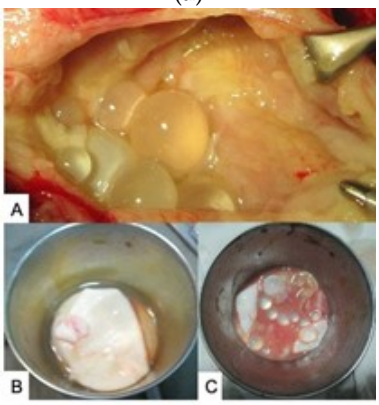
17.4. Echinococcal (Hydatid) Disease

Echinococcus granulosus and Echinococcus multilocularis are the two most frequent echinococcal pathogens. Although echinococcal disease of the CNS is uncommon, the dorsal spine is the most frequently afflicted area of the CNS (Neumayr et al. 2013). The symptoms of spinal involvement are nonspecific and are caused by spinal cord compression, which leads to myelopathy or radiculopathy (Majmundar et al. 2019). Cystic lesions in adjacent vertebral bodies, spondylitis, and bone lysis can all be seen on plain X-rays. Ultrasonography may aid in the detection of abdominal involvement. Osteolytic lesions of the vertebral bodies can be seen with a CT scan (Czermak et al. 2001). Intravenous contrast does not enhance the lesion (Prabhakar et al. 2005). The most sensitive neuroimaging modality for detecting spinal hydatid disease is MRI (Prabhakar et al. 2005). T1W scans typically show a cystic wall that is hypointense or isointense, but T2W images show a hypointense cystic wall with a hyperintense cyst (Pamir et al. 2002). Spinal tuberculosis, abscess, malignancy, and cystic lesions like spinal arachnoid cysts, epidermoids, or aneurysmal bone cysts are among the differential diagnoses (do Amaral et al. 2015). A definitive diagnosis can only be made through surgical exploration and histological testing. Tests for serodiagnosis are specific but not sensitive. The therapy of choice is surgery (Pamir et al. 2002). Though the necessity for spinal fusion should always be evaluated based on the amount of the lesion, the most commonly performed treatment is simple decompressive laminectomy (Figure 13a,b and Figure 14).

The majority of surgical operations are performed using the posterior approach (Pamir et al. 2002). In most cases, radical surgical excision is preferred. The use of scolicidal drugs intraoperatively to limit parasite dispersion during the operation has been recorded in cases of hydatid cyst excision in the abdomen and pelvis (Majmundar et al. 2019; Besim et al. 1998), but it might theoretically give a comparable protective advantage in cases of hydatid cyst removal in the spine.



(**a**)



(**b**)

Figure 13. (**a**) MRI of dorsal spine (sagittal sections) showing spinal hydatid cyst causing cord compression (**A**,**B**); MRI of dorsal spine (axial section) showing spinal, mediastinal, and intramuscular hydatid cyst (marked with an arrow) (**C**); MRI of chest and upper abdomen (coronal section) showing hydatid cysts in mediastinum and liver (**D**,**E**). (**b**) Intraoperative pictures of patient in (**a**). Daughter cysts in intramuscular hydatid cyst (**A**); hydatid cysts after removal (**B**,**C**). Source: Figure by authors.

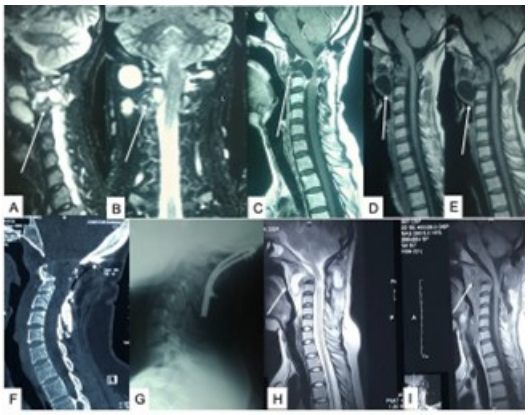


Figure 14. (**A**–**I**) MRI of CVJ showing hydatidosis (Echinococcosis) with instability and cord compression. Source: Figure by authors.

18. Conclusions

Spinal infections are not common, and presentation is usually nonspecific, so early diagnosis is difficult. Nevertheless, appropriate imaging modalities, like MRI with Gad enhancement, blood tests (ESR, CRP), biopsy, and histopathology can aid in making the diagnosis and can guide the proper management, either surgical or

nonsurgical, to preserve neurological function and spinal stability. Assessment of the remission of clinical features and of the normalization of ESR and CRP are part of the long-term follow-up. Recurrent infections frequently necessitate surgical intervention and long-term antibiotic therapy.

Author Contributions: Conceptualization, methodology, validation, formal analysis, investigation, resources, data curation, M.R.H. and F.H.C.; writing—original draft preparation, E.A.Z.; writing—review and editing, visualization, supervision, F.H.C. All authors have read and agreed to the published version of the manuscript.

Funding: This research received no external funding.

Conflicts of Interest: The authors declare no conflicts of interest.

References

Adeel, Ahmed Awad. 2015. Spinal cord schistosomiasis. *Sudanese Journal of Paediatrics* 15: 23–28.

Al Sebai, M. Wasef, M. Monir Madkour, and K. R. Al Moutaery. 2004. Surgical Management of Spinal Tuberculosis. In *Tuberculosis*. Edited by M. Monir Madkour. Berlin: Springer, pp. 493–534.

An, Howard S., Koichi Masuda, and Nozomu Inoue. 2006. Intervertebral disc degeneration: Biological and biomechanical factors. *Journal of Orthopaedic Science* 11: 541–52. [CrossRef]

Ashraf, Aabid, Altaf Kirmani, Abdul Bhat, and Arif Sarmast. 2013. A rare case of recurrent primary spinal echinococcosis. *Asian Journal of Neurosurgery* 8: 206–8. [CrossRef]

Benli, I. Teoman, Alper Kaya, and Emre Acaroglu. 2007. Anterior instrumentation in tuber- culous spondylitis: Is it effective and safe? *Clinical Orthopaedics and Related Research* 460: 108–16. [CrossRef]

Besim, H., K. Karayalçin, O. Hamamci, Ç. Güngör, and A. Korkmaz. 1998. Scolicidal agents in hydatid cyst surgery. *HPB Surgery* 10: 347–51. [CrossRef]

Broner, Frank A., Douglas E. Garland, and Jack E. Zigler. 1996. Spinal infections in the immunocompromised host. *Orthopedic Clinics of North America* 27: 37–46. [CrossRef]

Brown, R., K. McHugh, M. Hussain, V. Novelli, and D. Jones. 2001. Discitis in young children. *Bone & Joint Surgery British Volume* 83: 106–11. [CrossRef]

Butler, Joseph S., Martin J. Shelly, Marcus Timlin, William G. Powderly, and John M. O'Byrne. 2006. Nontuberculous pyogenic spinal infection inadults: A 12-year experience from a tertiary referral center. *Spine* 31: 2695–700. [CrossRef]

Carod Artal, Francisco Javier. 2012. Cerebral and spinal schistosomiasis. *Current Neurology and Neuroscience Reports* 12: 666–74. [CrossRef]

Carragee, Eugene J. 1997. Pyogenic vertebral osteomyelitis. *Journal of Bone and Joint Surgery American* 79: 874–80. [CrossRef]

Chen, Yi-Cheng, Ming-Chau Chang, Shih-Tein Wang, Wing-Kuang Yu, Chien-Lin Liu, and Tain-Hsiung Chen. 2003. One-stage posterior surgery for treatment of advanced spinal tuberculosis. *Journal of the Chinese Medical Association: JCMA* 66: 411–17.

Cheng, V. C., W. C. Yam, I. F. Hung, P. C. Woo, S. K. Lau, B. S. Tang, and K. Y. Yuen. 2004. Clinical evaluation of the polymerase chain reaction for the rapid diagnosis of tuberculosis. *Journal of Clinical Pathology* 57: 281–85. [CrossRef]

Cherasse, A., D. Martin, C. Tavernier, and J. F. Maillefert. 2003. Are blood cultures performed after disco-vertebral biopsy useful in patients with pyogenic infective spondylitis? *Rheumatology (Oxford)* 42: 913. [CrossRef]

Christodoulou, Anastasios G., Panagiotis Givissis, Dimitrios Karataglis, Panagiotis D. Symeonidis, and John Pournaras. 2006. Treatment of Tuberculous Spondylitis with Anterior Stabilization and Titanium Cage. *Clinical Orthopaedics and Related Research* 444: 60–65. [CrossRef]

Czermak, Benedikt V., Karin M. Unsinn, Thaddeus Gotwald, Andreas A. Niehoff, Martin C. Freund, Peter Waldenberger, Wolfgang Vogel, and Werner R. Jaschke. 2001. *Echinococcus granulosus* Revisited: Radiologic Patterns Seen in Pediatric and Adult Patients. *American Journal of Roentgenology* 177: 1051–56. [CrossRef]

Danner, Robert L., and Barry J. Hartman. 1987. Update on spinal epidural abscess: 35 cases and review of the literature. *Reviews of Infectious Diseases* 9: 265–74. [CrossRef]

de Lucas, Enrique Marco, Andrés González Mandly, Agustín Gutiérrez, Raúl Pellón, Laura Martín-Cuesta, Javier Izquierdo, Elena Sánchez, Eva Ruiz, and Fernando Quintana. 2009. CT-guided fine-needle aspiration in vertebral osteomyelitis: True usefulness of a common practice. *Clinical Rheumatology* 28: 315–20. [CrossRef]

Del Curling, O., Jr., David J. Gower, and Joe M. McWhorter. 1990. Changing concepts in spinal epidural abscess: A report of 29 cases. *Neurosurgery* 27: 185–92. [CrossRef]

Delafuente, Jeffrey C. 1991. Nutrients and immune responses. *Rheumatic Diseases Clinics of North America* 17: 203–12. [CrossRef]

Diehn, Felix E. 2012. Imaging of spine infection. *Radiologic Clinics* 50: 777–98. [CrossRef]

do Amaral, Lázaro Luís Faria, Renato Hoffmann Nunes, and Antonio Jose da Rocha. 2015. Parasitic and rare spinal infections. *Neuroimaging Clinics* 25: 259–79. [CrossRef] [PubMed]

Dobson, Jessie. 1972. Percivall Pott. *Annals of The Royal College of Surgeons of England* 50: 54–65. [PubMed]

Duarte, Rui M., and Alexander R. Vaccaro. 2013. Spinal infection: State of the art and management algorithm. *European Spine Journal* 22: 2787–99. [CrossRef]

Dufour, Véronique, Antoine Feydy, Ludovic Rillardon, Aimée Redondo, Laurence Le Page, Frédéric Bert, Nadia Belmatoug, and Bruno Fantin. 2005. Comparative study of postoperative and spontaneous pyogenic spondylodiscitis. *Seminars in Arthritis and Rheumatism* 34: 766–71. [CrossRef]

Eismont, Frank J., H. H. Bohlman, P. L. Soni, V. M. Goldberg, and A. A. Freehafer. 1983. Pyogenic and fungal vertebral osteomyelitis with paralysis. *Journal of Bone and Joint Surgery America* 65: 19–29. [CrossRef]

Fantoni, Massimo, Enrico Maria Trecarichi, Barbara Rossi, Valentina Mazzotta, G. Di Giacomo, L. A. Nasto, E. Di Meco, and E. Pola. 2012. Epidemiological and clinical features of pyogenic spondylodiscitis. *European Review for Medical & Pharmacological Sciences* 16: 2–7.

Faraj, A. A., and J. K. Webb. 2000. Spinal instrumentation for primary pyogenic infection report of 31 patients. *Acta Orthopaedica Belgica* 66: 242–47.

Fayazi, Amir H., Steven C. Ludwig, Michael Dabbah, R. Bryan Butler, and Daniel E. Gelb. 2004. Preliminary results of staged anterior debridement and reconstruction using titanium mesh cages in the treatment of thoracolumbar vertebral osteomyelitis. *The Spine Journal* 4: 388–95. [CrossRef]

Ferrari, Teresa Cristina A., and Paulo Roberto R. Moreira. 2011. Neuroschistosomiasis: Clinical symptoms and pathogenesis. *The Lancet Neurology* 10: 853–64. [CrossRef]

Frangen, T. M., T. Kälicke, M. Gottwald, S. Andereya, H.-J. Andress, O. J. Russe, E. J. Müller, G. Muhr, and C. Schinkel. 2006. Surgical management of spondylodiscitis: An analysis of 78 cases. *Der Unfallchirurg* 109: 743–53. [CrossRef]

García-García, Concepción, Federico Castillo-Álvarez, José M. Azcona-Gutiérrez, María J. Herraiz, Valvanera Ibarra, and José A. Oteo. 2015. Spinal cord toxoplasmosis in human immunodeficiency virus infection/acquired immunodeficiency syndrome. *Infectious Diseases* 47: 277–82. [CrossRef] [PubMed]

Garcia-Gubern, Carlos, Carmen R. Fuentes, Lissandra Colon-Rolon, and Daniel Masvidal. 2010. Spinal cord toxoplasmosis as an unusual presentation of AIDS: Case report and review of the literature. *International Journal of Emergency Medicine* 3: 439–42. [CrossRef] [PubMed]

Gasbarrini, A. L., E. Bertoldi, M. Mazzetti, L. Fini, S. Terzi, F. Gonella, L. Mirabile, G. Barbanti Bròdano, A. Furno, A. Gasbarrini, and et al. 2005. Clinical features, diagnostic and therapeutic approaches to haematogenous vertebral osteomyelitis. *European Review for Medical and Pharmacological Sciences* 9: 53–66. [PubMed]

Gasbarrini, A., Luca Boriani, Caterina Salvadori, S. Mobarec, J. Kreshak, C. Nanni, E. Zamparini, M. Alberghini, Pierluigi Viale, and U. Albisinni. 2012. Biopsy for suspected spondylodiscitis. *European Review for Medical & Pharmacological Sciences* 16: 26–34.

Gemmel, Filip, Nicolas Dumarey, and Christopher J. Palestro. 2006. Radionuclide imaging of spinal infections. *European Journal of Nuclear Medicine and Molecular Imaging* 33: 1226–37. [CrossRef]

Gemmel, Filip, Paul C. Rijk, James MP Collins, Thierry Parlevliet, Katrin D. Stumpe, and Christopher J. Palestro. 2010. Expanding role of 18F-fluoro-D- deoxyglucose PET and PET/CT in spinal infections. *European Spine Journal* 19: 540–51. [CrossRef]

Gepstein, R., and F. I. Eismont. 1990. Post-Operative Spine Infections. In *The Lumbar Spine*. Edited by James N. Weinstein and Sam W. Wiesel. Philadelphia: W.B. Saunders.

Gouliouris, Theodore, Sani H. Aliyu, and Nicholas M. Brown. 2010. Spondylodiscitis: Update on diagnosis and management. *Journal of Antimicrobial Chemotherapy* 65: iii11–iii24. [CrossRef]

Govender, S. 2005. Spinal infections. *The Journal of Bone & Joint Surgery British* 87: 1454–58. [CrossRef]

Govender, S., and A. H. Parbhoo. 1999. Support of the anterior column with allografts in tuberculosis of the spine. *The Journal of Bone & Joint Surgery British* 81: 106–9. [CrossRef]

Grados, Franck, François Xavier Lescure, Eric Senneville, René Marc Flipo, Jean Luc Schmit, and Patrice Fardellone. 2007. Suggestions for managing pyogenic(non-tuberculous) discitis in adults. *Joint Bone Spine* 74: 133–39. [CrossRef]

Hayes, A. J., M. Choksey, N. Barnes, and O. C. Sparrow. 1996. Spinal tuberculosis in developed countries: Difficulties in diagnosis. *Journal of the Royal College of Surgeons of Edinburgh* 41: 192–96. [PubMed]

Hee, Hwan T., Mohammad E. Majd, Richard T. Holt, and David Pienkowski. 2002. Bettertreatment of vertebral osteomyelitis using posterior stabilization and titanium mesh cages. *Clinical Spine Surgery* 15: 149–56.

Heller, J. G. 1992. Postoperative infections of the spine. In *The Spine.* Edited by Richard H. Rothman and Frederick A. Simeone. Philadelphia: W.B. Saunders.

Hodgson, A. R., F. E. Stock, H. S. Y. Fang, and G. B. Ong. 1960. Anterior spinal fusion: The operative approach and pathological findings in 412 patients with Pott's disease of the spine. *Journal of British Surgery* 48: 172–78. [CrossRef] [PubMed]

Jain, A. K. 2010. Tuberculosis of the spine: A fresh look at an old disease. *The Journal of Bone & Joint Surgery British Volume* 92: 905–13. [CrossRef]

Jain, Anil K., and Ish Kumar Dhammi. 2007. Tuberculosis of the spine: A review. *Clinical Orthopaedics and Related Research* 460: 39–49. [CrossRef]

Jevtic, Vladimir. 2004. Vertebral infection. *European Radiology* 14: E43–E52. [CrossRef]

Jimenez-Mejias, Manuel E., Juan de Dios Colmenero, Fernando J. Sánchez-Lora, Julián Palomino-Nicás, José M. Reguera, Joaquín García de la Heras, María A. García-Ordoñez, and Jerónimo Pachón. 1999. Postoperative spondylodiskitis: Etiology, clinical findings, prognosis, and comparison with nonoperative pyo-genic spondylodiskitis. *Clinical Infectious Diseases* 29: 339–45. [CrossRef]

Khanna, Rohit K., Ghaus M. Malik, Jack P. Rock, and Mark L. Rosenblum. 1996. Spinal epidural abscess: Evaluation of factors influencing outcome. *Neurosurgery* 39: 958–64.

Kim, Choll W., Andrew Perry, Brad Currier, Michael Yaszemski, and Steven R. Garfin. 2006. fungal infection of the spine. *Clinical Orthopedics and Related Research* 444: 92–99. [CrossRef]

Kornblum, Martin B., David P. Wesolowski, Jeffrey S. Fischgrund, and Harry N. Herkowitz. 1998. Computed tomography-guided biopsy of the spine: A review of 103 patients. *Spine* 23: 81–85. [CrossRef]

Kourbeti, Irene S., Sotirios Tsiodras, and Dimitrios T. Boumpas. 2008. Spinal infections: Evolving concepts. *Current Opinion in Rheumatology* 20: 471–79. [CrossRef] [PubMed]

Kraemer, Robert, Alexander Wild, Holger Haak, Jörg Herdmann, Rüdiger Krauspe, and Jürgen Kraemer. 2003. Classification and management of early complications in open lumbar microdiscectomy. *European Spine Journal* 12: 239–46. [CrossRef] [PubMed]

Krogsgaard, Michael R., Peter Wagn, and Jörgen Bengtsson. 1998. Epidemiology of acute vertebral osteomyelitis in Denmark: 137 cases in Denmark 1978–1982, compared to cases reported to the National Patient Register 1991–1993. *Acta Orthopaedica Scandinavica* 69: 513–17. [CrossRef] [PubMed]

Kulchavenya, Ekaterina. 2014. Extrapulmonary tuberculosis: Are statistical reports accurate? *Therapeutic Advances in Infectious Disease* 2: 61–70. [CrossRef]

Kumar, Raj, Rupant K. Das, and Ashok Kumar Mahapatra. 2010. Role of interferon gamma release assay in the diagnosis of Pott disease. *Journal of Neurosurgery: Spine* 12: 462–66. [CrossRef]

Lee, Sun-Ho, Joo-Kyung Sung, and Yeun-Mook Park. 2006. Single-stage transpedicular decompression and posterior instrumentation in treatment of thoracic and thoracolumbar spinal tuberculosis: A retrospective case series. *Journal of Spinal Disorders & Techniques* 19: 595–602. [CrossRef]

Lestini, William F., and Gordon R. Bell. 1990. Spinal infections: Patient evaluation. *Journal of Neurosurgery: Spine* 2: 244–56.

Li, Yun-Da, Chak-Bor Wong, Tsung-Ting Tsai, Po-Liang Lai, Chi-Chien Niu, Lih-Huei Chen, and Tsai-Sheng Fu. 2018. Appropriate duration of post-surgical intravenous antibiotic therapy for pyogenic spondylodiscitis. *BMC Infectious Diseases* 18: 468. [CrossRef]

Lillie, Patrick, Hiten Thaker, Peter Moss, Joy Baruah, Lorraine Cullen, Damien Taylor, and Gavin Barlow. 2008. Healthcare associated discitis in the era of antimicrobial resistance. *JCR: Journal of Clinical Rheumatology* 14: 234–37. [CrossRef]

Luk, K. D. K. 1999. Tuberculosis of the spine in the new millennium. *European Spine Journal* 8: 338–45. [CrossRef]

Majmundar, Neil, Purvee D. Patel, Vincent Dodson, Ashley Tran, Ira Goldstein, and Rachid Assina. 2019. Parasitic infections of the spine: Case series and review of the literature. *Neurosurgical focus FOC* 46: E12. [CrossRef] [PubMed]

Mallick, I. H., M. H. Thoufeeq, and T. P. Rajendran. 2004. Iliopsoas abscesses. *Postgraduate Medical Journal* 80: 459–62. [CrossRef] [PubMed]

Maslen, David R., Stephen R. Jones, Mark A. Crislip, Raymond Bracis, Ronald J. Dworkin, and Jeffrey E. Flemming. 1993. Spinal epidural abscess: Optimizing patient care. *Archives of Internal Medicine* 153: 1713–21. [CrossRef] [PubMed]

McGuire, Robert A., and Frank J. Eismont. 1994. The fate of autogenous bone graftin surgically treated pyogenic vertebral osteomyelitis. *Clinical Spine Surgery* 7: 206–15.

Michel, Sven C. A., Christian W. A. Pfirrmann, Norbert Boos, and Juerg Hodler. 2006. CT-guided core biopsy of subchondral bone and intervertebral space in suspected spondylodiskitis. *American Journal of Roentgenology* 186: 977–80. [CrossRef]

Mizuno, S. 1967. Posterior approaches in spondylotomy for spinal tuberculosis. *Seikei Geka* 18: 876–82. (In Japanese).

Mylona, Eleni, Michael Samarkos, Eleni Kakalou, Panagiotis Fanourgiakis, and Athanasios Skoutelis. 2009. Pyogenic vertebral osteomyelitis: A systematic review of clinical characteristics. *Seminars in Arthritis and Rheumatism* 39: 10–17. [CrossRef]

Natale, Massimo, M. Lama, Raffaele D'Avanzo, D. Squillante, and F. A. Cioffi. 1992. Postoperative lumbar discitis. *Minerva Anestesiologica* 58: 209–15.

Neumayr, Andreas, Francesca Tamarozzi, Sam Goblirsch, Johannes Blum, and Enrico Brunetti. 2013. Spinal cystic echinococcosis—A systematic analysis and review of the literature: Part 1. Epidemiology and anatomy. *PLoS Neglected Tropical Diseases* 7: e2450. [CrossRef]

Oniankitan, Owonayo, Eyram Fianyo, Kodjovi Kakpovi, Lama K. Agoda-Koussema, and Moustafa Mijiyawa. 2014. Sacrum Pott's disease: A rare location of spine tuberculosis. *The Egyptian Rheumatologist* 36: 209–11. [CrossRef]

Ozuna, Richard M., and Rick B. Delamarter. 1996. Pyogenic vertebral osteomyelitisand postsurgical disc space infections. *The Orthopedic Clinics of North America* 27: 87–94. [CrossRef] [PubMed]

Pamir, M. Necmettin, Koray Ozduman, and Ilhan Elmaci. 2002. Spinal hydatid disease. *Spinal Cord* 40: 153–60. [CrossRef] [PubMed]

Pawar, Uday M., Vishal Kundnani, Vikas Agashe, Amita Nene, and Abhay Nene. 2009. Multidrug-resistant tuberculosis of the spine—Is it the beginning of the end? A study of twenty-five culture proven multidrug-resistant tuberculosis spine patients. *Spine* 34: E806–E810. [CrossRef]

Pertuiset, Edouard, Johann Beaudreuil, Frédéric Lioté, Anne Horusitzky, Farid Kemiche, Pascal Richette, Dominique Clerc-Wyel, Isabelle Cerf-Payrastre, Henri Dorfmann, Jacques Glowinski, and et al. 1999. Spinal tuberculosis in adults: A study of 103 cases in a developed country, 1980–1994. *Medicine (Baltimore)* 78: 309–20. [CrossRef] [PubMed]

Prabhakar, Mukund, Apurv J. Acharya, Dhaval R. Modi, and Bhavin Jadav. 2005. Spinal hydatid disease: A case series. *The Journal of Spinal Cord Medicine* 28: 426–31. [CrossRef]

Rajasekaran, S., Rishi Mugesh Kanna, and Ajoy Prasad Shetty. 2014. Pathophysiology and treatment of spinal tuberculosis. *JBJS Reviews* 2: e4. [CrossRef]

Rankine, J. J., D. A. Barron, P. Robinson, P. A. Millner, and R. A. Dickson. 2004. Therapeutic impact of percutaneous spinal biopsy in spinal infection. *Postgraduate Medical Journal* 80: 607–9. [CrossRef]

Rasouli, Mohammad R., Maryam Mirkoohi, Alexander R. Vaccaro, Kourosh Karimi Yarandi, and Vafa Rahimi-Movaghar. 2012. Spinal tuberculosis: Diagnosis and management. *Asian Spine Journal* 6: 294–308. [CrossRef]

Ratcliffe, J. F. 1985. Anatomic basis for the pathogenesis and radiologic features of vertebral osteomyelitis and its differentiation from childhood discitis: A microarteriographic investigation. *Acta Radiologica Diagnosis* 26: 137–43. [CrossRef]

Rath, Stefan A., Ulrich Neff, Ortwin Schneider, and Hans-Peter Richter. 1996. Neurosurgical management of thoracic and lumbar vertebralosteomyelitis and discitis in adults: A review of 43consecutive surgically treated patients. *Neurosurgery* 38: 926–33. [CrossRef]

Razak, M. M., Z. H. Kamari, and S. Roohi. 2000. Spinal infection—An overview and the results of treatment. *The Medical Journal of Malaysia* 55: 18–28. [PubMed]

Rea, Gary L., John M. McGregor, Carole A. Miller, and Michael E. Miner. 1992. Surgical treatment of the spontaneous spinal epidural abscess. *Surgical Neurology* 37: 274–79. [CrossRef] [PubMed]

Sapico, Francisco L., and John Z. Montgomerie. 1990. Vertebral osteomyelitis. *Infectious Disease Clinics of North America* 4: 539–50. [CrossRef] [PubMed]

Schimmer, Ralph C., Christina Jeanneret, Pierce D. Nunley, and Bernard Jeanneret. 2002. Osteomyelitis of the cervical spine: A potentially dramatic disease. *Journal of Spinal Disorders & Techniques* 15: 110–17.

Schirmer, Patricia, Cybèle A. Renault, and Mark Holodniy. 2010. Is spinal tuberculosis contagious? *International Journal of Infectious Diseases* 14: e659–E666. [CrossRef]

Schuster, James M., Anthony M. Avellino, Frederick A. Mann, Allain A. Girouard, M. Sean Grady, David W. Newell, H. Richard Winn, Jens R. Chapman, and Sohail K. Mirza. 2000. Use of structural allografts in spinalosteomyelitis: A review of 47 cases. *Journal of Neurosurgery: Spine* 93: 8–14. [CrossRef]

Shang, H., D. Y. Wang, and G. H. Wen. 2010. I-Stage surgical treatment for thoracolumbar spinal tuberculosis with kyphosis. *Chinese General Practice* 32: 42.

Sharif, Hassan S. 1992. Role of MR imaging in the management of spinal infections. *AJR. American Journal of Roentgenology* 158: 1333–45. [CrossRef]

Shih, Robert Y., and Kelly K. Koeller. 2015. Bacterial, fungal, and parasitic infections of the central nervous system: Radiologic-pathologic correlation and historical perspectives. *Radiographics* 35: 1141–69. [CrossRef]

Skaf, Ghassan S., Nathalie T. Domloj, Michael G. Fehlings, Carmel H. Bouclaous, Amira S. Sabbagh, Zeina A. Kanafani, and Souha S. Kanj. 2010. Pyogenic spondylodiscitis: An overview. *Journal of Infection and Public Health* 3: 5–16. [CrossRef]

Sobottke, Rolf, Harald Seifert, Gerd Fätkenheuer, Matthias Schmidt, Axel Goßmann, and Peer Eysel. 2008. Current diagnosis and treatment of spondylodiscitis. *Deutsches Ärzteblatt International* 105: 181–87. [CrossRef] [PubMed]

Su, Shou-Hsin, Wen-Cheng Tsai, Chun-Yu Lin, Wei-Ru Lin, Tun-Chieh Chen, Po-Liang Lu, Pei-Ming Huang, Jong-Rung Tsai, Ya-Ling Wang, Ming-Chu Feng, and et al. 2010. Clinical features and outcomes of spinal tuberculosis in southern Taiwan. *Journal of Microbiology, Immunology and Infection* 43: 291–300. [CrossRef] [PubMed]

Tayles, Nancy, and Hallie R. Buckley. 2004. Leprosy and tuberculosis in Iron Age Southeast Asia? *American Journal of Physical Anthropology* 125: 239–56. [CrossRef]

Torabi, Amir M., Mary Quiceno, Dianne B. Mendelsohn, and Craig M. Powell. 2004. Multilevel intramedullary spinal neurocysticercosis with eosinophilic meningitis. *Archives of Neurology* 61: 770–72. [CrossRef] [PubMed]

Tuli, Surendra Mohan. 1993. *Tuberculosis of the Skeletal System*. New Delhi: JayPee Brothers Medical, pp. 144–53.

Tuli, Surendra Mohan. 2007. Tuberculosis of the spine: A historical review. *Clinical Orthopaedics and Related Research* 460: 29–38. [CrossRef]

Tyrrell, P. N. M., V. N. Cassar-Pullicino, and I. W. McCall. 1999. Spinal infection. *European Radiology* 9: 1066–77. [CrossRef]

Velayati, Ali Akbar, Mohammad Reza Masjedi, Parissa Farnia, Payam Tabarsi, Jalladein Ghanavi, Abol Hassan ZiaZarifi, and Sven Eric Hoffner. 2009. Emergence of new forms of totally drug-resistant tuberculosis bacilli: Super extensively drug-resistant tuberculosis or totally drug-resistant strains in Iran. *Chest* 136: 420–25. [CrossRef]

Wong-Chung, John K., Shaheed A. Naseeb, Sangam G. Kaneker, and Ali J. Aradi. 1999. Anterior disc protrusion as a cause for abdominal symptoms in childhood discitis. A case report. *Spine* 24: 918–20. [CrossRef]