

Painless Spontaneous Spinal Epidural Hematoma Mimicking Acute Ischemic Stroke: A Case Report

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AIM: Spontaneous spinal epidural hematoma is a rare neurological emergency. Its painless variant is particularly uncommon and can easily be misdiagnosed as acute ischemic stroke, especially in patients receiving anticoagulant therapy. This study aims to enrich the clinical recognition of painless spontaneous spinal epidural hematoma and emphasize its differential diagnosis from acute stroke in high-risk medicated patients.

CASE PRESENTATION: We report a rare case of painless spontaneous spinal epidural hematoma mimicking acute ischemic stroke in a woman in her early 60s receiving anticoagulation and antiplatelet therapies. She initially presented with neck tightness and bilateral limb numbness without pain, followed by transient right hemiplegia and rapid neurological deterioration. Magnetic resonance imaging (MRI) revealed a C4–T2 cervical epidural hematoma causing severe spinal cord compression.

RESULTS: The patient underwent emergent C4–C6 laminectomy with partial C7 laminectomy and hematoma evacuation. Despite partial strength recovery, extensive and prolonged cord compression may have contributed to the poor prognosis.

CONCLUSIONS: Painless spontaneous spinal epidural hematoma is an easily overlooked stroke-mimicking emergency. For patients receiving anticoagulant or antiplatelet therapy who present with acute bilateral limb weakness or stroke-mimicking neurological deficits without typical spinal pain, spontaneous spinal epidural hematoma should be included in the early differential diagnosis to avoid delayed treatment and unfavorable prognosis.

Keywords: spontaneous spinal epidural hematoma; lower extremity weakness; magnetic resonance imaging; painless; ischemic stroke; case report

Introduction

Spontaneous spinal epidural hematoma (SSEH) is a rare neurosurgical emergency associated with high rates of long-term disability. Its incidence is approximately 0.1 per 100,000 population, with a male-to-female ratio of approximately 1.4:1 [1,2]. It classically presents with acute severe neck/back pain, followed by sensory disturbances and motor deficits, such as paraparesis or quadriparesis [1,2]. However, painless SSEH is a rare clinical subtype without obvious neck or back pain, which easily delays early diagnosis [3,4]. Moreover, SSEH with hemiplegia is another extremely rare presentation, commonly misdiagnosed

as stroke [5,6,7]. Isolated painless presentation and isolated hemiparetic stroke-mimicking presentation are rare but have been previously reported; however, the combination of both is extremely uncommon [4,5]. Notably, additional antiplatelet administration in patients on warfarin may exacerbate hematoma expansion and neurological deterioration [7,8]. We herein report a rare case with two uncommon features, namely bilateral dorsal hematoma extension and possible aggravation of hemorrhage associated with subsequent antiplatelet therapy during ongoing anticoagulation. This case report summarizes the clinical course to highlight the atypical manifestations of SSEH and aims to enhance clinicians' awareness of this disease.

Case Presentation

This study complies with the Case Report Guidelines [9] to ensure the accuracy and completeness of the report (**Supplementary Material**). Written informed consent was obtained from the patient's family for the publication of the data in this case report. The clinical course of the patient is summarized in Fig. 1.

A woman in her early 60s presented to the Emergency Department (ED) of The Fourth Affiliated Hospital of School of Medicine, and International School of Medicine, Interna-

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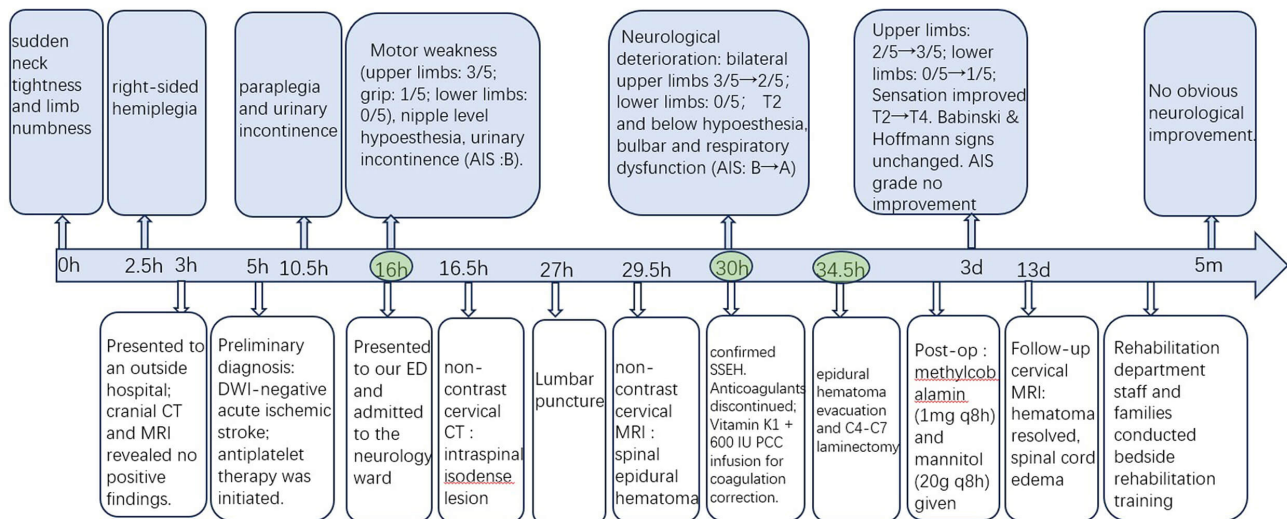


Fig. 1. Clinical course of the patient. CT, computed tomography; MRI, magnetic resonance imaging; DWI, diffusion-weighted imaging; ED, Emergency Department; SSEH, spontaneous spinal epidural hematoma; AIS, American Spinal Injury Association Impairment Scale; PCC, prothrombin complex concentrate.

tional Institutes of Medicine, Zhejiang University at 23:00, with a complaint of sudden neck tightness, limb numbness, and progressive neurological deficits 16 hours after an insect bite. She developed right-sided hemiplegia 2.5 hours after symptom onset. Her past medical history included cerebral infarction with no residual neurological deficits and atrial fibrillation, for which she had been on oral warfarin therapy for more than 4 years, with the INR previously maintained within the therapeutic range and no history of bleeding or thrombosis. She denied a history of recent trauma, hereditary bleeding disorders, or other accompanying symptoms. She was initially evaluated at Hengdian Wenrong Hospital, where brain computed tomography (CT) and brain magnetic resonance imaging (MRI) with diffusion-weighted imaging (DWI) were conducted; however, no significant abnormalities were identified. Considering the patient's presentation with right hemiplegia and National Institutes of Health Stroke Scale (NIHSS) score of 11 [10], acute ischemic stroke was highly suspected, for which she received antiplatelet therapy. However, the patient developed progressive bilateral limb weakness, lower extremity motor dysfunction, and urinary incontinence 10.5 hours after symptom onset.

Physical Examination on Admission

Her vital signs were within normal limits. Neurological examination revealed muscle strength of 3/5 in both upper limbs, 1/5 for bilateral grip strength, and 0/5 in both lower limbs. Tendon reflexes were normal in the upper extremities; present in the left lower limb but absent in the right. The left Babinski sign was equivocally positive, and bilateral Hoffmann signs were negative. Sensation above the nipple level was intact. Her American Spinal Injury As-

sociation Impairment Scale (AIS) grade was B [11]. The remainder of the physical examination was unremarkable.

Auxiliary Examinations

Coagulation testing showed markedly prolonged prothrombin time (PT, 30 s), activated partial thromboplastin time (APTT, 46 s), and INR of 3.01. Urinalysis revealed 3+ occult blood. Complete blood count revealed mild leukocytosis ($11.1 \times 10^9/L$) and neutrophilia ($9.8 \times 10^9/L$). Lumbar puncture was performed at the L3–L4 interspace 27 hours after symptom onset, with normal cerebrospinal fluid findings. Non-contrast cervical CT showed a faintly visible isodense lesion within the spinal canal (Fig. 2). Non-contrast cervical spine MRI performed at 29.5 hours after symptom onset showed a spinal epidural hematoma within the cervical spinal canal from C4 to T2, extending from the right to the left side with severe spinal cord compression. The lesion was isointense on T1-weighted images and mixed signal intensity on T2-weighted images (Fig. 3). Twelve-lead electrocardiogram (ECG) confirmed atrial fibrillation.

Diagnosis and Treatment Process

On admission, considering the patient's history of a prior insect bite-associated infection, infectious acute myelitis and Guillain-Barré syndrome were considered in the differential diagnosis. Lumbar puncture and cervical MRI were scheduled for the following day to further confirm the diagnosis.

Following cervical MRI, an urgent neurosurgical consultation was requested for definitive surgical management of the SSEH. Preoperatively, anticoagulants and antiplatelet agents were immediately discontinued. Coagulation parameters were promptly corrected and normalized using vi-



Fig. 2. Non-contrast cervical spine computed tomography (CT) findings. A faint isodense shadow (dashed line) is visible on the right dorsal aspect of the spinal canal, causing spinal cord compression.

tamin K1 (for antagonism) and 600 IU prothrombin complex concentrate by intravenous infusion. The patient experienced a decline in neurological function, with bilateral upper extremity strength decreased to 2/5, bulbar and respiratory dysfunction, and impaired deep and superficial sensation below the T2 level. Her AIS grade deteriorated from B to A.

The patient underwent emergency epidural hematoma evacuation and C4–C6 laminectomy with partial C7 laminectomy for decompression 34.5 hours after symptom onset. Under general anesthesia, the patient was placed in the prone position with cervical spine flexed and the head se-

cured in a head frame. After marking, disinfection and draping, a posterior cervical incision was made. The paravertebral muscles were dissected to expose the C4–C7 laminae. The C5–C6 spinous processes and laminae were resected via drilling and rongeur removal, with division of the interspinous ligament. An old dark-red epidural hematoma was identified, causing marked spinal cord compression and extending cranially and caudally. No obvious abnormal vessels were observed. Additional resection of the C4 and partial C7 spinous processes and laminae allowed full exposure of the hematoma margins. The hematoma was completely evacuated under a microscope.

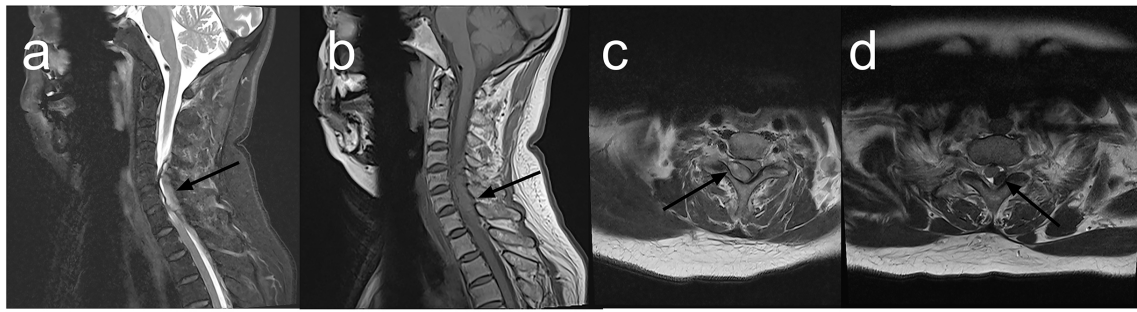


Fig. 3. Preoperative non-contrast cervical spine magnetic resonance imaging (MRI) findings (spontaneous spinal epidural hematoma). (a) Sagittal fat-saturated T2-weighted imaging (T2WI-FS) shows isointense and hyperintense lesions (arrow) at the cervicothoracic junction within the spinal canal. (b) Sagittal T1-weighted imaging (T1WI) reveals an isointense T1 signal at the same location (arrow). (c) Axial T2WI demonstrates a mixed-signal hematoma in the right spinal canal (arrow) with spinal cord compression and displacement. (d) Axial T2WI shows a hypointense hematoma in the left spinal canal (arrow), compressing the spinal cord.

After meticulous hemostasis, the operative field was covered with gelatin sponge, hemostatic gauze and fibrin glue. An epidural drain was placed, and layered closure was performed involving the muscle, fascia, subcutaneous tissue, and skin. The patient was transferred to the intensive care unit after wound dressing. No specimens were submitted for pathological examination.

Follow-up

Postoperatively, oral methylcobalamin (1 mg, q8h) was administered for neurotrophs, and intravenous mannitol (20 g, q8h) for spinal cord edema. Bilateral upper limb strength improved from 2/5 to 3/5, and lower limb strength improved from 0/5 to 1/5. Deep and superficial sensation improved from T2 to T4. The Babinski and Hoffmann signs remained unchanged. However, her AIS grade showed no improvement. Follow-up cervical MRI at 12 days postoperatively revealed linear T2 hyperintense signals within the cervical spinal cord spanning C5–T1, consistent with spinal cord edema and degeneration (Fig. 4).

The patient underwent bedside rehabilitation under the rehabilitation department; however, at the 5-month telephone follow-up, she reported no further improvement in bilateral muscle strength compared with the early postoperative status. The patient declined repeat cervical MRI due to financial concerns.

Discussion

Spontaneous spinal epidural hematoma (SSEH) is a rare but life-threatening neurological emergency that demands prompt diagnosis and intervention [1,2,4]. Classically, patients present with acute severe neck or back pain followed by motor paralysis or tetraparesis, which represents a pathological indicator for early diagnosis [5,12]. However, painless SSEH is an extremely rare variant, and the absence of cardinal symptoms often leads to significant diagnostic delay, misdiagnosis, and potentially irreversible neurological deficits [4].

Painless SSEH typically presents with acute stroke-like symptoms, hindering early differential diagnosis [8]. Most authorities agree that SSEH involving the cervical spine or cervicothoracic junction can mimic ischemic stroke, owing to spinal cord compression by the hematoma, rapid clinical deterioration, and early-onset ipsilateral neurological deficits [4,5,6]. However, most reported SSEH cases with stroke-mimicking manifestations present with neck or back pain [4,5,6]. The absence of pain in our patient increased the diagnostic difficulty, thereby leading to initial misdiagnosis and the administration of antiplatelet therapy before rapidly progressing to paraplegia at Hengdian Wenrong Hospital. This is consistent with the findings of Stetkarova et al. [8] that antithrombotic therapy after misdiagnosis may lead to severe consequences. On admission, the patient with a recent history of an insect bite exhibited paralysis, sensory deficits, and bladder-bowel dysfunction below the lesion level. These findings were consistent with acute myelitis and partially overlapped with Guillain-Barré syndrome. Lumbar puncture and cervical MRI were thus performed. Due to nighttime MRI shutdown, limited resources and the absence of a prioritized spinal MRI pathway, cervical MRI was obtained at 29.5 hours after symptom onset, subsequent to lumbar puncture at 27 hours. The aforementioned objective factors and the diagnostic challenges posed by atypical SSEH contributed to delayed diagnosis.

Our case is unique in that the patient presented with no neck or back pain but had transient right hemiplegia, bilateral dorsal epidural hematoma extension, and a dual exposure to anticoagulation and antiplatelet therapy. To our knowledge, only several painless SSEH cases have been reported thus far [3,4,13]. Unlike Senelick et al.'s [4] painless SSEH case with intermittent symptom remission, urinary retention, and an L1–L3 hematoma, our patient had progressive deterioration and urinary incontinence, with a hematoma extending from C4 to T2. In contrast to Jackson's [3] reported painless SSEH—an autopsy-confirmed complete cervical epidural hematoma without anticoagu-

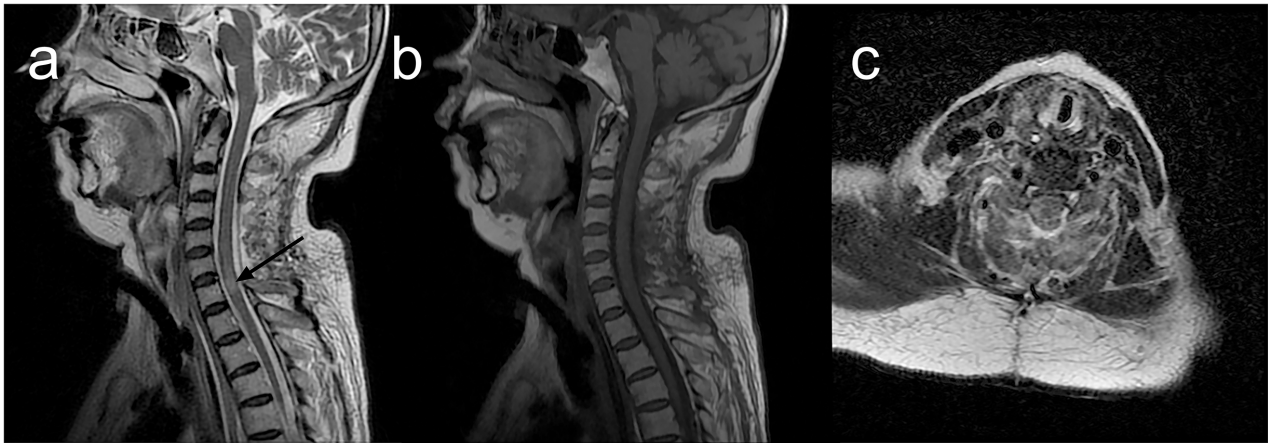


Fig. 4. Postoperative non-contrast cervical spine MRI findings. (a) Sagittal T2WI demonstrates relieved spinal cord compression, with hyperintense signal at the C7 level suggestive of spinal cord edema (arrow). (b) Sagittal T1WI reveals postoperative resection of the posterior elements from C4 to C7 postoperatively. (c) Axial T2WI shows resolution of spinal cord compression with heterogeneous spinal cord signal intensity.

lant use or hemiparesis—our patient presented with hemiparesis, received combined therapy consisting of anticoagulant and antiplatelet drugs, and was diagnosed with cervicothoracic epidural hematoma on MRI. The patient, initially misdiagnosed with acute ischemic stroke, received antiplatelet therapy alongside long-term anticoagulation, which may have aggravated hemorrhage [5,8,14]. Most of the other reported cases describe either a painless presentation or isolated stroke-mimicking manifestations, but not both. Most previously reported spinal epidural hematomas involve only unilateral dorsal extension, since bilateral dorsal involvement is exceedingly rare [1,2,3,4,5,6,7,8]. In contrast, the present case demonstrates a hematoma extending from the right to the left dorsal cervicothoracic spinal canal with bilateral involvement, a pattern that is exceptionally uncommon. This case therefore adds to the limited literature describing such rare clinical and anatomical presentations.

The etiology of SSEH is multifactorial, with major predisposing factors including coagulopathy, anticoagulant use, hypertension, vascular malformations, and iatrogenic causes [15,16]. Anticoagulant and antiplatelet therapies are among the most common interventions, particularly in elderly patients. SSEH represents a rare but critical complication of these treatments [8,16]. In this case, anticoagulant therapy was considered a definite predisposing factor for the epidural hematoma.

Spinal MRI represents the gold-standard approach to diagnosing SSEH, whereas CT is a preliminary emergency screening tool with lower sensitivity [17]. In our case, non-contrast cervical spine CT showed an isodense intraspinal mass at the cervicothoracic junction, obscured by bone artifacts and of indeterminate nature. Cervical spine MRI revealed the hematoma as hyperintense or isointense on T2WI and isointense on T1WI. Therefore, spinal MRI is strongly

recommended for suspected SSEH, as it clearly delineates the hematoma's size, location, extent, and severity of spinal cord compression to guide therapeutic management [18].

Treatment standards for SSEH remain inconsistent. However, most studies suggest that patients with mild neurological deficits (AIS D–E) managed conservatively may achieve symptomatic relief and functional recovery under close neurosurgical monitoring, whereas urgent surgery is recommended for those with severe or rapidly progressive neurological impairment (AIS A–C) once an epidural hematoma is confirmed on MRI [13,14,19]. Recurrent SSEH also warrants surgical intervention [20]. Our patient suffered from progressive neurological dysfunction with an AIS grade A, and emergency surgery was necessary.

Early surgery significantly enhances functional recovery and reduces the risk of permanent neurological injury [13,19]. Poor prognostic factors include severe preoperative neurological deficits, paraplegia duration exceeding 12 hours, and postoperative spinal cord edema [20]. Owing to atypical clinical manifestations, the patient was misdiagnosed at Hengdian Wenrong Hospital, and diagnosis was further delayed in our center, resulting in a 24-hour duration of preoperative paraplegia. The severity of preoperative spinal cord compression and canal compromise ratio are also key prognostic indicators; lower hematoma occupancy is associated with more favorable outcomes [8,21]. In this case, the American Spinal Injury Association Impairment Scale (AIS) was adopted to assess spinal cord compression severity. The patient's preoperative AIS grade was A, which indicated severe spinal cord compression. The spinal canal occupancy rate was calculated as the ratio of hematoma area to total spinal canal area, following the method proposed by Marhold et al. [20]. In their research, a cutoff value of 51% was identified to distinguish between favorable and poor clinical prognoses [20]. The

measured rate in our patient was 54.73%, may be associated with an unfavorable prognosis and was consistent with their reported findings. Postoperative MRI demonstrated spinal cord edema, suggestive of a dismal prognosis [20]. Collectively, these factors contributed to an unfavorable long-term prognosis. Besides, long-term anticoagulation combined with subsequent antiplatelet therapy may also contribute to a poor prognosis. Notably, the patient presented during nighttime hours when MRI services were unavailable, leading to diagnostic delay. Therefore, 24/7 availability of MRI and operating room facilities and the establishment of an emergency green channel for spinal MRI are highly recommended, as prompt diagnosis and urgent decompression are essential for optimal neurological recovery. This case report has several limitations, including the inherent inability of a single-case design to establish causal relationships, the reliance on telephone follow-up, and the absence of repeat MRI during follow-up, and the unavailability of detailed dosage information for the antiplatelet agent administered at an external hospital.

Conclusions

This case underscores the importance of recognizing the painless, stroke-mimicking subtype of SSEH. For patients with unexplained acute limb weakness without pain and a history of anticoagulant use, SSEH should be considered in the differential diagnosis, and urgent spinal MRI should be performed. Timely recognition and intervention can significantly improve neurological function and potentially reduce long-term neurological deficits.

Availability of Data and Materials

The data analyzed are available from the corresponding authors upon reasonable request.

Author Contributions

WJZ: conceptualization, data curation, writing – original draft. JS: conceptualization, data curation, writing – original draft, software. KWW: data curation, investigation, visualization. JBH: writing – review & editing, data curation, investigation. HYX: writing – review & editing, supervision, data curation. All authors have been involved in revising the manuscript critically for important intellectual content. All authors gave final approval of the version to be published. All authors have participated sufficiently in the work to take public responsibility for appropriate portions of the content and agreed to be accountable for all aspects of the work in ensuring that questions related to its accuracy or integrity.

Ethics Approval and Consent to Participate

The study was in accordance with the Declaration of Helsinki and its later amendments. The study was approved by the Ethics Committee of The Fourth Affiliated Hospi-

tal of Zhejiang University School of Medicine (approval number: K2026061; Date of approval: 13 February 2026). Written informed consent was obtained from the patient's family for the publication of the data included in this case report. All patient information has been fully anonymized to protect privacy.

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Conflict of Interest

The authors declare no conflict of interest.

Supplementary Material

Supplementary material associated with this article can be found, in the online version, at <https://doi.org/10.62713/ai.c.4729>.

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