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

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 sequela [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
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 15,500/100ml\(^3\), 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.
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.

<table>
<thead>
<tr>
<th>Cases</th>
<th>Gender, Age</th>
<th>Spine level</th>
<th>Presentation</th>
<th>Primary diagnosis</th>
<th>CSF</th>
</tr>
</thead>
<tbody>
<tr>
<td>[67] Fotaki 2018</td>
<td>M, 2.5 y</td>
<td>C3-T2</td>
<td>Fever, neck pain and stiffness</td>
<td>Meningitis</td>
<td>Pleocytosis, low glucose, elevated protein</td>
</tr>
<tr>
<td>[61] Horner 2016</td>
<td>M, 34 d</td>
<td>C3-C5</td>
<td>Fever, irritability, decreased oral intake</td>
<td>Meningitis</td>
<td>Pleocytosis (WBCs 2113 / mm³), Low glucose,</td>
</tr>
<tr>
<td>Cases</td>
<td>Gender, Age</td>
<td>Spine level</td>
<td>Presentation</td>
<td>Primary diagnosis</td>
<td>CSF</td>
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</tr>
<tr>
<td>[62] Paropanjan 2016</td>
<td>M, 3 wk</td>
<td>C4-C5</td>
<td>Irritability, paresis and areflexia of both arms.</td>
<td>-</td>
<td>Elevated protein</td>
</tr>
<tr>
<td>[60] Aycan 2016</td>
<td>F, 13 y</td>
<td>T12-L5</td>
<td>Fever, back pain, paraparesis, Fever, headache and back pain in lumbar-sacral region, bilateral leg weakness</td>
<td>Meningitis</td>
<td>Pleocytosis, glucose normal, protein elevated</td>
</tr>
<tr>
<td>[48] Vergori 2015</td>
<td>M, 15 y</td>
<td>T11-L2</td>
<td>Refusal to walk, areflexia of both arms, back pain.</td>
<td>-</td>
<td>pleocytosis, glucose normal, protein elevated</td>
</tr>
<tr>
<td>[58] Harris 2014</td>
<td>M, 21 m</td>
<td>L4-L5</td>
<td>Fever, refuse to walk</td>
<td>Septic arthritis</td>
<td>Not performed</td>
</tr>
<tr>
<td>[55] Hawkins 2013</td>
<td>F, 17 y</td>
<td>L1-L4</td>
<td>Fever, nausea, vomiting</td>
<td>-</td>
<td>Not performed</td>
</tr>
<tr>
<td>[55] Hawkins 2013</td>
<td>M, 3 y</td>
<td>T1-L2</td>
<td>Fever, stomachache</td>
<td>-</td>
<td>Not performed</td>
</tr>
<tr>
<td>[55] Hawkins 2013</td>
<td>M, 1.2 y</td>
<td>L3-L4</td>
<td>Refusal to walk, irritability, weakness</td>
<td>Acute myelitis, diskitis, meningitis</td>
<td>800 cells/mm³, 2% PMN, glucose 21 mg/dl</td>
</tr>
<tr>
<td>[56] Pathak 2013</td>
<td>M, 13 y</td>
<td>C7-T1</td>
<td>Transient fever, neck and upper back pain, tingling sensation in hands and feet, urine incontinence, abdominal distension, inability to sit and walk</td>
<td>-</td>
<td>WBCs &gt; 10,000/mm³, Undetectable glucose/protein</td>
</tr>
<tr>
<td>[57] Sales 2013</td>
<td>M, 15 y</td>
<td>L2-L3</td>
<td>Fever, urinary retention, Back pain, Left leg weakness</td>
<td>Low back pain and Not specified urinary retention</td>
<td>Not performed</td>
</tr>
<tr>
<td>[66] Hazeldon 2012</td>
<td>M, 16 d</td>
<td>C3-C4</td>
<td>Fever, irritability</td>
<td>-</td>
<td>180 PMN, 9900 red blood cells</td>
</tr>
<tr>
<td>[53] Mantadakis 2011</td>
<td>F, 11 y</td>
<td>T11-L4</td>
<td>Fever, lumbar pain</td>
<td>Back pain</td>
<td>Not performed</td>
</tr>
<tr>
<td>[54] Rook 2011</td>
<td>F, 15 y</td>
<td>T3-T8</td>
<td>Right scapular pain, fever, chills with night sweats, headache, photophobia</td>
<td>Right rhomboid muscle strain with spasm, acute febrile illness</td>
<td>Normal</td>
</tr>
<tr>
<td>[50] Tang 2001</td>
<td>F, 7 wk</td>
<td>T10-T12</td>
<td>Flaccid paraplegia</td>
<td>-</td>
<td>Not performed</td>
</tr>
<tr>
<td>[68] Kim 2011</td>
<td>F, 10 y</td>
<td>L3-L5</td>
<td>Fever, low back pain, radiating pain in both legs, saddle anesthesia, bladder and bowel dysfunction</td>
<td>-</td>
<td>Not performed</td>
</tr>
<tr>
<td>[63] Shawar 2017</td>
<td>F, 13 y</td>
<td>Not available</td>
<td>Fever, lumbar pain, headache, nausea, localized tenderness</td>
<td>Viral infection</td>
<td>WBCs &gt; 10,000/mm³</td>
</tr>
<tr>
<td>[65] Raus 2015</td>
<td>F, 3 m</td>
<td>C5-C6</td>
<td>Neck stiffness, irritability, right upper extremity hypotonia, exaggerated tendon reflexes</td>
<td>Meningoencephalitis</td>
<td>Not available</td>
</tr>
<tr>
<td>[49] Bair-Meritt 2000</td>
<td>F, 3 y</td>
<td>L5-S1</td>
<td>Fever, malaise, right hip pain</td>
<td>-</td>
<td>Not performed</td>
</tr>
<tr>
<td>[64] Rood 2014</td>
<td>M, 10 m</td>
<td>L5-S1</td>
<td>Fever, back pain, gait change</td>
<td>Bacterial infection of unknown location</td>
<td>Not performed</td>
</tr>
<tr>
<td>[59] Prasad 2014</td>
<td>F, 14 y</td>
<td>Not available</td>
<td>Abdominal tenderness</td>
<td>Appendicitis</td>
<td>Not performed</td>
</tr>
<tr>
<td>[52] Kiyymaz 2005</td>
<td>F, 10 y</td>
<td>C2-C3</td>
<td>Fever, rear back pain</td>
<td>Meningitis</td>
<td>Not performed</td>
</tr>
<tr>
<td>[51] Flikweert 2002</td>
<td>M, 7 y</td>
<td>T3-T7</td>
<td>Fever, abdominal pain</td>
<td>Appendicitis</td>
<td>Not performed</td>
</tr>
<tr>
<td>Our case</td>
<td>F, 11 y</td>
<td>T2-T10</td>
<td>Fever, chest/back pain, LE weakness</td>
<td>URTIs/Pneumonia/GBS</td>
<td>Not performed</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Cases</th>
<th>Positive blood culture</th>
<th>Etiology</th>
<th>Surgery</th>
<th>Antibiotics Duration</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>[67] Fotaki 2018</td>
<td>Yes</td>
<td>Group A Streptococcus</td>
<td>No surgery</td>
<td>Vancomycin, meropenem, rifampicin, 6 weeks</td>
<td>No deficit</td>
</tr>
<tr>
<td>[61] Horner 2016</td>
<td>Yes</td>
<td>Methicillin-sensitive Staphylococcus aureus</td>
<td>No surgery</td>
<td>Nafcillin 6 weeks</td>
<td>No deficit</td>
</tr>
<tr>
<td>[62] Paropanjan 2016</td>
<td>No</td>
<td>Group A Streptococcus</td>
<td>Drainage</td>
<td>Ampicillin, clindamycin, gentamycin, 3 weeks</td>
<td>No deficit</td>
</tr>
<tr>
<td>[60] Aycan 2016</td>
<td>Not available</td>
<td>Methicillin- resistant Staphylococcus aureus</td>
<td>Drainage</td>
<td>Vancomycin, penicillin, ceftriaxone 2 weeks</td>
<td>No deficit</td>
</tr>
<tr>
<td>[48] Vergori 2015</td>
<td>No</td>
<td>Methicillin-sensitive Staphylococcus aureus</td>
<td>Laminectomy, abscess drainage</td>
<td>Ceftriaxone, clindamycin 8 weeks</td>
<td>Thoracolumbar kyphosis and lumbar lordosis</td>
</tr>
<tr>
<td>[58] Harris 2014</td>
<td>No</td>
<td>Group A beta-hemolytic Streptococcus</td>
<td>L4-L5, Partial S1 Laminectomy and drainage</td>
<td>Ceftriaxone 6 weeks</td>
<td>No deficit</td>
</tr>
<tr>
<td>[55] Hawkins 2013</td>
<td>No</td>
<td>Unknown</td>
<td>No surgery</td>
<td>Doxycycline, doripenem (duration not available)</td>
<td>No deficit</td>
</tr>
<tr>
<td>[55] Hawkins 2013</td>
<td>Yes</td>
<td>Methicillin- resistant Staphylococcus aureus</td>
<td>Left laminectomy</td>
<td>Clindamycin, rifampin (duration not available)</td>
<td>No deficit</td>
</tr>
</tbody>
</table>
Cases | Positive blood culture | Etiology | Surgery | Antibiotics Duration | Outcome |
--- | --- | --- | --- | --- | --- |
[55] Hawkins 2013 | No | Unknown | 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 | Ceftazolin then cephalaxin 6 weeks | No deficit |
[66] Hazelton 2012 | Yes | Methicillin-sensitive Staphylococcus aureus | Right sided laminectomy drainage | Flucoxacinill, 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 |
[68] Kim 2011 | Not available | Staphylococcus aureus | No surgery | Ceftezeole, cefaclor 6 weeks | No deficit |
[63] Shawar 2017 | Yes | Methicillin-sensitive Staphylococcus aureus | No surgery | Oxacillin 6 weeks | Not available |
[65] Raus 2015 [49] Bair-Meritt 2000 | No | Not available | No surgery | Clindamycin, meropenen 5 weeks | Not available |
[64] Rood 2014 | Not available | Not available | No surgery | Gentamicin, oxacillin 6 weeks | No deficit |
[59] Prasad 2014 | Not available | Not available | Drainage | Ceftriaxone, metronidazole 3 weeks | No deficit |
[52] Kiymaz 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. |

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 abscesses 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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References


