





## CASE REPORT

# Extensive Spinal Epidural Abscess Resulting in Complete Paraplegia Treated by Selective Laminectomies and Irrigation

Tongshuai Xu, MM , Yukun Du, MD , Jianwei Guo, MD , Jianyi Li, MD , Cheng Shao, MM, Changfang Shi, BM, Xianfeng Ren, MD, Yongming Xi, MD

*Department of Spinal Surgery, The Affiliated Hospital of Qingdao University, Qingdao, China*

**Background:** Spinal epidural abscess (SEA) is an uncommon clinical entity that is often subject to delayed diagnosis and suboptimal treatment. Untreated disease leads to compression of the spinal cord, resulting in devastating complications.

**Case Presentation:** A 56-year-old man visited our hospital for progressive lower back and lower extremity pain of several days' duration. Significant pyrexia (39.5°C) and elevated C-reactive protein (89.2 mg/L) were detected during admission, but no positive neurological examination findings were observed. Magnetic resonance imaging revealed pyogenic discitis at L3–4. Despite the administration of directed antibiotic therapy, the patient's condition rapidly deteriorated, culminating in complete paraplegia secondary to an extensive SEA from L4 to C7. Emergency spinal decompression surgery was canceled due to his poor clinical condition and refusal of informed consent. After further deterioration, he consented to two-level selective laminectomies and irrigation.

**Conclusions:** In contrast with prior case reports, this case illustrates the natural history of an extensive SEA during conservative and late surgical treatment. Early diagnosis and timely surgical decompression are of great importance for extensive SEA.

**Key words:** irrigation; laminectomies; neurological infection; paraplegia; spinal epidural abscess

## Introduction

Spinal epidural abscess (SEA) is defined as extensive when it involves more than five vertebral levels. Several decades ago, the incidence of SEA was approximately 1 per 10,000 hospital admissions<sup>1,2</sup>. Recently, the incidence of SEA has increased to approximately 5.1 cases per 10,000 admissions<sup>3</sup>. The increasing incidence is multifactorial, relating to aging, improved diagnostics, and different study populations<sup>4</sup>. To the best of our knowledge, the predisposing factors for SEA include diabetes mellitus, concomitant infection, intravenous drug use, alcohol use, and immunocompromise.

Because SEA does not have a characteristic clinical presentation, it may initially be misdiagnosed. Without rapid

diagnosis and treatment, SEA may progress to spinal cord compression, leading to acute flaccid paralysis and eventually, death. Upon diagnosis of SEA, the major dilemma is the choice of treatment; most studies support surgical decompression combined with systemic antibiotics<sup>5</sup>. Patients with acute complete loss of neurological function secondary to SEA achieve good recovery with surgical debridement. Non-surgical treatment is preferred in patients with no or minimal neurological impairment, smaller abscesses, and prohibitive operative risk factors.

This report presents a rare case of a patient with extensive SEA treated with antibiotics and late surgical decompression and drainage<sup>6,7</sup>. Unfortunately, due to

**Address for correspondence** Yongming Xi and Xianfeng Ren, Department of Spinal Surgery, the Affiliated Hospital of Qingdao University, No. 59 Haier Road, Qingdao 266000, Shandong, China. Email: [xym700118@163.com](mailto:xym700118@163.com) and [austin26@sina.com](mailto:austin26@sina.com)

<sup>†</sup>Co-first author: Tongshuai Xu and Yukun Du. Both authors contributed equally to this project and manuscript preparation.

Received 10 November 2021; accepted 27 April 2022

uncontrollable factors, the SEA resulted in complete and permanent paraplegia.

## Case Report

### History and Examination

Written informed consent was obtained from the patient and his family for the publication of this case report and accompanying images.

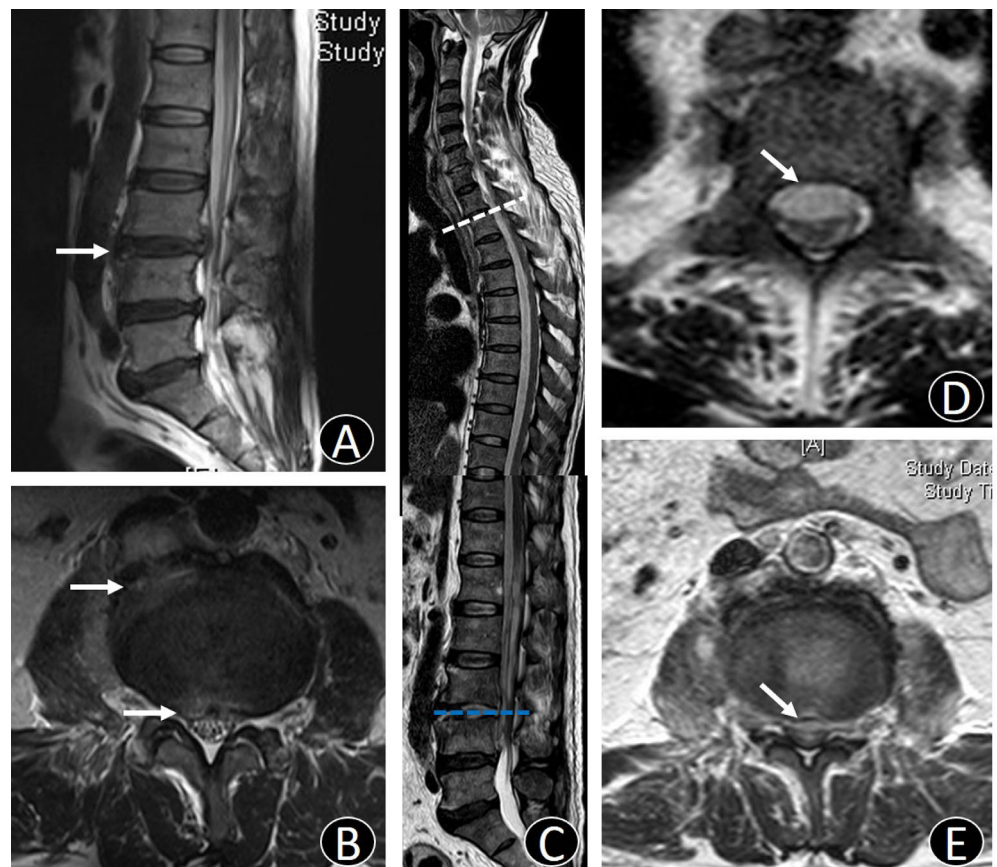
A 56-year-old Chinese man with an unremarkable medical history initially presented with lower back pain for 8 days. He was admitted to our hospital with a suspected lumbar muscle sprain or spondylodiscitis. He reported no lower extremity pain and was afebrile. Lumbar spine computed tomography showed non-specific degenerative changes. Outpatient physiotherapy and analgesia were recommended. However, the patient was readmitted after 2 weeks due to progression of his lower back pain, decreased range of motion in the lumbar spine, and new-onset lower extremity pain. Neurological examination findings were normal.

Two hours after admission, pyrexia (39.5°C) and tachycardia were noted. His blood pressure was 148/92 mm Hg. Laboratory tests showed a hemoglobin level of 138 g/L and white blood cell (WBC) count of  $12.2 \times 10^9/L$  with 80% neutrophils. The C-reactive protein level was 89.2 mg/L;

erythrocyte sedimentation rate (ESR), 61 mm/h; albumin, 30.9 g/L; alanine transaminase, 58 U/L; aspartate transaminase, 47 U/L; and blood glucose level, 10.8 mmol/L. Echocardiography did not show evidence of endocarditis. However, magnetic resonance imaging (MRI) of the lumbar spine revealed pyogenic discitis at L3–4 and abscesses disseminated throughout the anterior, right lateral, and epidural spaces of L3–4 (Figure 1(A),(B)). Suspecting *Staphylococcus aureus* infection, empirical intravenous antibiotic therapy with vancomycin (1000 mg twice daily) was administered before obtaining blood culture results. The trough concentration of vancomycin was monitored. Given the extent of the abscess, surgery was determined to be unsuitable until neurological deterioration was noted.

The results of blood culture and sensitivity testing revealed methicillin-resistant *S. aureus* sensitive to vancomycin. Thus, intravenous vancomycin was continued. After 1 week of intravenous vancomycin treatment, intermittent pyrexia up to 39°C was still noted, and laboratory tests revealed a hemoglobin level of 127 g/L and WBC count of  $17.74 \times 10^9/L$ . The C-reactive protein level was 132.68 mg/L; ESR, 85 mm/h; albumin, 23.5 g/L; alanine transaminase, 108 U/L; and aspartate transaminase, 110 U/L. Lower back pain and diffuse lower extremity pain progressed, but no lower extremity weakness was noted until 12 days post-admission, when the patient

**Fig. 1** (A) Sagittal and (B) axial T2-weighted magnetic resonance images show pyogenic discitis at L3–4 and abscesses (white arrows) disseminated in the anterior, right lateral, and epidural space of L3–4. (C) T2-weighted sagittal magnetic resonance image of the whole spine shows progressing epidural abscess predominating anteriorly continuous from L4 to T1. The white dotted line indicates T1–2 and the blue dotted line indicates L3–4, at which levels axial images are shown in (D) and (E), respectively. T2-weighted axial image at (D) T1–2 and (E) L3–4 levels show the huge epidural abscess (white arrows) is located in the ventral part of the spinal canal, critically compressing the spinal cord toward the posterior side



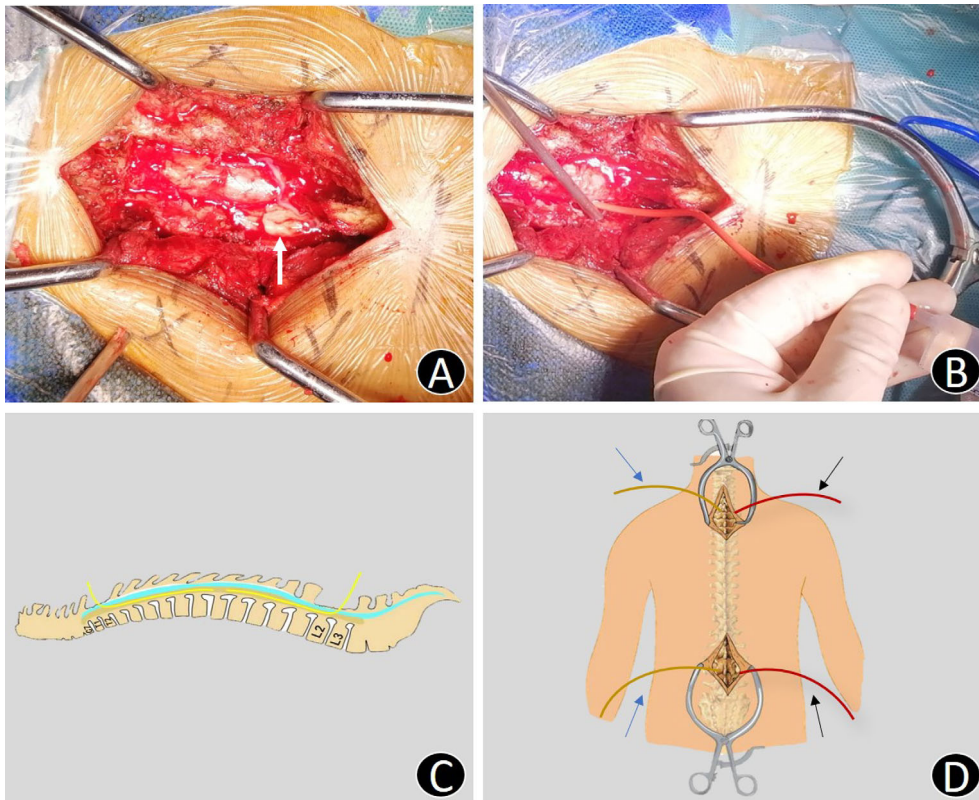
complained of bilateral lower extremity paresthesia. The muscle strength of the lower extremities had decreased to grade 3, without evidence of pyramidal tract involvement or meningitis. Cervical, thoracic, and lumbar spine MRI (Figure 1(C)) showed progression of the SEA, predominating anteriorly and continuous from L4 to T1. Laboratory tests revealed a hemoglobin level of 84 g/L; WBC count,  $9.75 \times 10^9/L$ ; C-reactive protein level, 103.5 mg/L; ESR, 97 mm/h; albumin, 22.3 g/L; alanine transaminase, 102 U/L; and aspartate transaminase, 68 U/L. Due to his neurological deterioration, emergency surgical decompression was suggested to the patient and his family, which was refused. The vancomycin trough concentration was 5.9  $\mu\text{g/mL}$  (recommended trough concentration, 15–20  $\mu\text{g/mL}$ ). Consequently, the dosage of intravenous vancomycin was modified to 1000 mg every 8 h.

Thirteen days after admission, the muscle strength of both lower extremities decreased to grade 0 with no sensation below the nipple line and continued intermittent pyrexia up to 38.5 °C. However, the patient and his family persistently refused surgical decompression. Based on the trough concentrations, the administration of intravenous vancomycin was increased to 1000 mg every 6 h. Seventeen days after admission, grip strength of the left hand decreased to grade 3. Repeat MRI of the cervical, thoracic, and lumbar spine showed extensive SEA continuous from L4 to C7. Postcontrast T1-weighted MRI showed enhancement of the margins of the abscess, with no enhanced contents (Figure 3(A),(B)). Laboratory tests revealed

a hemoglobin level of 82 g/L; WBC count,  $8.85 \times 10^9/L$ ; C-reactive protein, 22.79 mg/L; ESR, 44 mm/h; albumin, 27.5 g/L; alanine transaminase, 103 U/L; and aspartate transaminase, 57 U/L. After extensive discussion with the patient and his family, they finally consented to surgery.

### Operation

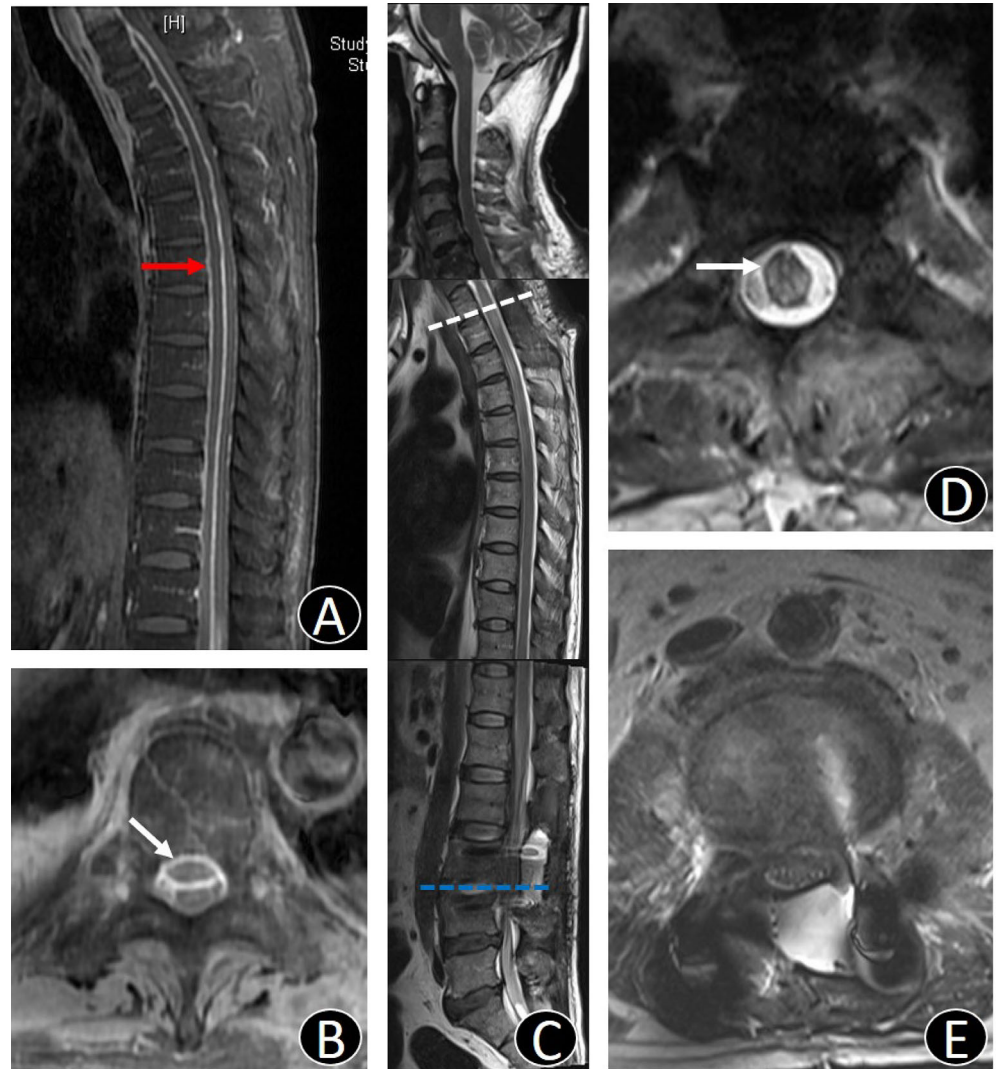
Two separate midline longitudinal incisions were made over the cervicothoracic and mid-lumbar areas. Laminectomies were performed at T1–2 in the thoracic spine and L2–3 in the lumbar spine. Liquid pus was suctioned from each laminectomy (Figure 2(A)). Subsequently, a ventricular catheter was inserted caudally into the epidural space from the thoracic level down toward the lumbar area. Another ventricular catheter was inserted cranially into the epidural space from the lumbar level up toward the thoracic area. The two catheters met at the level of T8–9. Normal saline was slowly injected through the catheter to irrigate the epidural abscess (Figure 2(B)). The intraspinal length of the catheters was approximately the length of the spine from T2 to L2 (Figure 2(C)). During irrigation, both ends were assessed for egress of irrigation fluid and pus. The dura was gently pulled at the opposite end to assist the egress of fluid. Approximately 3 L of normal saline was used for irrigation until clear fluid was obtained. Subsequently, debridement of the L3–4 disc space was performed, and pedicle screws were used to facilitate fusion. Samples were obtained and sent for Gram staining and culture. Two irrigation tubes were inserted



**Fig. 2** Anatomical exposure after laminectomies is performed. (A) Yellowish-white pus is noted (white arrow). (B) Normal saline is slowly injected through the catheter to irrigate the epidural abscess. (C) The two catheters meet at approximately the level of T8–9. The intraspinal length of the catheters is approximately the length of the spine from T2 to L2. (D) Blue arrows indicate flush tubes, and black arrows indicate drainage tubes



**Fig. 3** Preoperative magnetic resonance images: postcontrast T1-weighted magnetic resonance image (MRI) of the spine showing extensive epidural abscess (A, red arrow) continuous from L4 to C7, with enhancement of the margins of the abscess (B, white arrow). Postoperative magnetic resonance images: (C) T2-weighted sagittal MRI of the whole spine shows the complete disappearance of the epidural abscess. The white dotted line indicates T1–2 and the blue dotted line indicates L3–4, at which levels axial images are shown in (D) and (E), respectively. (D) An abnormal high signal (white arrow) is detected on T2-weighted axial MRI of T1–2. (E) Spinal epidural abscess and pyogenic discitis disappear on T2-weighted axial MRI of L3–4



into the epidural space, and two drainage tubes were left in the operative area for 7 days postoperatively (Figure 2(D)).

#### Postoperative Care

The microbiological examination of samples showed methicillin-resistant *S. aureus* with sensitivity to vancomycin. Postoperatively, the patient received neurotrophic drugs and actively strengthened his upper extremity through ball-holding training. His paraplegia was managed through massage and passive exercises. Four weeks after the operation, the strength of the upper extremity returned to normal with no sensory deficits, and MRI confirmed the resolution of the SEA (Figure 3(C)). Intravenous vancomycin administration was continued for 6 weeks. The postoperative ESR decreased to 5 mm/h. Unfortunately, his lower limb weakness did not significantly improve, despite treatment in a rehabilitation hospital for 2 months, and he became wheelchair bound. At the 1-year follow-up, muscle strength in the lower extremities was unchanged while sensory function had improved.

#### Discussion

The characteristic clinical features of SEA are back pain, fever, and neurologic deficit<sup>8,9</sup>. However, they only present in a minority of patients. In this case, lower back pain was the initial presentation. Given the ubiquity of back pain, our patient was initially misdiagnosed. Presently, MRI allows diagnosis in >91% of cases. Typically, SEA shows low or intermediate intensity on T1-weighted images and high or intermediate intensity on T2-weighted images. Gadolinium-enhanced MRI can aid in defining the consistency (liquid pus or granulated tissue) of the abscess. Liquid pus is associated with an area of low signal intensity on T1-weighted images and enhancement of the margins of the abscess after contrast injection. In our case, an enhanced margin of the abscess was noted in the postcontrast T1-weighted images of the spine, indicating liquid pus, described as the “double spinal cord sign” (Figure 3(B)).

The timing of surgical decompression is of great importance for extensive SEA. Although controversial, the first-line treatment for extensive SEA or SEA presenting with complete

**TABLE 1 SEA treated by surgery reported in the literature**

Reference	Infective etiology	Location	Sex/age	Symptom	Operation	Outcome
Elsamaloty, <i>et al.</i> <sup>13</sup>	Methicillin-susceptible <i>Staphylococcus aureus</i>	C1-L1	M/53y	Fever, neck pain, respiratory failure, and paralysis of all four limbs	Multilevel laminectomies at C5–C6, T2–T3, and L1–L2	6 months postoperative: strength were normal in the upper limbs, while weakness was in the lower limbs
Fujii, <i>et al.</i> <sup>15</sup>	Group G streptococcus	T6-L3	M/81y	Fever, back pain, and progressive muscle weakness in bilateral legs for 7 days	Fluoroscopy-guided percutaneous epidural drainage	Patient walked unassisted 20 days postoperatively
Urrutia J, Rojas C. <sup>16</sup>	Gram-positive coccus	From C2 to the sacrum	M/36y	Malaise, fever, and severe lumbar and neck pain for 15 days	Multilevel laminectomies at C3-C5, T5-T6, L4-L5	Motor and sensory examination of all four limbs were normal after surgery
Ansari, <i>et al.</i> <sup>17</sup>	<i>Staphylococcus aureus</i>	T5-T12 and C1/2	F/50y	Fever, headache and neck stiffness	Multilevel laminectomies at C1, C3/4, C7/T1, T4/5, and T7/8	Patient recovered to normal 30 days postoperatively

paralysis of less than 48 h should be urgent surgical decompression to prevent progression that could permanently compromise neurological function<sup>10</sup>. Because the rate of progression of neurological impairment is unpredictable, as illustrated by some patients who develop paralysis within hours of the onset of initial neurological symptoms, caution should be advocated when implementing the “watchful waiting” approach, a lesson exemplified by this case<sup>1,5,11,12</sup>. Although this patient’s neurological deterioration had progressed for >48 h, emergency surgical intervention was necessary in our case to preserve life.

Most literature reports advocate active surgical decompression and abscess drainage for patients with symptoms of nerve damage, and different surgical methods are used according to the location of the abscess and the patient’s condition (Table 1). Elsamaloty *et al.*<sup>13</sup> reported that an SEA extending from C1 to L1 in a patient with previously undiagnosed diabetes was successfully treated by multilevel laminectomies at C5–6, T2–3, and L1–2. Abd-El-Barr *et al.*<sup>14</sup> reported two patients with extensive SEA who were treated with “apical laminectomies” and irrigation of the epidural space with two pediatric tubes. A similar method was used in our case, adapted to avoid laminectomies in the thoracic apical area, thus reducing the risk of postoperative spinal deformity. Furthermore, Fujii *et al.*<sup>15</sup> reported that percutaneous epidural drainage, which is a minimally invasive treatment for SEA, extended from T6 to L3 under the guidance of fluoroscopy. However, percutaneous epidural drainage is only suitable for fluid abscesses located behind the dura mater, or in patients in a poor clinical condition, to reduce surgical trauma and shorten the operative time<sup>16,17</sup>.

With only two separate incisions, we effectively prevented disease progression in our patient in a poor

clinical condition. To ensure adequate irrigation and drainage, preoperative spine MRI should be used to visualize the distribution of the abscess and to identify segments for skip decompression. According to the preoperative MRI, the apex of the thoracic spine was at the level of T8–9, and the spinal curvature from T2 to T8–9 and from L2 to T8–9 was almost straight. Preoperative MRI also indicated liquid pus in the SEA. Thus, the irrigation catheter could be inserted to the level of T8–9, and the pus could be washed out. Considering that the resistance of a single irrigation tube across the whole spine is relatively large, the irrigation tube must be rigid, but because of this, it can easily damage the spinal cord. Thus, we used two relatively soft ventricular tubes to meet at the apex of the thoracic vertebra. The ventricular catheter, being relatively flexible, is suitable to prevent mechanical injury to the spinal cord.

In conclusion, this report presents a rare case of extensive SEA treated with antibiotics and late surgical decompression and drainage. Considering that the progress of neurological impairment and infection is unpredictable, minimally invasive selective laminectomy and irrigation can be considered early for patients in a poor general condition.

#### Acknowledgment

This study was supported by Taishan Scholar Project of Shandong Province, China (no. ts20190985).

#### Conflict of Interest

There are no conflicts of interest for this case report.

#### Author Contributions

Tongshuai Xu: conceptualization, investigation, writing of original draft, resources. Yukun Du: conceptualization,

writing—review and editing. Jianwei Guo: methodology. Jianyi Li: data curation. Cheng Shao: investigation. Changfang Shi: visualization. Xianfeng Ren: conceptualization, writing—review and editing, supervision. Yongming Xi: funding acquisition, supervision.

### Authorship Declaration

All authors listed in this manuscript meet the authorship criteria according to the latest guidelines of the International Committee of Medical Journal Editors, and all authors are in agreement with the manuscript.

### References

1. Patel A, Alton T, Bransford R, et al. Spinal epidural abscesses: risk factors, medical versus surgical management, a retrospective review of 128 cases. *Spine J*. 2014;14:326–30.
2. Reihnsaus E, Waldbaur H, Seeling W. Spinal epidural abscess: a meta-analysis of 915 patients. *Neurosurg Rev*. 2000;23:175–204.
3. Vakili M, Crum-Cianflone N. Spinal epidural abscess: a series of 101 cases. *Am J Med*. 2017;130:1458–63.
4. Erşahin Y. Spinal epidural abscess: a meta-analysis of 915 patients. *Neurosurg Rev*. 2001;24:156.
5. Proietti L, Ricciardi L, Noia G, et al. Extensive spinal epidural abscesses resolved with minimally invasive surgery: two case reports and review of the recent literature. *Acta Neurochir Suppl*. 2019;125:345–53.
6. Mann S, Schütze M, Sola S, et al. Nonspecific pyogenic spondylodiscitis: clinical manifestations, surgical treatment, and outcome in 24 patients. *Neurosurg Focus*. 2004;17:E3.
7. Koo D, Townson A, Dvorak M, et al. Spinal epidural abscess: a 5-year case-controlled review of neurologic outcomes after rehabilitation. *Arch Phys Med Rehabil*. 2009;90:512–6.
8. Akalan N, Ozgen T. Infection as a cause of spinal cord compression: a review of 36 spinal epidural abscess cases. *Acta Neurochir*. 2000;142:17–23.
9. Davis D, Wold R, Patel R, et al. The clinical presentation and impact of diagnostic delays on emergency department patients with spinal epidural abscess. *J Emerg Med*. 2004;26:285–91.
10. Tuchman A, Pham M, Hsieh P. The indications and timing for operative management of spinal epidural abscess: literature review and treatment algorithm. *Neurosurg Focus*. 2014;37:E8.
11. Lu C-H, Chang W-N, Lui C-C, Lee PY, Chang HW. Adult spinal epidural abscess: clinical features and prognostic factors. *Clin Neurol Neurosurg*. 2002;104:306–10.
12. Barner K, Yuquimpo K, McMillan D, et al. A unique presentation of a large, seven-segment, spinal epidural abscess in a patient with a pleural empyema. *Cureus*. 2019;11:e4084.
13. Elsamaloty H, Elzawawi M, Abduljabar A. A rare case of extensive spinal epidural abscess in a diabetic patient. *Spine*. 1976;2010(35):E53–6.
14. Abd-El-Barr M, Bi W, Bahluyen B, et al. Extensive spinal epidural abscess treated with “apical laminectomies” and irrigation of the epidural space: report of 2 cases. *J Neurosurg*. 2015;22:318–23.
15. Fujii M, Shirakawa T, Shime N, Kawabata Y. Successful treatment of extensive spinal epidural abscess with fluoroscopy-guided percutaneous drainage: a case report. *JA Clin Rep*. 2020;6:4.
16. Urrutia J, Rojas C. Extensive epidural abscess with surgical treatment and long term follow up. *Spine J*. 2007;7:708–11.
17. Ansari A, Davies D, Lohn J, et al. Extensive spinal epidural abscess associated with an unremarkable recovery. *Anaesth Intensive Care*. 2004;32:825–9.