

CASE REPORT

Hemiparesis in spontaneous spinal epidural haematoma: a potential stroke imitator

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SUMMARY

Spontaneous spinal epidural haematoma (SSEH) is a rare condition that requires urgent surgical intervention in order to prevent permanent neurological deficit. SSEH commonly presents as a paraparesis or tetraparesis. SSEH presenting as a hemiparesis is less common and in such situations, it can be mistaken for a cerebrovascular accident (CVA). Thrombolytic or anticoagulant treatment for CVA can potentially worsen the neurological deficit. We report one such case of SSEH misdiagnosed as a CVA. Treatment with tissue plasminogen activator led to worsening of his condition. On a subsequent cervical spine MRI, an epidural haematoma extending from C3 to C5 was detected and treated with laminectomy and evacuation. Surgical intervention led to significant improvement from American Spinal Injury Association Scale (ASIA) B to ASIA E. Presence of clinical features such as Horner's syndrome, Brown-Sequard syndrome and the absence of cranial nerve palsies in acute hemiparesis are indicative of SSEH rather than CVA.

BACKGROUND

Symptomatic spontaneous spinal epidural haematoma (SSEH) leading to spinal cord compression is a neurosurgical emergency that requires urgent decompression and haematoma evacuation. The clinical presentation depends on the location and the size of the SSEH. Patients with cervical SSEH usually present with axial neck pain that is occasionally associated with radicular arm pain, thus mimicking a prolapsed intervertebral disc. This is quickly followed by the onset of motor weakness, usually presenting as tetraparesis or paraparesis.¹⁻³ Hemiparesis is an extremely rare presentation of SSEH and is frequently misdiagnosed as a cerebrovascular accident (CVA).⁴⁻¹⁵ It is very important to consider cervical spinal cord compression in patients presenting with acute neurological deficit and to perform an MRI scan if there is any doubt.^{16 17} It has been suggested that early diagnosis and decompression lead to better neurological recovery and a good outcome.¹⁸

CASE PRESENTATION

A 51-year-old right-handed man with a history of hypertension presented with an acute onset of right-sided posterior neck pain in the early morning. The Visual Analogue Score for pain was 7. He had no weakness or loss of coordination, and he was able to walk. After about 45 min, he noticed the onset

of right upper limb and right lower limb weakness with drooping of the eyelids on right side. There were no visual disturbances, change of voice or difficulty in swallowing. He was quickly rushed to the tertiary care hospital where his blood pressure (BP) was recorded to be 157/95 mm Hg with a pulse rate of 62 beats/min. Neurological examination revealed weakness of grade 2/5 throughout the right upper limb, grade 3/5 power of right hip flexion and knee extension (L2, L3 myotomes) and grade 4/5 power of right ankle dorsiflexion, big toe dorsiflexion and ankle plantar flexion (L4, L5 and S1 myotomes, respectively). The left upper limb and left lower limb power was grade 5/5. Reduced sensation with a sensory level of T12 was noted with hyper-reflexia, ankle clonus and Babinski's sign in the right lower limb. The National Institutes of Health Stroke Scale score was 6 at the time of presentation. Hence, as per American Heart Association/American Stroke Association guidelines, a non-enhanced CT scan (NECT) and multiphasic CT angiography of the brain were done.¹⁹ A provisional diagnosis of acute territorial infarct of left middle cerebral artery was made with an Alberta Stroke Program Early CT Score of 9. However, no haemorrhage, no clot in the arterial system or any arterial dissection was detected on the CT-brain angiogram. In view of the strong clinical suspicion, no evidence of haemorrhage in brain and closing window of opportunity for effective thrombolysis tissue plasminogen activator (t-PA) was commenced 4.5 hours after the onset of symptoms.¹⁹ A subsequent MRI brain did not show any evidence of the brain infarct.

His neurological deficit then gradually deteriorated along with the onset of acute retention of urine and he was shifted to a tertiary spine institute next day. On examination in the emergency department, he had grade 0/5 power of the C5, C6 myotomes, grade 1/5 power of C7-T1, L4-L5 myotomes, grade 2/5 power in L2 and grade 5/5 power in L3, S1 myotomes (table 1), all on the right side. The left upper limb and left lower limb had grade 5/5 power. The sensory examination revealed hypoesthesia below T4 on the left side and hyperaesthesia in right upper limb which was suggestive of a Brown-Sequard syndrome. There was no saddle anaesthesia and the anal tone was intact. The American Spinal Injury Association scale was grade C. He was noted to have ptosis, miosis (anisocoria) and anhidrosis on the right side of the face indicative of a Horner's syndrome. The BP was 130/72 mm Hg, and the pulse rate was 59 beats/min.



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Table 1 Evaluation of patient as per American Spine Injury Association Score

Myotomal involvement	Before t-PA	Preoperative	6 weeks postoperative
Right C5 elbow flexion	2	0	4
Right C6 wrist extension	2	0	4
Right C7 elbow extension	2	1	4
Right C8 finger flexion	2	1	4
Right T1 finger abduction	2	1	3
Right L2 hip flexors	3	2	5
Right L3 knee extensions	3	5	5
Right L4 ankle dorsiflexion	4	1	5
Right L5 long toe dorsiflexion	4	1	5
Right S1 ankle plantar flexion	4	5	5

t-PA, tissue plasminogen activator; 0, absent; 1, visible contractions; 2, active movement only if gravity eliminated; 3, active movement through range of motion against gravity; 4, active movement through range of motion against resistance; 5, normal.

INVESTIGATIONS

Radiographs and a CT scan of the cervical spine showed no fracture or malalignment. MRI of the whole spine showed a fusiform heterogeneous non-enhancing extradural mass along the right posterolateral aspect of central canal at C3–C5 level measuring 8 mm × 1.7 mm in maximum axial dimension (figure 1). The mass was isointense on T1-weighted and T2-weighted images with severe narrowing of the central canal and increased signal intensity of the spinal cord. MR angiogram did not reveal any vascular lesion but it showed that the left vertebral artery was the dominant artery. Full blood count, coagulation profile, platelet count and biochemistry profile were normal.

DIFFERENTIAL DIAGNOSIS

- ▶ SSEH.
- ▶ Prolapsed intervertebral disc.
- ▶ Spondylodiscitis.
- ▶ Epidural abscesses.
- ▶ Transverse myelitis.
- ▶ Epidural tumours.
- ▶ Acute myocardial infarctions.
- ▶ Cerebrovascular events.
- ▶ Dissecting aortic aneurysms.

TREATMENT

Next morning (48 hours after onset of symptoms) the patient underwent bilateral spinous process C4 laminectomy and a right C3 and C5 hemilaminectomy (figures 2 and 3). The haematoma was evacuated and haemostasis was achieved. A malformed

venous plexus was found overlying the spinal cord at C5 level. The intraoperative blood loss was 350 mL. Fresh frozen plasma was kept ready but was not required as the haemostasis was adequate. Histological examination of the haematoma showed a well-formed and organised clot without any evidence of malignancy.

OUTCOME AND FOLLOW-UP

The neurological status improved remarkably over the next 6 weeks. His urinary incontinence recovered by day 14. The motor power of all the lower limbs muscles recovered to MRC grade 5/5, and the upper limb recovered to MRC grade 4/5 except for right T1 which was grade 3/5. He recommenced work by the 9th postoperative week.

DISCUSSION

Acute SSEH was first described by Jackson in 1869²⁰ and first treated surgically by Bain in 1897.²¹ The common sites for development of SSEH show a bimodal distribution between the lower cervical and thoracic regions with peaks at C6 and T12.²² The haematoma usually spans three to four cervical levels.^{23 24} The most common age at presentation is after fourth or fifth decade and the male-to-female ratio is 1.4:1.³ The cause of SSEH is idiopathic in up to 40% of cases and causes such as vascular malformation, coagulopathy, anti-platelet agents and excessive garlic ingestion have been reported in the rest.²⁵ The source of bleeding can be either the epidural veins or the epidural arteries. It is hypothesised that activities such as coughing, sneezing, sudden twisting or lifting a heavyweight can trigger a rise in venous pressure and precipitate haemorrhage in the epidural venous plexus.²⁶ A rise in intra-abdominal and intrathoracic pressure leads to an increase in spinal epidural venous pressure.^{22 25} Sudden increase of epidural venous pressure in this valveless system predisposes to a rupture. A pair of longitudinal veins in the posterior internal vertebral venous plexus is connected with multiple interconnecting veins and a rupture of these veins along the sides of interconnecting veins is believed to be the cause of SSEH.^{27 28} However, Beatty and Winston² believed that the pressure in the epidural veins is less than the intrathecal pressure and so any form of venous bleeding should be tamponaded. A tear of the lateral longitudinal arterial system due to traction of cervical nerves during stretching of upper limbs has also been proposed to cause SSEH.² It partially explains the higher incidences of SSEH at the mobile lower cervical spine and lower thoracic spine. This hypothesis is further supported by the fact that larger series of SSEH have shown a correlation between SSEH and arterial hypertension.²³



Figure 1 Fusiform heterogeneous T2-weighted isointense non-enhancing extradural mass (white arrows) along the right posterolateral aspect of central canal extending from levels C3/4 to C5 and compressing the cord from right. (A) Sagittal T2-weighted image, (B) axial T2-weighted image at C3/4, (C) axial T2-weighted image at C4 and (D) axial T2-weighted image at C5.

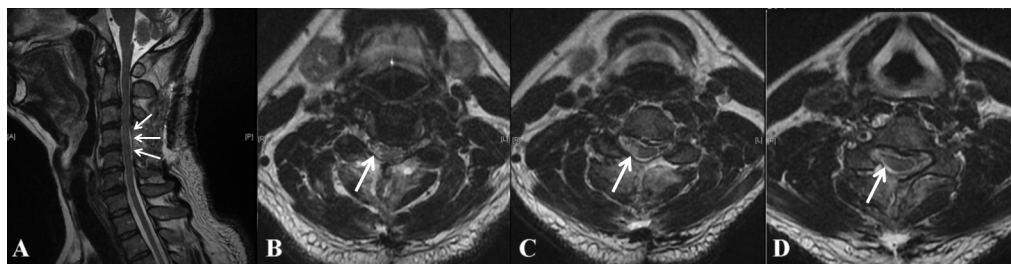


Figure 2 MRI after the spinous process preserving C4 laminectomy and C3, C5 right hemilaminectomy. Postoperative MRI shows no residual haematoma (white arrows) and decompressed spinal cord at the affected segments: (A) sagittal T2-weighted image, (B) axial T2-weighted image at C3-4, (C) axial T2-weighted image at C4 and (D) axial T2-weighted image at C5.

The differential diagnosis of SSEH includes a prolapsed intervertebral disc, infections such as spondylodiscitis, epidural abscesses and transverse myelitis, epidural tumours, acute myocardial infarctions, cerebrovascular events and dissecting aortic aneurysms.¹¹ SSEH with hemiparesis being mistaken for an acute ischaemic CVA is not uncommon and there are at least 11 such cases reported in the literature (table 2). Treatment with heparin, t-PA, aspirin or warfarin has been instituted in these patients with a severe risk of aggravating the clinical condition. In fact, mortality has been reported in one of the patients with SSEH that was misdiagnosed as a CVA.⁹ Several factors can lead to such a misdiagnosis. Hypertension has been associated with 3% of the SSEH patients.²³ Frequently, anxiety at the time of presentation can increase their BP. In such a scenario, the first differential that physicians suspect is CVA. Patients with SSEH presenting with hemiparesis may also have dysarthria but the cause of it is not known.¹³ Elderly patients with SSEH may be using dentures and during the acute phase of disease when the dentures are removed, their speech might sound slurred.¹¹

Thrombolysis and embolectomy are the two available treatments that have proven to be effective after CVA.^{19 29} Thrombolysis is non-invasive method that can be instituted early while embolectomy is an invasive procedure that is employed after extensive workup. There is a narrow window for administration of a thrombolytic agent for a CVA and this is the only available non-invasive treatment that has proven to be effective.^{19 29} Thus, a NECT brain with or without CT angiography or CT perfusion is the investigation of choice in the acute emergency setting to detect intracranial haemorrhage or early ischaemic CVA. In most centres, an MRI brain takes a longer time to organise. Thus, thrombolytic treatment is commenced in the presence of clinical suspicion of stroke after ruling out haemorrhagic CVA on NECT even though an MRI is found superior to a CT scan in diagnosing

a cerebral infarct.³⁰ Hence, in situations when the window of opportunity for t-PA is closing, an MRI brain is usually done after the commencement of thrombolytic treatment. An alternate diagnosis is considered only when subsequent MRI brain or a CT angiogram turns out to be normal.

Two issues are of importance in patients with SSEH: (1) to prevent a delay in diagnosis and (2) to anticipate and avoid blood loss during subsequent surgery. A careful assessment of the patient in the emergency department is important. SSEH should be considered when patients with hemiparesis also have back or neck pain with/without radiculopathy.¹⁵ Other factors that point towards SSEH rather than CVA are the presence of a Brown-Sequard syndrome,^{4 10 12} Horner's syndrome,^{7 12} the absence of cranial nerve palsies or facial weakness,^{4 11 15} a normal MRI brain,¹⁵ no improvement or a deterioration of neurology after starting thrombolytic therapy and a positive Lhermitte's sign.³¹ After diagnosing SSEH, an urgent surgical evacuation is warranted unless the symptoms are mild and non-progressive. Non-operative management is feasible if spontaneous recovery is seen or if the neurological deficits are mild.^{1 3 25} However, haematoma evacuation is preferred in patients with profound deficit as there is inverse correlation between time to surgery and recovery.¹ A study by Shin *et al* with a series of 14 patients showed that 84% of patients recovered if operated within 12 hours while only 64% of them recovered if operated between 12 and 24 hours. Only 47% of those who were operated more than 24 hours later showed recovery.¹⁶ Surgical decompression that is delayed more than 12 hours in the presence of a profound neurological deficit may lead to permanent neurological disability.²⁵ Reversal agents and appropriate blood products should be made available so surgery is not delayed.³² Surgeries carried out immediately after t-PA infusions are rare and guidelines for these situations do not exist. The half-life of t-PA is approximately 5 min

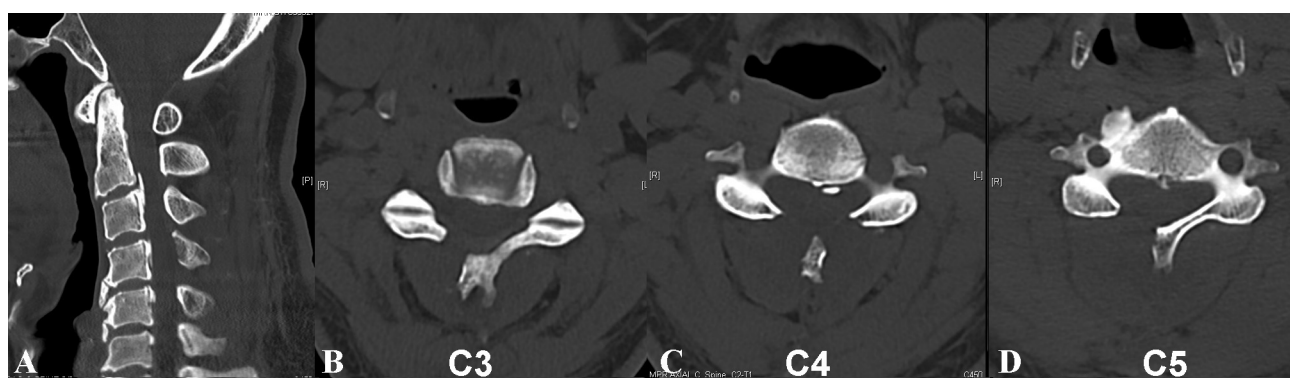


Figure 3 Postoperative CT scan showing (B) C3 right hemilaminectomy, (C) C4 laminectomy and (D) C5 right hemilaminectomy. The haematoma was decompressed through limited laminectomies preserving the spinous processes and posterior ligaments.

Table 2 Reported cases of SSEH that were misdiagnosed and treated as stroke

Author	Age	Sex	Presenting symptoms	Horner's syndrome	Cranial nerve palsy	Brown-Sequard syndrome	Treatment offered for misdiagnosis	Subsequent events	Timing of surgical intervention after the onset of symptoms	Outcome	Location
Morimoto <i>et al</i> ¹⁵	71	M	Left hemiparesis; neck pain	–	No	–	t-PA administered 2 hours after onset	Worsening of neurology, urinary retention	Was operated after 24 hours of t-PA	Good recovery	C4–C6
Tsukita <i>et al</i> ¹⁶	64	F	Right hemiparesis, neck pain	–	No	–	Oral aspirin for 3 days	No change for 3 days	Conservative	Complete recovery	C4–C6
Ueba <i>et al</i> ¹⁷	84	M	Left lower limb weakness	No	No	–	Antiplatelet therapy	Left shoulder pain and weakness of left upper limb extremity	Conservative	Good recovery	C3–C7
Yurter <i>et al</i> ¹³	69	F	Left hemiparesis	–	No	–	t-PA	Tetraparesis	Underwent C1–T1 decompression and C2–C7 posterior instrumented fusion same day	Complete recovery	C1–T1
Son <i>et al</i> ⁶	63	M	Left hemiparesis, neck pain, shoulder pain	–	No	–	Thrombolytic treatment started within 2 hours 30 min of onset	Stopped within 1 hour as MRI shows SSEH	Clotting screen was normal and was operated within 2 hours 45 min of t-PA	Incomplete recovery	C4–T2
Wang <i>et al</i> ⁴	69	M	Right hemiparesis with neck and shoulder pain	No	No	Yes	Heparin	Worsening of hemiparesis, new appearance of sensory deficits and exaggerated DTR	Was operated after 27 hours of onset	Complete	C4–C5
So <i>et al</i> ¹²	42	F	Left hemiparesis and neck pain	Yes	No	Yes	Aspirin	Urinary retention, Horner's syndrome and C6 BS	Laminoplasty done after 32 hours	Moderate disability	C4–C7
Tanaka <i>et al</i> ⁷	74	F	Left hemiparesis and neck pain	Yes	No	–	Argatroban	Newly presented Horner's syndrome	Conservative	Good recovery	C3–C4
Usami <i>et al</i> ⁸	80	M	Neck pain, right hemiparesis, right paraesthesia and speech disorder	No	No	–	Warfarin	Worsened to tetraparesis, articulation disorder improved after mouth care	Laminoplasty after 18 hours	Incomplete recovery; residual right hemiparesis	C2–T1
Kadota <i>et al</i> ⁹	74	F	Right hemiparesis, neck pain, shoulder pain, nausea	No	No	–	Not described	Worsening of hemiparesis and new sensory deficits	Laminoplasty after 4 days	Dead due to aspiration pneumonia	C6–C7
Hseih <i>et al</i> ¹⁴	65	M	Right hemiparesis, neck pain	No	No	Yes	Heparin 32 hours after onset	Nil	Laminectomy after 55 hours	Incomplete recovery	C4–C5
Adamson <i>et al</i> ¹⁰	66	M	Right hemiparesis, neck pain	–	No	Yes	Heparin	Worsening of hemiparesis and BS	FFP, protamine, dexamethasone	Good recovery	
Sakamoto <i>et al</i> ⁵	75	F	Neck pain, shoulder pain, right hemiparesis	–	No	–	Antiplatelet agent was started	Urinary retention developed within 12 hours of starting antiplatelet	FFP and vitamin K, operated on the same day	Complete recovery	C3–C4

FFP, fresh frozen plasma; SSEH, spontaneous spinal epidural haematoma; t-PA, tissue plasminogen activator; BS, Brown-Sequard syndrome; DTR, Deep Tendon Reflex.

but the fibrinolytic activity persists for 24 hours.³³ Adamson *et al* used fresh frozen plasma, protamine and dexamethasone before surgery,¹⁰ whereas Son *et al* operated on SSEH about 2 hours and 45 min after t-PA infusion without using any supplements as the coagulation profile remained normal.⁶ In our case report, the coagulation profile was normal. Hence, the patient could be managed without any supplementation of clotting factors. In an immediate surgery following t-PA infusion when the clotting factors are deranged, administration of fresh frozen plasma or cryoprecipitate can be used for effective haemostasis.³⁴

For patients presenting with hemiparesis, the emergency physicians and neurologists are under pressure to institute thrombolytic treatment within 3 hours as it is the only effective non-invasive treatment for ischaemic CVA. Hence, our suggestion to consider SSEH in a small subgroup may not always be justified. However, it is prudent to continue monitoring a patient on thrombolytic therapy and an urgent MRI cervical spine should be sought in those who deteriorate after thrombolysis or who present with atypical features of stroke. A proactive consideration of SSEH as a differential diagnosis for CVA is essential.¹¹ A high index of suspicion, prompt and appropriate investigation and immediate intervention can reduce morbidity when SSEH mimics CVA.

Learning points

- ▶ Spontaneous spinal epidural haematoma should be considered in patients presenting with hemiparesis and neck or back pain with or without radiculopathy.
- ▶ Epidural haematoma should be ruled out before initiating thrombolytic treatment.
- ▶ Presence of Horner's syndrome, Brown-Sequard syndrome, absence of cranial nerve palsies, positive Lhermitte's sign and a normal brain MRI are indicators of a spinal pathology in patients presenting with hemiparesis.

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