Case Report

Catastrophic spontaneous spinal epidural hematoma following thrombolysis: An intersection of neurosurgical and cardiological challenges – An institutional experience

ABSTRACT

Catastrophic spontaneous spinal epidural hematoma (SSEH) following thrombolysis poses a complex intersection of neurosurgical and cardiological challenges. This case report presents the institutional experience of a 66-year-old female who developed rapid-onset compressive myelopathy after thrombolysis for inferior wall myocardial infarction with injection streptokinase. SSEH, although rare, demands prompt recognition due to its potential for permanent neurologic injury and mortality. The discussion highlights the clinical significance, anatomical considerations, and multidisciplinary approach requisite for accurate diagnosis and effective management of SSEH. The conclusion underscores the necessity for clinicians, particularly cardiologists administering thrombolytic therapies, to consider SSEH in postthrombolysis patients presenting with neurological deficits.

Keywords: Compressive myelopathy, multidisciplinary approach, neurological symptoms, spontaneous spinal epidural hematoma, thrombolysis

INTRODUCTION

Spontaneous intraspinal hemorrhage is rare, marked by sudden back pain due to hematomas in epidural, subdural, or subarachnoid spaces, resulting in compression of the underlying neural elements. Intraspinal hemorrhages can also manifest within the cord parenchyma independently of trauma, vascular lesions, or tumors. Individuals with coagulopathy face an elevated risk of both nontraumatic and traumatic hemorrhage. Coagulopathy arising from primary hematologic disorders is prevalent, with 25%–30% of cases associated with anticoagulants.[1] Spontaneous spinal epidural hematoma (SSEH) is a dreaded complication following thrombolysis and can lead to permanent neurologic injury which in turn results in mortality. We are presenting a case of spontaneous long-segment posterior epidural hematoma following thrombolysis for inferior wall myocardial infarction (MI) with injection streptokinase, resulting in rapid onset compressive myelopathy with cord edema with subsequent neurological symptoms development.

Access this article online	
Website: www.jcvjs.com	Quick Response Code
DOI: 10.4103/jcvjs.jcvjs_62_24	

CASE REPORT

A 66-year-old female presented to the neurosurgery department with a recent onset of bilateral upper and lower limb weakness for 1 day. Two days before admission, the patient experienced chest pain and breathlessness at rest, prompting a visit to a private hospital where she was diagnosed with an inferior wall MI and atypical viral pneumonia. As a life-saving intervention, the patient

Tushar V. Soni, Shreyansh J. Patel, Varshesh K. Shah, Kavan M. Joshipura

Department of Neurosurgery, Smt. NHL Municipal Medical College, Ahmedabad, Gujarat, India

Address for correspondence: Dr. Shreyansh J. Patel, Department of Neurosurgery, Smt. NHL Municipal Medical College, Ahmedabad, Gujarat, India.
E-mail: shreyanshpatel1110@gmail.com

Submitted: 21-Apr-24 **Accepted:** 01-May-24
Published: 12-Sep-24

How to cite this article: Soni TV, Patel SJ, Shah VK, Joshipura KM. Catastrophic spontaneous spinal epidural hematoma following thrombolysis: An intersection of neurosurgical and cardiological challenges – An institutional experience. J Craniovert Jun Spine 2024;15:371-4.

underwent thrombolysis using an injection streptokinase on the day of chest pain. Following this cardiac intervention, the patient developed a sudden onset of bilateral upper and lower limb weakness, leading to referral to our institute for further neurosurgery and cardiology evaluation. The patient has a known history of hypertension and diabetes mellitus for the past 10 years, on regular treatment. On presentation, the patient was hemodynamically stable, with a pulse of 90 beats per minute, blood pressure measuring 150 / 90 mmHg, and oxygen saturation of 96% on room air. Neurologically, the patient was conscious, bilateral pupils measuring 3 mm reacting to light with decreased breath-holding time, and quadriplegia graded as 0 on the Medical Research Council (MRC) scale. The patient demonstrated ASIA Grade A and had a modified Rankin Scale score of 5. Bilateral plantar reflexes were mute. The diagnostic evaluation included a magnetic resonance imaging (MRI) of the cervico-dorsal spine [Figures 1 and 2] conducted at the previous hospital. The imaging revealed long segment epidural hematoma and cord compression of maximum thickness of 7 mm extending from C3 to D2. This collection resulted in indentation over the posterior surface of the cord, accompanied by long segment intramedullary signal intensity. On an emergency basis, the patient underwent C3 to D2 *en mass* laminectomy [Figure 3] with evacuation of the spinal epidural hematoma [Figure 4] and reposition of the lamina flap. Postoperatively, the patient was managed in the neurosurgical intensive care unit, receiving single antiplatelet therapy and injectable low-molecular-weight heparin. Despite immediate emergent intervention following presentation, postoperative neurological recovery remained partial, manifesting as MRC Grade 1 in bilateral upper limbs. Unfortunately, there was no discernible improvement in the MRC grade of bilateral lower limbs. This outcome is attributed to the extensive long-segment hematoma and the exceedingly rapid onset of neurological disability, as indicated by the preoperative poor ASIA grade.

DISCUSSION

In the present case, the patient experienced spontaneous long-segment posterior epidural hematoma following thrombolysis for inferior wall MI with injection streptokinase,

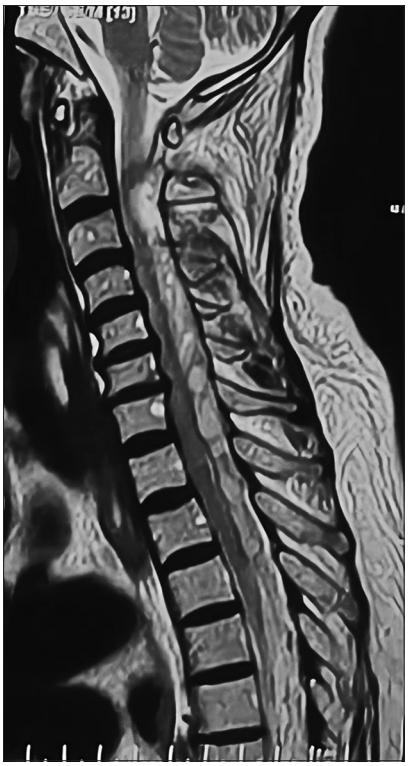

Figure 1: Magnetic resonance T2-weighted image of cervico-dorsal spine showing posterior spinal epidural hematoma

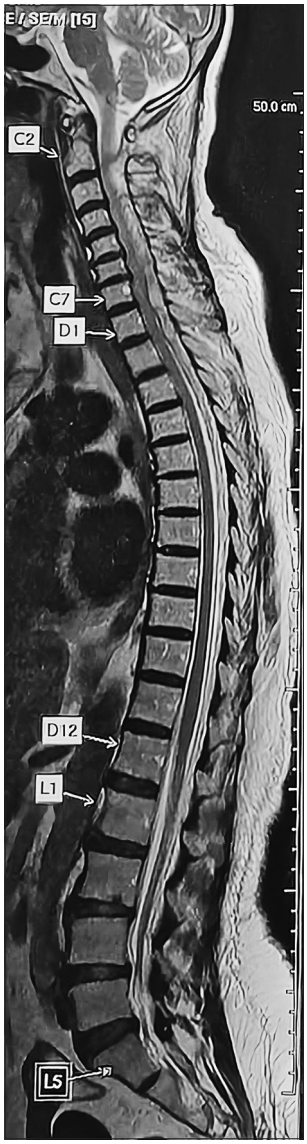

Figure 2: Magnetic resonance T2-weighted image of whole spine showing long segment posterior spinal epidural hematoma

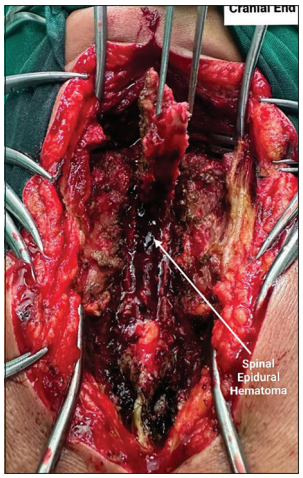

Figure 3: Intraoperative image showing (white arrow) C3 to D2 en-mass laminectomy with posterior spinal epidural hematoma before evacuation

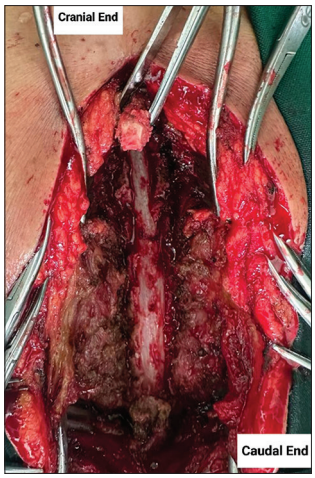

Figure 4: Intraoperative image showing postevacuation of spinal epidural hematoma

resulting in rapid onset compressive myelopathy with cord edema with subsequent neurological symptoms development. Thrombolytic and antithrombin agents are the mainstays of treatment in the management of acute coronary syndrome. The most common side effect of this agent is bleeding, out of which the most life-threatening is intracranial hemorrhage. SSEH is another dreaded complication following thrombolysis and can lead to permanent neurologic injury which in turn results in mortality. Spontaneous intraspinal hemorrhage is a rare event. This entity was first described by Blauby in 1808 according to Mayer.[2] Scholarly reviews of the subject were published in 1972 by Jellinger[3] and in 1976 by Bruyn and Bosma.[4] The spinal epidural space is truly an intradural space between two leaves of the spinal dura mater. The cranial dura mater splits into two laminae at the foramen magnum: the internal lamina forms the dural sac and the external lamina lines the inner wall of the bony spinal canal. The external lamina is clearly defined in infants but rarely distinguishable in adults. The space external to the internal lamina of the spinal dura mater (between the two laminae) is the epidural space by traditional usage. The anterior portion of the dural tube is closely approximated to the bony canal and is fixed to the posterior longitudinal ligament by connective tissue strands. The posterior spinal epidural space is filled with fatty tissue that extends laterally to surround the nerve roots. The posterior epidural space is thinnest in the thoracic region; it may be as wide as 14 mm in the lumbosacral area and 3–6 mm in the cervical region. Small arteries and a complex venous network traverse the epidural fat. Large-caliber longitudinal veins run in the anterolateral epidural space, anastomoses from side to side through an anterior and posterior transverse network, and contribute to the internal vertebral plexus, which anastomoses in turn with the external vertebral plexus. The epidural venous plexus anastomoses through the segmental veins with the inferior vena cava and azygos and hemiazygos veins. The internal lamina of the spinal dura mater is supplied by two types of arterial networks originating from lateral spinal arteries: a longitudinal-oriented network on the posterior surface, which is more prominent in the cervical and lumbar regions, and small vascular clusters in the thoracic region.[5] The initial back pain is localized at the level of the lesion, and radicular pain may occur simultaneously with the episode of bleeding or develop minutes to hours later. Signs of neural compression appear rapidly in most cases.[5] If left untreated, frequently complete and permanent neurological deficits can occur. The evaluation and treatment of patients with the acute onset of back pain and progressive neural dysfunction must proceed on an emergent basis. Recovery from spinal epidural hematoma is independent of age, although children seem to recover more readily than adults. Prognosis depends on the length of time between the first clinical symptoms and the onset of sensorimotor deficit: it worsens with the increasingly rapid development of cord dysfunction. The length of time between the onset of sensorimotor deficit and surgical decompression is critical. Recovery also varies directly with the severity of the neurological deficit at the time of decompression.[5] Useful motor recovery occurs in $<$50% of patients if paralysis is present for longer than 36 h.[6] The least favorable prognosis was in patients in whom the sensorimotor deficit developed rapidly and the clot extended over more than one segment in the thoracic region. The best outcome is in patients with lumbar hematomas.[7] A hallmark indicator of pretherapy neural deficit is the ASIA score. Across the 12 studies, it was observed that 30% of patients who presented with an ASIA score of A did not improve with surgery. However, every SSEH patient who presented with an ASIA score of C or D improved with surgery. This validates the necessity to collect an ASIA score immediately upon presentation of SSEH-like symptoms, as

it aids in developing a prognosis and determining the time and type of intervention needed.[8] Treatment consists of prompt evacuation of the clot and decompression of the dural tube, usually by laminectomy. Isolated cases of recovery without operation have been reported in patients having mild symptoms, presented late in their course, and were beginning to recover spontaneously.[9,10] The urgent nature of the clinical problem usually requires evacuation of the hematoma even if a vascular malformation is suspected; angiography is then performed electively during the postoperative period.[8] In view of the acute deterioration of neurological deficits and the emergent nature of the disease, it should be evaluated with spinal computed tomography (CT) scanning or MRI as soon as possible. On spinal CT scanning, SSEH can be viewed as an intraspinal biconvex and hyperdense lesion with a density equivalent to blood.[11,12] Spinal CT scanning, however, may be nondiagnostic in the thoracic spine where resolution is poorer than in the lumbar and cervical spine because of the high contrast between the lung parenchyma and vertebral bone.[13] Therefore, in the modern era, CT scanning has been replaced by MRI, which provides a noninvasive and clear view of the precise location of the lesion, its position, size, probable nature, and the degree of cord compression. On T1-weighted imaging, SSEH usually displays an isointense signal to the spinal cord within 24 h after symptom onset and a hyperintense signal to the cord after 36 h.[14,15]

CONCLUSION

SSEH represents an uncommon yet clinically significant condition. Thrombolytic agents, crucial in managing acute coronary syndrome, pose a risk of bleeding complications, with intracranial hemorrhage being a well-known life-threatening side effect. However, SSEH, although rare, is another serious complication that clinicians should be vigilant about, as it can lead to permanent neurologic injury and increased mortality. The discussion of the case emphasizes the anatomy of the spinal epidural space, the rarity of spontaneous intraspinal hemorrhage, and the critical importance of prompt evaluation and intervention in cases of acute onset back pain and progressive neural dysfunction. The prognosis of SSEH depends on various factors, including the duration between symptom onset and decompression, the severity of neurological deficit, the level of the hematoma, and on presentation ASIA score. Treatment primarily involves surgical evacuation of the hematoma and decompression of the dural tube, usually through laminectomy. While there have been isolated cases of spontaneous recovery without surgery, the urgent nature of SSEH typically necessitates prompt intervention. The conclusion underscores the need for physicians, particularly cardiologists dealing with thrombolytic therapies, to consider SSEH as a differential diagnosis in patients presenting with hemiparesis or quadriparesis postthrombolysis. To conclude, this case report contributes to the understanding of SSEH and emphasizes its complexity, necessitating a multidisciplinary approach for accurate diagnosis and effective management.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient (s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initial s will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

REFERENCES

1. Zuccarello M, Scanarini M, D'Avella D, Andrioli GC, Gerosa M. Spontaneous spinal extradural hematoma during anticoagulant therapy. Surg Neurol 1980;14:411-3.
2. Mayer JA. Extradural spinal hemorrhage. Can Med Assoc J 1963;89:1034-7.
3. Jellinger K. Traumatic vascular disease of the spinal cord. In: Vinken PJ, Bruyn GW, editors. Handbook of Clinical Neurology. Vascular Diseases of the Nervous System. Vol. 12. Part II. Amsterdam: North-Holland; 1972. p. 556-630.
4. Bruyn GW, Bosma NJ. Spinal extradural haematoma. In: Vinken PJ, Bruyn GW, editors. Handbook of Clinical Neurology. Vol. 26. Amsterdam: North-Holland; 1976. p. 1-30.
5. Wilkins RH, Rengachary SS. Neurosurgery. 2nd ed., Vol. 2. New York: McGraw-Hill, Health Professions Division; 1996. p. 2559-65.
6. McQuarrie IG. Recovery from paraplegia caused by spontaneous spinal epidural hematoma. Neurology 1978;28:224-8.
7. Mattle H, Sieb JP, Rohner M, Mumenthaler M. Nontraumatic spinal epidural and subdural hematomas. Neurology 1987;37:1351-6.
8. Raasck K, Habis AA, Aoude A, Simões L, Barros F, Reindl R, *et al.* Spontaneous spinal epidural hematoma management: A case series and literature review. Spinal Cord Ser Cases 2017;3:16043.
9. Dawson BH. Paraplegia due to spinal epidural haematoma. J Neurol Neurosurg Psychiatry 1963;26:171-3.
10. Pear BL. Spinal epidural hematoma. Am J Roentgenol Radium Ther Nucl Med 1972;115:155-64.
11. Beatty RM, Winston KR. Spontaneous cervical epidural hematoma. A consideration of etiology. J Neurosurg 1984;61:143-8.
12. Post MJ, Seminer DS, Quencer RM. CT diagnosis of spinal epidural hematoma. AJNR Am J Neuroradiol 1982;3:190-2.
13. Avrahami E, Tadmor R, Ram Z, Feibel M, Itzhak Y. MR demonstration of spontaneous acute epidural hematoma of the thoracic spine. Neuroradiology 1989;31:89-92.
14. Boukobza M, Guichard JP, Boissonet M, George B, Reizine D, Gelbert F, *et al*. Spinal epidural haematoma: Report of 11 cases and review of the literature. Neuroradiology 1994;36:456-9.
15. Chen CJ, Hsu WC. Imaging findings of spontaneous spinal epidural hematoma. J Formos Med Assoc 1997;96:283-7.