Pediatric Spinal Epidural Abscess: A 9-Year Institutional Review and Review of the Literature

abstract

Spinal epidural abscess (SEA) is a rare condition that requires prompt diagnosis and initiation of treatment for optimal outcome. Treatment generally consists of surgical intervention and systemic antibiotics. We present 1 of the largest cohorts of nontuberculous pediatric SEA in the English literature, emphasizing the outcomes of conservative (ie, nonoperative) management. We retrospectively identified 9 pediatric patients (≤18 years of age) with SEAs at Our Lady of the Lake Children’s Hospital from 2002 to 2011. Cases were reviewed for demographic, clinical, diagnostic, and treatment characteristics and outcomes. The diagnosis of SEA was made by MRI in all cases, and methicillin-resistant Staphylococcus aureus (MRSA) was the only identified pathogen, isolated via blood culture in 6 of 9 patients. Although every patient received systemic antibiotics, only 2 had neurosurgical intervention. Four of the 7 patients treated conservatively received computed tomography-guided needle drainage. All patients recovered without significant sequelae. SEA is a potentially fatal illness that necessitates a heightened clinical awareness for diagnosis and treatment. Although official recommendations regarding management in pediatrics are lacking, treatment has generally been surgical decompression and drainage in combination with antibiotics; recent reports have suggested that antibiotic therapy alone may be successful in select patient populations. Although the adult literature has suggested that such management can be trialed in specific situations, only a handful of cases in the pediatric literature have reported this nonoperative approach. We present one of the largest reviews in support of successfully treating SEA with nonsurgical therapy.

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abscess, infection, pediatrics

ABBREVIATIONS:
CRP—C-reactive protein
CT—computed tomography
ESR—erythrocyte sedimentation rate
MRSA—methicillin-resistant Staphylococcus aureus
SEA—spinal epidural abscess

Dr Hawkins conceptualized and designed the study, designed the data collection instruments, performed the review of the literature and interpretation of data, and drafted the initial manuscript; Dr Bolton conceptualized and designed the study, conducted data analyses, and reviewed and revised the manuscript; both authors approved the final manuscript as submitted.

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Spinal epidural abscess (SEA) is an infrequent diagnosis requiring prompt initiation of treatment for optimal outcome. SEAs are potentially increasing in incidence due to a rise in risk factors (spine trauma, previous skin infections, intravenous drug use, and immunodeficiency) and enhancement of imaging studies allowing increased detection.1–3 Treatment has traditionally been surgical decompression and drainage in combination with antibiotics4–6; recent reports suggest that antibiotic therapy alone can be successful in select patient populations.1,3,7–10 We present an institutional review revealing successful outcomes in patients who historically have had significant morbidity and mortality.

METHODS
Institutional review board approval was obtained before review of medical records (classified as exempt). Nine pediatric patients (=18 years of age) with spinal epidural abscess (International Classification of Disease, Ninth Revision diagnosis code 324.1 as primary or secondary diagnosis) were identified at Our Lady of the Lake Children’s Hospital (a 95-bed hospital within a regional medical center) from 2002 to 2011. Cases were reviewed for the following characteristics: demographics, number of medical visits before diagnosis, length of stay, readmission rates, clinical features, diagnostic data (including vertebral body involvement, culture results, and concurrent osteomyelitis), treatments, and outcomes.

RESULTS
The incidence of SEA was 1.5 cases per 10,000 admissions from 2002 to 2011. The average patient age was 9.2 years with a male predominance (56%). Sixty-seven percent of patients presented with fever; 33% had fever as the chief complaint. Six of the 9 cases had an identifiable risk factor for SEA: recent skin/soft tissue infections or trauma. Four patients reported ≥1 skin or soft tissue infection in the preceding 6 months, 1 patient had lower-extremity cellulitis presumably secondary to Bartonella henselae infection, and 1 patient suffered a fall with resulting back pain 1 week before presentation. No patient had evidence of immunodeficiency. Four cases presented with weakness, and 1 patient had bowel and bladder incontinence, although none of the patients presented with or experienced paralysis (Tables 1 and 2).

The groups were similar in age, length of stay, white blood cell count, and number of involved vertebral bodies. Inflammatory markers were obtained in every patient; average erythrocyte sedimentation rate (ESR) was 82.9 mm/hr and C-reactive protein (CRP) was 150.5 mg/L. Diagnosis was made by MRI in all cases. The average number of vertebral bodies involved was 6.7 (range 2–14). All but 2 patients had multiple medical visits before SEA was diagnosed.

Methicillin-resistant Staphylococcus aureus (MRSA) was the only pathogen cultured (all via blood culture) in 6 of 9 cases (Table 3). Three patients had negative cultures, although 1 patient had epidemiologic factors and serologic evidence of B henselae infection. B henselae infection has been described in association with vertebral osteomyelitis and SEA infection in 3 children in reports published from 2005 to 20099 and presumably was the source of infection in this child.

Seven of 9 SEA patients were successfully treated with conservative (nonoperative) management. Of these 7 patients, 4 had computed tomography (CT)-guided needle drainage of their abscesses (1 of the 7 patients had an exploratory laparotomy for abdominal pain on presentation). Only 2 of the 9 patients underwent neurosurgical intervention consisting of incision and drainage and laminectomy. All patients received systemic antibiotics (Table 4). There were no significant sequelae in any patient upon documented follow-up. The rarity of this illness prohibited statistical analyses between surgical and nonsurgical groups.

DISCUSSION
The classic presentation of SEA is a triad of back pain, fever, and neurologic deficits. However, only a minority of cases present with all 3 symptoms,1,9 and children are less likely to have aforementioned SEA risk factors.4 Consistent with previous results, our patients presented with fever, refusal to bear weight, pain with movement, and back pain; although none presented with all symptoms. Diagnosis in children is especially challenging because of nonspecific symptoms and frequently delayed until neurologic symptoms develop.11–13

The diagnosis is based on clinical suspicion, increased inflammatory markers, and neurologic imaging.10 Hematogenous spread is the most common cause of infection in children as supported by the 6 positive blood cultures in our cohort.4,11,13,16,17 Untreated disease has variable progression, especially in young children.14,15

Acute-phase reactants (CRP and ESR) can provide insight into the level of inflammation and tissue involvement.5,18 ESR has been shown to be a strong indicator of disease, more so than leukocyte count.3,5,8,9 Table 3 demonstrates the rates of relatively normal white blood cell counts on presentation in contrast to the remarkable elevations of both CRP and ESR. Evaluation of inflammatory markers in a febrile patient with localized back pain should be considered as significant elevations may suggest SEA and may warrant diagnostic imaging.5 As suggested by this review, neurologic deficit does not need to be present to have heightened
clinical suspicion because these findings do not typically present until later in the disease progression.10 All patients underwent MRI, the imaging modality of choice for the detection of SEA.1,3–5,13,19 Advances in such imaging have led to increased and earlier detection, more precise delineation of location and extent of infection (aiding in surgical technique planning), and improved evaluation of disease progression and resolution.2

The most common organism causing pediatric SEA is S. aureus.2,4,7,9,11,12,16,17,19–22 There is increasing prevalence of community-acquired MRSA infections in children without risk factors.23,24 A thorough literature review performed by Auletta et al indicates high rates of methicillin-sensitive S. aureus, in contrast to our review in which the 6 culture-positive patients grew MRSA, reinforcing the changing prevalence and invasiveness community-acquired MRSA can display.3,25 The incidence of MRSA could explain our higher CRP values but does not explain why only 22% of our patients required neurosurgical intervention (compared with 75% in previous reports4) and did not have disease progression after initiation of antibiotics. The outcome of no permanent neurologic effects was surprising in contrast to the expected worsened pathogenicity from MRSA in comparison with methicillin-sensitive S. aureus.

The prompt initiation of treatment necessitates a broad-spectrum empiric regimen, although coverage should

### TABLE 1 Summary of Clinical Data on 9 Patients With SEA: Historical Information

<table>
<thead>
<tr>
<th>Pt</th>
<th>Gender, Age (y)</th>
<th>Chief Complaint</th>
<th>Past Medical History</th>
<th>Past Surgical History</th>
<th>SEA Risk Factors</th>
<th>Previous Antibiotic Use</th>
<th>Number of Medical Visits Before Diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>M, 1.2</td>
<td>Refusal to walk</td>
<td>Frequent otitis media</td>
<td>Tymanostomy tube placement at 6 mo</td>
<td>None</td>
<td>None</td>
<td>2</td>
</tr>
<tr>
<td>2</td>
<td>F, 1.8</td>
<td>Fever, tachypnea, pneumonia</td>
<td>Ingrown toenail 4 mo earlier, RSV infection at 5 mo</td>
<td>None</td>
<td>Left knee pain 1 week before presentation</td>
<td>Ceftriaxone for pneumonia</td>
<td>2</td>
</tr>
<tr>
<td>3</td>
<td>M, 3</td>
<td>Fever, stomach ache</td>
<td>Asthma</td>
<td>Circumcision during neonatal period</td>
<td>None</td>
<td>None</td>
<td>1</td>
</tr>
<tr>
<td>4</td>
<td>F, 10</td>
<td>Flank pain</td>
<td>None</td>
<td>None</td>
<td>Left lower extremity cellulitis, exposure to kittens</td>
<td>Cephalexin for cellulitis</td>
<td>3</td>
</tr>
<tr>
<td>5</td>
<td>M, 10</td>
<td>Back pain for 5–7 d, abdominal pain, shortness of breath</td>
<td>Asthma</td>
<td>None</td>
<td>Fall 1 week earlier with resulting back pain</td>
<td>Amoxicillin for unknown indication</td>
<td>5</td>
</tr>
<tr>
<td>6</td>
<td>M, 13</td>
<td>Influenza-like symptoms</td>
<td>None</td>
<td>None</td>
<td>Recent soft tissue abscess lanced with needle at home</td>
<td>None</td>
<td>2</td>
</tr>
<tr>
<td>7</td>
<td>M, 14</td>
<td>Difficulty walking</td>
<td>None</td>
<td>None</td>
<td>Soft tissue abscess within previous 6 mo</td>
<td>None</td>
<td>5</td>
</tr>
<tr>
<td>8</td>
<td>F, 17</td>
<td>Fever, nausea, vomiting</td>
<td>None</td>
<td>Tonsillectomy and adenoidectomy</td>
<td>None</td>
<td>None</td>
<td>1</td>
</tr>
<tr>
<td>9</td>
<td>F, 13</td>
<td>Back pain for 2 wk</td>
<td>None</td>
<td>None</td>
<td>Soft tissue abscess within previous 6 mo</td>
<td>None</td>
<td>3</td>
</tr>
</tbody>
</table>

F: female; M: male; Pt: patient; RSV, respiratory syncytial virus.

### TABLE 2 Summary of Clinical Data on 9 Patients With SEA: Symptoms on Presentation

<table>
<thead>
<tr>
<th>Pt</th>
<th>Fever</th>
<th>Irritability</th>
<th>Weakness</th>
<th>Enuresis/Encopresis</th>
<th>Musculoskeletal</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>None</td>
<td>Yes</td>
<td>Yes, bilateral lower extremities</td>
<td>Yes, constipation</td>
<td>Refusal to bear weight or sit up</td>
</tr>
<tr>
<td>2</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes, left lower extremity</td>
<td>None</td>
<td>Slight left lower extremity limp</td>
</tr>
<tr>
<td>3</td>
<td>Yes</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>None</td>
</tr>
<tr>
<td>4</td>
<td>Yes</td>
<td>None</td>
<td>Brisk reflexes bilateral lower extremities</td>
<td>None</td>
<td>None</td>
</tr>
<tr>
<td>5</td>
<td>Yes</td>
<td>None</td>
<td>Yes, bilateral lower extremities</td>
<td>None</td>
<td>Bilateral flank pain</td>
</tr>
<tr>
<td>6</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>None</td>
</tr>
<tr>
<td>7</td>
<td>None</td>
<td>None</td>
<td>Decreased strength in bilateral hip flexors, increased reflexes bilateral lower extremities with clonus, left scapular protrusion</td>
<td>None</td>
<td>Neck pain</td>
</tr>
<tr>
<td>8</td>
<td>Yes</td>
<td>Ill appearing</td>
<td>None</td>
<td>None</td>
<td>None</td>
</tr>
<tr>
<td>9</td>
<td>Yes</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>Back pain with significant decreased range of motion, mild swelling of thoracic area, alteration of gait</td>
</tr>
</tbody>
</table>

Pt, patient.
eventually be tailored to the offending organism. Due to increased rates of MRSA, vancomycin should be used for antistaphylococcal coverage as was done for all but 1 patient in this cohort.1,10 Duration of intravenous antibiotic therapy should be a minimum of 4 to 6 weeks.6 The length of therapy is dependent on the level of immunocompetence, clinical improvement, and response to treatment demonstrated by subsequent ESR and MRI.8 The typical treatment of SEA is surgical decompression and drainage in combination with several weeks of antibiotics,4,13,26 although no consensus on management has been reported.2 Some studies have shown improved outcomes with surgery,5,16,17,22,26 whereas others have shown success with alternative treatment options in select adult populations.2,7–9,27,28 There are limited data pertaining to the pediatric population. Enberg reviewed

<table>
<thead>
<tr>
<th>Pt</th>
<th>Laboratory Results</th>
<th>Radiologic Results</th>
<th>Imaging Received</th>
<th>Description of SEA Location and Features</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>White Blood Cell/μL</td>
<td>Platelet Count, cells/μL</td>
<td>ESR, mm/h</td>
<td>CRP Level, mg/L</td>
</tr>
<tr>
<td>1</td>
<td>13 800</td>
<td>425 000</td>
<td>104</td>
<td>N/A</td>
</tr>
<tr>
<td>2</td>
<td>6600</td>
<td>212 000</td>
<td>41</td>
<td>137.6</td>
</tr>
<tr>
<td>3</td>
<td>20 000</td>
<td>335 000</td>
<td>114</td>
<td>N/A</td>
</tr>
<tr>
<td>4</td>
<td>12 900</td>
<td>425 000</td>
<td>105</td>
<td>174.5</td>
</tr>
<tr>
<td>5</td>
<td>6400</td>
<td>212 000</td>
<td>140</td>
<td>N/A</td>
</tr>
<tr>
<td>6</td>
<td>9800</td>
<td>197 000</td>
<td>61</td>
<td>N/A</td>
</tr>
<tr>
<td>7</td>
<td>10 000</td>
<td>448 000</td>
<td>10</td>
<td>182</td>
</tr>
<tr>
<td>8</td>
<td>15 800</td>
<td>130 000</td>
<td>88</td>
<td>178.2</td>
</tr>
<tr>
<td>9</td>
<td>11 100</td>
<td>320 000</td>
<td>80</td>
<td>N/A</td>
</tr>
</tbody>
</table>

N/A, not available; PET, positron emission tomography; Pt, patient.
46 pediatric cases described in the literature spanning 1835 to 1972. In this review, 8 of 13 patients who received surgery before paralysis had full recovery (although 1 developed kyphoscoliosis 6 months later). Outcomes in cases when surgery was performed within 24 hours of neurologic deficit were also favorable, consistent with other reports. Unfortunately, nonsurgical management was fatal in 10 of 10 cases spanning 1835 to 1961. In Rubin’s review of 57 pediatric cases before 1993, 50 patients (88%) received surgery, the remaining 7 did not undergo surgery because of poor clinical condition or an incorrect diagnosis, and 6 of these patients died. In Auletta’s review of 8 pediatric cases from 1984 to 1999, 6 received surgical drainage, 1 received CT-guided needle drainage, and 1 received no surgical intervention; no patients died, and only 2 had minor neurologic sequelae. Our review of 9 cases from 2002 to 2011 demonstrates successful outcomes in all 9 patients with only 2 patients receiving nonsurgical intervention and 4 patients undergoing CT-guided needle drainage. All patients in our review presented before onset of paralysis, which historically has greatly improved outcomes compared with presentation after the onset of neurologic symptoms.

There are 4 scenarios based on adult data in which nonsurgical management may be considered: no neurologic deficit because these patients are likely less advanced in the disease course, extensive disease to the entire spinal column that would require a multilevel laminectomy, >48 hours of paraplegia (reports have shown much less chance of neurologic recovery), and extensive comorbid conditions that preclude safely undergoing surgery. The absence of neurologic deficit in our review accompanied by a favorable outcome lends support to the aforementioned nonsurgical approach. There is a risk of deterioration with conservative treatment even in those without neurologic symptomatology. Reviews of the adult literature have shown that 19% to 23% of patients with nonsurgical management develop worsening neurologic symptoms despite appropriate antibiotic therapy. Nonsurgical management can include serial neurologic exams, inflammatory marker evaluation, and repeat MRI to ensure resolving infection.

The length of time before initiation of treatment and the severity of neurologic deficit are the 2 factors most predictive of outcome. There is less neurologic recovery with prolonged neurologic deficit before initiation of therapy; paralysis for >24 hours is commonly irreversible. Although data are limited, children tend to have improved outcomes compared with adults.

In rare disease processes such as SEA, it is not feasible to form a clinical trial to compare interventions; therefore, we must rely on past experiences reported and tailor management decisions based on the details of each case. We present 1 of the largest cohorts of pediatric SEA from a single institution successfully managed with nonoperative treatment. This pediatric review demonstrates the potential success of such management (ie, minimally invasive drainage techniques, serial laboratory studies, and imaging) in combination with systemic antimicrobial treatment and may be a prudent approach in select children.

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REFERENCES


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