

Spinal Epidural Abscess in Immunocompetent Child: A Case Report and Review of Literature

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Abstract

Spinal epidural abscess (SEA) is uncommon and rare condition in immunocompetent population and even more rare in pediatric group. The incidence of spinal epidural abscess appears to be increasing and comprises up to 2 per 10,000 hospital admissions. The presentation is variable and diagnosis can be easily missed on first visit. The diagnosis is established by history, clinical examination finding, increased inflammatory markers and neurological imaging. Surgical decompression and drainage in combination with antibiotic for four to six weeks are the typical treatment for SEA. An alternative treatment with parenteral antibiotic only is an alternative treatment. We reported an 11-year-old girl presented fever, chest and back pain she was found to have unsteady gait and lower extremity weakness. Spinal MRI showed heterogeneous enhancing collection in the posterior epidural space from the level of T2 vertebra to T10 vertebra. She was treated with antibiotic for 6 weeks without complications.

Keywords

Spine, Epidural, Abscess, Immunocompetent, Pediatric

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1. Background

Spinal epidural abscess (SEA) is uncommon and rare condition in immunocompetent population and even more rare in pediatric group. This is unusual bacterial infection that requires prompt diagnoses and early intervention to prevent devastating neurologic sequelae [1-3]. Spinal epidural abscess usually occurs in subjects with risk factors including diabetes mellitus, cancer, advanced age, immunodeficiency, chronic renal failure, alcoholism, intravenous drug abuse, neurosurgical intervention to spine, acupuncture or mucocutaneous trauma [2, 4-7]. Predisposing factors can be

absent in around 10-20% of SEA patients [8-10]. The access of bacteria to spinal epidural space can be through contiguous spread, hematogenous dissemination or the source of infection is unknown as it is stated in 20-40% of previously reported cases [2, 8, 11-14]. The most causative agent of SEA is *Staphylococcus aureus* followed by Gram negative bacteria, streptococci and anaerobic bacteria [2, 8, 11, 14-16]. The presentation is variable and diagnosis can be easily missed on first visit [3]. A triad of back pain, neurological deficits and fever is the classic presentation of SEA. However, only a few number of cases present with the classical triad [4, 5, 17]. Previous studies revealed that most

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of untreated SEA patients undergo into four stages of the disease. Stage one present with fever, lumbar pain and local tenderness; stage two present with radicular pain, changes in the reflexes and nuchal rigidity; stage three consist of motor and sensory abnormalities and bowel and bladder dysfunction and in stage four, paralysis with permanent complication [2, 12, 13]. The diagnosis is established by history, clinical examination finding, increased inflammatory markers and neurological imaging [4, 18]. Surgical decompression and drainage in combination with antibiotic for four to six weeks are the typical treatment for SEA [2-4, 19-21]. An alternative treatment with parenteral antibiotic only was successful in specific population [6, 22-26]. Hawkins *et al.*, reported in their review of 11 pediatrics SEA that there is potential therapeutic success by using systemic antimicrobial treatment in combination with minimally invasive drainage techniques, serial laboratory studies, and imaging in pediatric group [4].

2. Case Report

An 11-year-old girl was hospitalized in our hospital on August 2018. She had been well since she was born - there was no previous medical or surgical history and no previous hospital admission -until one week earlier, when she started to have fever, chest and back pain. There was being well since she was born and there was no previous medical or surgical history. She was seen in local health center and was managed as a case of upper respiratory tract infection conservatively. She was seen in another hospital as she was not feeling any improvement. She was investigated there and discharged on oral antibiotics with the impression of pneumonia. Two days later she was called as her labs showed positive culture methicillin-sensitive *Staphylococcus aureus*, neutrophilic leukocytosis and bilateral infiltrate in chest x-ray. She was examined by our physician in the day of admission with fever, chest pain, lower back pain and lower limbs weakness. Birth and development history were unremarkable. Past medical was unremarkable. At hospitalization, she was febrile with temperature of 39C, pulse 111/min, respiration 36/min. She Looked well-nourished but lethargic. The positive findings included crepitations in both lung fields, tenderness in lumbar spinal region, unsteady gait and lower extremity weakness with power 4 right side and 3 in the left. Planter flexion 1/5 in both side.

Her white blood cell 15,500/100ml³, absolute neutrophil count 10 and C-reactive protein 500 mg/L. Urine analysis was unremarkable. Repeated blood cultures taken. Lumbar puncture was not performed as parent were totally refusing this test. She was started on IV ceftriaxone 2 grams twice

daily and 1 gram daily. Whole spine-MRI was performed and showed heterogeneous enhancing collection in the posterior epidural space from the level of T2 vertebra to T10 vertebra (Figure 1). It measures 6mm in thickness causing compression and anterior displacement of the spinal cord with evidence of high signal at T9 and T10. Diffuse subcutaneous collection and edema noted in the posterior aspect of the lumbar spine.

During the period of admission, she was improving clinically and the inflammatory markers were decreasing. In the third day following antibiotics, her weakness started to improve. She retained back to her baseline by day eight. Following MRI after 3-weeks form initiation of treatment was done (Figure 2). She continued the same antibiotic for six weeks. She was discharge home clinically and vitally stable with follow up after 4 weeks in the clinic.



Figure 1. Whole-spine T2-weighted magnetic resonance imaging with contrast extensive epidural collection extending from T2 to T10. Spinal cord is compressed anteriorly with normal signal.



Figure 2. Whole-spine T1-weighted magnetic resonance imaging with contrast 3 weeks after parenteral antibiotic showing complete resolution of epidural abscess.

3. Discussion

Spinal infections are uncommon but significant causes of morbidity and hospitalization in the pediatric population [27]. Spinal epidural abscess (SEA) is a rare condition that requires prompt diagnosis and initiation of treatment for optimal outcome [28]. The incidence of spinal epidural abscess appears to be increasing and comprises up to 2 per 10,000 hospital admissions [29]. Diagnosis is particularly challenging in patients who present with sepsis of unexplained origin; any complaint of back or neck pain should be urgently investigated with a MRI [30-33]. Risk factors for SEA include diabetes, malignancy, dialysis-dependent chronic renal disease, AIDS, and steroid use, immunosuppression and previous epidural procedures.

Despite a low incidence of SEA, clinicians must maintain low thresholds of suspicion for spinal epidural abscess to diagnose and treat prior to development of irreversible deficits [28]. As found in our case, *Staphylococcus* has been the most frequently causative organism for spinal epidural abscess which represents 63% of the cultured organisms [28]. MRI is the study of choice for detecting spinal epidural abscesses [28]. CT-myelogram are equivalent in their sensitivities to SEA in patients with contraindications for gadolinium enhanced MRI. Therapy for spinal epidural abscess therefore focuses on 3 goals: preservation of normal neurologic function, prevention of worsening of existing neurologic deficits, and optimization of opportunities for improvement and return of function [28]. Urgent surgical decompression has been the treatment of choice for spinal epidural abscess, and this has been confirmed in numerous series to date [34-39]. Appropriate intravenous antibiotics are administered for 4 to 6 weeks [35, 36]. Pediatric cases raise concerns regarding development of post-laminectomy kyphosis [40]. Alternative surgical techniques (e.g., laminotomy) [41] and percutaneous drainage [42] may address this issue. Several investigators, both in case reports [43, 44] and in case series [45-47], have advocated antibiotic therapy alone for SEA cases. In a recent series of 75 cases of spinal epidural abscess [29], 22 (29%) patients were treated conservatively. In our case, antibiotic therapy alone was used as treatment strategy.

Including our report, we found a total of 24 pediatric SEA cases without predisposing factors, 12 were males and 12 were female, age range 16 days–17 years (Table 1). Most cases presented with fever and non-specific symptoms including neck and back pain, vomiting and irritability. Two cases were initially diagnosed with acute appendicitis. Lumbar puncture was done in 8 cases only, 1 case had normal LP results. In 8 cases, blood culture was done as part of patient initial workup. *Staphylococcus aureus* was the most common cause of SEA (n=14) [Methicillin-sensitive *Staphylococcus aureus* 45.8%, Methicillin-resistant *Staphylococcus aureus* 8.3%]. Surgical intervention was done in 14 cases; 11 had laminectomy and 3 drainage of the SEA. Ten cases received conservative management with antibiotics only. Seventy-nine percentage of cases received combination for more than one antibiotic agent based on culture and sensitivity. The average of therapy duration was 6 weeks. In four cases the duration was not specified.

Table 1. Summary of 24 cases of spinal epidural abscess in immunocompetent Children published in literature.

Cases	Gender, Age	Spine level	Presentation	Primary diagnosis	CSF
[67] Fotaki 2018	M, 2.5 y	C3-T2	Fever, neck pain and stiffness	Meningitis	Pleocytosis, low glucose, elevated protein
[61] Horner 2016	M, 34 d	C3-C5	Fever, irritability, decreased oral intake	Meningitis	Pleocytosis (WBCs 2113 / mm ³) Low glucose,

Cases	Gender, Age	Spine level	Presentation	Primary diagnosis	CSF
[62] Paropanjani 2016	M, 3 wk	C4-C5	Irritability, paresis and areflexia of both arms.	-	elevated protein Pleocytosis
[60] Aycan 2016	F, 13 y	T12-L5	Fever, back pain, paraparesis,	-	Not performed
[48] Vergori 2015	M, 15 y	T11-L2	Fever, headache and back pain in lumbar-sacral region, bilateral leg weakness	Meningitis	Pleocytosis, glucose normal, protein elevated
[58] Harris 2014	M, 21 m	L4-L5	Fever, refuse to walk	Septic arthritis	Not performed
[55] Hawkins 2013	F, 17 y	L1-L4	Fever, nausea, vomiting	-	Not performed
[55] Hawkins 2013	M, 3 y	T1-L2	Fever, stomachache	-	Not performed
[55] Hawkins 2013	M, 1.2 y	L3-L4	Refusal to walk, irritability, weakness	-	Not performed
[56] Pathak 2013	M, 13 y	C7-T1	Transient fever, neck and upper back pain, tingling sensation in hands and feet, urine incontinence, abdominal distension, inability to sit and walk	Acute myelitis, diskitis, meningitis	800 cells/mm ³ , 2% PMN, glucose 21 mg/dl
[57] Sales 2013	M, 15 y	L2-L3	Fever, urinary retention, Back pain,	Low back pain and Not specified urinary retention	Not performed
[66] Hazelton 2012	M, 16 d	C3-C4	Fever, irritability	-	180 PMN, 9900 red blood cells
[53] Mantadakis 2011	F, 11 y	T11-L4	Fever, lumbar pain	Back pain	Not performed
[54] Rook 2011	F, 15 y	T3-T8	Right scapular pain, fever, chills with night sweats, headache, photophobia	Right rhomboid muscle strain with spasm, acute febrile illness	Normal
[50] Tang 2001	F, 7 wk	T10-T12	Flaccid paraplegia	Neoplasia	Not performed
[68] Kim 2011	F, 10 y	L3-L5	Fever, low back pain, radiating pain in both legs, saddle anesthesia, bladder and bowel dysfunction	-	Not performed
[63] Shawar 2017	F, 13 y	Not available	Fever, lumbar pain, headache, nausea, localized tenderness	Viral infection	WBCs > 10,000/mm ³ Undetectable glucose/protein
[65] Raus 2015	F, 3 m	C5-C6	Neck stiffness, irritability, right upper extremity hypotonia, exaggerated tendon reflexes	Meningoencephalitis	Not available
[49] Bair-Meritt 2000	F, 3 y	L5-S1	Fever, malaise, right hip pain	-	Not performed
[64] Rood 2014	M, 10 m	L5-S1	Fever, back pain, gait change	Bacterial infection of unknown location	Not performed
[59] Prasad 2014	F, 14 y	Not available	Abdominal tenderness	Appendicitis	Not performed
[52] Kiyamaz 2005	F, 10 y	C2-C3	Fever, neck pain stiffness	Meningitis	Not performed
[51] Flikweert 2002	M, 7 y	T3-T7	Fever, abdominal pain	Appendicitis	Not performed
Our case	F, 11 y	T2-10	Fever, chest/back pain, LE weakness	URTIs/Pneumonia/GBS	Not performed

Table 1. Continued.

Cases	Positive blood culture	Etiology	Surgery	Antibiotics Duration	Outcome
[67] Fotaki 2018	Yes	Group A Streptococcus	No surgery	Vancomycin, meropenem, rifampicin, 6 weeks	No deficit
[61] Horner 2016	Yes	Methicillin-sensitive Staphylococcus aureus	No surgery	Nafcillin 6 weeks	No deficit
[62] Paropanjani 2016	No	Group A Streptococcus	Drainage	Ampicillin, clindamycin, gentamycin, 3 weeks	No deficit
[60] Aycan 2016	Not available	Methicillin-resistant Staphylococcus aureus	Laminectomy	Vancomycin, penicillin, ceftriaxone 2 weeks	No deficit
[48] Vergori 2015	No	Methicillin-sensitive Staphylococcus aureus	Laminectomy, abscess drainage L4-L5,	Ceftriaxone, clindamycin 8 weeks	Thoracolumbar kyphosis and lumbar lordosis
[58] Harris 2014	No	Group A beta-hemolytic Streptococcus	Partial S1 Laminectomy and drainage	Ceftriaxone 6 weeks	No deficit
[55] Hawkins 2013	No	Unknown	No surgery	Doxycycline, doripenem (duration not available)	No deficit
[55] Hawkins 2013	Yes	Methicillin-resistant Staphylococcus aureus	Left laminectomy	Clindamycin, rifampin (duration not available)	No deficit

Cases	Positive blood culture	Etiology	Surgery	Antibiotics Duration	Outcome
[55] Hawkins 2013	No	Unknown	T5–L2 No surgery	Nafcillin, vancomycin Then clindamycin (+vancomycin on readmission)	Standing with assistance on discharge
[56] Pathak 2013	No	Unknown	No surgery	Ceftriaxone, vancomycin 6 weeks (duration not available)	No deficit
[57] Sales 2013	Not available	Staphylococcus aureus	L2–L3 laminectomy drainage	Cefazolin then cephalexin 6 weeks	No deficit
[66] Hazelton 2012	Yes	Methicillin-sensitive Staphylococcus aureus	Right sided laminectomy drainage	Flucloxacillin, ampicillin 6 weeks	No deficit
[53] Mantadakis 2011	No	Methicillin-sensitive Staphylococcus aureus	Drainage	Clindamycin+ ceftriaxone then clindamycin + cefuroxime 7.5 weeks	No deficit
[54] Rook 2011	Yes	Methicillin-sensitive Staphylococcus aureus	Thoracic laminectomy, drainage	Ceftriaxone 4 weeks	Headache and paraspinal pain for subsequent 3 years
[50] Tang 2001	Not available	Staphylococcus aureus	T9–T12 laminectomy	Not specified anti-staphylococcal antibiotics (duration not available)	No deficit
[68] Kim 2011	Not available	Staphylococcus aureus	Hemilaminotomy and drainage	Ceftazole, cefaclor 6 weeks	No deficit
[63] Shawar 2017	Yes	Methicillin-sensitive Staphylococcus aureus	No surgery	Oxacillin 6 weeks	Not available
[65] Raus 2015	No	Not available	No surgery	Clindamycin, meropenem 5 weeks	Not available
[49] Bair-Meritt 2000	Yes	Oxacillin-sensitive Staphylococcus aureus	No surgery	Gentamicin, oxacillin 6 weeks	No deficit
[64] Rood 2014	Not available	Not available	No surgery	Ceftriaxone, metronidazole 3 weeks	No deficit
[59] Prasad 2014	Not available	Not available	Drainage	Not available	No deficit
[52] Kiyamaz 2005	No	No microorganism isolated initially, 2 months later Streptococcus anginosus	Laminectomy	Vancomycin, amikacin, cefotaxime, 6 weeks	Two months later: symptom relapse, MRI: abscess, treated with antibiotics. 12 month follow up all normal.
[51] Flikweert 2002	Not available	Group A Streptococcus	Laminectomy	Penicillin 4 weeks	No deficit
Our case	Yes	Methicillin-sensitive Staphylococcus aureus	No surgery	Ceftriaxone + vancomycin 6 weeks	No deficit

showed during the conduct of the paper.

4. Conclusion

SEA is a rare condition that requires prompt diagnosis and initiation of treatment for optimal outcome. Despite a low incidence of SEA, clinicians must maintain low thresholds of suspicion for spinal epidural abscess to diagnose and treat prior to development of irreversible deficits. MRI is the study of choice for detecting spinal epidural abscesses. Therapies for spinal epidural abscess focus on 3 goals: preservation of normal neurologic function, prevention of worsening of existing neurologic deficits, and optimization of opportunities for improvement and return of function.

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