

Spinal Subdural Hematoma : A Complication of Intracranial Surgery

Tae-Wan Kim, M.D., Weon Heo, M.D., Hwa-Seung Park, M.D., Dong-Youl Rhee, M.D.

Department of Neurosurgery, Wallace Memorial Baptist Hospital, Busan, Korea

Spinal subdural hematoma(SSDH) is rare disease. Furthermore, it rarely occurs as a complication of intracranial surgery. There are few case reports which describing SSDH after craniotomy. Although the exact pathogenetic mechanism is obscure, some investigators propose that downward migration of intracranial hematoma by the effect of gravity is one of the cause of SSDH, and which is commonly suggested. But others propose that cerebrospinal fluid(CSF) hypotension is an another possible mechanism. In this paper, we report two cases of SSDH after clipping of an aneurysmal neck.

KEY WORDS : Spinal subdural hematoma · Aneurysmal clipping · Cerebrospinal fluid hypotension.

Introduction

Spinal subdural hematoma(SSDH) is not frequently seen in clinical practice. But the incidence of SSDH have been gradually increased due to accelerating rates of spinal trauma, anticoagulants therapy and diagnostic use of magnetic resonance image(MRI). It can be caused by several well-known precipitating factors, including coagulation abnormality, use of anticoagulants and iatrogenic causes such as lumbar puncture or spinal anesthesia and etc. It may also occur as a complication of intracranial surgery, but the incidence is extremely rare.

We report two cases of SSDH after clipping of an aneurysmal neck which have no clearly identifiable predisposing factors, and discuss the possible mechanism of SSDH following craniotomy.

Case Report

Case 1

A 44-year-old male visited our emergency room due to severe headache. Neurologic and physical examination were normal except mild neck stiffness. Computed tomography(CT) of brain showed subarachnoid hemorrhage(SAH), and right middle cerebral artery(MCA) aneurysm was identified on cerebral angiography. Perioperative routine laboratory analyses,

including platelet count, prothrombin time(PT), partial prothrombin time(PTT) and international normalized ratio (INR) were normal. We performed an emergency operation with clipping of aneurysmal neck through pterional approach. Postoperative neurological findings were normal. We recommended early ambulation from 4th postoperative day(POD). From 11th POD, he complaint low back pain without radiating leg pain. But neurologic examination was normal. In spite of medication, low back pain was more aggravated. MRI of the lumbar spine was checked at 16th POD, a subdural hematoma(SDH) was detected on the low lumbar area. There was no vascular anomaly in the whole spinal cord. Signal intensity of hematoma was high on T1-weighted image(T1WI) and slightly low on T2-weighted image(T2WI). As neurologic symptoms produced by a hematoma were absent, we thought that surgical evacuation of the hematoma is not necessary. The patient recovered completely after conservative treatment and discharged at 26th POD without any neurologic deficit.

Case 2

A 67-year-old male admitted for evaluation of a vascular anomaly which was incidentally detected. Neurologic and physical examination were normal. Brain CT showed normal, and right MCA aneurysm was identified on cerebral angiography. Perioperative routine laboratory analyses were normal. We perf-

• Received : May 3, 2005 • Accepted : July 26, 2005

• Address for reprints : Dong-Youl Rhee, M.D., Department of Neurosurgery, Wallace Memorial Baptist Hospital, 374-75 Namsan-dong, Geumjeong-gu, Busan 609-340, Korea Tel : +82-51-580-1281, Fax : +82-51-582-6682, E-mail : rdy@wmbh.co.kr

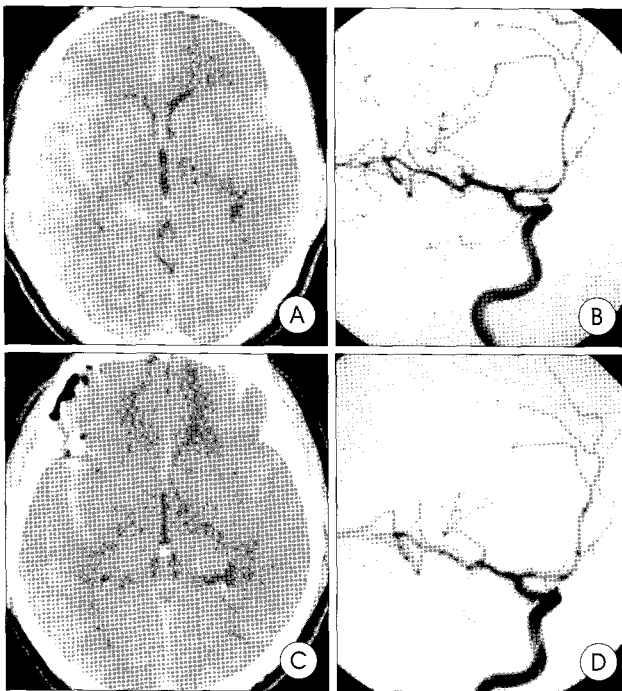


Fig. 1. Perioperative evaluations. A : Preoperative computed tomography scan of brain shows a interhemispheric and right sylvian subarachnoid hemorrhage secondary to a right middle cerebral artery(MCA) bifurcation aneurysm rupture. B : Preoperative cerebral angiography shows right MCA bifurcation aneurysm. C : Postoperative computed tomography scan of brain shows a aneurysm clip, and remained interhemispheric and right sylvian subarachnoid hemorrhage. D : Postoperative cerebral angiography shows no remnant aneurysm sac on right MCA bifurcation.

formed an operation with clipping of aneurysmal neck through pterional approach. There was no intraoperative rupture of aneurysm or excessive bleeding. Postoperative neurological findings were normal. Also, there was no intracranial hematoma on postoperative brain CT. We recommended early ambulation from 4th POD. From 16th POD, patient complaint low back pain without radiating leg pain. Neurologic examination was normal. MRI of the lumbar spine was checked at 17th POD, a SSDH was detected on the low lumbar area. Also, there was no vascular anomaly in the whole spinal cord. Signal intensity of hematoma was high on T1WI and slightly low on T2WI. The patient recovered completely after conservative treatment only and discharged at 24th POD without any neurologic deficit.

Discussion

Although the SSDH has various causative factors^{2,5)}, including a defect in hemostatic mechanism (such as coagulopathy or use of anticoagulants), history of spinal puncture, intraspinal tumor, vascular anomaly, trauma, certain conditions (such as toxemia of pregnancy, diabetes mellitus and hemodialysis), it is usually associated with bleeding diathesis and

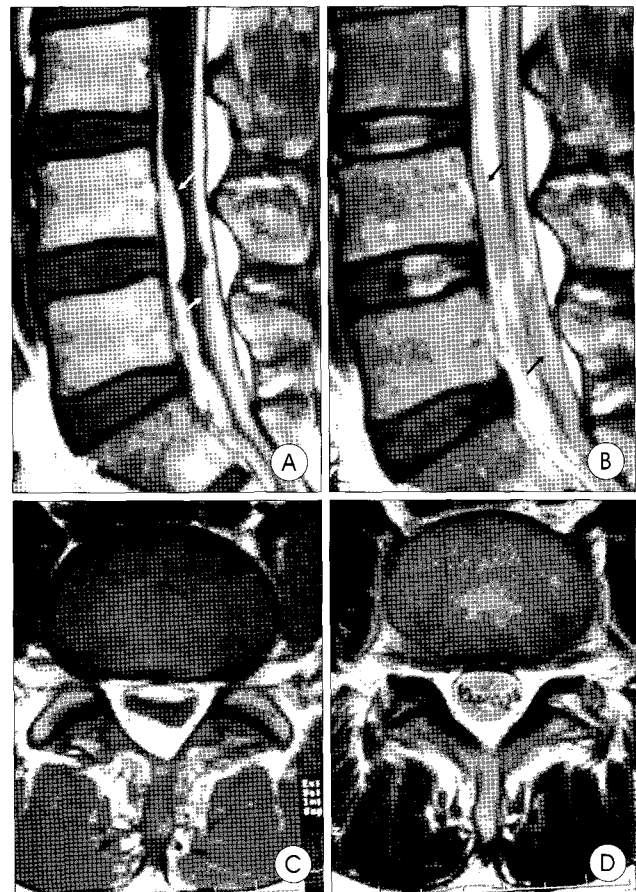


Fig. 2. Lumbar spinal magnetic resonance image shows spinal subdural hemorrhage of early subacute stage. A : Sagittal T1-weighted image shows high signal intensity in spinal subdural space (white arrows). B : Sagittal T2-weighted image shows low signal intensity in spinal subdural space (black arrows). C : Axial T1-weighted image shows high signal intensity in spinal subdural space. D : Axial T2-weighted image shows low signal intensity in spinal subdural space (white arrows indicates arachnoid membrane).

iatrogenic causes such as spinal puncture. Domenicucci et al²⁾ reviewed 106 cases of nontraumatic acute SSDH and demonstrated that there was a defect in hemostatic mechanism such as coagulopathy or use of anticoagulants in 54% of the cases, and history of spinal puncture in 47%. In 15% of the cases, there was no apparent cause. On the other hand, SSDH may occur as a complication of intracranial surgery. Until now, only 14 cases of SSDH-through 9 paper reports-were reported^{5,6,8-10)}.

It is difficult to determine the source of blood in SSDH. Some authors¹⁾ suggest that spontaneous SSDH is the isolated result of subarachnoid hemorrhage. Spinal subdural space is a potential avascular space. Thus, SSDH may come from an extradural or subarachnoid source. Indeed, just beneath the dura mater, spinal subdural space has small anastomosing network of extremely delicate vessels, which directed longitudinally along the lateral margins of the dura mater. But their size is so small as to makes them as a source of hemorrhage. Within the subarachnoid space, the only substantial vessels

are the major radiculomedullary artery of Adamkiewicz and its corresponding vein. Rader⁷⁾ proposed that spinal subdural hemorrhage resulted from an indirect application of force to the intraspinal vessels. A sudden great increase in abdominal and thoracic pressure can increase the intravascular pressure of vessels in the spinal subdural and subarachnoid spaces. This force would not be neutralized by a simultaneous increase in spinal fluid pressure because of shielding effect of the spinal column and its ligaments. During a extremely short interval, therefore, there will be such a great momentary disparity between intravascular and extravascular pressure that can rupture these vessels⁷⁾. At first, hemorrhages developed from these vessels are subarachnoid hemorrhage. The diluting and redistributing effect of CSF usually prevents subarachnoid blood from clotting. If bleeding is massive or if there is anatomic block of normal CSF flow, the hemorrhage can form the hematoma. In turn, the blood mass ruptures through the thin and brittle arachnoid into the subdural space. The blood then dilutes and redistributes from the CSF in the subarachnoid space, leaving the isolated SDH¹⁾.

The exact pathogenesis and the source of blood in SSDH after intracranial surgery is obscure. Some authors⁶⁾ hypothesize that the CSF hypotension is one of the possible pathogenetic mechanism. Wurm et al⁹⁾ suggested that SSDH which originated independently from intracranial subdural blood was caused by lower pressure in the whole craniospinal subdural area after overdrainage of CSF. During and after intracranial surgery, it is possible that low CSF pressure which was induced by various causes could predispose the tearing of bridging veins of intracranial space. In that case, decreased CSF pressure may predispose the development of a intracranial subdural hematoma(SDH). However, in CSF hypovolemia syndrome, the pattern of spinal abnormalities differs from that which was seen in intracranial cavity for anatomical reasons. In the spinal canal, dura is not adherent to the bone, and therefore collapse of dural sac and dilatation of the epidural venous plexus could occur rather than SDH⁵⁾.

Recently, most of authors⁵⁻¹⁰⁾ proposed that the downward migration of the blood from the intracranial subdural space into that of spine under the influence of gravity is the most reasonable mechanism of the SSDH. This hypothesis could be supported by the various factors, including existence of postoperative SDH, anatomical loc-

ation of hematoma and signal intensity of hematoma^{6,8-10)}. In most cases, postoperative SDH had occurred and hematoma was present consistently in the lower part of spinal canal, and signal intensity of hematoma on MRI suggested the subacute or chronic stage. This means that the hematoma is liquefied form and it can migrate through the potential subdural space. It also means that the hematoma was not from de novo bleeding in the spinal canal which could result in acute blood clot. The continuity between the intracranial subdural space and that of spine has been proven⁵⁾. Haines et al³⁾ examined the dura-arachnoid junction at the electron microscopic level and demonstrated that although the external dural layer is strong, the inner dural or meningeal dural layer is structurally weak and vulnerable to injury. Under normal conditions, there is no space at the dura-arachnoid junction. When the pathologic or traumatic process does happen, the space is identifiable. So it is called "potential space" subsequently.

In the review of our cases, there were no defect in hemostatic mechanism, no use of anticoagulants, no history of spinal puncture or epidural block, no intraspinal tumor, no spinal vascular anomaly, no history of trauma, and no other causative conditions such as diabetes mellitus or hemodialysis. There were no recognizable SDH during perioperative period. And also there was no other form of bleeding intraoperatively and postoperatively. Furthermore, in case 2, we operated unruptured aneurysm. Thus, we think that it is unreasonable that downward migration of hematoma by gravity assume the only pathogenetic mechanism. So, we hypothesize that both of the CSF hypotension and the downward migration under the influence of gravity take the part together in forming the SSDH. During the intracranial operation, although we don't want it to happen, overdrainage of CSF may induce excessive shrinkage

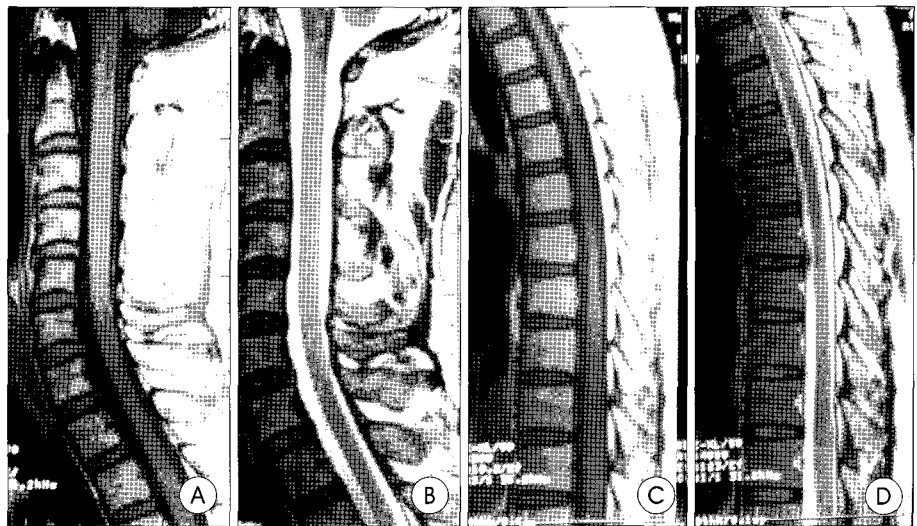


Fig. 3. Cervical spinal magnetic resonance(MR) image of sagittal T1-weighted image (A) and T2-weighted image (B), thoracic spinal MR image of sagittal T1-weighted image (C) and T2-weighted image (D) show no specific lesion in spinal cord.

of brain. Shrunken brain may open the “potential space” and leads fluid to collect in this space. If fluid collection occurs, it moves downward by gravity along the spinal potential space⁴. This process may damage small anastomosing network of extremely delicate spinal dural vessels, and finally symptomatic hematoma may be formed.

Since the first MRI findings of SSDH were reported in 1990, MRI is the best way to diagnosis and follow-up. It provides better visualization of hematoma compared with CT, and is also helpful to decide therapeutic modality by differentiation of SSDH from other spinal lesions^{2,5}

The incidence of spinal subdural hematoma(SSDH) is extremely rare, however it does occur, it may lead to irreversible neurologic damage in a short time without proper management. So most acute spinal subdural hematomas are a neurosurgical emergency that needs a prompt decompressive laminectomy, but some cases of spontaneous resolution have been reported^{5,6,8,10}. In case of acute deterioration and severe neurological deficit, emergent surgical decompression is the best treatment option. Conservative management may be indicated when symptoms improve quickly during the acute phase with treatment of spinal edema and the severity of initial neurologic symptom is grade D or better on the Frankel's grading scale, the degree of extension precludes surgical treatment or if associated with coagulopathy⁵. Lumbar spinal puncture and drainage of hematoma is another therapeutic modality. But it does not seem to contribute to the outcome⁶. Because initial neurological state is grade E on the Frankel's grading scale in all our two cases, and they showed good response to medication, we achieved excellent result with conservative management.

Conclusion

Because most spinal subdural hematomas progress rapidly and their prognosis are poor, the disastrous result of delayed diagnosis and treatment warrant recognition of this disease entity and a high index of suspicion in any case with similar symptom. Although the exact pathogenetic mechanism and prevalence is not certain, neurosurgeon should consider the possibility of SSDH in a patient in whom back pain develops after intracranial surgery.

References

1. Calhoun JM, Boop F : Spontaneous spinal subdural hematoma : case report and review of the literature. *Neurosurgery* 29 : 133-134, 1991
2. Domenicucci M, Ramieri A, Ciappetta P, Delfini R : Nontraumatic acute spinal subdural hematoma : report of five cases and review of the literature. *J Neurosurg (Suppl 1)* 91 : 65-73, 1999
3. Haines DE, Harkey HL, Al-Mefty O : The “subdural” space : a new look at an outdated concept. *Neurosurgery* 32 : 111-120, 1993
4. Kim HW, Cho KY, Yim SG, Park SK : Migration of Intracranial Subdural Hematoma to Spinal Canal. *J Korean Neurosurg Soc* 35 : 433-435, 2004
5. Kim MS, Chung CK, Hur JW, Lee JW, Seong SO, Lee HK : Spinal subdural hematoma following craniotomy : case report. *Surg Neurol* 61 : 288-292, 2004
6. Lee JI, Hong SC : Spinal subdural haematoma as a complication of cranial surgery. *Acta Neurochir* 145 : 411-415, 2003
7. Rader JP : Chronic subdural hematoma of the spinal cord : report of a case. *N Engl J Med* : 374-376, 1955
8. Shimizu S, Tachibana S, Maezawa H, Fujii K, Kan S : Lumbar spinal subdural hematoma following craniotomy : case report. *Neurol Med Chir* 39 : 299-301, 1999
9. Wurm G, Pogady P, Lungenschmid K, Fischer J : Subdural hemorrhage of the cauda equina. A rare complication of cerebrospinal fluid shunt. Case report. *Neurosurg Rev* 19 : 113-117, 1996
10. Yamaguchi S, Hida K, Akino M, Yano S, Iwasaki Y : Spinal subdural hematoma : a sequela of a ruptured intracranial aneurysm : *Surg Neurol* 59 : 408-412, 2003