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Review Article Spinal infections in children: A review

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ABSTRACT

Spinal infections are uncommon but significant causes of morbidity and hospitalization in the paediatric population. These infections encompass a broad range of conditions, from discitis to osteomyelitis and spinal epidural and intramedullary abscesses. Paediatric spinal infections can be caused by a range of bacterial, viral, fungal and parasitic agents. Ultrastructural differences of the vertebrae and associated structures result in distinct mechanisms of pathogenesis of spinal infections in children compared to adults. The non-specific nature of symptoms produced by them can cause considerable diagnostic delays. Magnetic Resonance (MR) imaging can facilitate early identification of the disease, and distinguish it from other spinal pathologies. The association of antimicrobial resistant bacterial strains from some of the cases appears worrisome; as is the increasing incidence of Kingella kingae infections causing spinal infections. Rest and immobilization are the general treatment, and prompt initiation of antimicrobial therapy is warranted to ensure optimal clinical outcome. Most patients generally have a good prognosis; however, early identification and prompt initiation of antimicrobial therapy is essential to achieve the best therapeutic response.

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1. Introduction

Spinal infections include infections primarily affecting (a) the spinal cord, (b) the nerve roots and meninges, or (c) the vertebrae, intervertebral discs, and epidural space.¹ They are broadly classified as pyogenic or non-pyogenic, with the former category including vertebral osteomyelitis and discitis, while parasitic, fungal, and tuberculous infections constitute the latter category.^{2,3} These infections are rare but significant causes of morbidity and hospitalization in the pediatric population. Though accurate statistics are unavailable from most parts of the world, data from the developed countries reveal an incidence of 0.3 per 100,000 cases among individuals aged less than 20 years^{4,5} (Table 1).⁶⁻¹⁰ Spinal infections can contribute to prolonged hospitalization, unnecessary diagnostic tests and antibiotic use, lost school days, caregiver absenteeism at work, and considerable healthcare expenditure. Corresponding with the increase in general population, the number of individuals with immune compromising conditions, intravenous drug use, and spinal instrumentation, the incidence of these infections is likely to increase in future. These

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infections deserve special clinical attention as they exhibit distinct pathogenic mechanisms, exhibit non-specific symptoms and can be potentially devastating.

In most children, the average age at diagnosis of spinal infections is between 2 and 7 years.¹¹ However, neonates can be affected as well (Table 2).^{11–16}

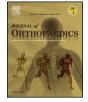
A number of risk factors exist for the acquisition of these infections, and are summarized in Table 3.9,10,13,17-22,26

1.1. Infections of the spinal cord

Spinal infections may be caused by a number of bacterial, viral, fungal, and parasitic agents (Table 4). Intramedullary spinal cord abscesses due to bacterial pathogens are rare in children, though some reviews reported that 20-50% of all cases occurred in children.¹ The reported risk factors include congenital heart disease, immune disorders, long-term use of vascular access devices, spinal cord tumors, and dermal sinuses. Abscesses within and outside the spinal cord may result from dermal sinuses.^{10,24-26} The patients generally have an antecedent history of infection elsewhere, and the spinal lesions may develop from hematologic or lymphatic spread. The condition initially presents as myelitis, and can progress to abscess formation if left untreated.¹

Viral infections often disrupt protective mucosal barriers, promoting the entry and spread of oropharyngeal flora leading







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Table 1

Incidence of spinal infections in children.

Type of infection and age	Incidence	References
All spinal infections, across all ages	2.4 per 100,000 cases per year	4,5
All spinal infections in persons aged ≤20 years	0.3 per 100,000 cases per year	
Spinal epidural abscesses, across all ages	0.2–2.0 per 10,000 hospital admissions per year	6-8
Spinal epidural abscesses	0.6 per 10,000 hospital admissions per year	9
Intramedullary spinal cord abscesses	Extremely rare	10

Table 2

Age distribution of spinal infections in children.

Type of infection	Observation with respect to age	References
All spinal infections Osteomyelitis	Mean age at diagnosis: 2–7 years Rare in children aged \leq 3 years	11 12
Discitis	Rare in children aged \geq 8 years	12
Spinal epidural abscesses	Mostly in children aged 7.6–8 years	13,14
	Occasionally in neonates	15
Spinal subdural abscesses	Mostly in infants and toddlers	16
Intramedullary spinal	Mean age at diagnosis: 36 months	10
cord abscesses	(range, 18 days to 17 years)	

Table 3

Risk factors associated with spinal infections in children.

Type of infection	Risk factor	References
All infections	Hematologic seeding from infectious focus in skin, genitourinary tract, gastrointestinal tract, oral cavity, respiratory tract	2
Spinal epidural abscesses	Underlying disease (in 1/3rd of children)	13
	Immunocompromised states (sickle cell anemia, polymyositis, leukemia)	9,17
	Tuberculosis	18
	Vertebral osteomyelitis	19
	Trauma and hematoma (in 17–24% of cases)	20,21
Spinal subdural abscesses	Midline congenital dermal sinuses and hematogenous spread of infection	22
Intramedullary spinal	Dermal sinus tracts (53% of cases)	26
cord abscesses	Hematogenous spread of infection from foci in vulva, urinary tract, lungs, middle ear, endocardium, kidney and sagittal sinus	10,26
	Intramedullary spinal cord tumors, trauma, bacterial meningitis	10,25,26

to respiratory infections. The ensuing bacteremia may seed the organisms within the joint space, bones or intervertebral discs, creating foci of suppurative infection.²⁷ Reports have suggested that varicella infection may increase the risk for spinal epidural abscesses due to secondary bacterial.^{28–30}

Infection with poliovirus, herpes virus, and cytomegalovirus (especially in patients with HIV) can also result in myelitis. With interruption of wild poliovirus circulation from many parts of the world, myelitis associated with it has decreased in the recent years. However, an earlier study reported an increase in herpes zoster myelitis.³¹

Fungal pathogens including *Candida* and *Aspergillus* can also cause spinal infections in children.¹

Table 4	4
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Common pathogens associated with spinal infections in children.

Category	Name of the pathogen
Bacterial	Staphylococcus aureus (including Methicillin Resistant Staphylococcus aureus, MRSA) Group B streptococci Escherichia coli Listeria spp. Haemophilus influenzae Neisseria meningitidis Pneumococcus Neisseria meningitides Brucella spp. Kingella kingae
Viral	Herpes viruses Polio viruses Cytomegalovirus
Fungal	Aspergillus spp. Candida spp.
Parasitic	Taenia solium Schistosoma

Among parasitic agents, *Schisotosoma* sp. is frequently reported in children.¹ Rarely, cysticercosis can also be a cause of spinal masses in children, especially in endemic areas.³² It can manifest in extraspinal (vertebral) or intraspinal (epidural, subdural, arachnoid or intramedullary) forms.

About 44% of children with intramedullary spinal cord abscesses may be afebrile, and localizing neurologic signs may be absent. The major symptoms include weakness, numbness, paralysis, paresthesias, and incontinence of bowel/bladder.²³

1.2. Infections of the nerve roots and meninges

Bacterial meningitis is the commonest spinal infection in children, and the infectious process might involve the dura, leptomeninges, and CSF. Spinal invasion mostly follows hematologic seeding, traumatic inoculation, local extension from sinusitis, mastoiditis, otitis, brain abscesses, etc., and may subsequently spread along the peripheral nervous system. Group B streptococcal infections account for about 50% cases in infants in neonates, followed by *Escherichia coli* and *Listeria*. About 40–60% of cases in younger infants are caused by *Haemophilus influenzae*, followed by *Neisseria meningitidis* and *Pneumococcus*. The predominant isolates from older children and adults include *Pneumococcus*, *N. meningitidis*, and *Staphylococcus aureus*.¹

1.3. Infections of the vertebrae, discs, and epidural space

Childhood spondylodiscitis may encompass a spectrum of spinal infections ranging from discitis to vertebral osteomyelitis, with associated soft tissue abscesses.¹² A recent study reported 12 cases of spondylodiscitis among 540 pediatric patients presenting with back pain.³³ The condition may involve any location within the spine, but commonly occurs in the lumbar region in children less than 3 years of age.³⁴

1.3.1. Vertebral osteomyelitis

Vertebral osteomyelitis is uncommon in children, and accounts for only 1–2% of all cases. The mean age at presentation is usually over 7 years. Most infections typically involve the lumbar or thoracic regions. The pathogen most frequently associated with vertebral osteomyelitis in children is *S. aureus*.²³

1.3.2. Bacterial discitis

Discitis is most common between children aged 1–5 years. Studies indicate that spinal infections in children begin in the vertebral body near the end plate, in the form of microabscesses.³⁵ Rupture of the focus of infection may then spread the infection from the end-plate to the disk through the perforating vascular channels, and its further extension to the paravertebral area, epidural space, and vertebral bodies in proximity.^{1,36} Compression of the spinal cord or nerve root may also follow from spinal instability caused by erosive changes in the disk, bone, and posterior elements.³⁷

The symptoms of discitis include inability or failure to walk, abdominal pain, chronic back-ache, irritability, fever, and local tenderness.^{1,38} The symptoms may occur gradually over 2–4 weeks. The primary site of involvement in children is the L3–4 and L4–5 interspaces. X-rays may be normal or show disk space narrowing after 2–4 weeks. Bone scans are useful in localizing the pathology, but need to be followed up by additional imaging.³⁹

Diagnosis is often delayed due to vague symptoms in toddlers like inability to walk, mild fever and irritability, and spondylodiscitis is identified upon MR imaging.^{39–44}

Most cases resolve spontaneously, but there are differing opinions on avoiding antibiotic therapy. With time, the lesions may remain stable or progress further, with the latter indicating failure of the therapeutic regimen.

The patients usually have normal WBC counts but elevated inflammatory markers.^{33,39}

The microbiological etiology of bacterial discitis is not exactly known, as laboratory confirmation is not attempted routinely, and therapy is guided by clinical and neuroimaging findings. Identification of the infecting organism may not be feasible in about one-third of patients.⁴⁴ S. *aureus* and streptococcus species predominate in culture-positive cases, while gram-negative bacilli form the major isolates in intravenous drug abusers. Infections by Mycobacterium tuberculosis, fungi, and parasites are rare, and occur mainly in immune-compromised individuals. Strains of coagulasenegative staphylococci (CONS) and Streptococcus viridans may cause chronic infections. Immunocompromised individuals and children with sickle-cell anemia may develop salmonella discitis and osteomyelitis.^{1,45} Incidence of osteomyelitis has decreased in the recent years, compared to discitis.¹ Strains of Kingella kingae (a gram-negative coccobacillus of low virulence) producing a potent cytotoxin called RTX as significant causes of self-limiting spondylodiscitis in children under 3 years of age.³⁴

The most severe form of childhood spondylodiscitis occurs in infants under 6 months, and is often associated with multiple foci of infection and septicemia. The affected infants manifest severe vertebral damage and destruction, and major kyphosis can result from involvement of the thoracic spine. Neurologic manifestations occur frequently, and *S. aureus* is the major isolate from over 80% of cases.^{46–48} About 60% of childhood spondylodiscitis occurs in children aged 6 months to 4 years, and most yield sterile biopsies or grow *Kingella kingae*.^{34,49} Among patients older than 4 years, there is a greater risk for vertebral osteomyelitis, and the *S. aureus* is the predominant isolate. Gower's sign (pathognomonic of Duchenne muscular dystrophy) can be helpful leading to a diagnosis in young children (aged 3.3–8 years) with lumbar discitis, and it resolves upon appropriate therapy.⁵⁰

1.4. Tubercular spondylodiscitis

Tuberculosis of the spine is a significant health burden in developing countries, though it is also an emerging problem in the developed world. In contrast to adults, the disease is more aggressive in children, who may develop large abscesses. The affected children usually present with pain and signs of chronic infection. Thoracolumbar region is most commonly affected in children.⁵¹ Paraplegia, however, is rare in children, compared to adult patients with spinal tuberculosis. The disease more

commonly follows pulmonary or genitourinary tuberculosis in children, but can also be the initial manifestation.

In a case series, 76% of the affected children were less than 5 years of age, 50% presented with neurological deficits, and paraspinal abscesses developed in 62%. The authors reported recovery within 6 months in 50% of the patients, with prompt therapy.⁵²

Three forms of the disease are distinguished, as anterior, paradiscal, and central types.¹ In the anterior type, the disease progresses from the anterior and inferior vertebral bodies and under the anterior longitudinal ligament to adjacent vertebrae. The disk space may be intact, show narrowing but does not show enhancement. The vertebral lesions may give rise to large pre- or para-vertebral abscesses. Paradiscal form of the disease is comparatively rare in children and usually commences from the lateral sides of the discs, with resultant narrowing of disk space, and development of large abscesses from it. The central type is characterized by infection arising from the middle of the vertebral body, often producing a vertebra plana, leading to acute angle kyphosis. It tends to progress posteriorly to the spinal canal, with thecal sac compression.

1.4.1. Spinal epidural abscess

An epidural abscess manifests as a collection of pus between the bone and dura mater,³⁵ and occurs more frequently in girls than in boys. It generally follows discitis or osteomyelitis of pyogenic or tubercular etiology.⁵³ A usual origin is by hematogenous spread of infection from the urogenital tract, skin, lungs, or teeth. The patients usually exhibit the triad of fever, excruciating pain and rapid progression of neurologic symptoms, warranting prompt identification and treatment.⁹ The diagnosis can be overlooked in afebrile neonates and infants showing non-specific symptoms. Often, neurologic symptoms may be absent or masked, especially in children who received antibiotics earlier. Epidural abscesses may develop days to weeks after spinal instrumentation or surgery in children. Worsening pain at sites of surgery or instrumentation, along with purulent discharge, may be useful clinical pointers in this entity.

1.4.2. Spinal subdural abscess

Fever and meningism may be present in 50% of the cases.⁵⁴ Symptoms usually progress gradually, localised back pain with sphincter involvement can be seen in about 25% of the cases. Rapid onset of neurologic deficit, symptoms suggestive of transverse myelitis, high fever and backache in children with dermal sinuses may indicate intradural pathology.¹¹ Cauda equina syndrome may develop with increase in size of the lesion.

2. Diagnostic evaluation

Estimation of WBC count, ESR, and CRP yields only non-specific information in pediatric spondylodiscitis.^{11,12,33} Invasive diagnostic procedures such as needle aspiration and biopsy have attendant procedural and anesthetic risks, and the indication for these are not firmly established at present.^{34,49} Magnetic resonance imaging can facilitate early diagnosis of pediatric spinal infections.¹

In infections of the spinal cord, MR generally reveals hyperintense T2 signals and expansion of the cord. A capsule may be present around the lesion, occurring as a thin hypointense strip. Signal enhancement may or may not be present, upon contrast administration, depending on the stage of inflammation. Following therapy, the signal intensity of T2 weighted images decreases, and ring enhancement is seen, which subsequently declines.¹

Sensory deficits associated with characteristic vesicular rash aids clinical diagnosis in herpes myelitis, and MR may reveal focal, rounded, hyperintense signals with or without contrast enhancement, on ipsilateral half of the spinal cord, in T2-weighed images. Diffuse arachnoiditis is characteristic of acute bacterial meningitis. The surface of the brain and spinal cord may show a purulent exudate, yielding contrast-enhanced signals upon MR imaging. Clinical and physical examination generally suffice to make a clinical diagnosis, while neuroimaging studies may facilitate early diagnosis, evaluation of response to therapy, and identification of potential complications.

In bacterial discitis, early MR imaging studies may reveal a reduction in the height of the intervertebral disk, and swelling of the annulus, which may appear hyperintense in T2-weighted images. Contrast enhancement is often noted. Initially, the vertebral plates and subchondral regions may show only minor changes, but with progression, the end plates may become irregular and blurred. Later, the end plates and vertebrae become bright on T2-weighted images. With subsequent spread of infection, the adjacent vertebrae may show abnormal signal intensity in the canal for the basivertebral vein.¹

MR has a sensitivity of 90% in detecting epidural abscesses. Imaging usually reveals a prominent epidural space with intermediate signal intensity in T1- and T2-weighted images, and shows homogeneous enhancement with gadolinium. Prompt antibiotic therapy suffices in these cases, and surgery is not warranted. Cases with abscess formation may show a rimenhancing epidural lesion. The non-enhancing center is suggestive of pus. Such lesions warrant surgical drainage. The length of the abscess may span 2–4 vertebral bodies. Cord edema and poor venous drainage following involvement of Batson plexus may contribute to hyperintensity in T2-weighted images, at locations above, below and at the level of the lesion.

Percutaneous biopsy under CT guidance gives variable success in finding organism (36–91%) as it depends on initial antibiotic treatment and the technique. Overall biopsy has high specificity but low sensitivity.⁵⁶

3. Management

Spinal stabilization by immobilization is recommended in discitis, and epidural, subdural, and intramedullary abscesses. Analgesics may be provided for pain control. Usually, parenteral antibiotics to cover *S. aureus* are given, and a biopsy may be considered if symptoms persist after 1–2 weeks. Surgical intervention may be necessary in cases with abscesses or progression of neurologic deficits. In cases of subdural abscesses managed with antibiotic therapy alone, a low threshold for surgical intervention is to be maintained, in the setting of persistent fever or progressive symptoms.²³ Epidural abscess are located behind the vertebral body; hence, a laminectomy is appropriate for decompression. Instrumentation with fusion is reserved for the cases where posterior elements are affected.⁵⁷

4. Conclusion

Spinal infections remain uncommon entities in pediatric clinical practice. Due to non-specific clinical presentations, they may lead to missed or delayed diagnoses. Clinicians need to maintain awareness of these rare and potentially fatal infections and attempt to identify and treat them at the earliest in order to preserve the best therapeutic outcomes.

Conflicts of interest

The author has none to declare.

References

 Rossi A. Pediatric spinal infection and inflammation. Neuroimaging Clin N Am. 2015;25(2):173–191.

- Wiley A, Trueta J. The vascular anatomy of the spine and its relationship to pyogenic vertebral osteomyelitis. J Bone Jt Surg Br. 1959;41–B:796–809.
- Ratcliffe J. Anatomic basis for the pathogenesis and radiologic features of vertebral osteomyelitis and its differentiation from childhood discitis: a microarteriographic investigation. Acta Radiol Diagn (Stoch). 1985;26(2):137–145.
- Krogsgaard MR, Wagn P, Bengtsson J. Epidemiology of acute vertebral osteomyelitis in Denmark: 137 cases in Denmark 1978–82, compared to cases reported o the National Patient Register 1991–1993. Acta Orthop Scand. 1998;69(5):513–517.
- Grammatico L, Baron S, Rusch E, et al. Epidemiology of vertebral osteomyelitis (VO) in France: analysis of hospital-discharge data 2002–2003. *Epidemiol Infect*. 2008;136(5): 653–660.
- Kurpad S. Infections of the spinal canal. In: Hall W, McCutcheon I, eds. In: *Neurosurgery*. Par Ridge, Illinois: The American Association of Neurological Surgeons; 2000:65–80.
- Rigamonti D, Liem D, Sampath P, et al. Spinal epidural abscess: contemporary trends in etiology, evaluation and management. *Surg Neurol.* 1999;52(2): 189–196.
- Wheeler D, Keiser P, Rigamonti D, Keay S. Medical management of spinal epidural abscesses: case report and review. *Clin Infect Dis.* 1992;15(1):22–27.
- Ghia A. Infections of the spinal axis. In: Albright AL, Abeoson PD, eds. In: Principles and Practice of Pediatric Neurosurgery 5th ed. New York: Thieme; 2008:1204–1206.
- Simon JK, Lazareff JA, Diament MJ, Kennedy WA. Intramedullary abscess of the spinal cord in children: a case report and review of the literature. *Pediatr Infect Dis J.* 2003;22(2):186–192.
- Fernandez M, Carrol C, Baker CJ. Discitis and vertebral osteomyelitis in children: an 18-year review. *Pediatrics*. 2000;105(6):1299–1304.
- Early SD, Kay RM, Tolo VT. Childhood diskitis. J Am Acad Orthop Surg. 2003;11(6): 413–420.
- Auletta JJ, John CC. Spinal epidural abscesses in children: a 15-year experience and review of the literature. *Clin infect Dis.* 2001;32(1):9–16.
- Martin RJ, Yuan HA. Neurosurgical care of spinal epidural, subdural and intramedullary abscesses and arachnoiditis. Orthop Clin N Am. 1996;27(1):125–136.
- Paro-Panjan D, Grcar LL, Pedaric-Meglic N, Tekavcic I. Epidural cervical abscess in a neonate. Eur J Pediatr. 2006;165(10):730–731.
- Mohindra S, Gupta R, Chhabra R, et al. Infected intraparenchymal dermoids: an underestimated entity. J Child Neurol. 2008;23(9):1011–1016.
- Bowen DK, Mitchell LA, Burnett MW, Rooks VJ, Martin JE. Spinal epidural abscess due to tropical pyomyositis in immunocompetent adolescents. *J Neurosurg Pediatr.* 2010;6(1):33–37.
- Kumar A, Singh AK, Badole CM, Patond KR. Tubercular epidural abscess in children: report of two cases. *Indian J Tuberc*. 2009;56(4):217–219.
- Baker AS, Ojemann RG, Swartz MN, Richardson Jr EP. Spinal epidural abscess. N Engl J Med. 1975;293(10):463–468.
- Jacobsen FS, Sullivan B. Spinal epidural abscesses in children. Orthopedics. 1994;17(12):1131–1138.
- Rubin G, Michowiz SD, Ashkenasi A, Tadmor R, Rappaport ZH. Spinal epidural abscess in the pediatric age group: case report and review of literature. *Pediatr Infect Dis J.* 1993;12(12):1007–1011.
- 22. Wright R. Surgical management of intracranial and intraspinal infections. In: Sweet W, Schmidek HH, eds. In: Operative Neurosurgical Techniques: Indications, Methods and Results. Philadelphia: Saunders; 1995:1673–1679.
- International Society for Pediatric Neurosurgery. The ISPN Guide to Pediatric Neurosurgery. http://ispn.guide/book [accessed 15.02.16].
- Dev R, Husain M, Gupta A, Gupta RK. MR of multiple intraspinal abscesses associated with congenital dermal sinus. AJNR. 1997;18:742–743.
- Koppel B, Daras M, Duffy K. Intramedullary spinal cord abscess. *Neurosurgery*. 1990;26(1):145–146.
- Morandi X, Mercier P, Fournier HD, Brassier G. Dermal sinus and intramedullary spinal cord abscess. Report of two cases and review of the literature. *Childs Nerv* Syst. 1999;15(4):202–207.
- Yagupsky P. Kingella kingae: from medical rarity to an emerging paediatric pathogen. Lancet Infect Dis. 2004;4(6):358–367.
- Cossu G, Farhane MA, Daniel RT, Messerer M. Spinal epidural abscess from group A Strepococcus after varicella infection: a case report and review of the literature. Childs Nerv Syst. 2014;30(12):2129–2133.
- Quach C, Tapiero B, Noya F. Group a streptococcus spinal epidural abscess during varicella. *Pediatrics*. 2002;109(1):E14.
- Ziebold C, von Kries R, Lang R, Weigl J, Schmitt HJ. Severe complications of varicella in previously healthy children in Germany: a 1-year survey. *Pediatrics*. 2001;108(5): E79.
- Friedman DP. Herpes zoster myelitis: MR appearance. AJNR Am J Neuroradiol. 1992;13(5):1404–1406.
- Homans J, Khoo L, Chen T, Commins DL, Ahmed J, Kovacs A. Spinal intramedullary cysticercosis in a five-year-old child: case report and review of the literature. *Pediatr Infect Dis J.* 2001;20(9):904–908.
- Spencer SJ, Wilson NIL. Childhood discitis in a regional childrens hospital. J Pediatr Orthop. 2012;21(3):264–268.
- 34. Ceroni D, Cherkaoui A, Ferey S, Kaelin A, Schrenzel J. Kingella kingae osteoarticular infections in young children: clinical features and contribution of a new specific real-time PCR assay to the diagnosis. J Pediatr Orthop. 2010;30(3): 301–304.
- Wenger DR, Davids JR, Ring D. Discitis and osteomyelitis. In: Weinstein SL, ed. In: The Pediatric Spine, Principles and Practice. New York: Raven; 1994:813–816.
- 36. Tay B, Deckey J, Hu SS. Spinal infections. J Am Acad Orthop Surg. 2002;10(3): 188–197.
- 37. Govender S. Spinal infections. Bone Jt J. 2005;87-B(11):1454-1458.

- Karabouta Z, Bisbinas I, Davidson A, Goldsworthy LL. Disitis in toddlers: a case series and review. Acta Pediatr. 2005;94(10):1516–1518.
- Chandrasenan J, Klezl Z, Bommireddy R, Calthorpe D. Spondylodiscitis in children. J Bone Jt Surg Br. 2011;93-B(8):1122–1125.
- Arthurs OJ, Gomez AC, Heinz P, Set PA. The toddler refusing to weight-bear: a revised imaging guide from a case series. *Emerg Med J.* 2009;26(11):797–801.
- **41.** van den Huel R, Hertel M, Gallagher J, Naido V. A toddler who refused to stand or walk: lumbar spondylodiscitis. *BMJ Case Rep.* 2012.
- Lim S, Sinnathamby W, Noordeen H. Refusal to walk in an afebrile well toddler. Postgrad Med J. 2002;78:568.
- Ramphul M, Chawla K. Not walking? An uncommon spinal pathology. BMJ Case Rep. 2015.
- Maslen DR, Jones SR, Crislip MA, Bracis R, Dworkin RJ, Flemming JE. Spinal epidural abscess. Optimizing patient care. Arch Intern Med. 1993;153(14):1713–1721.
- Broner FA, Garland DE, Zigler JE. Spinal infections in the immunocompromised host. Orthop Clin N Am. 1996;27(1):37–46.
- Eismont FJ, Bohlman HH, Soni PL, Goldberg VM, Freehafer AA. Vertebral osteomyelitis in infants. J Bone Jt Surg Br. 1982;64(1):32–35.
- Garron E, Viehweger E, Launay F, Guillaume JM, Jouve JL, Bollini G. Nontuberculous spondylodiscitis in children. J Pediatr Orthop. 2002;22(3):321–328.

- Tsirikos AI, Tome-Bermejo F. Spondylodiscitis in infancy: a potentially fatal condition that can lead to major spinal complications. J Bone Jt Surg Br. 2012;94(10): 1399–1402.
- 49. Ceroni D, Kampouroglou G, Valaikaite R, Anderson della Llana R, Salvo D. Osteoarticular infections in young children: what has changed over the last years? J Pediatr Orthop. 2014;144:w13971.
- Mirovsky Y, Copeliovich L, Halperin N. Gowers' sign in children with discitis of the lumbar spine. J Pediatr Orthop. 2005;14(2):68–70.
- Shanley DJ. Tuberculosis of the spine: imaging features. AJR Am J Roentgenol. 1995;164(3):659–664.
- Sharif HS. Role of MRI in the management of spinal infections. AJR Am J Roentgenol. 1992;158(6):1333–1345.
- Ruiz A, Post MJ, Ganz WI. Inflammatory and infectious processes of the cervical spine. *Neuroimaging Clin N Am.* 1995;5(3):401–426.
- Leite CC, Jinkins JR, Escobar BE, et al. MR imaging of intramedullary and intraduralextramedullary spinal cysticercosis. AJR Am J Roentgenol. 1997;169(6):1713–1717.
- Kizilkilic O, Hanci M. Percutaneous biopsy for diagnosis of spinal infections. World Spinal Column J. 2015;6(January (1)).
 Govender S. Spinal infections. J Bone Jt Surg. 2005;87-B:1454-1458. http://
- Govender S. Spinal infections. J Bone Jt Surg. 2005;87-B:1454–1458. http:// dx.doi.org/10.1302/0301-620X.87B11. 16294.