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Spinal Epidural Abscess in Immunocompetent Child

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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: Abscess; Epidural; Immunocompetent; Pediatric; Spine

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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 require prompt diagnoses and early intervention to prevent devastating neurologic sequelae [1,2,3]. 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 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].

Case Report

A 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 500mg/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.

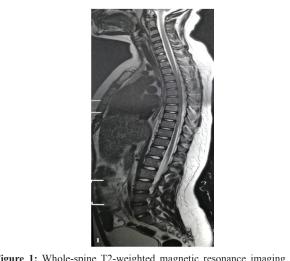


Figure 1: Whole-spine T2-weighted magnetic resonance imaging with contrast extensive epidural collection extending from T2 to T10. Spinal cord is compressed anteriorly with normal signal.

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.

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

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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.



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

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.

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Cases	Gender, Age	Spine Level	Presentation	Primary Diagnosis	CSF	Positive Blood Culture	Etiology
Fotaki [48]	М, 2.5 у	C3-T2	Fever, neck pain and stiffness	Meningitis	Pleocytosis, low glucose, elevated protein	Yes	Group A Streptococcu
Horner [49]	M, 34 d	C3-C5	Fever, irritability, decreased oral intake	Meningitis	Pleocytosis (WBCs 2113/mm ³) Low glucose, elevated protein	Yes	Methicillin-sensitive Staphylococcus aureu
Paro-panjan [50]	M, 3 wk	C4-C5	Irritability, paresis and areflexia of both arms.	-	Pleocytosis	No	Group A Streptococcu
Aycan [8]	F, 13 y	T12-L5	Fever, back pain, paraparesis,	-	Not performed	Not available	Methicillin- resistant Staphylococcus aureu
Vergori [2]	М, 15 у	T11-L2	Fever, headache and back pain in lumbar-sacral region, bilateral leg weakness	Meningitis	Pleocytosis, glucose normal, protein elevated	No	Methicillin-sensitive Staphylococcus aureu
Harris [3]	M, 21 m	L4-L5	Fever, refuse to walk	Septic arthritis	Not performed	No	Group A beta-hemoly ic Streptococcus
Hawkins [4]	F, 17 y	L1-L4	Fever, nausea, vomiting	-	Not performed	No	Unknown
Hawkins [4]	M, 3y	T1-L2	Fever, stomachache	-	Not performed	Yes	Methicillin- resistan Staphylococcus aurei
Hawkins [4]	M, 1.2 y	L3-L4	Refusal to walk, irritability, weakness	-	Not performed	No	Unknown
Pathak [51]	М, 13 у	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/mmc, 2% PMN, glucose 21 mg/dl	No	Unknown
Sales [52]	М, 15 у	L2-L3	Fever, urinary retention, Back pain,	Low back pain and Not specified urinary retention	Not performed	Not available	Staphylococcus aurei
Hazelton [53]	M, 16 d	C3-C4	Fever, irritability	-	180 PMN, 9900 red blood cells	Yes	Methicillin-sensitive Staphylococcus aureu
Mantadakis [46]	F, 11 y	T11-L4	Fever, lumbar pain	Back pain	Not performed	No	Methicillin-sensitive Staphylococcus aureu
Rook [47]	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	Yes	Methicillin-sensitive Staphylococcus aureu
Tang [43]	F, 7 wk	T10-T12	Flaccid paraplegia	Neoplasia	Not performed	Not available	Staphylococcus aurer
Kim [54]	F, 10 y	L3-L5	Fever, low back pain, radiating pain in both legs, saddle anesthe- sia, bladder and bowel dysfunction	-	Not performed	Not available	Staphylococcus aurei
Shawar [55]	F, 13 y	Not available	Fever, lumbar pain, headache, nausea, localized tenderness	Viral infection	WBCs>10,000/mm ³ , Undetect- able glucose/protein	Yes	Methicillin-sensitive Staphylococcus aurei
Raus [56]	F, 3 m	C5-C6	Neck stiffness, irritability, right upper extremity hypotonia, exag- gerated tendon reflexes	Meningoencephalitis	Not available	No	Not available
Bair-Meritt [42]	F, 3 y	L5-S1	Fever, malaise, right hip pain	-	Not performed	Yes	Oxacillin-sensitive Staphylococcus auren
Rood [57]	M, 10 m	L5-S1	Fever, back pain, gait change	Bacterial infection of unknown location	Not performed	Not available	Not available
Prasad [58]	F, 14 y	Not available	Abdominal tenderness	Appendicitis	Not performed	Not available	Not available
Kiymaz [45]	F, 10 y	C2-C3	Fever, neck pain stiffness	Meningitis	Not performed	No	No microorganism isolated initially, 2 months later <i>Strepto</i> <i>coccus anginosus</i>
Flikweert [44]	М, 7 у	T3-T7	Fever, abdominal pain	Appendicitis	Not performed	Not available	Group A Streptococc
Our case	F, 11 y	T2-10	Fever, chest/back pain, LE weakness	URTIs/Pneumonia/GBS	Not performed	Yes	Methicillin-sensitive Staphylococcus aurer

Conclusion

Spinal Epidural Abscess (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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