Pediatric spinal infections

Raj Kumar[1,2], Rajnish Arora[2]

Departments of Neurosurgery, [1]SGPGIMS, Lucknow, Uttar Pradesh, [2]All India Institute of Medical Sciences, Rishikesh, Uttarakhand, India

ABSTRACT

The infections of the spinal axis in children are rare when compared with adults. They encompass a large spectrum of diseases ranging from relatively benign diskitis to spinal osteomyleitis and to the rapidly progressive, rare, and potentially devastating spinal epidural, subdural, and intramedullary spinal cord infections. We present a comprehensive review of the literature pertaining to these uncommon entities, in light of our experience from northern India. The most prevalent pediatric spinal infection in Indian scenario is tuberculosis, where an extradural involvement is more common than intradural. The craniovertebral junction is not an uncommon site of involvement in children of our milieu. The majority of pyogenic infections of pediatric spine are associated with congenital neuro-ectodermal defects such as congenital dermal sinus. The clinico-radiological findings of various spinal infections commonly overlap. Hence the endemicity of certain pathogens should be given due consideration, while considering the differential diagnosis. However, early suspicion, rapid diagnosis, and prompt treatment are the key factors in avoiding neurological morbidity and deformity in a growing child.

Key words: Pediatric, Pott's disease, spinal epidural infections, spinal infections

INTRODUCTION

Spinal infections are relatively rare in the pediatric population as compared to adults.[1-3] There have been various published series in the world literature; however, the Indian scenario is relatively less well-explored.

The pediatric spinal infections can be broadly classified into extradural, intradural extramedullary, or intramedullary. Many of them are associated with congenital conditions peculiar to the pediatric spine such as neuro-ectodermal defects, most notably congenital dermal sinus; others are due to certain endemic conditions such as tuberculosis (TB), while few have been associated with iatrogenic causes such as instrumentations and lumbar punctures, etc. Though a variety of etiological agents, including mycobacteria, pyogenic bacteria, fungi, parasites and viruses are responsible for spinal infections, the clinical presentations are usually overlapping and include backache, fever, irritability, motor and sensory deficits, bladder-bowel involvement and spinal deformity, in various combinations. The diagnosis is particularly challenging in neonates and infants. The presence of a dermal sinus should be checked for in all pediatric patients as part of physical examination, especially in those with dermal stigmata as it may be associated with devastating spinal infection.[4]

The endemicity of pathogens should be taken into account while making a differential diagnosis. A high index of suspicion, prompt clinical evaluation and appropriate diagnosis are essential for the treatment of pediatric spinal infections, and these are critical for prevention of permanent neurologic damage or development of late bony deformity. The authors have attempted to review the scenario, clinical presentation and management principles of pediatric spinal infections, in view of the vast experience of the senior author (RK) in treating such infections at a premier tertiary care institute of northern India.

Access this article online	
Quick Response Code:	Website: www.ijns.com
	DOI: 10.4103/2277-9167.138891

EXTRADURAL INFECTIONS

The extradural compartment infections can be vertebral; vertebral with pus/granulation; epidural abscess or empyema; diskitis or a combination of these. Due to the involvement of the developing spine, spinal deformities are of a significant concern in the patients of this age group. Early diagnosis and adequate treatment for vertebral osteomyelitis are mandatory to prevent severe complications and lifelong disabilities.[5] In developing

Address for correspondence: Dr. Raj Kumar,
All India Institute of Medical Sciences, Rishikesh, Uttarakhand, India. E-mail: rajkumar1959@gmail.com

countries like India, TB remains the most common spinal infection even in the pediatric population.

Tubercular osteomyelitis (also called as Pott's disease)

Tuberculosis is caused by *Mycobacterium tuberculosis* and *Mycobacterium bovis* in most cases. Atypical mycobacterial infections are not very rare in our country. Though the disease is endemic in certain parts of the world, but now it is considered as a common disease worldwide. The skeletal involvement is present in 1-3% of patients suffering from TB affecting any body tissue, and spinal spondylitis is the most common (50%) manifestation of skeletal TB.[6,7] Most commonly spinal TB affects thoracic and thoracolumbar spine and is a leading cause of paraplegia in developing countries.[8] Craniovertebral junction (CVJ) TB is not a rare presentation in India.[9,10] Although no age is immune to the disease but there is a controversy regarding the age predilection of the disease. It is documented that Pott's is a disease of adults in affluent countries, and disease of the first three decades in other regions.[11] In our country, it is seen in the first decade in 50% of cases and is encountered in only 25% cases after 20 years of age.[9] There have been rare case reports of Pott's spine in infants[12,13] resulting from maternal genitourinary TB.

Etiopathogenesis

The spinal TB is a result of hematogenous dissemination from the primary focus in the lungs or the lymph nodes. The central type of vertebral TB spreads along with Batson's plexus of veins, while paradiscal infection spreads through the arteries. The anterior type of vertebral body TB results from the extension of the abscess beneath the anterior longitudinal ligament and periosteum. The pathogenesis of Pott's disease has been divided to following stages: (1) inflammatory edema and exudation stage, (2) the necrosis and cavitation stage (3) the destruction and deformation stage, (4) the healing and repair stage. Morphologically, two types of bone and joint TB have been recognized: the caseous type and exudative type with abscess formation. The exudative type is more common in children while the granular type is frequent in adults.[11]

Clinical presentation

The clinical symptoms of spinal TB in children are often insidious and include back pain, fever, paraparesis, sensory disturbance, bowel/bladder dysfunction[6] and local swelling due to the formation of a cold abscess. However, in our experience the initial presentation of a cold abscess can be far away from the site of lesion.[14] Majority of patients with spinal TB have evident collapse of vertebrae and destruction of adjacent tissue, probably due to delay in diagnosis, sometimes mimicking neoplasms. The disease may result in bone destruction, spinal deformity, and neural complications such as arachnoiditis, intramedullary tuberculoma, and spinal cord compression from epidural abscesses [Figure 1]. Approximately, 3% of children with spinal TB develop severe kyphosis (>60°). Risk factors for the development of severe kyphotic deformity are as follows: Age <10 years; involvement of >3 vertebral bodies; and thoracic spine localization. Severe kyphosis is cosmetically unacceptable and results in spinal cord compression over the apex of the deformity and cardiopulmonary dysfunction from restrictive lung disease.[6]

Diagnosis

None of the investigative modalities individually is efficacious enough to make a diagnosis of TB of the spine with such a degree of certainty as to obviate the need for tissue diagnosis or bacteriological confirmation. A combination of magnetic resonance imaging (MRI), bone scan, ELISA and the Quantiferon assay provides reasonably certain diagnosis in 90% of cases, thereby providing the grounds for starting a safe trial of antituberculous chemotherapy.[15] MRI is extremely useful in diagnosing the difficult and rare sites of disease like the CVJ. It detects the marrow changes, exudative and granulation types, extra- and intra- dural disease, and radiological response to treatment in the early follow-up period around 6-8 weeks.[11] For confirmatory diagnosis of Pott's disease, we recommend that, the combined results of Ziehl-Neelsen microscopy, BACTEC culture and histopathology are most appropriate. However, for rapid confirmation, the combination of PCR amplification and histopathology offer a better prospect.[16]

Treatment

Early TB spondylitis without spinal cord compression or neurological deficits can be treated conservatively

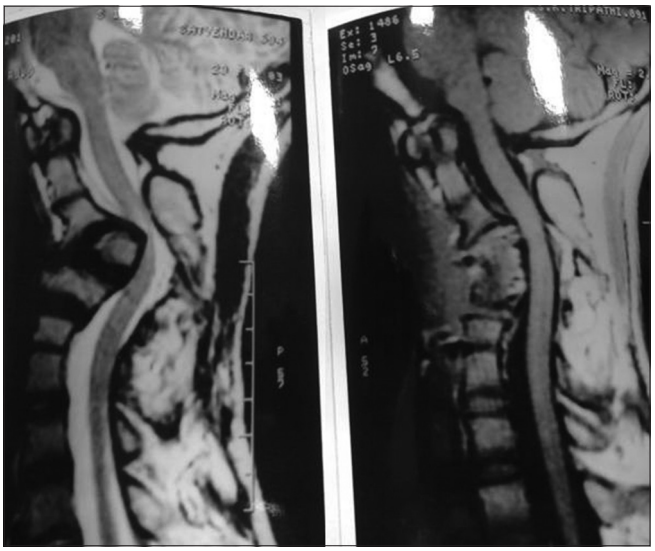

Figure 1: Illustrations of upper cervical Pott's spine, showing vertebral collapse, associated collections, thecal compression and kyphotic deformities

with chemotherapy (antitubercular therapy [ATT]). Traditionally, the antitubercular drugs have been classified into first and second line drugs. The first line drugs are most effective group of agents against the mycobacteria and include isoniazid, rifampicin, pyrazinamide, ethambutol and streptomycin. Second line drugs are less effective, more toxic and more expensive than the first line drugs (e.g. amikacin, kanamycin flouroquinolones [ciprofloxacin, ofloxacin, moxifloxacin, levofloxacin, gatifloxacin], paraaminosalicylic acid cycloserine, terizidone, ethionamide, protionamide, clofazamine, linezolid, amoxicillin/clavulunate, thioacetazone, cilastin/imipenem, clarithromycin). Isoniazid (H) is typically used in a dose of 5 (4-6) mg/kg/day and can cause side effects like peripheral neuropathy and reversible hepatotoxicity. The dose of rifampicin (R) is 10 (8-12) mg/kg/day and commonly observed side effects are hepatotoxicity, flu-like syndrome, orange discoloration of body and body fluids. Pyrazinamide (Z) is recommended at 25 (20-30) mg/kg/day. It can cause hyperuricemia, hepatotoxicity, and arthralgia. Ethambutol (E) is given in the dose of 15 (13-17) mg/kg/day. Retrobulbar optic neuritis is the most serious side effect, and this drug is contraindicated in children who might not report the visual changes at an early stage. Streptomycin (S) is an aminoglycoside used in dose of 15 (12-18) mg/kg/day and can adversely affect both vestibular and hearing functions and can cause nephrotoxicity.[17]

Spinal TB is the severe extrapulmonary form of TB and the scheduled WHO category-I treatment is 2 months of intensive phase with 4 drugs (HRZE) followed by 4 months of the continuation phase of two (HR) drugs.[17] However, because of the serious risk of disability and mortality and because of difficulties of assessing treatment response, WHO recommends 9 months of treatment for TB of bones or joints.[18] The American Thoracic Society recommends 6 months of chemotherapy for spinal TB in adults and 12 months in children.[19] Many experts still prefer the durations of 12-24 months or until radiological or pathological evidence of regression of disease occurs.[20-23]

Surgery in cases of Pott's spine is recommended for patients with spinal cord compression or neurological compromise, significant spinal deformity, persistent severe axial pain, or disease progression on maximal medical therapy. Various surgical approaches have been reported for treatment of TB spondylitis in the pediatric age group. Any surgical approach must take into account the skeletal immaturity of young children and should minimize the number of fused segments (especially in the thoracic spine) to avoid complications such as iatrogenic short trunk, crankshaft deformity, and pulmonary hypoplasia from restricted growth of the rib cage.[6]

We have surgically treated 74 cases of pediatric Pott's spine between 2002 and 2010.[9] Follow up of >6 months is available for 64 cases. The most common site of involvement was thoracic region (33%) followed by CVJ in 26%, thoracolumbar in 15% and cervical and lumbosacral region in 9% cases each. Extradural involvement (79%) predominated over intradural or intramedullary location. 69% had a poor neurological grade[24] at presentation. In our series, transthoracic approach was the most common approach performed in 28% patients. Other approaches used were transmanubrial, transoral, posterolateral and retroperitoneal approach. The choice of the surgical approach depends upon the site of maximum compression, involvement of bony element and soft tissue inflammation/abscess, degree of compression, and stability of spine [Figure 2a, b and Table 1].

The short and long-term clinical outcomes of more extensive surgery are similar compared with debridement alone, but the correction of kyphosis and deformity was better in children undergoing extensive surgery with anterior reconstruction than in patients undergoing debridement alone. In addition, extensive surgery seems to decrease the length of drug therapy, because clinical and radiographic outcomes were similar for chemotherapeutic regimens prescribed for 6, 9, or 18 months when combined with extensive surgery.[6,25]

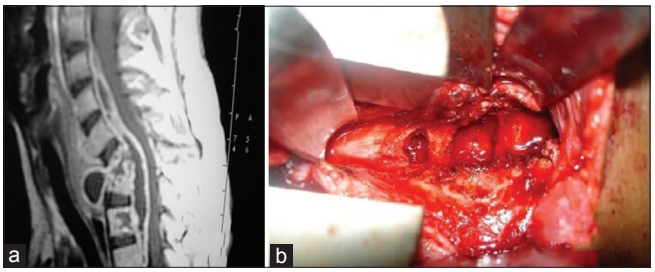

Figure 2: (a) Sag contrast enhanced T1-weighted magnetic resonance imaging of a 13-year-old girl showing Pott's spine affecting upper dorsal spine. She had spastic paraparesis of 13 month duration. She was operated by transmanubrial approach. (b) intraoperative photograph

Table 1: surgical approach used in 64 cases of pott's spine[9]

Surgical approach	Number (%)
Transoral+post fusion	12 (18)
Post fusion	5 (7.5)
Transmanubrial	4 (6)
Transthoracic	18 (28)
Postero-lateral	10 (15)
Laminectomy	11 (16.5)
Retroperitoneal	4 (6)

However, we always prefer minimum treatment for 18 months or even 2 years in selected cases. Overall, in our series 72% of the cases showed some improvement, 25% remained same, while 3% worsened.[9] A significant improvement is possible even in the patients belonging to the poor grade with judicious use of the surgical approach and ATT.[9] The good prognostic factors are partial cord compression, short duration of neurological symptoms, young age and good general condition of the patient.

Another important point to be taken into consideration is about the multidrug resistant-TB (MDRTB). India is one of the global leaders for MDRTB cases. India reports of 2.3% and 17.2% of MDR cases from new and previously treated cases, respectively.[26,27] Among the first line drugs, the maximum resistance is observed to isoniazid, rifampicin, streptomycin respectively and least to pyrazinamide. It is recommended that culture sensitivity of pus/granulation tissue should be performed in each suspected case of MDRTB. However this facility may not be available at each center. When culture sensitivity is not available or when there is progression of disease in spite of standard ATT and in situations of repeated negative cultures, an objectively designed empirical treatment should be considered consisting of at least four new drugs-not taken by patient in past. It is not recommended to add a single drug to failing regime. No drugs of same pharmacological group or drugs with similar adverse effect profile should be clubbed together in regime. Only pyrazinamide from first line therapy should be reused. ATT should not be stopped by fixed time frame and we need to evaluate spinal lesions clinically, hematologically and radiologically. All drug resistance patients should be serially monitored with 3 months complete blood count, erythrocyte sedimentation rate C-reactive protein and MRI along with drug toxicity effects.[26-30]

Pyogenic vertebral osteomyelitis and diskitis

Pyogenic vertebral osteomyelitis is rare in children as compared to adults.[31] The mean age of diagnosis of pyogenic spinal infections ranges from 2 to 7 years.[32] Osteomyelitis is uncommon in children 3 years of age or younger, and diskitis is rare in children older than 8 years of age.[33] The lumbar spine is the most commonly involved region. Vertebral osteomyelitis affects more frequently the vertebral body, and involvement of posterior elements is rare.[34] The diagnosis should be considered in a child who develops a limp or in an infant who displays a postural or positional preference.[35]

Most common cause of vertebral osteomyelitis is due to hematogenous spread from other sites of infection such as soft tissue, skin or genitourinary tract; however it may occur due to direct extension from retropharyngeal/retroperitoneal and abdominal abscess. Some cases occur following trauma. Pyogenic spinal infections are typically the result of methicillin-sensitive *Staphylococcus aureus* that spreads hematogenously. The incidence of methicillin-resistant *S. aureus* has increased in recent years. Gram-negative rods are other important pathogens. Rare causes include *Clostridium difficile*[36] and Cat scratch disease.[37] Inflammation of the intervertebral disk is an uncommon entity. Noniatrogenic cases of diskitis are seen almost exclusively in children and the etiology remains controversial. However, the causative organism is identifiable in 60-82% of cases via direct biopsy.[38]

Blood cultures should be obtained for children with vertebral osteomyelitis because it is the definite guide for providing accurate treatment. Computed tomographic-guided aspiration of abscess should be considered for patients with negative blood cultures. Plain radiographs, bone scintigraphy, and MRI are useful for making the diagnosis. Although MRI is the imaging method of choice for vertebral osteomyelitis and discitis, however in the early stages, it may show subtle, nonspecific endplate subchondral changes and a repeat examination may be required to show the typical features.[39]

Antimicrobial therapy for 6-12 weeks is usually successful, and an early transition to oral form does not increase the risk of treatment failure.[5,40] A 3-6 weeks course of antibiotics and supportive therapy is effective in majority of cases of diskitis.

Postoperative surgical site infections (SSI) also deserve a special mention. The incidence in pediatric spinal fusion procedures has been reported as high as 3.4%. Significant risk factors for deep SSI include inappropriate timing of preoperative antibiotic prophylaxis, previous spine surgery, presence of a complex underlying medical condition, age, >10 vertebrae fused, and an increased estimated blood loss per kilogram body weight.[41] Debridement with implant removal may be required, especially for late-onset infections associated with previous spinal surgery.[5]

Fungal osteomyelitis

The fungal osteomyelitis occurs rarely. Candida osteomyelitis have been reported[42] in children on immunomodulating agents, while invasive aspergillosis have been reported in an immunocomptent child.[43] These infections require prolonged supervised treatment with newer agents like azoles (fluconazole, itraconazole, voriconazole and posaconazole), polyenes (amphotericin deoxycholate and liposomal amphotericin B), flucytosine (5-fluorocytosine),

and echinocandins (caspofungin, micafungin and anidulafungin).[44]

Among azoles, fluconazole is least toxic. Others may have hepatic and cardiac side effects, voriconazole is contraindicated in renal failure and these (voriconazole and posaconazole) require monitoring of blood levels.[45] The polyenes are used for Salvage therapy and empiric use due to broad activity. Nephrotoxicity and infusion reactions are their major side effects. Flucytocine is used in combination with other antifungal agents as monotherapy using this agent causes rapid development of resistance. It is only given orally and has bone marrow toxicity.

Candida osteomyelitis requires fluconazole (6 mg/kg) daily for 6-12 months or a lipid formulation of amphotericin B, 3-5 mg/kg daily for several weeks, followed by fluconazole for 6-12 months.[46] Echinocandins may also be used as first line therapy, while flucytosine can be used in combination of amphotericin in severely ill patients.

Voriconazole has the best clinical activity against *Aspergillus*, and is considered first-line therapy for infections caused by this pathogen. Amphotericin B products can be used as second-line agents in patients who cannot tolerate or are failing voriconazole therapy, and echinocandins can be used as alternative or salvage therapy.[45]

Spinal epidural empyema and abscess

A spinal epidural abscess (SEA) or empyema constitutes a neurosurgical emergency in which early diagnosis and prompt decompression are necessary to avoid permanent cord damage. This entity has an incidence of 0.2-1.2 per 10,000 hospital admissions.[47] Pediatric population is affected even rarely.[38] Average age of diagnosis in pediatric population is 7.6-8 years,[48] but cases have been reported even in neonatal period, where diagnosis is a challenge.[47] The chances of misdiagnosis are quite high, because of which severe cord compression results in suboptimal recovery. The prognosis is guarded in cases with longstanding history and chronic cord compression.

Majority of cases are due to hematogenous spread from an antecedent infection and due to direct spread from a primary infective source (vertebral osteomyelitis/dermal sinus). Iatrogenic cases of pediatric epidural abscess have been reported following epidural catheter placement.[49] SEA can also be associated with pyomyositis.[50]

The most commonly affected site in SEA is dorsal (33%), lumba (27%) and then dorsolumbar (15%). The abscess is located commonly over the posterior aspect of epidural space (86%), other locations are less common (7% laterally, 5% anteriorly and 2% circumferential). The average length of collection is 7 segments. Performing a lumbar puncture may yield frank pus in more than half of cases (52%). However, cerebrospinal fluid (CSF) may be normal in upto 10% of cases whereas, a few cases have raised proteins as the only abnormality.[51] The most common organisms associated with of SEA are *S. aureus*, followed by *Streptococci.* In our cases, staphylococci were encountered in 70%, streptococci in 8%, while enterobacteria and anaerobes in about 6% cases [Figure 3].

Antibiotics should be started as soon as there is a suspicion of SEA and should be directed against the causative organism as described. It can be treated by various types of surgical decompression procedures, including needle aspiration, laminotomy, laminectomy, flavotomy and irrigation[47] or corpectomy.[52] The outcome is mainly determined by the preoperative neurological condition and the localization of abscess. The recurrence rate is dependent on the longitudinal extent of the mass and the intra-operative finding of granulation tissue, but not on the placement of postoperative suction-irrigation drainage. The interlaminar decompression should be considered instead of laminectomy in lumbar SEA and in impending anterior column instability due to spondylitis. Intra-operative ultrasound is a beneficial aid for the determination of the extent of decompression during surgery and is practicable even through a narrow interlaminar bony window. After immediate surgical decompression, appropriate antibiotic treatment should be initiated according to culture sensitivity and maintained for at least 6 weeks (2 weeks intravenous + 4 weeks of oral antibiotics). It may be extended if osteomyelitis is also present. Spinal stability should be a concern after treatment, during ambulatory outpatient visits.[51]

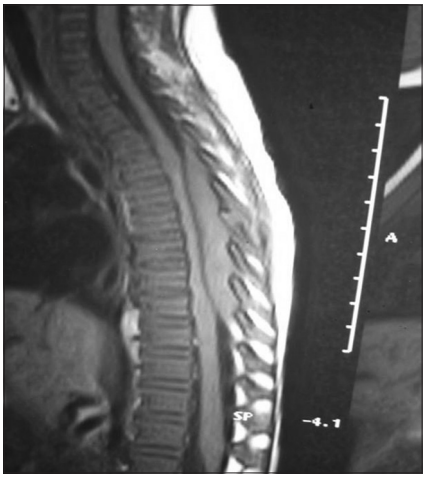

Figure 3: Spinal epidural abscess extending over 7 segments in dorsal region

Early recognition with a combination of surgical decompression and antibiotic therapy provide excellent results with full recovery of neurological function. There are reports in literature where conservative management alone has been successful.[53-55] However, caution should be exercised to closely monitor the patient's recovery as any deterioration in the neurological state warrants surgery.

Spinal subdural abscess

SSA is a relatively rare entity occurring predominantly in 5th through 7th decade of life and it is even rarer in children. A total of 73 cases of pediatric spinal subdural infections have been reported till 2013.[56] Among the most recent cases, the age range is from 4 weeks to 20 years (mean, 6.5 years), and over half (53%) of spinal subdural infections in children are associated with spinal dysraphism or other congenital abnormalities of the spine. The commonest organism to infect the spinal subdural space in children is *M. tuberculosis* and the thoracic spinal region is most commonly infected. The etiology is known to be hematogenous spread, iatrogenic contamination (lumbar puncture, previous surgical history) and local extension.[56-58] The predisposing factors are anatomical abnormalities of the spinal cord, congenital dermal sinus and dermoid cyst,[59] AIDS, Diabetes mellitus, and repetitive meningitis.[60] A chronic subdural abscess can mimic an intra-dural extramedullary mass (IDEM).[61] MRI is the investigation of choice [Figure 4]. However it may not be able to differentiate between epidural and subdural collection in all cases. But as both these entities require a surgical procedure for definitive treatment, it is only a relative liability. The management is similar to SEA.

Intramedullary lesions

Pyogenic intramedullary abscess

An intramedullary abscess of the spinal cord (IASC) represents a rare disease associated with a potentially devastating outcome.[62] IASC may present at any age, but children under the age of 5 years are more likely affected.[63] The thoracolumbar region is by far the most common site of involvement though it can affect any part of spinal cord. *S. aureus* is usually the most common cultured organism in this group; however, other pathogens may also be implicated but less frequently. Specific infections, such as Acid Fast Bacilli, *Brucella melitensis*/brucellosis are to be considered mainly in endemic areas.[63,64]

The source of IASC can be classified into hemopoietic spread, contagious spread, and spread from an unknown source (cryptogenic). Contrary to the mechanism found in adults, the dermal sinus is the leading cause of IASC in children. It has been found to be the source of infection in 45% of children compared with 24% in all age groups.[65] Still, an IASC is the least common infection-related complication of a dermal sinus. Iatrogenic cases following lumbar puncture have also been reported.[66] Once infection develops in the spinal cord, it has a tendency to spread longitudinally along the fibers of the cord itself.[67] The mean range of involvement is three to six levels[68] and involvement of the entire cord is exceedingly rare.[65,67,69,70]

In an acute presentation, symptoms of infection (e.g. fever, backache, malaise) are common. Chronic cases might mimic features of an intramedullary tumor presenting with neurological symptoms.[71] MRI helps in surgical planning and also demonstrates the co-existence of congenital anomalies. Localized abscess with variable enhancement may resemble neoplasms. In children, limited laminectomy with myelotomy focused over the most bulbous segment of the spinal cord for drainage of the abscess and adequate antibiotics is the treatment of choice [Figures 5a and b]. Timely surgical drainage along with adequate antibiotics should be performed before irreversible spinal cord damage can occur from vascular compromise and local mechanical pressure within a restricted and nonexpansible space. The holocord abscess may pose a management challenge

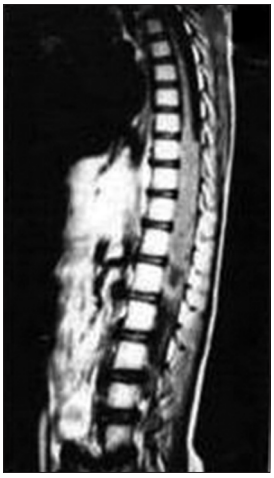

Figure 4: Intradural extramedullary spinal cord empyema in a child

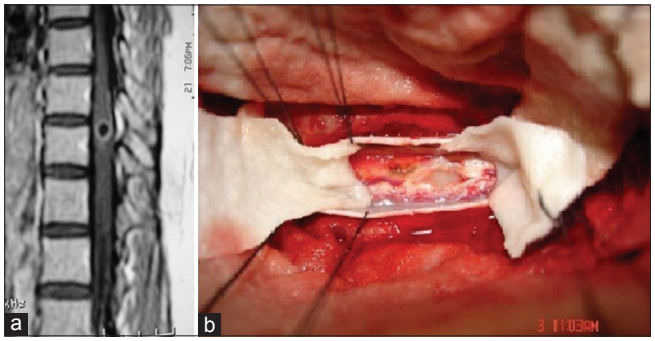

Figure 5: Sagittal contrast enhanced magnetic resonance imaging (a) and intraoperative image (b) of an intramedullary abscess

but the above mentioned strategy has been found to be effective. Bunyaratavej *et al*. have successfully treated holocord intramedullary abscess due to a dermal sinus in a 2-month-old child with limited myelotomy and aspiration.[65] It is highly recommended to surgically excise the dermal sinus once it is found, in order to eliminate potentially debilitating and serious central nervous system (CNS) infection.

Tubercular intramedullary abscess

Intramedullary and subarachnoidal tubercular abscesses are rare forms of spinal TB as compared with extradural collections secondary to vertebral TB. Less than 10 cases of intradural tubercular abscesses have been reported till date.[67,72-75] Intramedullary and subarachnoid tubercular abscesses present with acute or subacute, progressive motor and sensory deficits. Tubercular abscesses are iso to hypointense in T1 weighted images, iso to hyperintense in T2 -weighted MR images and show ring enhancement after contrast administration. There can be enhancement of the overlying meninges. However, these imaging features may be shared by bacterial abscesses, demyelination, and intramedullary tumors. The presence of an IDEM mass at the lower end of the spinal cord associated with holocord T2 hyperintensities of the spinal cord has been described and should raise the suspicion of intramedullary tubercular abscess. Biopsy along with microscopic examination and culture are essential to confirm the diagnosis. As the number of tubercular abscesses reported in the literature is limited, definitive guidelines cannot be laid down for their management. Surgery is aimed for biopsy and safe maximal decompression as complete resection is rather difficult. This is to be followed by a full course of antituberculous chemotherapy. Complete drainage of the abscess is not required. Antitubercular drugs can help in the resolution of the residual abscess.[72,76-78]

Intra medullary granulomas

Intramedullary spinal tuberculomas (IMT) are rare, even in geographical areas where TB is endemic. In a large series from a tertiary care center in India, the incidence of spinal intramedullary tuberculoma was 1 in 175 cases of spinal TB and 1 in 1600 cases of central nervous system TB.[77] In endemic regions, in patients with evidence of TB elsewhere, intramedullary spinal lesion can be of tuberculous etiology [Figure 6].

Gadolinium-diethylenetriamine pentaacetic acid enhanced MRI can significantly increase the diagnostic accuracy of IMT in conjunction with radiological and serological evidence of TB. Follow-up MRI becomes essential not only to assess the efficacy of treatment but also to detect any paradoxical increase of size of the lesion. These lesions usually have a good response to medical treatment, and early diagnosis avoids any unnecessary surgical intervention. On treatment with chemotherapy the lesion regresses in size and disappears, to be replaced by an area of gliosis, which probably is seen as an area of hypointensity on MRI.[77]

We have managed three cases of intramedullary tuberculomas in pediatric age group, and in our experience, if the cord is severely inflamed and compressed in such cases, the deficits are severe and recovery remains guarded.

Fungal intramedullary abscess is usually found in immune-compromised patients e.g. acute leukemia, and causative agents include Cryptococcosis, Aspergillosis, *Blastomycosis, and Coccoidiodomycoses*. A rapid diagnosis followed by aggressive antifungal therapy for 6 weeks is recommended [Figure 7].

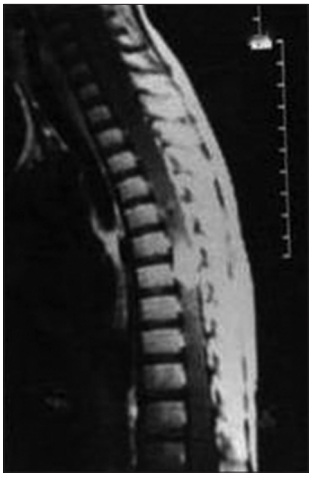

Figure 6: Intramedullary tubercular granuloma affecting dorsal cord in a pediatric patient

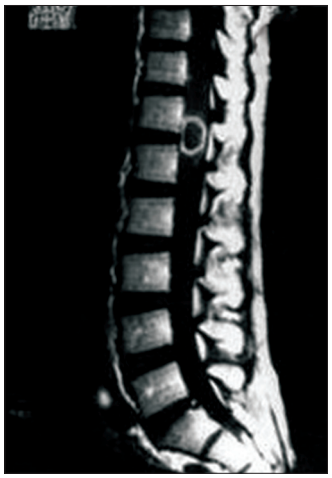

Figure 7: CEMR shows a ring enhancing intramedullary lesion at conus. It was found to be a fungal abscess at conus

PARASITIC INFESTATIONS OF SPINE

These include Cysticercosis, hydatid disease, and Schistosomiasis. Uncommon protozoan like *Balantidium coli* have been reported to cause osteomyelitis and resultant myelopathy.

Cysticerci

Neurocysticercosis (NCC) is the commonest infestation of CNS.[79,80] Spinal involvement is present in 1-5% cases of nervous system Cysticercosis. Commonly it affects the adults, however cases of Intramedullary NCC have been reported in children as young as 5 years.[81] Spinal cysticercosis has been classified anatomically as extraspinal (vertebral)[82] or intraspinal (epidural, subdural, arachnoid, or intramedullary). Most (80%) spinal NCC occurs in the subarachnoid space.[83] The thoracic region is most commonly affected followed by cervical. Intramedullary Cysticercosis (IMC) is far less common with only 53 cases have been reported until 2010.[84] IMC can cause neurological symptoms/deficitis by mechanical compression by cyst, inflammatory changes and cord edema, (secondary to metabolic products/degenerative larval remains) and gliosis due to vasculopathy.[85]

On MRI, IMC is usually a cystic lesion within the spinal cord. The differential diagnosis of an intramedullary cystic lesion is extensive, such as arachnoid cyst, ependymal cyst, neurenteric cyst, sarcoidosis, neoplasms such as ependymoma, and infections such as abscess.[80] Presumptive preoperative diagnosis of spinal cysticercosis is easy when there is associated Cerebral Cysticercosis. The CSF examination and cysticercal antibodies in CSF and as well as in serum help in diagnosis.

The current therapy for spinal NCC with cord or root compression is laminectomy and resection followed by medical management (albendazole - 15 mg/kg/day for 4-6 weeks in conjuction with steroids).[79] In cases that total resection is not feasible due to holocord involvement, the limited resection with cyst removal and chemotherapy for the treatment of spinal NCC may be an option. Surgery can eliminate only compressive element and this should be performed before the cord changes take place.[82] Except for being used after surgery, albendazole also could be used independently in the conservative treatment for the patients whom are highly suspected as IMC and whose clinical courses are stable.[85] The potential advantages of medical therapy alone include avoidance of surgery and treatment of surgically unreachable and multifocal cysticercus.

Hydatid disease

Hydatid disease is caused by the larvae of the tapeworm *Echinococcus granulosus* and *Echinococcus multilocularis* and is endemic in certain parts of world including Indian subcontinent.[86] Spinal involvement has been reported in only 0.5-1% of the cases where 90% of them are confined to the vertebrae and the epidural space [Figure 8]. Spinal hydatid disease has been classified by Braithwaite and Lees into five types: (1) primary intramedullary hydatid cyst, (2) intradural extramedullary hydatid cyst, (3) extradural intraspinal hydatid cyst, (4) hydatid disease of the vertebrae and (5) paravertebral hydatid disease. The first three types are rare-occasional reports are there.[87-89]

The diagnosis of hydatid disease is mainly established by combination of serological tests and imaging findings. MRI features can also help differentiate between a viable and a succumbed cyst. En bloc removal surgery is the treatment of choice in hydatid cyst disease. Careful puncture and aspiration of cyst contents, followed by total extirpation of the cyst wall, can be performed when total removal is difficult and hazardous. Chemotherapy usually includes well known antihelmintic drugs: albendazole or mebendazole and have to be given for a long period (3 month to 1-year).[89]

Schistosomiasis

Schistosomiasis (or bilharziasis) is an endemic parasitic disease caused by Trematoda platyhelminthes of the genus *Schistosoma*. Neuroschistomiasis typically affects young males, who, with no other complaints of schistosomal infection, present with a flaccid paraplegia secondary to a conus medullaris or cauda equine lesion or transverse myelitis, and are bedridden at presentation (70%). Although the onset of the disease may vary from sudden

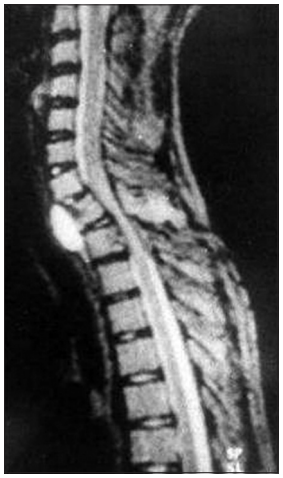

Figure 8: Sagittal T2-weighted magnetic resonance imaging of a pediatric patient shows upper dorsal hydatid infestation

to progressive, it arises in most instances in a subacute or chronic manner, with progressive worsening of the deficits. Treatment requires drugs like praziquantal and steroids and surgery in some cases. Schistosomiasis is an endemic disease in Africa, South America like in Brazil and the Caribbean Islands, and should be considered in differential diagnosis of transeverse myelitis in travelers or emigrants returning from endemic areas.[90]

CONCLUSION

The pediatric spinal infections are relatively rare when compared to adults. TB is the most prevalent pediatric spinal infection in Indian scenario. The extradural involvement is more common than intradural spinal TB and CVJ is not an uncommon site of involvement in children of our mileau. Majority of pyogenic infections of pediatric spine are associated with congenital ectodermal defects such as congenital dermal sinus. As a part of routine clinical examination; all newborns should be checked for the presence of dermal sinus or other congenital defects. Once it is discovered; prophylactic surgical resection is recommended regardless of age, to avoid serious neurosurgical complications. Fungal infections are usually found in immuno-compromised children. As the clinico-radiological findings commonly overlap in many cases of pediatric spinal infections, endemicity of certain pathogens should be given due consideration while considering differential diagnosis. Early suspicion, rapid diagnosis and prompt treatment are important to avoid neurological morbidity and deformity in a growing child.

REFERENCES

1. Ghia A. Infections of the spinal axis. In: Albright AL, Adelson PD, editors. Principles and Practice of Pediatric Neurosurgery. 5th ed. New York: Thieme; 2008. p. 1204-6.
2. Grammatico L, Baron S, Rusch E, Lepage B, Surer N, Desenclos JC, *et al.* Epidemiology of vertebral osteomyelitis (VO) in France: Analysis of hospital-discharge data 2002-2003. Epidemiol Infect 2008;136:653-60.
3. Krogsgaard MR, Wagn P, Bengtsson J. 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 Orthop Scand 1998;69:513-7.
4. Morimoto K, Takemoto O, Nakamura H, Takeuchi M. Spinal dermal sinus associated with intramedullary abscess and dermoid. Pediatr Neurosurg 2003;39:225-6.
5. Wu SY, Wei TS, Chen YC, Huang SW. Vertebral osteomyelitis complicated by iliopsoas muscle abscess in an immunocompetent adolescent: Successful conservative treatment. Orthopedics 2012;35:e1576-80.
6. Rangel-Castilla L, Hwang SW, Whitehead WE, Curry DJ, Luerssen TG, Jea A. Surgical treatment of thoracic Pott disease in a 3-year-old child, with vertebral column resection and posterior-only circumferential reconstruction of the spinal column: Case report. J Neurosurg Pediatr 2012;9:447-51.
7. Gautam MP, Karki P, Rijal S, Singh R. Pott's spine and paraplegia. JNMA J Nepal Med Assoc 2005;44:106-15.
8. Tuli SM. Results of treatment of spinal tuberculosis by "middle-path" regime. J Bone Joint Surg Br 1975;57:13-23.
9. Kumar R, Srivastava AK, Tiwari RK. Surgical management of Pott's disease of the spine in pediatric patients: A single surgeon's experience of 8 years in a tertiary care center. J Pediatr Neurosci 2011;6:S101-8.
10. Kalra SK, Kumar R, Mahapatra AK. Tubercular atlantoaxial dislocation in children: an institutional experience. J Neurosurg 2007;107:111-8.
11. Kumar R. Spinal tuberculosis: With reference to the children of northern India. Childs Nerv Syst 2005;21:19-26.
12. Kumar A, Ghosh SB, Varshney MK, Trikha V, Khan SA. Congenital spinal tuberculosis associated with asymptomatic endometrial tuberculosis: A rare case report. Joint Bone Spine 2008;75:353-5.
13. Peterson RR, Agarwal I, Gibikote S. Spinal tuberculosis in an infant associated with maternal urinary tuberculosis. Indian J Tuberc 2012;59:168-70.
14. Kumar R, Chandra A. Gluteal abscess: A manifestation of Pott's spine. Neurol India 2003;51:87-8.
15. Kumar R, Das RK, Mahapatra AK. Role of interferon gamma release assay in the diagnosis of Pott disease. J Neurosurg Spine 2010;12:462-6.
16. Kumar M, Kumar R, Srivastva AK, Nag VL, Krishnani N, Maurya AK, *et al.* The efficacy of diagnostic battery in Pott's disease: A prospective study. Indian J Orthop 2014;48:60-6.
17. Rajasekaran S, Khandelwal G. Drug therapy in spinal tuberculosis. Eur Spine J 2013;22 Suppl 4:587-93.
18. WHO. Treatment of Tuberculosis: guidelines. 4th ed. Who/htm/tb/2009.420. Geneva: WHO; 2009.
19. Bass JB Jr, Farer LS, Hopewell PC, O'Brien R, Jacobs RF, Ruben F, *et al.* Treatment of tuberculosis and tuberculosis infection in adults and children. American Thoracic Society and The Centers for Disease Control and Prevention. Am J Respir Crit Care Med 1994;149:1359-74.
20. Kotil K, Alan MS, Bilge T. Medical management of Pott disease in the thoracic and lumbar spine: A prospective clinical study. J Neurosurg Spine 2007;6:222-8.
21. Cormican L, Hammal R, Messenger J, Milburn HJ. Current difficulties in the diagnosis and management of spinal tuberculosis. Postgrad Med J 2006;82:46-51.
22. van Loenhout-Rooyackers JH, Verbeek AL, Jutte PC. Chemotherapeutic treatment for spinal tuberculosis. Int J Tuberc Lung Dis 2002;6:259-65.
23. Donald PR. The chemotherapy of osteo-articular tuberculosis with recommendations for treatment of children. J Infect 2011;62:411-39.
24. Kumar R, Kalra SK, Mahapatra AK. A clinical scoring system for neurological assessment of high cervical myelopathy: Measurements in pediatric patients with congenital atlantoaxial dislocations. Neurosurgery 2007;61:987-93.
25. Upadhyay SS, Saji MJ, Yau AC. Duration of antituberculosis chemotherapy in conjunction with radical surgery in the management of spinal tuberculosis. Spine (Phila Pa 1976) 1996;21:1898-903.
26. WHO. Multidrug and Extensively Drug-Resistant TB (M/XDR-TB): 2010 Global Report on Surveillance and Response. Who/htm/tb/2010.3. Geneva: WHO; 2010.
27. WHO. Anti-tuberculosis Drug Resistance: Report No. 4, the WHO/IUALTD Global Project on Anti-Tuberculosis Drug Resistance Surveillance. Who/htm/tb/2008.394. Geneva: WHO; 2008. p. 1-142.
28. Mohan K, Rawall S, Pawar UM, Sadani M, Nagad P, Nene A, *et al.* Drug resistance patterns in 111 cases of drug-resistant tuberculosis spine. Eur Spine J 2013;22 Suppl 4:647-52.
29. Pawar UM, Kundnani V, Agashe V, Nene A, Nene A. 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 (Phila Pa 1976) 2009;34:E806-10.
30. Jain AK, Srivastava A, Saini NS, Dhammi IK, Sreenivasan R, Kumar S. Efficacy of extended DOTS category I chemotherapy in spinal tuberculosis based on MRI-based healed status. Indian J Orthop 2012;46:633-9.
31. Ogden AT, Kaiser MG. Single-stage debridement and instrumentation for pyogenic spinal infections. Neurosurg Focus 2004;17:E5.
32. Fernandez M, Carrol CL, Baker CJ. Discitis and vertebral osteomyelitis in children: an 18-year review. Pediatrics 2000;105:1299-304.
33. Early SD, Kay RM, Tolo VT. Childhood diskitis. J Am Acad Orthop Surg 2003;11:413-20.
34. Reiss-Zimmermann M, Hirsch W, Schuster V, Wojan M, Sorge I.

Pyogenic osteomyelitis of the vertebral arch in children. J Pediatr Surg 2010;45:1737-40.
35. Germain ML, Krenzer KA, Hasley BP, Varman M. 11-month-old child refuses to sit up. Pediatr Ann 2008;37:290-3.
36. Al-Najjar A, Al-Rawahi GN, Hoang LM, Kollmann TR. Clostridium difficile vertebral osteomyelitis. Pediatr Infect Dis J 2013;32:1030-2.
37. Tasher D, Armarnik E, Mizrahi A, Liat BS, Constantini S, Grisaru-Soen G. Cat scratch disease with cervical vertebral osteomyelitis and spinal epidural abscess. Pediatr Infect Dis J 2009;28:848-50.
38. Ghia A, Proctor MR. Infections of the spinal axis. In: Albright AL, Pollack IF, Adelson PD, editors. Principles and Practice of Pediatric Neurosurgery. 2nd ed. New York: Thieme; 2008. p. 1198-9.
39. Dunbar JA, Sandoe JA, Rao AS, Crimmins DW, Baig W, Rankine JJ. The MRI appearances of early vertebral osteomyelitis and discitis. Clin Radiol 2010;65:974-81.
40. Glazer PA, Hu SS. Pediatric spinal infections. Orthop Clin North Am 1996;27:111-23.
41. Milstone AM, Maragakis LL, Townsend T, Speck K, Sponseller P, Song X, *et al.* Timing of preoperative antibiotic prophylaxis: a modifiable risk factor for deep surgical site infections after pediatric spinal fusion. Pediatr Infect Dis J 2008;27:704-8.
42. Huang A, Huang C, Kugathasan S. Vertebral osteomyelitis due to Candida parapsilosis in a child with Crohn disease while receiving anti-TNF therapy. J Pediatr Gastroenterol Nutr 2013;56:e23-6.
43. Karthik K, Shetty AP, Rajasekaran S. Spontaneous cord transection due to invasive aspergillus spondylitis in an immunocompetent child. Eur Spine J 2011;20 Suppl 2:S188-92.
44. Fisher BT, Zaoutis TE. Treatment of invasive candidiasis in immunocompromised pediatric patients. Paediatr Drugs 2008;10:281-98.
45. Kriengkauykiat J, Ito JI, Dadwal SS. Epidemiology and treatment approaches in management of invasive fungal infections. Clin Epidemiol 2011;3:175-91.
46. Pappas PG, Kauffman CA, Andes D, Benjamin DK Jr, Calandra TF, Edwards JE Jr, *et al.* Clinical practice guidelines for the management of candidiasis: 2009 update by the Infectious Diseases Society of America. Clin Infect Dis 2009;48:503-35.
47. Tovar-Spinoza Z, Bode M. Spinal epidural abscess in a neonate. J Neurosurg Pediatr 2011;7:205-8.
48. Auletta JJ, John CC. Spinal epidural abscesses in children: A 15-year experience and review of the literature. Clin Infect Dis 2001;32:9-16.
49. Lin YC, Greco C. Epidural abscess following epidural analgesia in pediatric patients. Paediatr Anaesth 2005;15:767-70.
50. Bowen DK, Mitchell LA, Burnett MW, Rooks VJ, Martin JE. Spinal epidural abscess due to tropical pyomyositis in immunocompetent adolescents. J Neurosurg Pediatr 2010;6:33-7.
51. 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;12:1007-11.
52. Paro-Panjan D, Grcar LL, Pecaric-Meglic N, Tekavcic I. Epidural cervical abscess in a neonate. Eur J Pediatr 2006;165:730-1.
53. Leys D, Lesoin F, Viaud C, Pasquier F, Rousseaux M, Jomin M, *et al.* Decreased morbidity from acute bacterial spinal epidural abscesses using computed tomography and nonsurgical treatment in selected patients. Ann Neurol 1985;17:350-5.
54. Mampalam TJ, Rosegay H, Andrews BT, Rosenblum ML, Pitts LH. Nonoperative treatment of spinal epidural infections. J Neurosurg 1989;71:208-10.
55. Messer HD, Lenchner GS, Brust JC, Resor S. Lumbar spinal abscess managed conservatively. Case report. J Neurosurg 1977;46:825-9.
56. Sandler AL, Thompson D, Goodrich JT, van Aalst J, Kolatch E, El Khashab M, *et al.* Infections of the spinal subdural space in children: A series of 11 contemporary cases and review of all published reports. A multinational collaborative effort. Childs Nerv Syst 2013;29:105-17.
57. Wright R. Surgical management of intracranial and intraspinal infections. In: Sweet W, Schmidek HH, editors. Operative Neurosurgical Techniques: Indications, Methods, and Results. Philadelphia: Saunders; 1995. p. 1673-9.
58. Mohindra S, Gupta R, Chhabra R, Gupta SK, Pathak A, Bal AK, *et al.* Infected intraparenchymal dermoids: An underestimated entity. J Child Neurol 2008;23:1011-6.
59. Chen CY, Lin KL, Wang HS, Lui TN. Dermoid cyst with dermal sinus tract complicated with spinal subdural abscess. Pediatr Neurol 1999;20:157-60.
60. Bako W, Raczkowska-Kozak J, Liberek A, Góra-Gebka M. Subdural empyemas - a rare complication of meningococcal cerebrospinal meningitis in children. Med Sci Monit 2000;6:1008-12.
61. Lim HY, Choi HJ, Kim S, Kuh SU. Chronic spinal subdural abscess mimicking an intradural-extramedullary tumor. Eur Spine J 2013;22 Suppl 3:S497-500.
62. Gerlach R, Zimmermann M, Hermann E, Kieslich M, Weidauer S, Seifert V. Large intramedullary abscess of the spinal cord associated with an epidermoid cyst without dermal sinus. Case report. J Neurosurg Spine 2007;7:357-61.
63. Al Barbarawi M, Khriesat W, Qudsieh S, Qudsieh H, Loai AA. Management of intramedullary spinal cord abscess: Experience with four cases, pathophysiology and outcomes. Eur Spine J 2009;18:710-7.
64. Vajramani GV, Nagmoti MB, Patil CS. Neurobrucellosis presenting as an intra-medullary spinal cord abscess. Ann Clin Microbiol Antimicrob 2005;4:14.
65. Bunyaratavej K, Desudchit T, Pongpunlert W. Holocord intramedullary abscess due to dermal sinus in a 2-month-old child successfully treated with limited myelotomy and aspiration. Case report. J Neurosurg 2006;104:269-74.
66. da Silva PS, de Souza Loduca RD. Intramedullary spinal cord abscess as complication of lumbar puncture: A case-based update. Childs Nerv Syst 2013.
67. Desai KI, Muzumdar DP, Goel A. Holocord intramedullary abscess: An unusual case with review of literature. Spinal Cord 1999;37:866-70.
68. Byrne RW, von Roenn KA, Whisler WW. Intramedullary abscess: A report of two cases and a review of the literature. Neurosurgery 1994;35:321-6.
69. David C, Brasme L, Peruzzi P, Bertault R, Vinsonneau M, Ingrand D. Intramedullary abscess of the spinal cord in a patient with a right-to-left shunt: Case report. Clin Infect Dis 1997;24:89-90.
70. Erlich JH, Rosenfeld JV, Fuller A, Brown GV, Wodak J, Tress BP. Acute intramedullary spinal cord abscess: Case report. Surg Neurol 1992;38:287-90.
71. Guzel N, Eras M, Guzel DK. A child with spinal intramedullary abscess. Childs Nerv Syst 2003;19:773-6.
72. Bommakanti K, Alugolu R, Chittem LR, Patil M, Purohit AK. Fulminant holocord intramedullary tubercular abscess with enigmatic presentation. Surg Neurol Int 2013;4:32.
73. Achouri M, Hilmani S, Sami A, Ouboukhlik A, el Kamar A, el Azhari A, *et al.* Intradural extramedullary tuberculous abscess. Apropos of a case. Neurochirurgie 1996;42:306-8.
74. Hanci M, Sarioglu AC, Uzan M, Islak C, Kaynar MY, Oz B. Intramedullary tuberculous abscess: A case report. Spine (Phila Pa 1976) 1996;21:766-9.
75. Khalid M, Khalid S, Mittal S, Ahmad U. Intramedullary tubercular abscess with syrinx formation. J Pediatr Neurosci 2012;7:61-3.
76. Tewari MK, Devi BI, Thakur RC, Pathak A, Khandelwal N, Kak VK. Intramedullary spinal cord abscess: A case report. Childs Nerv Syst 1992;8:290-1.
77. Devi BI, Chandra S, Mongia S, Chandramouli BA, Sastry KV, Shankar SK. Spinal intramedullary tuberculoma and abscess: A rare cause of paraparesis. Neurol India 2002;50:494-6.
78. Chittem L, Bommanakanti K, Alugolu R. 'Precipitation sign': A new radiological sign for spinal intramedullary tubercular abscess. Spinal Cord 2014;52 Suppl 1:S1-2.
79. Sinha S, Sharma BS. Neurocysticercosis: A review of current status and management. J Clin Neurosci 2009;16:867-76.
80. Mathuriya SN, Khosla VK, Vasishta RK, Tewari MK, Pathak A, Prabhakar S. Intramedullary cysticercosis: MRI diagnosis. Neurol India 2001;49:71-4.
81. 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;20:904-8.
82. Furtado SV, Dadlani R, Ghosal N, Rao AS. Solitary thoracic vertebral body cysticercosis presenting with progressive compressive myelopathy. J Neurosurg Spine 2013;18:394-7.

83. Lim BC, Lee RS, Lim JS, Cho KY. A case of neurocysticercosis in entire spinal level. J Korean Neurosurg Soc 2010;48:371-4.
84. Qi B, Ge P, Yang H, Bi C, Li Y. Spinal intramedullary cysticercosis: A case report and literature review. Int J Med Sci 2011;8:420-3.
85. Chhiber SS, Singh B, Bansal P, Pandita KK, Razdan S, Singh J. Intramedullary spinal cysticercosis cured with medical therapy: Case report and review of literature. Surg Neurol 2009;72:765-8.
86. Senol MG, Tekeli H, Kendirli MT, Kaya S, Turhan V, Sonmez G, *et al.* Intramedullary hydatid cyst of the cervical spine. Indian J Med Microbiol 2012;30:480-1.
87. Kalkan E, Cengiz SL, Ciçek O, Erdi F, Baysefer A. Primary spinal intradural extramedullary hydatid cyst in a child. J Spinal Cord Med 2007;30:297-300.
88. Ley A Jr, Marti A. Intramedullary hydatid cyst. Case report. J Neurosurg 1970;33:457-9.
89. Sharma A, Mittal RS, Arora R, Gandhi A. Recurrent multiple hydatid cysts of the spine with paraparesis: A case report. J Spinal Surg 2012;3:814-6.
90. Algahtani HA, Aldarmahi AA, Al-Rabia MW, Baeesa SS. Acute paraplegia caused by Schistosoma mansoni. Neurosciences (Riyadh) 2014;19:47-51.

How to cite this article: Kumar R, Arora R. Pediatric spinal infections. Indian J Neurosurg 2014;3:75-85.
Source of Support: Nil, **Conflict of Interest:** None declared.