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*Case Report*

# Hemiparesis to Quadriparesis Following Alteplase: A Case of Spontaneous Spinal Epidural Haematoma Mimicking Acute Stroke

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## Abstract

New-onset neurological deficit is a common presentation in the hospital emergency department. It is crucial to differentiate an acute stroke from a stroke mimic, as thrombolysis is widely used for acute ischaemic stroke. Here we discuss a patient who presented with sudden-onset hemiparesis and neck pain and was initially offered thrombolysis. He later deteriorated neurologically and was found to have a spontaneous spinal epidural haematoma. Following urgent surgical decompression, he showed gradual improvement in motor power and was subsequently referred for rehabilitation.

**Keywords:** Acute ischaemic stroke, stroke mimic, hemiparesis, spontaneous spinal epidural haematoma, thrombolysis

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## Introduction

Spinal epidural haematoma (SEDH) is a relatively rare condition encountered in clinical practice. The first published case dates back to 1869 [1], and the annual incidence is approximately one per million [2]. In many patients, no underlying aetiology is identified. When SEDH is not associated with trauma or a medical intervention, it is termed a spontaneous spinal epidural haematoma (SSEDH) [3].

SEDH should be considered in the hospital emergency department, as it can present as a sudden-onset neurological deficit that mimics an acute stroke [4,5]. Clinical manifestations of SEDH can vary, ranging from simple radiculopathic neck pain to profound quadriparesis [3]. If the patient presents in the acute stage, within the first four and a half hours after symptom onset, distinguishing SEDH from an acute stroke is challenging. As illustrated by our case, this may lead to the administration of thrombolytic therapy despite a normal non-contrast CT scan of the brain.

If an unrecognised spontaneous spinal epidural haematoma is treated with thrombolysis, the haematoma may expand, leading to further compression of the spinal cord and worsening neurological deficits. There are only a few reported cases of worsening weakness following the administration of recombinant tissue plasminogen activator (rtPA) due to an expanding cervical epidural haematoma [6,7]. To our knowledge, this is the first reported case from Sri Lanka. Therefore, early suspicion is crucial for this relatively rare entity, both before and after the administration of rtPA, particularly in a neurologically deteriorating patient with normal brain imaging. Early recognition may facilitate timely surgical decompression and a favourable neurological outcome.

## Case report

A 66-year-old male, a known patient with hypertension and chronic kidney disease stage 3, presented to the

Emergency Treatment Unit, National Hospital Kandy, with a three-hour history of sudden-onset right-sided upper and lower limb weakness. An urgent non-contrast CT (NCCT) scan of the brain was arranged, and the neurology team was informed to assess his eligibility for thrombolysis.

The on-call registrar in neurology immediately attended to the patient. Upon further inquiry, the patient revealed that he had developed sudden-onset lower neck and interscapular pain approximately 30 minutes before the onset of right-sided weakness. This had occurred while travelling by bus, and he could not recall any acceleration, deceleration or trauma to the neck. On examination, the patient was conscious and alert with no visual disturbances, and his speech was normal. Neurological examination revealed power of 4/5 in the right upper limb and 4/5 in the right lower limb. Sensory examination and power in the left upper and lower limbs were normal. His pulse was regular, and his blood pressure was 180/100 mmHg in both upper limbs. There was no radio-radial delay, and neck movements were not restricted. Together with his vascular risk factors, elevated blood pressure and a National Institute of Health Stroke Scale (NIHSS) score of 5, the clinical presentation was considered most consistent with an acute ischaemic stroke.

Meanwhile, the NCCT brain was reviewed, and the only abnormality detected was a left fronto-temporal arachnoid cyst. His chest radiograph did not show mediastinal widening. As the onset of weakness was still within four and a half hours, it was decided to administer intravenous alteplase after lowering the blood pressure with a single bolus of intravenous labetalol. Over the next several hours, the patient developed progressive neurological deterioration with involvement of the left upper and lower limbs. By the following morning, he had progressed to quadriplegia. Throughout this period, there was no cranial nerve involvement, and the patient remained conscious and alert. All limb reflexes were diminished, and a sensory level at C4 was demonstrated. An episode of bradycardia and hypotension was also observed on continuous monitoring.

The preceding neck pain, progressive neurological deterioration and preserved higher cortical function raised the suspicion of cervical cord compression due to an expanding epidural haematoma. An urgent MRI of the cervical and dorsal spine was requested. His coagulation profile was normal, with an INR of 1.1 and an activated partial thromboplastin time (APTT) of 32 seconds. The complete blood count showed a haemoglobin of 10.4 g/dL, platelet count of 241 × $10^9$/L and white cell count of 7.13 × $10^9$/L. His serum creatinine was 129.1 µmol/L. The MRI demonstrated a spinal epidural haematoma extending from C2 to C4, causing eccentric canal stenosis and spinal cord compression (Figure 2).

The neurosurgical team subsequently took over the patient's care and performed an emergency C1–T1 laminectomy with evacuation of the spinal epidural haematoma. Multiple tissue biopsies, including adjacent bone, were obtained during surgery. Histopathological examination was negative, and tissue cultures showed no growth. The absence of an underlying structural or pathological cause supported the diagnosis of spontaneous spinal epidural haematoma. The preceding neck pain and initial hemiparesis suggested that the haematoma had developed before thrombolysis, while the subsequent neurological deterioration was most likely due to expansion of the haematoma following alteplase administration.

Following surgical decompression, the patient demonstrated gradual improvement in motor power during the postoperative period. He was subsequently referred for rehabilitation for further neurological recovery. The timeline of events is briefed in Table 1.

**Discussion**

Spinal epidural haematoma is a relatively rare condition that can be associated with tumours, coagulation abnormalities, arteriovenous malformations or cavernous angiomas. SSEDH is diagnosed after excluding these underlying causes and is considered idiopathic [3]. In approximately 40–50% of cases, no underlying cause is identified, and these cases are classified as SSEDH [3,8]. In our patient, coagulation studies were normal, and MRI findings and direct intraoperative observation did not reveal any vascular malformations, tumours or other causative pathology. The clinical presentation, with neck pain preceding the onset of hemiparesis, suggests that the spontaneous spinal epidural haematoma was already present before thrombolysis. Administration of rtPA most likely contributed to the expansion of the pre-existing haematoma, resulting in neurological deterioration from hemiparesis to quadriparesis.

**Table 1:** Timeline of the patient's clinical course

| Time | Clinical event |
|---|---|
| 0 hours | Sudden onset of lower neck and interscapular pain while travelling by bus |
| 30 minutes later | Sudden onset of right-sided upper and lower limb weakness |
| 3 hours after symptom onset | Presentation to the Emergency Treatment Unit |
| Shortly afterwards | NCCT brain showed no acute intracranial pathology; patient assessed as eligible for thrombolysis |
| Within 4.5-hour therapeutic window | Intravenous alteplase administered after blood pressure control |
| Over the next several hours | Progressive neurological deterioration with involvement of the left upper and lower limbs |
| Following morning | Quadriplegia with sensory level at C4, diminished reflexes, bradycardia and hypotension |
| Immediately thereafter | MRI cervical spine demonstrated C2–C4 spinal epidural haematoma with cord compression |
| Same day | Emergency C1–T1 laminectomy and evacuation of the haematoma |
| Postoperative period | Gradual improvement in motor power |
| Discharge | Referred for rehabilitation |



**Figure 1:** Non-contrast computed tomography (NCCT) scan of the brain demonstrating no evidence of acute intracranial haemorrhage or early ischaemic changes. An incidental left fronto-temporal arachnoid cyst is noted.

The location of SSEDH can vary. Typically, it exhibits a bimodal distribution, occurring at cervical spinal levels C1–C3 and C6–T12 [5,9]. In our patient, the haematoma extended from C2 to C4. It is usually located on the dorsal aspect of the spinal cord and has a mean length of 3.6 spinal segments [3,5,9]. Cervical epidural haematomas are typically acute in onset and spontaneous in aetiology, whereas lower spinal haematomas usually present subacutely and follow a more prolonged course [3]. Although SEDHs may originate from either arterial or venous bleeding, the commonly accepted source is the epidural venous plexus. As these veins are valveless, they are susceptible to sudden increases in transmitted pressure resulting from raised intrathoracic or intra-abdominal pressure, which may lead to rupture [3,10]. However, arterial bleeding has also been proposed as the source of cervical epidural haematomas because of the presence of anastomotic arteries within the epidural space [3]. In our patient, no provoking factors such as coughing, sneezing or trauma were identified. Therefore, together with the upper cervical location and the acute presentation, an arterial source of bleeding is a possible explanation. The subsequent neurological deterioration following thrombolysis most likely reflected expansion of the pre-existing haematoma rather than its initial development.

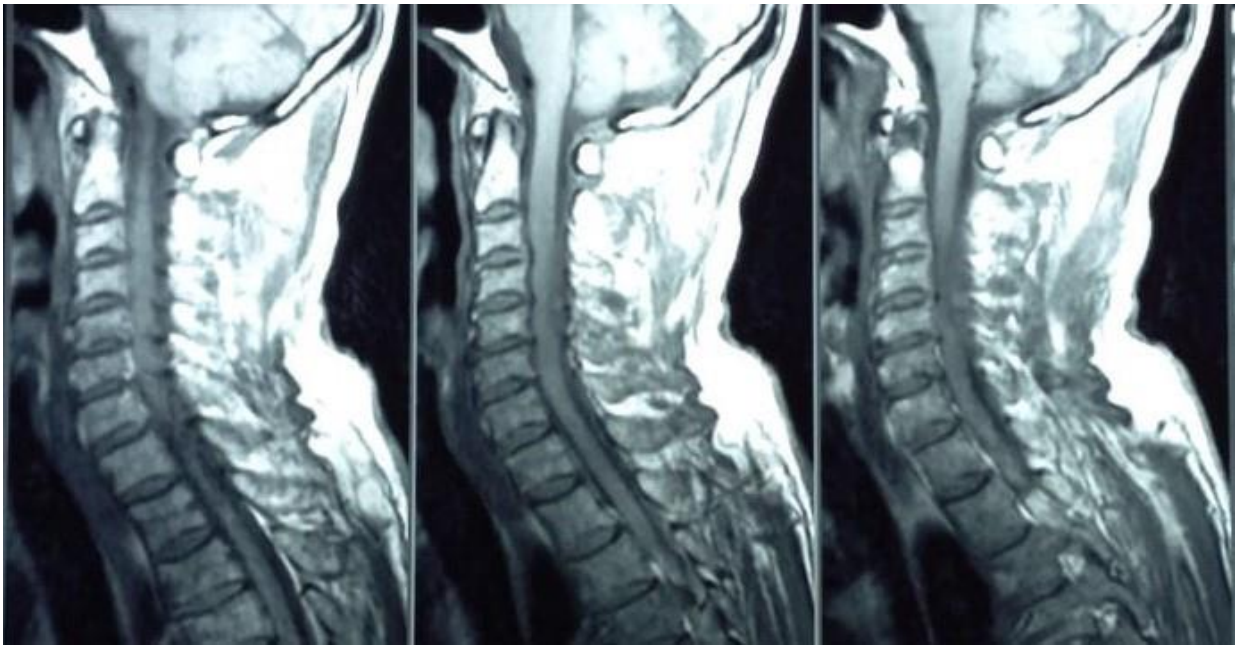


**Figure 2:** Magnetic resonance imaging (MRI) of the cervical spine demonstrating a spinal epidural haematoma extending from C2 to C4, causing eccentric spinal canal stenosis and compression of the cervical spinal cord.

The clinical presentation of SEDH can range from radiculopathic pain to profound quadriparesis. This depends on the degree of spinal cord compression and the location of the haematoma relative to the corresponding spinal segments [5]. In our patient, the initial hemiparesis was most likely due to unilateral compression of the corticospinal tract at the C2–C4 level. Subsequent expansion of the haematoma led to progressive spinal cord compression and neurological deterioration to quadriparesis. Depending on the severity of the haematoma, approximately 37% of patients present with complete sensorimotor deficits, while the remainder present with milder sensory, motor or combined sensorimotor deficits [3]. The initial degree of neurological impairment is an important prognostic indicator, with better neurological outcomes observed in patients who retain greater residual neurological function at presentation [3,9].

In the emergency department, particularly in the Emergency Treatment Unit, it is crucial to differentiate SEDH from acute cerebral infarction. According to the current literature, several clinical features may favour the diagnosis of SEDH and should prompt further evaluation before thrombolysis is considered in the presence of normal brain imaging. The most consistent clinical feature is acute-onset neck or back pain, which usually precedes the onset of hemiparesis or other neurological deficits [3,5–7]. Other suggestive features include a positive Lhermitte's sign and the absence of cranial nerve palsies. Horner's syndrome and Brown-Séquard syndrome have also been reported in some patients [6,7].

In the presence of these clinical features and a high index of suspicion for SEDH, CT angiography may be performed to exclude vertebral artery dissection, another important differential diagnosis in patients presenting with neck pain and stroke-like symptoms. More importantly, an MRI of the cervical and thoracic spine should be performed whenever feasible. However, this approach has limitations, particularly in resource-limited settings where MRI is not readily available. This may lead either to an unacceptable delay in administering thrombolysis to patients with acute ischaemic stroke or to inadvertent administration of rtPA to patients with SSEDH, resulting in potentially life-threatening neurological deterioration.

The current recommendation is to administer intravenous alteplase within four and a half hours of stroke symptom onset, provided that intracranial haemorrhage has been excluded by a non-contrast CT scan of the brain [11,12]. However, spontaneous spinal epidural haematoma remains a rare but important stroke mimic. In patients presenting with acute neck pain followed by hemiparesis, particularly when cranial nerve involvement is absent and brain imaging is normal, clinicians should maintain a high index of suspicion for SSEDH. Where feasible, MRI of the spine should be performed before administering rtPA to avoid inadvertent thrombolysis and the potential expansion of a pre-existing spinal epidural haematoma.

## Conclusion

Spontaneous spinal epidural haematoma is a rare but important stroke mimic that should be considered in patients presenting with acute neck pain followed by hemiparesis and normal brain imaging. A high index of suspicion is essential before administering intravenous thrombolysis, particularly in the absence of cranial nerve involvement. Early recognition and timely MRI of the spine, when clinically feasible, may facilitate prompt surgical decompression and prevent neurological deterioration associated with inadvertent thrombolysis.

**Patient Consent:** Informed consent for publication of this case report and the accompanying images was obtained from the patient. All identifying information has been omitted to protect patient confidentiality.

**Data Availability Statement:** All data generated or analysed during this study are included in this published article. No additional datasets were generated.

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**Competing Interests:** The authors declare that they have no competing interests.

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