

CASE REPORT

Spinal epidural empyema – an unusual cause of focal back pain in a teenager

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ABSTRACT

A spinal epidural empyema (SEE) is a collection of pus between the bones of the vertebral column and the dura covering the spinal cord. It is an uncommon disease in the pediatric population. It requires prompt diagnosis and targeted treatment to prevent permanent disability. The gold standard of management involves surgical treatment and systemic antibiotic therapy.

We present the clinical case of a 15-year-old boy with focal back pain in the lower thoracic region, causing limitations of spinal mobility, but no neurological dysfunction. Due to the patient's history, elevated inflammatory parameters and magnetic resonance imaging, epidural empyema was detected. The patient was operated on with the evacuation of pus from epidural space and temporary drainage placement and following administration of intravenous antibiotics. A complete recovery was achieved.

The non-specificity of the SEE symptoms poses a significant challenge for clinicians. Increased vigilance is therefore necessary when diagnosing seemingly benign disorders for a favorable clinical outcome.

KEY WORDS:

back pain, pediatrics, spinal epidural abscess, MSSA, spinal epidural empyema.

INTRODUCTION

Spinal epidural empyema (SEE) is a very rare infectious disease in the pediatric population [1–3]. Due to its oligosymptomatic course, it poses an extremely significant challenge to the clinician. It represents an even greater threat because rapid implementation of treatment is necessary to achieve a positive therapeutic outcome and avoid permanent neurological deficits, such as symptoms of para- and tetraplegia or paresis, as well as death [1, 2].

In order to make a diagnosis of SEE, a detailed history should be taken, taking into account risk factors.

A thorough physical examination is important, including a complete neurological evaluation and additional tests that take into account inflammatory parameters. In the typical clinical and radiological picture, the diagnosis can be made on the basis of contrast-enhanced magnetic resonance imaging (MRI) [1–4]. Intraoperative assessment of the lesion serves as definitive confirmation of the SEE diagnosis.

The remarkably low incidence of SEE among children and the non-specificity of clinical symptoms provided the basis for preparing a case report of this disease, which developed in a previously healthy teenager.

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CASE REPORT

Fifteen-year-old boy presented to the hospital because of persistent back pain in the lower thoracic region at T10–T12, lasting for several days. The pain was exacerbated in the supine position. The patient did not have a fever, and there had been two episodes of vomiting in the previous few days. The patient's history of trauma, infectious and chronic diseases, stimulants, and ongoing medications was negative. The patient was vaccinated according to the current immunization schedule. Critical parameters such as blood pressure, heart rate and saturation remained normal. On physical examination, there were no abnormalities. Laboratory tests were notable for leukocytosis (21.80 thousand/ μ l), elevated C-reactive protein (CRP) (8.9 mg/dl with normal cut-off value < 0.5 mg/dl) and creatine kinase (1097 U/l) with normal values of procalcitonin, troponin I and the MB isoenzyme of creatine kinase.

During the first days of hospitalization, both complaints of back pain and inflammatory parameters (leukocytosis and CRP – 20 mg/dl with normal < 0.5 mg/dl) were found to be increasing. A neurological re-examination on the second day of hospitalization revealed stiff neck, Kernig's sign and bilateral Lasague's sign with increasing, painful limitation of thoracic spine mobility. There were no motor or sensory deficits or other neurological symptoms. A review computed tomography (CT) scan of the thoracic and lumbosacral spine showed no abnormalities (Figure 1). An ultrasound (US) scan of the paravertebral tissues was insignificant. Due to the suspicion of an inflammatory process affecting the thoraco-lumbar region of the vertebral column, the patient was transferred to a higher referral center, for further diagnosis and treatment.

Magnetic resonance imaging examination of the thoraco-lumbar region revealed a dense fluid reservoir



FIGURE 1. Computed tomography scans of the thoracic and lumbosacral region showing no abnormalities in the spinal column

of max. 10 × 17 mm located in the epidural space at the level of T1–T12. The lesion compressed the spinal cord and spinal cone on the dorsal side with reduction of the fluid reserve of the meningeal sac (Figure 2). In view of the overall clinical picture and the results of additional tests, the diagnosis of SEE was made. The patient was evaluated by a neurosurgeon and scheduled for surgical intervention. A partial evacuation of the SEE was performed via a left T12/L1 fenestration with subsequent insertion of a catheter into the epidural space, and next connected with a Redon bottle without a vacuum. Empirical parenteral antibiotic therapy (ceftriaxone and vancomycin) was administered.

A follow-up MRI performed 2 days after surgery showed a marked reduction in the size of the abscess compared to the previous study – a 5 × 11 mm thick fluid reservoir was visualized, located at the T1–T12 level, the lesion slightly displacing the spinal cord ventrally, with no signs of compression. The fluid reserve was preserved but reduced (Figure 3).

The amount of liquid in the Redon drain was minimal (less than 5 ml per day) and stopped on the fifth day after the surgery, so the drainage was evacuated.

After obtaining cultures of the pus collected during the surgical procedure, the etiological agent was identified: *Staphylococcus aureus* – a methicillin-susceptible strain. Treatment was maintained as consistent with the antibiogram, with gradual improvement in the patient's con-

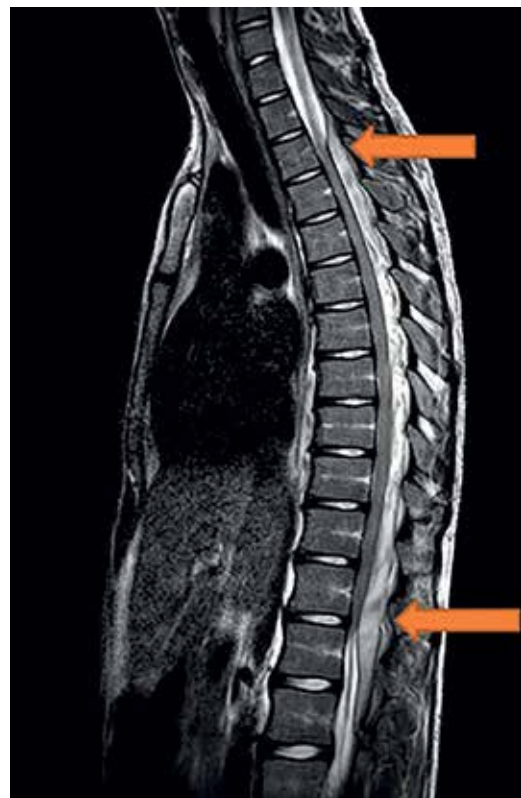


FIGURE 2. Magnetic resonance imaging performed before surgical intervention. T2-weighted image of the thoracic region (arrows indicate the upper [T1] and lower [T12] limits of the epidural empyema)



FIGURE 3. Magnetic resonance imaging performed two days after surgical intervention. T2-weighted image of the thoracic region (arrows indicate the upper [T1] and lower [T12] limits of the epidural empyema)

dition and reduction in inflammatory parameters. After three weeks, a repeat MRI scan of the thoracic spine was performed, showing no visible abnormalities (Figure 4).

Despite clinical and radiological improvement, one week later there was a recurrence of thoracolumbar region pain of lesser severity than before with accompanying vomiting and stomach ache. After basic blood analysis, features of acute kidney injury (AKI) were found. The analysis was expanded to include the serum concentration of vancomycin, leading to the confirmation of AKI due to antibiotic therapy. Such side effects are sometimes difficult to avoid with the aggressive antibiotic therapy that is required in severe infectious diseases. It is important to keep in mind the possibility of their occurrence, especially with long-term therapy. It is vital to monitor both the clinical condition of the patient and the function of the internal organs. In the case of vancomycin, it is crucial to monitor blood creatinine levels, in order to detect early deterioration of renal function and prevent renal failure. It may be useful to monitor the serum concentration of the drug during pharmacotherapy, with the aim of possibly implementing renal replacement therapy or by changing the antibiotic. In the described case, treatment with vancomycin was terminated, while ceftriaxone was continued. After symptomatic treatment, mainly intensive hydration, normalization of renal parameters was achieved.

The diagnosis was expanded to search for the primary source of infection, which could be the starting point for



FIGURE 4. Magnetic resonance imaging performed three weeks after surgical intervention and systemic antibiotic therapy. T2-weighted image of the thoracic region with no visible abnormalities

the formation of the SEE; however, no clear cause could be established. The boy did not present immunodeficiency (immunoglobulin levels, complement components, anti-nuclear antibodies, HIV, granulocyte chemiluminescence measurement remained normal). Echocardiography excluded a leaking heart defect.

After a six-week hospitalization, the patient was discharged home with the recommendation of oral antibiotic therapy for another two weeks (amoxicillin with clavulanic acid in combination with clindamycin) and neurosurgical follow-up after two months. He re-appeared as recommended, and reported no worrisome complaints. On physical examination, he presented no abnormalities. The next MRI examination was performed, revealing no further abnormalities.

DISCUSSION

Spinal epidural empyema primarily affects the adult population, with the highest incidence in the fifth to seventh decades of life [1]. To date, only a few dozen cases of SEE have been described in the pediatric population (0–18 years) [1]. The empyema usually develops by blood-borne spread from another source of infection. For this reason, it is important to have a history of recent urinary tract infections and respiratory infections and also to extend the diagnosis to infective endocarditis. Other possible routes of abscess formation are: infection

by continuity and iatrogenic. The main causative pathogen of SEE is considered to be the gram-positive bacterium *Staphylococcus aureus*. Gram-negative bacteria are isolated less frequently; they include *Escherichia coli* and *Pseudomonas aeruginosa* [1, 2, 4]. For the adult population, many risk factors for abscess have been described, which should also be considered when taking a history with a pediatric patient, including immunosuppression, diabetes mellitus, local skin infections, trauma, and spine surgery. Despite the lower prevalence of factors such as drug addiction (primarily with intravenous drug use) and alcoholism in this age group, these elements should not be overlooked when taking a history [1–3].

The typical clinical picture of SEE includes the characteristic triad of symptoms: fever, back pain and neurological deficits [1–3]. The above symptoms rarely occur simultaneously in a given patient, and their occurrence may instead be spread over time, depending on the phase of the disease [1, 2]. The clinical course commences with the phase of empyema formation, during which fever and local back pain are most often the predominant symptoms [2]. The next phase is manifested by neck stiffness and the appearance of root pain, which suggests mechanical and chemical irritation of the meninges in the process of empyema formation and enlargement. The next stage is the emergence of neurological symptoms in the form of both spinal syndromes and function disorder of structures innervated by the affected part of the central nervous system [1, 2]. Among the most common are bowel and bladder dysfunction [2]. This is related to the increased tendency for the empyema to be localized in the thoracolumbar region of the vertebral column, above the medullary cone of the spinal cord [1].

In the present case, the clinical presentation was oligosymptomatic, with no fever, either during hospitalization or in the pre-admission history. The patient presented only localized back pain in the area of the empyema. This is consistent with previous reports in the literature regarding the rare co-occurrence of the classic triad of SEE symptoms [1, 3, 4]. In the following days of hospitalization, meningeal symptoms and root pain were added, raising the suspicion of a neurological cause of the complaints. The progression of symptoms was probably due to the development of the next phase of empyema formation [3, 4]. The simultaneous presence of all the symptoms characteristic for SEE constitutes an unfavorable prognostic factor regardless of the implementation of targeted treatment [4]. With the delay in diagnosis and appropriate treatment, there is a considerable risk of paralysis of the function of internal organs, complete paralysis of the limbs, and even death as a result of spreading neuroinfection [1, 2, 4].

After taking a thorough history as to the temporal dynamics of symptoms, risk factors, and possible sources of infection, additional tests are required to verify the clinical suspicion of an inflammatory process. Laboratory

tests consider particular emphasis on the evaluation of peripheral blood count, and elevated levels of acute phase proteins such as CRP, fibrinogen and procalcitonin. Identification of the spinal canal microbiota is necessary [2]. Paravertebral soft tissue US of the pain-affected spinal region may be helpful in the differential diagnosis [5]. A definitive diagnosis of SEE is possible after imaging with intravenous contrast administration of the area clinically suspected to have a lesion. Magnetic resonance imaging is the preferred test, with sensitivity and specificity both greater than 90% [2]. However, in the case of atypical image of SEE, neurosurgery remains a crucial procedure, not only for treatment but also to confirm the final diagnosis [5–7].

In the present case, there were significantly elevated inflammatory parameters, raising the suspicion of an infectious cause of the observed symptoms. Imaging studies such as CT of the spine and US of the soft tissues of the paravertebral region did not reveal abnormalities. The false-negative outcome of the CT scans might be attributable to the lack of contrast enhancement, as it was performed as a screening test before the patient's admission to the higher referral center. Another factor affecting the outcome of the study was the location of the lesion within the vertebral canal. The computed tomography imaging of this region is not a dedicated diagnostic test due to the high density of bony structures surrounding the spinal cord, which underestimates the resolution of the CT when evaluating soft-tissue or fluid structures. A recommended MRI of the spine with contrast allowed the diagnosis to be made.

The gold standard of management to date is surgical drainage of the empyema with simultaneous decompression of nerve structures [1, 2, 8–10]. At the same time, causal treatment implies the implementation of systemic empirical intravenous antibiotic therapy, which needs to be correlated and possibly modified after obtaining an antibiogram from the material collected during surgery [11].

Different guidelines are present for both the route of administration and the length of antibiotic therapy. To ensure optimal bioavailability and penetration of the antibiotic in the tissue, intravenous antibiotic therapy is recommended in all cases, especially at the beginning of treatment [2]. The duration of intravenous treatment ranges from 3–4 [8] to 6–8 weeks [2], depending on the center, as well as the patient's general condition. After that, sequential therapy is possible. On the other hand, according to the European Society of Clinical Microbiology and Infectious Diseases, the recommended duration of antimicrobial treatment is 6–8 weeks. No recommendation is offered for early transition to oral antimicrobials because of a lack of data, and oral consolidation treatment after ≥6 weeks of intravenous antimicrobials is not routinely recommended [11].

In the described case, the patient's treatment modality was discussed with pediatricians and infectious disease

physicians, and intravenous treatment for 4 weeks was considered optimal, pending confirmation of a normal MRI of the spine. The patient's hospitalization was prolonged to 5 weeks by an episode of AKI. Subsequently, it was decided to use oral treatment for 3 weeks, so that the total treatment time was 8 weeks – the upper limit for the length of treatment in most guidelines.

Lumbar puncture is not recommended because of the possible spread of infection to the subarachnoid space and the risk of iatrogenic induction of the neuroinfection [2].

In the pediatric population, SEE poses a special challenge in terms of both diagnosis and treatment. Despite its extremely rare occurrence, it should be considered in any case of local back pain with concurrent fever or elevated inflammatory parameters [12–16]. Prompt and accurate diagnosis remains crucial for successful treatment, but it is this stage that appears to be the most difficult. The oligosymptomatic course, non-specificity of symptoms and sometimes difficult communication with the pediatric patient present a number of obstacles for the clinician during the diagnostic process [17].

In contrast to adults, focal back pain is not a common complaint in pediatric patients and always requires special attention. Increased clinical vigilance is required when the pain is exacerbated at night or in a horizontal position, suggesting disturbances in the circulation of cerebrospinal fluid. Similar complaints, with predominant focal back pain, may also manifest in inflammatory diseases of the vertebral column, neoplastic proliferation, disc herniation or degenerative changes associated with the spectrum of juvenile spondyloarthropathies [1, 9–13].

CONCLUSIONS

Due to the risk of permanent neurological deficits and also burdensome treatment for the patient, SEE is one of the most serious, although extremely rare, conditions manifesting as local back pain in the pediatric population.

DISCLOSURES

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