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*Case Report*

# Spontaneous cervical epidural hematoma: Insight into this occurrence with case examples

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## ABSTRACT

**Background:** First characterized in the 19th century, spontaneous spinal epidural hematoma (SSEH) is known as the idiopathic accumulation of blood within the spinal canal's epidural space, causing symptoms varying from general back pain to complete paraplegia. With varying etiologies, a broad spectrum of severity and symptoms, a time-dependent resolution period, and no documented diagnosis or treatment algorithm, SSEH is a commonly misunderstood condition associated with increasing morbidity. While SSEH can occur at any vertebrae level, 16% of all SSEH cases occur in the cervical spine, making it a region of interest to clinicians.

**Case Description:** Herein, the authors present two case examples describing the clinical presentation of SSEH, while also reviewing the literature to provide a comprehensive overview of its presentation, pathology, and treatment. The first case is a patient with nontraumatic sudden onset neck pain with rapidly progressing weakness. The second case is a patient with painless weakness that developed while taking 325 mg of aspirin daily.

**Conclusion:** Clinicians should keep SSEH in their differential diagnosis when seeing patients with nontraumatic sources of weakness in their extremities. The appropriate steps should be followed to diagnose and treat this condition with magnetic resonance imaging and surgical decompression if there are progressive neurological deficits. There is a continued need for more extensive database-driven studies to understand better SSEHs clinical presentation, etiology, and ultimate treatment.

**Keywords:** Epidural hematoma, Idiopathic hematoma, Spinal epidural hematoma, Spontaneous hematoma, Spontaneous spinal epidural hematomas

## INTRODUCTION

Spontaneous spinal epidural hematomas (SSEHs) are rare and complex spinal pathologies that have led to devastating neurologic outcomes among its patient population that has otherwise not had devastating trauma or explainable causes. Namely, SSEH is a condition where blood accumulates in the epidural space of the spinal column and ultimately compresses the spinal cord and nerve roots, leading to symptoms ranging from back pain to quadriplegia.[21] It is considered spontaneous as a reference to its idiopathic etiology, as opposed to some other SEH, which could occur through traumatic injury, tumors, or complications from invasive neurological procedures.[15] SSEHs can occur along at any spinal cord level, with the thoracic

spine being historically the most compromised region in adults while the lumbar being the most commonly affected in children.[17,20] Regarding the area of interest, the cervical spine, 16% of SSEH cases are within this region, with much of these cases continuing into the upper thoracic spine.[17] With its increasingly high morbidity, the SSEH diagnosis potentially requires immediate surgical care, as the condition could cause potentially irreversible neurological deficits by the hour.[2]

Despite its profound clinical consequences, SSEH is a highly misunderstood condition that requires proper identification by clinicians and patients alike to avoid devastating neurologic sequelae.[18] Increasingly so, neurological research has been investigating SSEH for clinical patterns.[18] However, with its low incidence rate, it is hard for researchers to determine risk factors and collect clinical data to better characterize this serious condition. Moreover, there has yet to be an algorithm that summarizes the necessary steps to diagnose and treat SSEH, as it is reported to be regularly misdiagnosed. At present, it is treated as any other SEH: active surveillance unless there is radiographic and clinical progression. Therefore, we present two independent SSEH cases from our institution along with a review of literature to provide an understanding of the SSEH pathology, presentation, and treatment. We also formulated a descriptive process to help diagnose and treat suspected SSEH cases within 24 h symptomatic onset, with the hopes of aiding clinicians to avoid catastrophic misdiagnosis and preserve the neurologic status of SSEH patients.

## CASE EXAMPLES

### Case 1

#### *Clinical presentation*

We present a 52-year-old right-handed Caucasian male with a medical history of HIV, hepatitis C, hypertension, and type II diabetes with a chief complaint of sudden onset severe upper back pain. The patient stated that the upper back pain started while he was conducting a Zoom lecture as a math teacher. He denied any trauma or injuries. He also denied any regular use of anticoagulant medications, but did take aspirin 81 mg daily for general cardiovascular health. He also denied any subjective fever or chills. He stated that the pain was 8/10 in severity. Within hours, he started to notice weakness in his right and right hand which prompted him to call the ambulance and was brought to the hospital. As the weakness became more severe, he began to develop numbness in the right hand and right leg. The patient was admitted and a magnetic resonance imaging (MRI) of the cervical spine was performed.

Radiographically, MRI of the cervical spine without contrast revealed an epidural fluid collection identified posterior to the cord at the C4-C7 levels measuring up to 1 cm thick [Figure 1]. The cord appeared compressed and was most pronounced at the C6 and C7 levels with mild increased cord signal. The signal characteristics were compatible with epidural hematoma.

The patient was emergently taken to the operating room for posterior C5-C7 decompressive laminectomy and evacuation of intra-SEH. On removal of the lamina and ligamentum flavum, there was visualization of epidural hematoma. The purple, clotted, thick, gelatinous material was completely evacuated and sent to pathology for confirmation. There was no obvious vascular malformation noted and there did not appear to be any trauma in the adjacent soft tissues. There did not appear to be any unusual bleeding problems intraoperatively. Complete evacuation of hematoma and decompression was achieved. The histopathological specimen consisted of a 2 × 2 × 1 cm aggregate of dark brown-purple clotted blood fragments. The pathological diagnosis confirmed SHE with no malignancy cells. The patient was returned to his hospital room for continued recovery and physical therapy rehabilitation. He was subsequently discharge to acute rehabilitation facility. The above patient represents a case of idiopathic SSEH.

### Case 2

#### *Clinical presentation*

We present a 72-year-old right hand dominant Caucasian female transferred from outside hospital status – post sudden onset severe left neck pain and left arm pain. She stated that her neck pain started the night prior and suddenly started radiating into her left shoulder and arm which was associated with progressive weakness. Her pain was made worse by any movement. She denied any preceding trauma. She denies any medical history other than hypertension. Of note, she stated that she does take 325 mg aspirin daily for

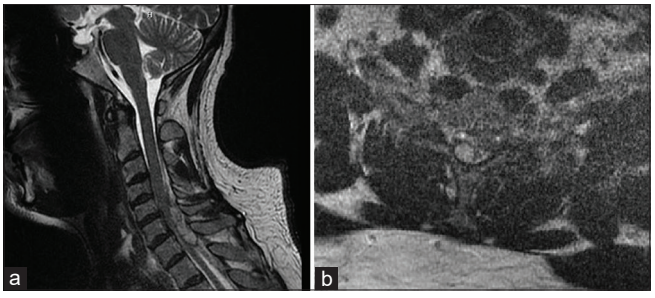


**Figure 1:** T2-weighted MRI of the cervical spine showing hyperintense epidural fluid collection with low T2 signal rim identified posterior to the cord on (a) sagittal view and (b) eccentric toward the right on axial view, at the C4-C7 levels measuring up to 1 cm thick. The cord appears compressed most pronounced at the C6 and C7 levels with mild increased cord signal.

joint pain. She denies having any known bleeding disorder but stated that she tends to bruise easily. Her coagulation laboratories indicated aspirin therapeutic response causing platelet dysfunction: platelet EPI >300 (normal 72–184) and platelet function aspirin 560 (normal 570–675). Her platelet count was 400 and other laboratory work noncontributory. Her physical examination was significant for having motor deficits as follows: left upper extremity: 4/5 deltoid, 4/5 bicep, 3/5 triceps, 2/5 wrist extension, 2/5 wrist flexion, 4/5 handgrip, and 2/5 interosseous muscles, left lower extremity: 4+/5 hip flexion, 5/5 knee flexion, 5/5 knee extension, 4+/5 dorsiflexion, 5/5 plantar flexion, and 5/5 extensor hallucis longus. She also had sensory deficits in the left upper extremity which displayed decreased sensation to pinprick in a nondermatomal distribution.

Radiographically, MRI of the cervical spine showed an epidural hematoma dorsal to the cord at the C3-T2 levels measuring up to 6 mm thick [Figure 2]. It lied eccentric toward the left with mild-to-moderate compression on the cord. There was no definite cord edema, abnormal enhancement, or major ligamentous injury. MRA of the neck was negative for any abnormal vascularity.

She was taken to surgery within hours of presentation for emergent posterior C3-C7 decompressive laminectomies and evacuation of intra-SEH. There were findings of dorsal purple clotted epidural hematoma that was eccentric to the left. Operatively, there was complete decompression achieved and hematoma was entirely evacuated. Baseline intraoperative neuromonitoring showed decreased motor signals in bilateral upper extremities, left worse than right. The SSEP sensory signals were normal and stable throughout the surgery. After decompression and evacuation, there was identifiable improvement in bilateral upper extremity motor signals on intraoperative neuromonitoring.

Postoperatively, within the first 24 h period, she had near resolution of her weakness with only residual weakness of 4+/5 wrist extension, 4+/5 wrist flexion, 4/5 handgrip, and 4/5 interosseous muscle. She continued to have left upper extremity decreased sensation to pinprick testing in nondermatomal fashion that was stable. She worked with the physical and occupational therapy teams and was subsequently discharged home with outpatient physical therapy and recommendations to discontinue her daily aspirin use. The above case represents SSEH likely resultant from daily aspirin usage.

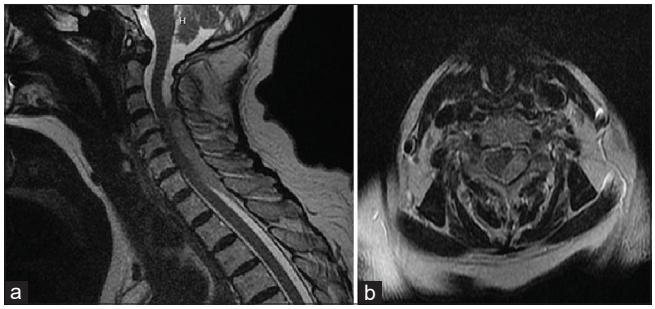


**Figure 2:** T2-weighted MRI of the cervical spine showing (a) sagittal view of compressive epidural hematoma and (b) axial view of the left eccentric epidural hematoma causing spinal cord compression.

## DISCUSSION

### Etiology

The etiology of SSEH is generally unknown, yet it has been widely investigated.[5] The general accepted theory is that these hematomas present following a rupture of the epidural venous plexus which are valveless vessels, but the source of the spontaneous bleeding is debated as originating from an arterial or venous source.[15] Other etiological sources of SEH have been discussed as vascular anomalies, arteriovenous malformations, vertebral hemangiomas, obstetrical birth trauma, lumbar puncture, spinal manipulation, epidural procedures, and medication side effect.[1,16] Coagulopathies, vascular surgeries, and surgical complications have also been discussed as potential causes of SSEH, yet this is not widely accepted as some authors would refuse to label the condition as "spontaneous" if they do not come from an idiopathic source.[5,22] Nevertheless, surgical bleeding, missile injuries, hypertension, physical exertion, and abnormal bleeding tendencies have also been reported to result in SSEH.[7] Long-term aspirin use has also been associated with SSEH in prior case series.[4,12,14] Of note, both patients presented here had long-term exposure to aspirin, and the SSEH described in Case 2 was likely pharmacologically associated.

### Epidemiology

Historical epidemiological literature suggests that SEHs have been clinically investigated since the mid-to-late 19$^{th}$ century.[5] However, since its description in autopsies in 1862, along with its first surgical treatment in 1897, SSEHs have remained an enigma in spinal care.[5,17] Much of this complexity is because of the idiopathic nature of this pathology, along with the growing speculation surrounding its pathogenesis. Nevertheless, the majority of SSEH is most commonly found in men between the ages of 52 and 70.[13,17] With an increasing age, the epidural space becomes smaller, providing conditions suitable for the SSEH environment. SSEH tends to be most prevalent around the C6 and T12 levels, with spinal hematomas generally measuring approximately 3.6 vertebral levels in length and found dorsal to the spinal cord.[5] There is no clear correlation between SSEH to race or gender, however, there are reported predisposing factors such as coagulopathies, anticoagulation, disc herniations, vascular malformations, and neoplasms to go with potential idiopathic causes.[9] SSEHs most prominent

risk factor is the presence of disorders of coagulation systems such as liver disease, alcoholism, thrombocytopenia, or pharmacologic anticoagulation.[11] SSEHs have also been considered to be more prevalent in individuals with rheumatologic pathologies, yet the reason for this is not clear. The pathology itself is considered to be very rare, with an incidence of 0.1 from 100,000 cases per year.[9] However, the introduction of MRI technology into mainstream clinical practice is creating a gradual prevalence increase due to increased detection capability.[9] Globally, SEHs are generally not widely represented. Asian countries such as South Korea and Japan do not have treatment codes that represent SSEH, indicating a need for greater global recognition of SSEHs epidemiological data. Nevertheless, its current data present enough information to create models and understanding for treatment across the globe.

## Pathophysiology

At present, it is uncertain whether SSEHs originate from the arterial or venous system. Both theories have been proposed, however, venous origins are more widely accepted due to the fact that spinal epidural veins are valveless, making them susceptible to damage from abdominal or thoracic pressure.[5] The venous etiology theory by Bruyn and Bosma proposed that increased thoracic and abdominal pressure can lead to an increase in intravenous pressure, and with no valves to displace the blood flow along with the epidural veins being naturally thin, blood accumulates in the epidural space leading the mechanical compression of the spinal cord and rupture of the locus minoris resistentiae.[5] This venous etiology theory is said to occur during events that increase intravenous pressure such as sneezing, coughing, defecation, weight lifting, or pregnancy. However, this theory seems less plausible in cervical areas due to the fact that there is low venous epidural pressure in the cervical spine.[5] With the rapid development of a cervical SEH, arterial etiology is hypothesized. Beatty and Winston hypothesized that the cause of the arterial bleeding in the cervical spine is free anastomotic arteries that run in the epidural space that subsequently connects with radicular arteries and causes blood accumulation. Essentially, the theory is based on the principle that venous pressure is less than the intrathecal pressure.[19] Because the majority of these hematomas are along the C6-7 region (a highly mobile segment), it is believed that drastic movements can lead to free arteries to stretch beyond their range limits of tolerance and cause them to rupture and potentially compress the spine.[6]

## Management principles

At the moment, there is no conclusive answer regarding the best practice for SSEH workup and treatment. With a low prevalence, unknown etiology, and a variety of symptoms, treating such a condition has raised profound challenges that prevent a simple generalized treatment. Nevertheless, SSEH is managed using two methods: conservative or surgical. The traditional treatment for SSEH has been through surgical intervention, with decompressive laminectomy with hematoma evacuation being the common procedure. However, several reports do find that conservative methods have led to spontaneous resolution of SSEH when patients are not experiencing neurological deficits.

### Conservative methods

The conservative method has been shown to be growing increasingly among patients who are treated for SSEH. Namely, conservative management means treating back and neck conditions using nonsurgical methods. This includes the use of injections, drug therapy, or physical therapy. In a SSEH case report, pharmacological conservative management was performed using steroids by giving the patient a dexamethasone loading dose of 10 mg IV followed by a 4 mg IV or PO every 4 h and tapering down.[8] This method is usually attributed to patients suffering from a more mild presentation, yet its clinical success has led to differences in opinions.[15] Some reports claim that conservative treatment may worsen the diagnosis, with increase in hematoma size and neural deficits being reported in some SSEH cases.[15] Moreover, certain reports claim that conservative therapy leads to clinical deterioration and will eventually require surgery and that delayed intervention could prove harmful.[5,15] Nevertheless, successful outcomes have been reported among patients treated conservatively. A study by Groen found that 84% of patients treated using conservative measures had a complete resolution of hematoma and resolved symptoms, however, these results had an element of bias as the majority of patients in these studies had a more mild onset of the disease.[15] Conservative management poses risks of an increase in neurological deficits, increase in hematoma size, and deteriorating America Spinal Cord Injury Association (ASIA) scores.[15] Therefore, conservative management should only be done when there is a lack of neurological deficits and in centers with spinal surgeons who have access to readily available MRI.

### Surgical methods

As mentioned, surgical management has been the preferred method for definitive treatment of SEEH with the standard operative procedure being a decompressive laminectomy with hematoma evacuation.[15] A decompressive laminectomy is a surgical procedure to lamina and ligamentum flavum to decompress the spinal cord, and thus, allowing for hematoma evacuation. Operative care is usually immediately done on patients who have more severe cases of SSEH, usually determined by the ASIA score system.[5,15] Most SSEH

patients who improve with operative care have a ASIA score between C and D.[15] The surgical time frame is of essence in SSEH patients, as Raasck *et al*. reported that patients who received operative attention within 12 h of symptoms had 84% recovery rate compared to patients who were operated 24 h after symptoms who had 47% recovery rate.[15]

**Diagnosis/prognosis**

Unfortunately, SSEH is a rare condition that is commonly misdiagnosed.[3] We propose a standard approach to evaluating patients with suspected SSEH including an initial clinical evaluation and confirmatory MR imaging studies. All patients should first be evaluated with a thorough history and physical focused on assessing for neurologic deficits. Typically, the presenting symptom is sustained acute axial back pain or neck pain that radiates to corresponding dermatomes with eventual neurologic deficits. This pain is often described as severe that mimics that of a disc herniation given its compressive nature.[15] The pain typically worsens through activities that increase intraspinal pressure such as coughing, sneezing, and weight lifting. Other presenting signs and symptoms could include lower extremity weakness, urinary or fecal incontinence, unilateral or bilateral weakness, sensory deficits with or without radicular paresthesia, alterations in deep tendon reflexes, and alteration of anal/sphincter tone.[11] In some cases, the hematoma could be asymptomatic, but in severe cases, the hematoma could compress the spinal cord and lead to devastating neurological consequences such as paraparesis or quadriparesis.[11] When diagnosing SSEH, it is paramount to be aware of present and worsening neurological deficits, as assessing the presence of any pathological entities related to SSEH is crucial to differentiating it from other conditions. The differential diagnosis associated with SSEH includes epidural abscess, spinal cord disease, neoplasm, or acute disk herniation, anterior spinal artery syndrome, exacerbation of a preexisting neurologic disorder, and presentation of a previously undiagnosed neurological condition.[11]

Additional laboratories and diagnostic tests should be performed when finding SSEH. CBC (complete blood count) with platelets is done to determine and rule out the presence of an infection while assessing hemorrhagic risk regarding platelet count. Prothrombin time (PT)/activated partial thromboplastin time could also be ordered to determine bleeding diathesis.[15] Toxicology and serum chemistries should also be assessed for significance as they could also complicate the clinical course. MRI has been historically the best imaging tool for emergency spinal procedures due to its noninvasive, rapid, and comprehensive evaluation of the spinal canal.[5]

For SSEH, MRI can accurately portray the presence of the hematoma while also finding any possible vascular malformations or epidural abscess. The MRI can also estimate the age and extent of the hematoma along with the degree of cord compression and myelomalacia.[11] Radiographic criteria for diagnosis have been proposed by Matsumura *et al*.[10] When assessing the MRI in the first 24 h, the hematoma is isointense on T1 images while being hyperintense on T2 images. By 48 h, the hematoma appears hyperintense on both T1 and T2 images.[11] T2 hyperintensity within the spinal cord within 24 h is a sign of poor clinical outcomes as it identifies myelomalacia and radiographic spinal cord injury.

Liao *et al*. proposed an algorithm to describe severity of cases, with progressive deterioration and ASIA scores between B-D and ASIA A scores lasting more than hours requiring "emergent surgery." ASIA A scores lasting less than 12 h are considered "urgent," while improving cases with ASIA D score are deemed to be solely monitored.[15] Nevertheless, the relationship between symptom onset time and clinical outcome is highly correlated in these severe cases. In cases of ASIA scores between C and D, critical care is still highly recommended, however, time is less of a factor which allows for considering conservative options.[15]

## CONCLUSION

SSEH will continue to be investigated as one of the most curious pathologies in spinal care. Moreover, with a wide clinical presentation spectrum, it is important that patients identify and seek immediate neurologic attention when experiencing symptoms such as sudden radiating back or neck pain, lower extremity weakness, or motor/sensory dysfunctions. From the clinical standpoint, it is paramount that suspected SSEH patients undergo the proper screening in a timely fashion. MRI showing T2 hyperintensity within the spinal cord within 24 h is a poor prognostic indicator. Depending on the patient's ASIA score, along with the time interval between symptom manifestation and treatment, conservative or surgical management techniques could be performed. Surgical management is considered the gold standard in resolving cases of SSEH with neurological deficits, however, conservative management has also shown success in patients with radicular symptoms or more mild pathological courses.

SSEH is sometimes wrongly misdiagnosed. Its rarity brings obstacles to neurologic research because it becomes harder to create a randomized and controlled trial when the incidence being so little while also hampering the collection of data and clinical outcomes. Further research is warranted for SSEH, where more risk factors are identified and parameters are created to determine treatment success. At present, there are no clear published data on recovery prognostication or their associated time periods. In conclusion, SSEH will remain to be a complex and serious diagnosis, and by further investigating its pathology, clinicians can aim for better treatment and education of its future patients.

### Declaration of patient consent

Institutional Review Board permission obtained for the study.

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### Conflicts of interest

There are no conflicts of interest.

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