

Case Report

Acute Spinal Subdural Hematoma Presenting with Spontaneously Resolving Hemiplegia

Seung-Hun Oh, M.D.,¹ In-Bo Han, M.D.,² Young-Ho Koo, M.D.,¹ Ok-Joon Kim, M.D.¹

Department of Neurology,¹ and Neurosurgery,² CHA Bundang Medical Center, CHA University College of Medicine, Seongnam, Korea

Although prompt diagnosis and emergent surgical intervention are important in acute spinal subdural hematoma (SSDH), some cases with spontaneous remission of symptom and hematoma without surgery have been reported. We present a case of acute nontraumatic SSDH presenting with transient left hemiplegia for 4 hours. A magnetic resonance imaging study of cervical spine confirmed SSDH with C3-6 cervical cord compression at the left side. The patient had conservative management without recurrence. Although hemiplegia is an unusual clinical manifestation of SSDH, it should be differentiated from that of cerebrovascular origin promptly. Conservative management may be an alternative therapeutic option for selective cases with transient neurological deficits.

KEY WORDS : Spinal subdural hematoma · Hemiplegia · Cervical.

INTRODUCTION

Acute spinal subdural hematoma (SSDH) is a rare spinal vascular disorder causing compression of the spinal cord or cauda equina. The cause of SSDH may be spontaneous or iatrogenic (lumbar or cervical spine puncture, spinal surgery, trauma), and most patients are frequently associated with underlying coagulopathy or those receiving anticoagulant or antiplatelet agent¹⁸. A typical clinical presentations are sudden onset of spinal or radicular pain, and myelopathy such as paraplegia or sensory level. However, unusual manifestations such as pure hemiparesis may lead to misdiagnosis initially, delaying the timing of treatment in some cases with spinal hematoma of subdural or epidural origin^{4,7,15}. Neurological deficits due to cord compression by hematoma are known to be a neurosurgical emergency. However, some cases with spontaneous remission of symptoms have also been reported, and conservative management could be an option in some cases with mild or transient neurological deficits^{5,8,10,12}.

We present a case of acute SSDH with hemiplegia, which was rapidly improved spontaneously. His symptom initially

was misdiagnosed as cerebrovascular accident (CVA).

CASE REPORT

A 59-year-old woman was admitted due to sudden onset of neck pain and motor weakness in the left arm and leg for 3 hours. The patient had anti-hypertensive and anti-hyperlipidemic medication for 6 years without previous history of trauma or bleeding diathesis. Neurological examination revealed motor weakness of MRC grade 2 in proximal and 3 in distal left arm, and grade 4 in proximal and distal left leg, respectively. She complained of mild neck pain radiating to left arm but no sensory abnormalities including touch, pain and vibration were found. Her mental status and cognitive functions were normal. The cranial nerve examination disclosed no abnormalities. Cerebellar functions were intact in the right side, and equivocal in the left side due to weakness. Deep tendon reflexes were decreased at left side but no Babinski sign were noted. Fortunately, her motor weakness and cervical pain were resolved spontaneously within 4 hours after symptom onset.

The brain computed tomography (CT) scan on admission was not remarkable. Tentative diagnosis of CVA including transient ischemic attack (TIA) due to vertebral arterial (VA) dissection was initially made. Immediate intravenous (IV) heparin was administrated. Brain magnetic resonance image (MRI) including diffusion-weight image on 12 hours

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• Address for reprints : Ok-Joon Kim, M.D.

Department of Neurology, CHA Bundang Medical Center, CHA University, 351 Yatap-dong, Bundang-gu, Seongnam 463-712, Korea
Tel : +82-31-780-5481, Fax : +82-31-780-5269
E-mail : okjun77@cha.ac.kr

after symptom onset confirmed no cerebral lesion. Brain MR angiography showed no stenotic lesion in extra- and intracranial artery. Based on the transient cervical pain and negative radiological finding of brain, cervical myelopathy was then suspected. The cervical MRI on 20 hours after symptom onset showed mass-like lesion at the dorsal portion of spinal cord extending from C3 down to C6 level longitudinally, with compression of the posterolateral portion of spinal cord at left side (Fig. 1A). The lesion was hypointense on T2-weighted image and isointense on T1-weighted image (Fig. 1B), suggesting acute stage of hemorrhage. On T1-weighted image after Gadolinium injection, the lesion was not enhanced, but there were enhancement in outer dura and inner arachnoid membrane (Fig. 1C). The lesion was

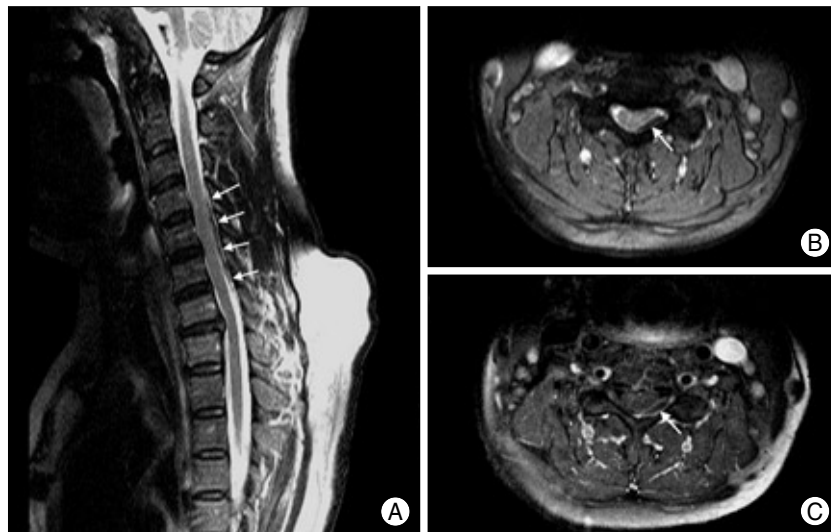


Fig. 1. Cervical magnetic resonance image on 20 hours after symptom onset. Sagittal T2-weighted image shows longitudinal mass-like lesion at the level of C3-C6 segments (A). The lesion is hypointense on T2-weighted image, compressing spinal cord (B). The non-enhanced isointense lesion on T1-weighted image is located between the enhanced dural and arachnoid membrane after Gadolinium injection (C).

located at the extramedullary and intradural space, indicating that the lesion was of subdural origin. All the laboratory studies, including complete blood counts and coagulation screen (platelet : 237,000/uL, prothrombin time : 103 %, activated partial thromboplastin (aPTT) time : 30 sec, protein C : 109%, protein S : 79%, D-dimer : 337 ng/mL, fibrinogen : 338 mg/dL, FDP : 40 ug/mL, antithrombin III : 92%), autoimmune test (rheumatoid factor, anti-nuclear antibody, anti-ds DNA antibody, lupus anticoagulant, anticardiolipin IgG) were negative.

After confirmatory diagnosis of acute SSDH was made, heparin was stopped immediately and conservative management was performed. The maximal aPTT was 54.3 second during IV heparin treatment. She was free of symptom, and discharged on 7 days after symptom onset. The neurological condition remained stable for 1 year, and the patient refused repeated MRI evaluation.

DISCUSSION

Our case presented spontaneous acute cervical SSDH confirmed by MRI. The most common site of SSDH is in the thoracolumbar (TL) or lumbar segment (L). The SSDH at cervical (C) or cervicothoracic (CT)- SSDH was rare. In meta-analysis of 106 cases with non-traumatic SSDH, there were only 12 cases with C- and CT-SSDH¹⁾. Demographic and clinical characteristics of 12 cases with C/CT-SSDH^{2,9,11,13,14,16,17,19-23)} in literature are

Table 1. Demographical and clinical characteristics of previously reported cases with non-traumatic SSDH at the cervical or cervicothoracic level

Case no.	Sex/Age	Level	Cause		Symptoms	Surgery	SAH	Outcome
			Underlying factor	Spinal tap				
1 ⁹⁾	M/47	C3-7	-	-	CP, QP, SL	+	+	Poor
2 ¹⁶⁾	M/57	C4-8	-	-	QP, SL	-	-	Death
3 ¹¹⁾	M/15	Oc-C4	Leukemia, coagulopathy	Cervical	QP, respiratory distress	+	-	Good
4 ¹⁷⁾	M/57	C	-	Cervical	Coma, decerebrate rigidity	-	-	Death
5 ¹³⁾	F/51	C5-T2	Anticoagulation	Lumbar	QP, BD	+	-	Good
6 ²¹⁾	F/59	C7-T2	Anticoagulation, cord tumor	-	CP, QP, SL, BD	+	-	Poor
7 ²⁰⁾	F/74	C4-T1	Cord tumor	-	CP, QP, SL, BD	+	-	Poor
8 ²³⁾	M/1	C3-T9	Cystic fibrosis	-	QP, SL, BD	+	+	Poor
9 ⁹⁾	F/55	C6-T4	Anticoagulation	-	QP, SL, BD	+	+	Poor
10 ¹⁴⁾	M/59	CT	Anticoagulation	-	CP, QP, SL, BP	-	-	Good
11 ²²⁾	M/68	C5-7	Cord tumor	-	CP, QP, BD	+	-	Good
12 ⁹⁾	F/59	C6-T6	Aneurysm of radicular artery	-	CP, paraplegia	+	+	Good

C : cervical, T : thoracic, CT : cervico-thoracic, Oc : occipital, CP : cervical pain, QP : quadriplegia, SL : sensory level, BD : bladder dysfunction, + : present, - : absent, SAH : subarachnoid hemorrhage

summarized in Table 1. Most of them were adult onset (above 50 years old), and about half of patients had predisposing factors including coagulopathy or history of spinal tap. Initial motor weakness was profound enough to perform emergent surgery in most cases. Most patients usually presented with spinal or radicular pain followed by myelopathy including motor, sensory and autonomic dysfunctions. The most common form of motor symptom was quadriplegia (Table 1). Comparing with previously reported cases with C/CT- SSDH, our case is unique since the symptom presented with hemiparesis and the symptom disappeared very quickly (within 4 hours). Unilateral involvement of motor or sensory disturbance is extremely rare in SSDH, therefore being initially misdiagnosed as CVA⁷. In this case, transient unilateral motor weakness and absence of sensory or autonomic dysfunction made it difficult to diagnose the SSDH immediately, and led us to misdiagnose as transient ischemic attack (TIA) due to vertebral artery dissection. We cannot totally exclude the possibility that neurological deficit of our case was from TIA, and asymptomatic SSDH after IV anticoagulation was co-incidentally found irrespective of symptoms. However, the findings of normal brain imaging study, and positive relationship between clinical and radiological findings of cervical cord indicate that symptoms resulted from the cervical origin itself. Furthermore, our case showed no further deterioration of symptom during IV anticoagulation. This finding suggests that IV anticoagulation appears not to be directly related to hematoma formation. Although anticoagulation did not affect the deterioration of symptom in this case, it could lead to rapid expansion of hematoma with complete cord compression and deterioration of clinical symptom in some cases with SSDH⁴. Therefore, the physicians should keep in mind that SSDH should be promptly differentiated from CVA in a case presenting with unilateral hemiplegia. Prompt cervical neuroimaging study is necessary if the result of brain neuroimaging study is negative.

Emergent surgical decompression is a treatment of choice for acute SSDH. In meta-analysis by Domecucci, et al., the rate of poor outcome (58% vs. 58%) and mortality rate (17% vs. 20%) between C/CT- and TL/L- SSDH were similar, indicating that lesion location itself is not a critical parameter for prediction of outcome¹. It is generally accepted that prompt diagnosis and emergent surgical treatment is the most important prognostic factor. However, there were several previous reports regarding spontaneous resolution of neurological deficits, who had mild neurological symptoms or rapid improvement of disable symptoms during clinical course^{1,5,8,10,12}. Most of them showed clinical improvement at least 2 days after symptom onset. The exact

mechanism of spontaneous resolution of symptom is unclear. Several factors including small volume of hematoma, prompt cease of active bleeding, flexibility of spinal cord, and appropriate width of spinal canal may influence on the recovery of the symptom. Rapid redistribution of hematoma with rostrocaudal expansion within subdural space may avoid the transverse expansion of hematoma, subsequently reducing the degree of cord compression. Another possible mechanism was recently suggested that redistribution of SSDH could occur due to the rapid dilution of hematoma intermingled with subarachnoidal cerebrospinal fluid (CSF) in the presence of arachnoidal tearing¹². Unlike the intracranial subdural space, the spinal subdural space is an avascular space,³ thereby assuming that hemorrhage in subdural space might come from subarachnoid source. The finding of accompanying subarachnoid hemorrhage (SAH) in several cases with SSDH supports this hypothesis (Table 1). Hemorrhage from the subarachnoid space extends to the subdural space in the situation of rupture of arachnoid membrane, and isolated SSDH may remain after a more rapid resolution of SAH by dilution with CSF^{6,12}. In this situation, the direction of the movement of hemorrhage might be reversed, namely from subdural to subarachnoid space. In our case, although hematoma still compressed spinal cord on cervical MRI on 1 day after symptom onset, no symptom recurred over the following 1 year, allowing us to perform only observation without surgery.

CONCLUSION

We report an unusual case with acute SSDH presenting with transient hemiplegia. We suggest that acute SSDH should be considered even in patients with hemiplegia, being commonly misdiagnosed as CVA. If neurological symptoms are transient, conservative management may be an alternative therapeutic option in selective patients.

References

1. Domenicucci M, Ramieri A, Ciappetta P, Delfini R : Nontraumatic acute spinal subdural hematoma : report of five cases and review of the literature. *J Neurosurg* 91 : 65-73, 1999
2. Gabl M, Kostron H : Acute spinal subdural haematoma. *Neurochirurgia (Stuttgart)* 31 : 99-100, 1988
3. Gillilan LA : Veins of the spinal cord. Anatomic details; suggested clinical applications. *Neurology* 20 : 860-868, 1970
4. Hsieh CF, Lin HJ, Chen KT, Foo NP, Te AL : Acute spontaneous cervical spinal epidural hematoma with hemiparesis as the initial presentation. *Eur J Emerg Med* 13 : 36-38, 2006
5. Kang HS, Chung CK, Kim HJ : Spontaneous spinal subdural hematoma with spontaneous resolution. *Spinal Cord* 38 : 192-196, 2000
6. Kirkpatrick D, Goodman SJ : Combined subarachnoid and subdural spinal hematoma following spinal puncture. *Surg Neurol* 3 : 109-

- 111, 1975
7. Küker W, Thiex R, Friese S, Freudenstein D, Reinges MH, Ernemann U, et al : Spinal subdural and epidural haematomas: diagnostic and therapeutic aspects in acute and subacute cases. *Acta Neurochir (Wien)* 142 : 777-785, 2000
 8. Kulkarni AV, Willinsky RA, Gray T, Cusimano MD : Serial magnetic resonance imaging findings for a spontaneously resolving spinal subdural hematoma : case report. *Neurosurgery* 42 : 398-400; discussion 400-401, 1998
 9. Langmayr JJ, Ortler M, Dessl A, Twerdy K, Aichner F, Felber S : Management of spontaneous extramedullary spinal hematomas : results in eight patients after MRI diagnosis and surgical decompression. *J Neurol Neurosurg Psychiatry* 59 : 442-447, 1995
 10. Maeda M, Mochida J, Toh E, Nishimura K, Nomura T : Nonsurgical treatment of an upper thoracic spinal subdural hemorrhage. *Spinal Cord* 39 : 657-661, 2001
 11. Mapstone TB, Rekatte HL, Shurin SB : Quadriplegia secondary to hematoma after lateral C-1, C-2 puncture in a leukemic child. *Neurosurgery* 12 : 230-231, 1983
 12. Mavroudakos N, Levivier M, Rodesch G : Central cord syndrome due to a spontaneously regressive spinal subdural hematoma. *Neurology* 40 : 1306-1308, 1990
 13. Messer HD, Forshan VR, Brust JC, Hughes JE : Transient paraplegia from hematoma after lumbar puncture. A consequence of anticoagulant therapy. *JAMA* 235 : 529-530, 1976
 14. Nighoghossian N, Ruel JH, Ffrench P, Froment JC, Trouillas P : [Cervicodorsal subdural hematoma caused by coumarinic rodenticide poisoning.] *Rev Neurol* 146 : 221-223, 1990
 15. Phookan G, Lehman RA, Kuhlengel KR : Cervical spinal epidural haematoma: the double jeopardy. *Ann Med* 28 : 407-411, 1996
 16. Reynolds AF Jr, Turner PT : Spinal subdural hematoma. *Rocky Mt Med J* 75 : 199-200, 1978
 17. Rogers LA : Acute subdural hematoma and death following lateral cervical spinal puncture. Case report. *J Neurosurg* 58 : 284-286, 1983
 18. Russell NA, Benoit BG : Spinal subdural hematoma. A review. *Surg Neurol* 20 : 133-137, 1983
 19. Schwartz FT, Sartawi MA, Fox JL : Unusual hematomas outside the spinal cord. Report of two cases. *J Neurosurg* 39 : 249-251, 1973
 20. Smith RA : Spinal subdural hematoma, neurilemmoma, and acute transverse myelopathy. *Surg Neurol* 23 : 367-370, 1985
 21. Toledo E, Shalit MN, Segal R : Spinal subdural hematoma associated with anticoagulant therapy in a patient with spinal meningioma. *Neurosurgery* 8 : 600-603, 1981
 22. Vázquez-Barquero A, Pascual J, Quintana F, Figols J, Izquierdo JM : Cervical schwannoma presenting as a spinal subdural haematoma. *Br J Neurosurg* 8 : 739-741, 1994
 23. Zochodne D, Hinton G, Del Maestro R, Coates R, Ecclestone E : Intradural spinal hematoma in an infant with cystic fibrosis. *Pediatr Neurol* 2 : 311-313, 1986