

CASE REPORT

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Conservative management of spontaneous thoracolumbar epidural hematoma: a case report

Menglong Jia^{1,2†}, Shan Gao^{1†}, Longtan Yu¹ and Guangbin Ma^{1*}

Abstract

Background Spontaneous spinal epidural hematoma (SSEH) is an exceedingly rare clinical entity often associated with neurological deficits, for which surgical intervention remains the mainstay of treatment. We report a case successfully managed through complete conservative therapy.

Case presentation A 59-year-old Chinese male with a medical history of hypertension, chronic smoking, and alcohol use presented to our institution with acute-onset low back pain accompanied by bilateral lower limb weakness and hypoesthesia persisting for 30 min. Diagnostic imaging (CT and MRI) revealed a thoracolumbar epidural hematoma (T11-L1 distribution). Contrary to conventional surgical management paradigms, we implemented a comprehensive conservative management protocol. Remarkably, complete resolution of symptoms and spontaneous hematoma absorption were achieved within 20 days.

Conclusion This case demonstrates that select SSEH cases may achieve favorable outcomes through conservative management without surgical decompression.

Keywords Spontaneous spinal epidural hematoma, Spontaneous absorption, Non-surgical treatment

Background

Spinal epidural hematoma (SEH), classically considered a well-documented complication of spinal procedures, primarily results from inadequate intraoperative hemostasis or compromised postoperative drainage [1]. In contrast, spontaneous spinal epidural hematoma (SSEH) represents a rare clinical entity with an estimated annual incidence of 0.1 per 100,000 individuals, characterized

by idiopathic onset and poorly understood pathophysiological mechanisms [2]. Typical manifestations include acute-onset cervicodorsal or lumbosacral pain accompanied by rapidly progressive neurological deficits secondary to spinal cord compression. This condition's non-specific presentation often leads to significant diagnostic challenges. The clinical manifestations can easily confound attending physicians, as similar symptoms may also present in other clinical entities, including but not limited to cerebrovascular accidents, myocardial infarction, and meningitis, potentially leading to irreversible neurological sequelae when therapeutic intervention is delayed. Current management paradigms prioritize surgical decompression as the gold-standard intervention for SSEH [3], with limited literature documenting successful non-surgical management. Notably, we present a

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case of spontaneous thoracolumbar epidural hematoma (T11-L1 distribution) that achieved complete clinical resolution through conservative measures. The patient demonstrated full neurological recovery (ASIA Impairment Scale improvement from Grade D to E) and radiological hematoma clearance within a 20-day observation period, as confirmed by serial MRI examinations. This case highlights the potential viability of watchful waiting protocols in select SSEH presentations, particularly when surgical contraindications exist or in resource-limited settings.

Case presentation

A 59-year-old man with a history of hypertension and a long-term habit of smoking and drinking presented with acute thoracolumbar pain accompanied by lower

limb weakness. These symptoms occurred when he was doing morning activities 30 min ago, and then he was transported to the emergency department by ambulance. The pain intensity reached a significant score of 8 on the Visual Analogue Scale (VAS) (with a full score of 10). CT imaging reveals a band-like soft tissue density posterior to the spinal cord at the T11-L1 level (Fig. 1), with an average reference CT value of approximately 69 Hounsfield Units (HU). MRI demonstrates that at the T11-L1 level, a band-like lesion is observed within the posterior epidural space of the spinal canal (Fig. 2). The lesion demonstrates high signal intensity on T2-weighted imaging (T2WI) and isointense signal on T1-weighted imaging (T1WI), with mildly heterogeneous internal signal. It measures approximately 5.7 cm craniocaudally. The

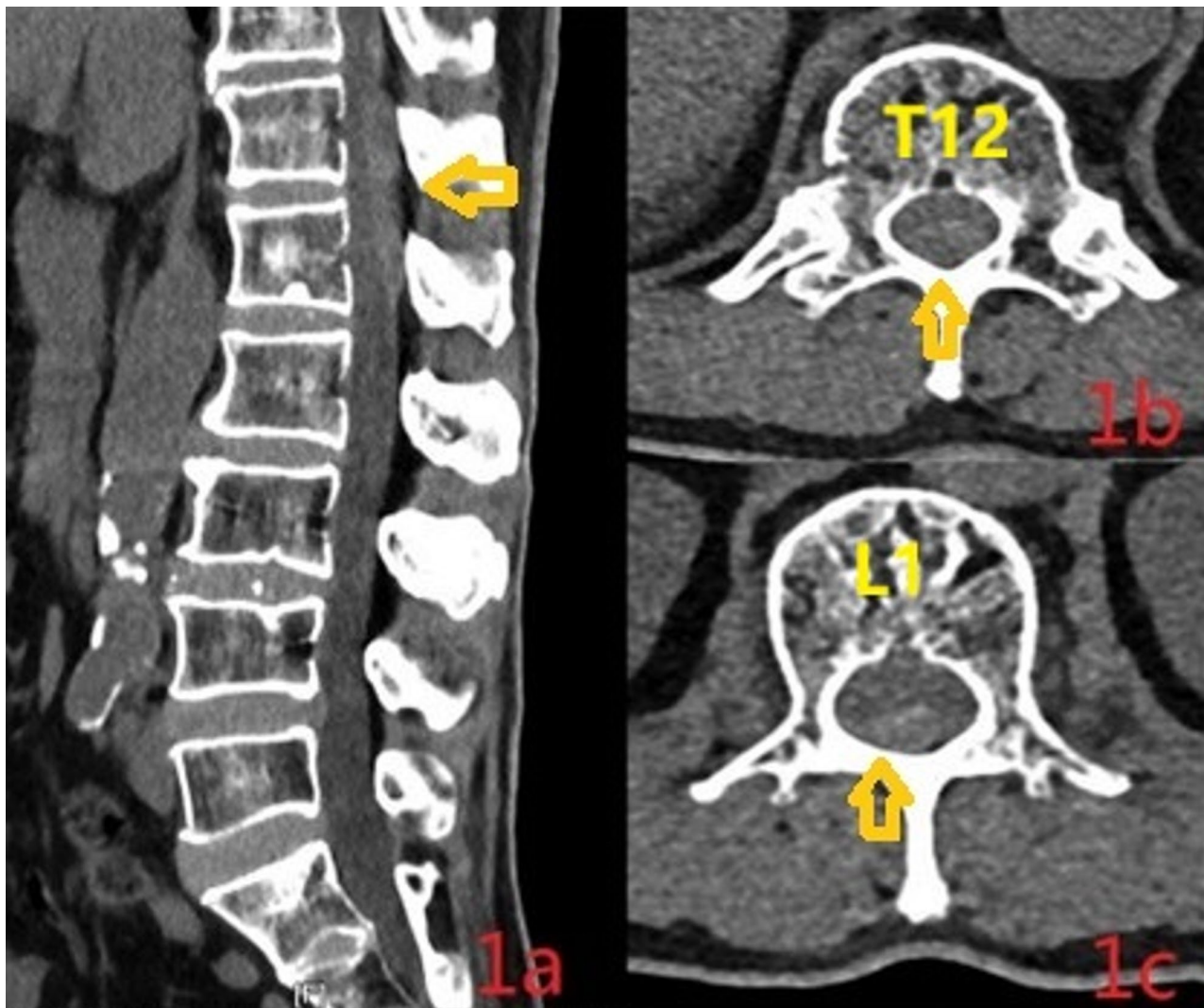


Fig. 1 Lumbar computer tomography imaging without contrast (on the day of symptom onset), arrow: epidural hematoma at the T11-L1 levels occupies the spinal canal and compresses the spinal cord. **1a:** A band-like soft tissue density is observed posterior to the spinal canal at the T11-L1 level, the average reference CT value was approximately 69 HU. **1b:** A soft tissue density lesion is identified dorsally within the spinal canal at the T12 level, resulting in a reduction of the effective spinal canal volume, and demonstrating a well-defined margin with the adjacent dural sac. **1c:** A soft tissue density lesion is identified dorsally within the spinal canal at the L1 level, its extent is observed to be smaller compared to that at the T12 level

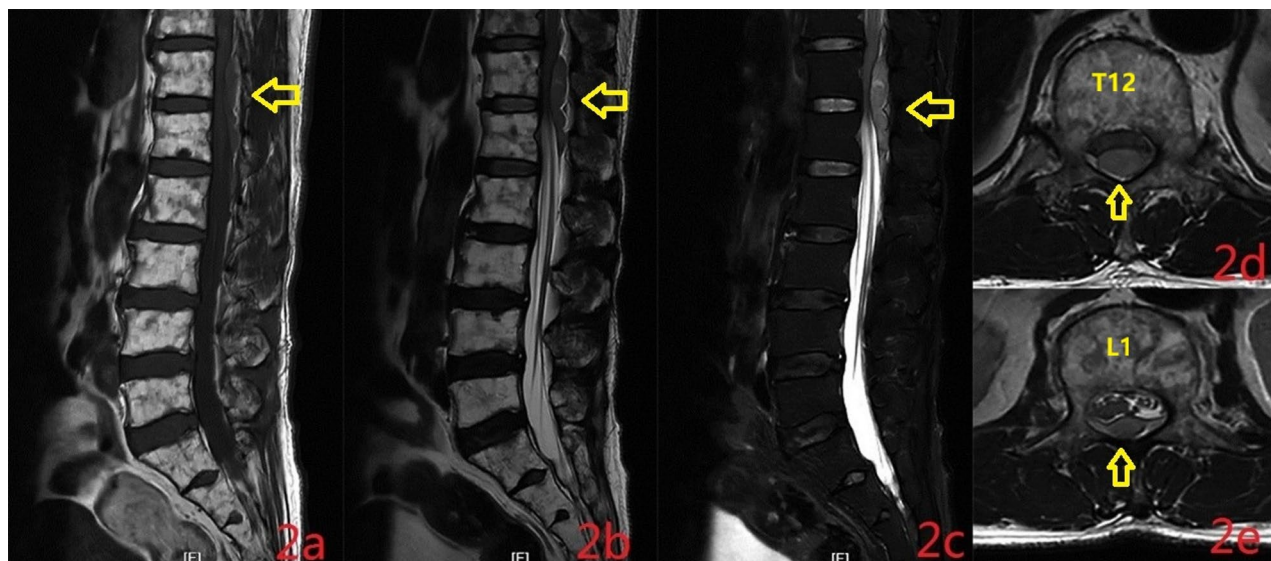


Fig. 2 Lumbar magnetic resonance imaging without contrast (on the day of symptom onset), arrow: epidural hematoma occupies the spinal canal and compresses the spinal cord. **2a:** A band-like isointense lesion on T1WI was observed within the posterior epidural space at the T11-L1 spinal level. **2b:** A band-like hyperintense lesion on T2WI was observed within the posterior epidural space at the T11-L1 spinal level, demonstrating heterogeneous internal signal characteristics. **2c:** A band-like hyperintense lesion on T2WI + Fat-Suppressed (T2WI + FS) was observed within the posterior epidural space at the T11-L1 spinal level, demonstrating heterogeneous internal signal characteristics. **2d:** On the axial T2WI, at the T12 level within the spinal canal, a space-occupying lesion is observed on the dorsal side. This lesion occupies approximately 60% of the spinal canal's cross-sectional area, resulting in significant compression of the spinal cord. **2e:** On the axial T2WI, at the L1 level within the spinal canal, a space-occupying lesion is observed on the dorsal side. This lesion occupies approximately 30% of the spinal canal's cross-sectional area, resulting in compression of the conus medullaris

adjacent dural sac is compressed and narrowed, while the spinal cord and conus medullaris appear compressed and thinned. Notably, the signal intensity of the spinal cord and conus medullaris remains within normal limits. Contrast-enhanced MRI demonstrates no significant enhancement of the lesion (Fig. 3). Laboratory investigations revealed mild erythrocytopenia (red blood cell count: $4.19 \times 10^{12}/L$; reference range: $4.3\text{--}5.8 \times 10^{12}/L$). Notably, the prothrombin time was 10.63 s (slightly below the lower limit of the reference range: 10.7–14.0 s), while other coagulation parameters remained within normal limits. Despite presenting with significant neurological impairment (right/left lower limb muscle strength grades 4/3, the grading of the muscles is performed according to the Medical Research Council (MRC) grading for muscle strength [4]), conservative management was initiated due to stable neurological status, involving strict bed rest, methylprednisolone (80 mg IV), mannitol, sodium aescinate, and adjunctive traditional Chinese medicine hot compresses. Meanwhile, Guide the patient in performing gentle flexion and extension exercises for the hip, knee, and ankle joints, ensuring smooth and controlled movements throughout each motion. The patient subsequently demonstrated remarkable recovery: within 24 h, pain significantly subsided (VAS score: 8→4). By day 6, pain resolved completely with restored ambulation. Complete neurological recovery was achieved by day 20, evidenced by an upgrade from ASIA Impairment Scale Grade D to

E. Follow-up MRI at 20 days confirmed complete resolution of the epidural hematoma (Fig. 4). Neurological examinations were performed daily until discharge. Key recovery milestones are detailed in Table 1.

This study was reviewed and approved by the Ethics Committee of Weifang Hospital of Traditional Chinese Medicine and has been conducted with the informed consent of participating patient. (Ethics Approval Code:2025YX562)

Discussion and conclusions

Spontaneous spinal epidural hematoma (SSEH) has been observed across all age groups, with reported cases ranging from infants as young as 6 months to elderly patients over 80 years of age. But, SSEH predominantly affects middle-aged and elderly populations with a slight male predominance. The spinal canal may be affected at any level; however, the hematoma demonstrates a bimodal distribution pattern, with predilection sites at the lower cervical spine and thoracolumbar junction. Current evidence suggests multifactorial etiologies including hypertension, coagulation disorders, anticoagulant use, and arteriovenous malformations, though the precise pathomechanisms remain elusive [5–9]. The presented case involved a hypertensive patient with irregular medication adherence (admission BP: 190/110 mmHg), yet Groen et al. [10] demonstrated no statistical correlation between hypertension prevalence in SSEH cohorts



Fig. 3 Thoracolumbar magnetic resonance imaging with contrast (on day 2 after symptom onset), arrow: epidural hematoma occupies the spinal canal and compresses the spinal cord. **3a**: Non-contrast T1-weighted imaging (T1WI) of the thoracolumbar spine. **3b**: Post-contrast imaging following Gd-DTPA administration demonstrated no significant enhancement within the lesion. **3c**: The axial post-contrast T1-weighted image demonstrates no enhancement in the dorsally located lesion within the spinal canal at the T12 level. **3d**: The axial post-contrast T1-weighted image demonstrates no enhancement in the dorsally located lesion within the spinal canal at the L1 level

versus controls, suggesting acute pain-induced blood pressure elevation might better explain such observations. Hemorrhagic origins remain debated: arterial rupture typically manifests rapid neurological deterioration, while venous bleeding often stabilizes early [11]. The most widely accepted hypothesis concerning the origin of bleeding points to the venous system. Unlike other vascular structures, the spinal epidural veins lack protection from fluctuations in abdominal or thoracic pressure. Consequently, any increase in these pressures results in a corresponding rise in intravascular pressure, which can ultimately lead to vascular rupture [12]. Papadimitriou et al. also support this hypothesis. They documented a case of SSEP during pregnancy, attributing the etiology to elevated intra-abdominal/intra-thoracic pressure and increased central venous pressure (CVP). This pressure readily propagates to the valveless epidural venous system, thereby inducing spontaneous hemorrhage [13]. Bakker et al. [14] anatomically favors venous sources, particularly emphasizing posterior vertebral plexus prominence in lower thoracic regions of elderly patients—consistent with our case's non-progressive neurological deficits indicative of venous origin.

SSEH typically presents with acute axial pain accompanied by sensorimotor deficits in clinical settings,

necessitating comprehensive differential diagnosis from conditions like intracranial lesions and aortic dissection. Although clinical manifestations may provide preliminary diagnostic clues, imaging modalities such as CT or MRI remain indispensable for definitive confirmation, underscoring their pivotal role as the gold standard in clinical decision-making [15]. MRI remains diagnostic gold standard, demonstrating longitudinal epidural masses; emergency CT may reveal hyperdense lesions from hemosiderin deposition, as observed in this patient (T11-L1 hyperdensity) accompanied by mild erythrocytopenia ($4.19 \times 10^{12}/L$), collectively supporting acute hematoma formation. Contrast-enhanced MRI further confirmed non-enhancing lesions, excluding vascular malformations.

Therapeutic consensus prioritizes surgical decompression within 48 h via traditional laminectomy or emerging endoscopic techniques [16, 17]. However, several clinical reports have documented the spontaneous resolution of SSEH without surgical intervention. Kim et al. reported favorable recovery in 4 of 5 patients with SSEH managed conservatively. Conservative management may be a viable option for patients who present with a neurological status classified as ASIA Scale E, or for those in whom an early functional recovery has been observed, falling

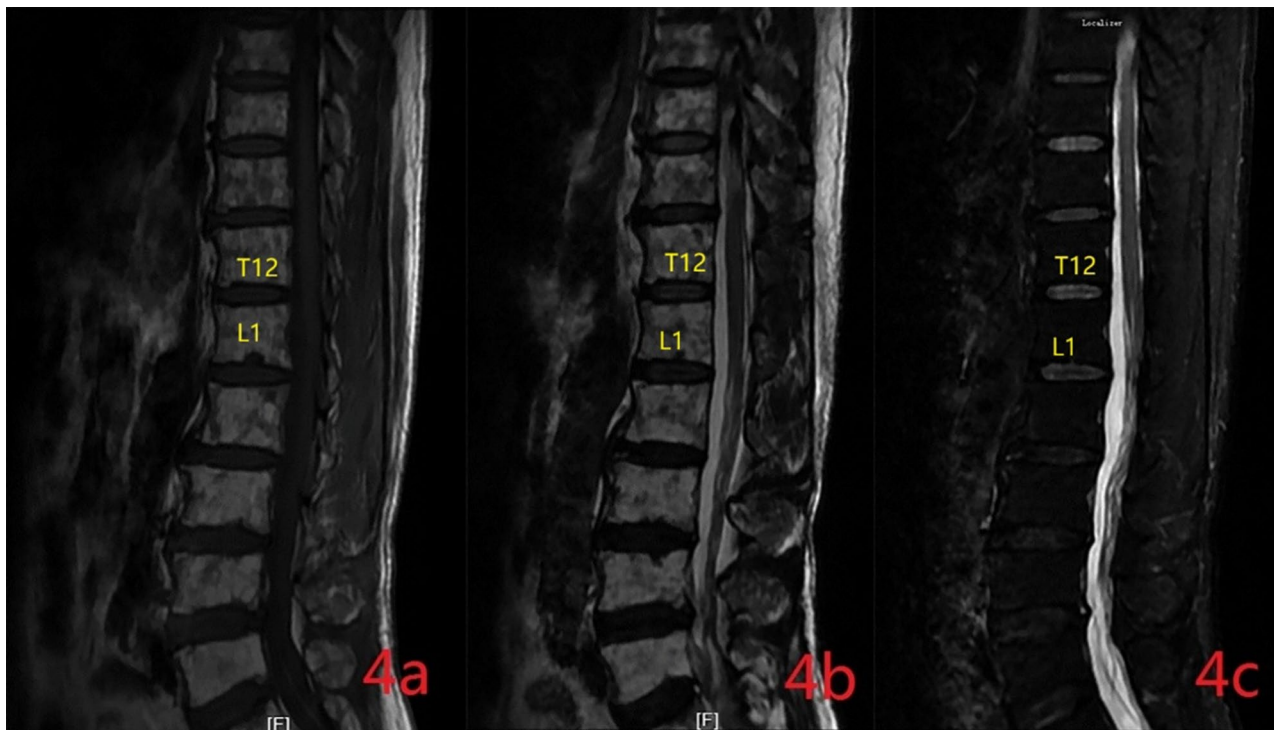


Fig. 4 Thoracolumbar magnetic resonance imaging without contrast (on day 20 after symptom onset). **4a:** T1WI of the thoracolumbar spine demonstrates that the previously observed epidural hematoma has now completely resolved. **4b:** T2WI of the thoracolumbar spine demonstrates that the previously observed epidural hematoma has now completely resolved. **4c:** T2WI + FS of the thoracolumbar spine demonstrates that the previously observed epidural hematoma has now completely resolved

Table 1 The patient's recovery status across various time points

Evaluation\ Time point	VAS	Muscle strength(R/L)	ASIA grade
At onset	8	4/3	D
24 h post-onset	4	4/3	D
6 days post-onset	0	4/4	D
20 days post-onset	0	5/5	E

under ASIA Scale C or D [18]. So, conservative management proves viable for neurologically stable cases (ASIA Scale D in this report). Critical conservative measures include: (1) Continuous neurological monitoring with surgical readiness; (2) Risk factor mitigation (BP stabilization, anticoagulant cessation); (3) Pharmacotherapy combining analgesics, osmotic agents (mannitol), corticosteroids, and neurotrophins, despite ongoing debates regarding high-dose steroid protocols; (4) Adjunctive hyperbaric oxygen and rehabilitation therapies [18]. Our patient achieved complete recovery through strict bed rest, methylprednisolone (80 mg IV), and multimodal therapy, demonstrating conservative potential in select cases. The role of high-dose methylprednisolone in the treatment of acute spinal cord injury continues to spark considerable debate within the medical community. Despite the ongoing controversy, a substantial number of scholars remain steadfast in advocating its use under

specific clinical circumstances. However, recent research has shed light on an intriguing alternative: low-dose methylprednisolone regimens have demonstrated significant therapeutic potential, offering a promising option that may mitigate some of the concerns associated with high-dose protocols [18]. In addition to corticosteroids, other pharmacological agents have gained prominence in the management of this complex condition. For instance, anti-edema agents such as mannitol have emerged as critical components in the recommended pharmacopeia, providing clinicians with expanded strategies to address the multifaceted challenges posed by acute spinal cord injuries. Collectively, these developments underscore the importance of tailoring treatment approaches to individual patient needs while continuing to refine our understanding of optimal therapeutic interventions [15, 19].

Diagnostic urgency mandates immediate MRI for suspected SSEH. Current evidence supports conservative trials for ASIA C-D cases, while ASIA A-B warrants prompt surgery. However, significant questions remain regarding the time frame for hematoma absorption in patients undergoing conservative treatment. Several studies have suggested that epidural hematomas may be absorbed within a time frame of 10 to 31 days. Unexpectedly, there exists a documented case of cervical epidural hematoma wherein the hematoma resolved entirely

within just 2 days post-onset [5]. Nitta et al. conducted a more specific analysis focusing on the outcomes of conservative treatment in 22 patients with SSEH. Their findings revealed that regression of the hematomas could be detected via MRI re-examination as early as 5 days post-onset. Based on this observation, they proposed a recommendation: if there is no deterioration in neurological function, an MRI re-examination can be performed 5 days after the onset of symptoms. Furthermore, once a reduction in hematoma volume is confirmed, patients may be permitted to resume physical activity [19]. In our case, a follow-up MRI was conducted 20 days after the onset of the disease. Notably, the patient's symptoms had already demonstrated significant improvement as early as 6 days into the course of the illness. This observation suggests the possibility that the hematoma may have started to regress by that time. Nevertheless, hematoma resorption mechanisms and optimal conservative duration require further investigation to refine management protocols. The degree of preoperative neurological deficit stands as the most critical factor influencing prognosis. Additionally, the time interval between disease onset and surgical decompression significantly affects the outcomes for patients with severe neurological impairments. Whether the location of the hematoma can influence the prognosis remains a topic of debate and controversy [7, 13, 14]. When clinicians encounter patients manifesting neurological deficits without a history of trauma in the emergency department, spontaneous epidural hematoma must be considered as a potential diagnosis regardless of the presence of associated risk factors [20].

In conclusion, this case highlights the necessity of individualized treatment plans, balancing neurological preservation against surgical risks. Select patients with SSEH can achieve full recovery through conservative management. Prompt development of personalized strategies based on comprehensive clinical evaluation is essential. Our study proposes a non-surgical approach and advocates further high-quality trials to validate its efficacy and reliability, strengthening evidence-based practice.

Author contributions

Menglong Jia and Shan Gao Co-first author and contributed equally to this work. All authors read and approved the final manuscript.

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Data availability

Not applicable.

Declarations

Ethics approval and consent to participate

The case report has been approved for publication by the Research Ethics Committee of Weifang Hospital of Traditional Chinese Medicine.

Consent for publication

The patient has signed the consent.

Competing interests

The authors declare no competing interests.

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