Case Report

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Pediatric Spinal Epidural Abscess: A Case Report of a 12-year-old Girl Without Risk Factors

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Abstract

A 12-year-old girl presented with a spinal epidural abscess (SEA), an unusual emergent infectious disease that often requires surgical intervention. Its nonspecific symptoms and rarity in pediatric patients frequently delay the diagnosis until neurologic symptoms develop. This patient had only fever without back pain or neurologic symptoms at the first visit. Whole-body CT later diagnosed a SEA, which was treated by antibiotics only. No neurologic deterioration was observed, and the fever and now-present pain gradually diminished. Early diagnosis of a SEA is important to avoid the development of symptoms. Despite its irradiation to the patient, CT might be useful for providing an early diagnosis in the absence of neurologic symptoms.

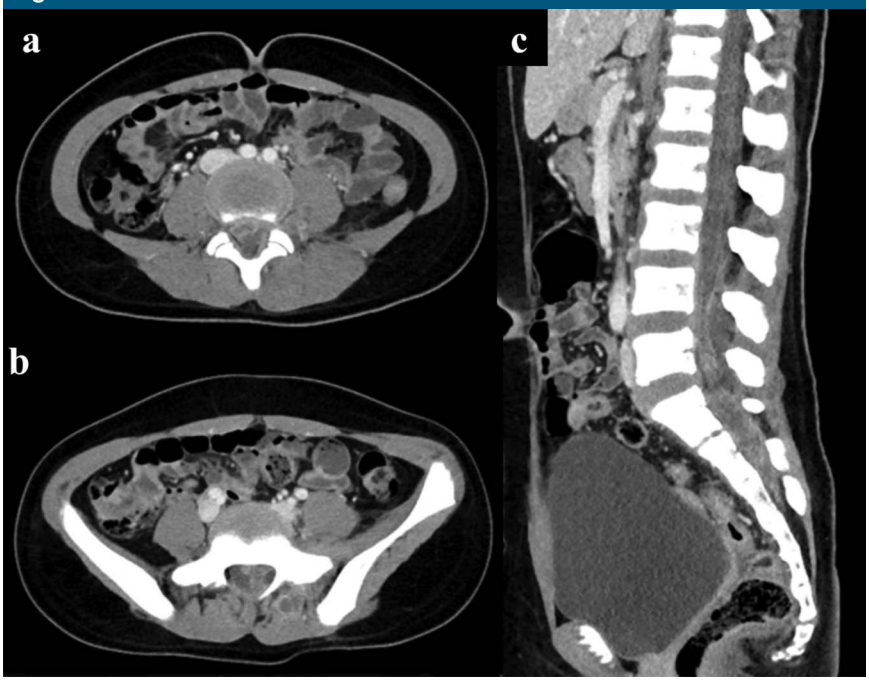
Spinal epidural abscess (SEA) is an unusual emergent infectious disease that often requires surgical intervention.[1] The classic presentation of SEA is a triad of back pain, fever, and neurologic deficits, although they are found in only a small number of the patients.[2,3] Predisposing risk factors have been reported, including previous trauma, infection, intravenous drug use, instrumentation of the epidural space, and immunodeficiency,[4,5] although they have been reported in only 65% to 81% of adults with SEA and 35% to 67% of pediatric patients with SEA.[4-6] Thus, it is rare even to suspect a SEA, especially in pediatric patients, and its diagnosis is frequently delayed until neurologic symptoms develop.[6,7]

We report a pediatric patient without risk factors in whom SEA was immediately diagnosed by chance using CT and was treated successfully using antibiotics without the development of neurologic complications.

Case Report

A 12-year-old girl was referred to the pediatrics department in our institution because of a 7-day history of fever (maximum 39.0°C) of unknown origin. No previous infections, trauma, or surgery, and remarkable medical history were found. On examination, her vital signs were normal except for the fever. No respiratory or central neurologic symptoms were observed. The rest of the physical examination performed by a pediatrician was negative. Laboratory evaluations included the following: white blood cell count (WBC) $10{,}200 \times 10^2/\mu L$ (76.1% neutrophils), C-reactive protein (CRP) 17.8 mg/dL, procalcitonin 0.2 ng/mL, and negative influenza. Two sets of blood cultures were collected at the time of admission.

Figure 1



CT scan showing a large epidural mass in the paramedian left spinal canal with intravenous contrast. **A**, An axial CT image of the L4/5. **B**, An axial CT image of the L5/S. **C**, A sagittal CT image that shows an epidural mass existing L3 to S1.

She was admitted to the pediatrics department the same day with the suspicion of infection, neoplasm, or collagen disease. Empirical treatment with intravenous cefotaxime was prescribed. On day 2 after admission, she reported of left buttock pain, but without neurologic symptoms. On day 3, the fever was still present, and blood culture revealed the presence of *Staphylococcus aureu*s. To detect any focus of infection immediately, pediatricians performed echocardiography and CT at thoracic-to-pelvic levels to identify the origin of the fever. Echocardiography showed no vegetation. CT with intravenous contrast, however, revealed a large epidural mass in the paramedian left spinal canal, from L3 to S1 (Figure 1). On day 4, gadolinium-enhanced MRI of the lumbar spine revealed a large mass with ring enhancement in the same area (Figure 2).

Our department was consulted on the same day when SEA was diagnosed. The patient still reported of left buttock pain and had local knocking pain on her lower back. On neurologic examination, the straight leg raising test showed a tension sign in her left leg (80° on the right, 70° on the left), but no motor weakness or sensory disturbance was observed. In addition, no weakness of the deep tendon reflexes and no bladder or bowel dysfunction were observed.

Considering the almost normal neurologic examination, we continued antimicrobial treatment with intravenous cefotaxime, to which the *S aureus* was sensitive. No deterioration of the neurologic examination was observed, and the fever and pain gradually disappeared. On day 11, MRI revealed a complete resolution of the SEA (Figure 3), and a normal CRP result was obtained on day 13. Consequently, the antimicrobial treatment was modified empirically, switching to intravenous cefazolin. However, on day 19, her temperature had risen to 38.4°C with the presence of cough and mucus. Her WBC was $9400 \times 10^2/\mu L$, and her CRP was 5.6 mg/dL. With the suspicion of SEA recurrence, we performed MRI of her spine, which revealed no recurrence. The fever spontaneously disappeared, and her WBC and CRP levels became consistently normal at follow-up examinations.

She was discharged with no symptoms on day 41 but with oral antibiotics continued for another 4 weeks (Figure 4). At 3 months after discharge, she displayed no symptoms, sequelae, or recurrence of the SEA on MRI at a clinical follow-up visit.

Discussion

SEA is an uncommon infectious disease with a reported incidence in pediatric patients of 0.6 to 1.5 per 10,000 hospital admissions.[4,5] However, the classic symptomatic triad associated with SEA—back pain (70% to 100%), fever (50%), and neurologic manifestations (33%)—are found in only 8% of the patients.[6,8] Because of the rarity of the disease, the few patients with the complete prognostic classic triad, and the low percentage of predisposing risk factors, the diagnosis of SEA in pediatric patients is challenging and frequently delayed, which allows the development of neurologic deficits even in pediatric

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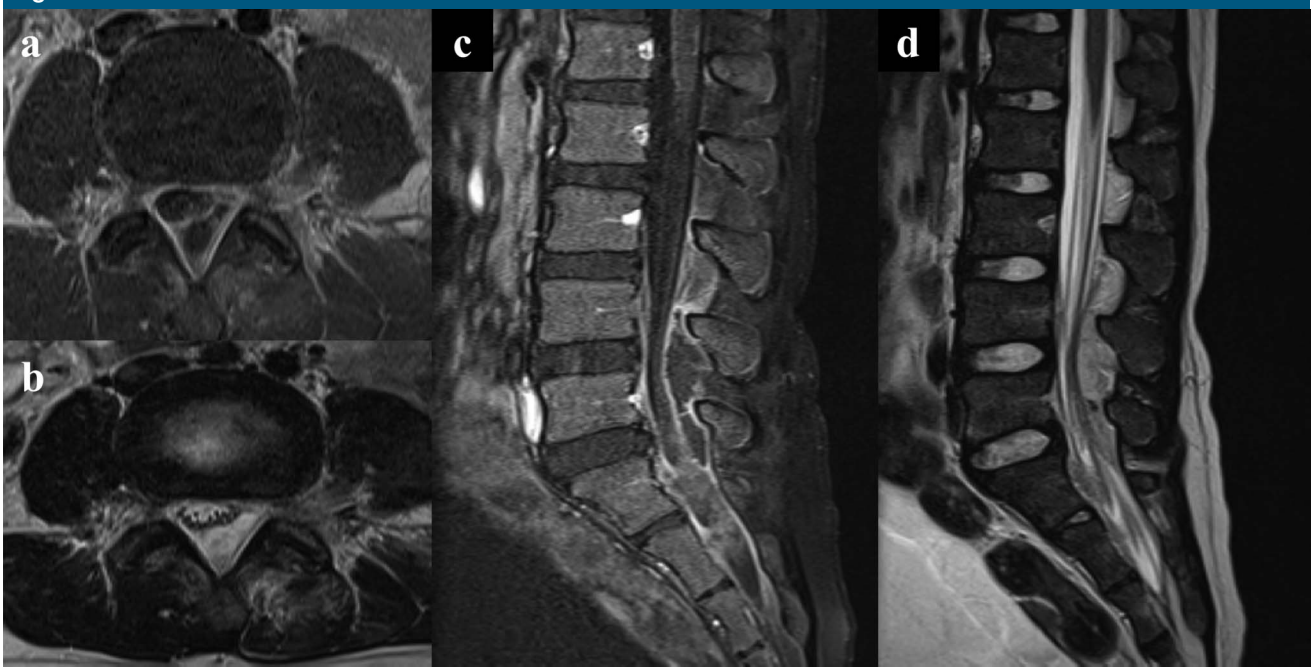
Figure 2



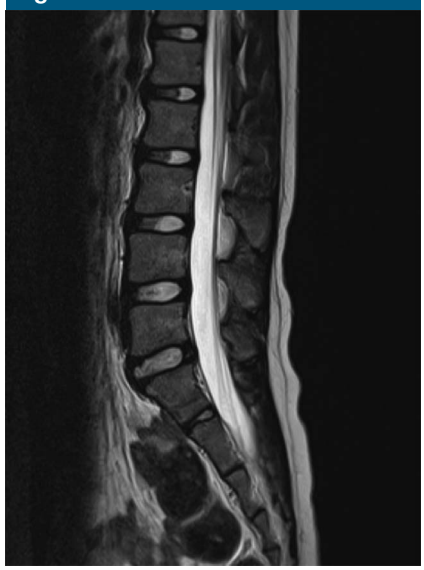
Gadolinium-enhanced MRI showing a large spinal epidural abscess. **A**, An axial MRI image of L4/5 with the T1 gadolinium-enhancement. **B**, An axial MRI image of L5/S with the T2 enhancement. **C**, A sagittal MRI image with the T1 gadolinium-enhancement. **D**, A sagittal MRI image with the T2 enhancement.

patients (15% to 25%).[4,6,7] In addition, most of the patients who did not undergo surgical treatment died because of poor clinical condition or misdiagnosis in the literature before 1990s.[1,9]

Early diagnosis of SEA is important to avoid the development of identifiable symptoms. The benchmark modality for detecting SEA is gadolinium-enhanced MRI, with >90% sensitivity and specificity.[8] For earlier diagnosis using MRI, an algorithm for SEA diagnosis is proposed for adult patients with severe back pain.[8] According to this algorithm, the patients with back pain are encouraged to undergo emergent or urgent MRI when they experience a neurologic deficit, any of the risk factors, and/or an elevated erythrocyte sedimentation rate or CRP level. Although back pain is the major symptom of the clinical triad, some patients with SEA (including the patient in this case) have no back pain or risk factors. Therefore, this algorithm for SEA diagnosis using MRI may not be useful, especially in pediatric patients. If MRI is not available, CT with intravenous contrast is an alternative diagnostic modality, although its sensitivity is low.[6,8] There have been reports on the usefulness of bone scans or positron emission tomography–CT in pediatric patients with fevers of unknown origin.[10,11] To narrow the clinical investigation and rule out neoplasms or other diseases, a whole-body CT (with/without positron emission tomography) or bone scan might be performed in the pediatric patient with fever or low back pain, but whose origin of symptoms is unknown, although these modalities are associated with irradiation.

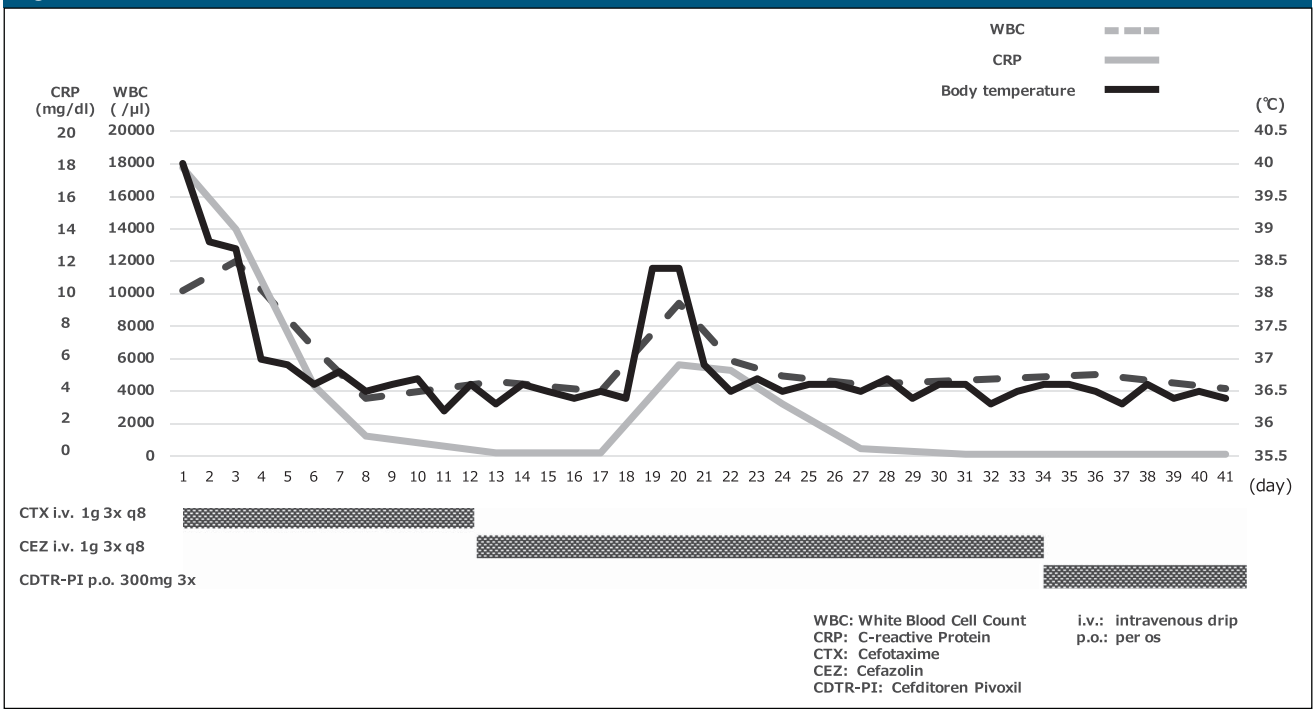
So as not to overlook SEA in a differential diagnosis, the most important factor to know is that some patients with SEA have no risk factors. In the reviews of the SEA, 5 of 8 patients (63%) had no medical conditions and surgical conditions that

Figure 3



MRI on day 11 revealing a complete resolution of the spinal epidural abscess.

Figure 4



Flowchart showing the clinical course during admission.

predisposed them to infection, as proposed by Auletta and John,[4] and 3 of 9 (33%) had no risk factors, as proposed by Hawkins and Bolton.[5] Even cases of SEA may exist that are treated using empirical antibiotics and consequently cured without any diagnosis. Pediatricians should include spinal infectious disease in the differential diagnosis of fever of unknown origin. Orthopaedic surgeons should not hesitate to perform neurologic examinations in pediatric patients with low back pain or fever and should allow them to undergo some spinal diagnostic modality or refer them for consultation at a better equipped institution.

The predictive factors for the outcome of patients with SEA are the length of time before treatment and the severity of the neurologic deficit.[5] Furthermore, proper treatment including surgical intervention is an important factor for prognosis. In the reviews of the adult literature in the 1990s, 19% to 23% of patients treated with only antibiotic therapy developed worsening neurologic symptoms despite appropriate antibiotics.[12,13] However, the success rate of nonsurgical treatment increased from 12.5% in the 1990s to 33% in the 2000s in patients without risk factors or medical conditions.[4,5] The advancement of antibiotics and infectious disease medicine and prompt diagnostic modality such as MRI may improve the treatment of SEA, especially without risk factors, and the higher success rate of nonsurgical treatment may be expected in recent years.

In this case, we would not have been aware of the presence of SEA without whole-body CT scans because the patient had no specific symptoms or risk factors. CT with intravenous contrast performed to identify the origin of the patient's fever revealed a spinal disease. Because of the anatomic field limitations of MRI, CT might be a more useful modality if SEA is suspected in pediatric patients and/or to identify other diseases in patients with nonspecific symptoms, providing an early diagnosis before neurologic deficits develop. In addition, nonsurgical treatment might be successful fortunately because this patient had no risk factors or medical conditions. We recommend nonsurgical treatment at first in pediatric patients with SEA without risk factors and neurologic symptoms, when SEA is diagnosed earlier by some modality.

Conclusion

We reported a pediatric patient without risk factors in whom a SEA was diagnosed by chance using a CT scan. Despite taking into consideration its irradiation, CT might still have the advantage of diagnosing a SEA before neurologic deficits appear. Nonsurgical treatment should be considered in patients without risk factors and neurologic symptoms.

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