

Case Report

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A spinal subdural hematoma induced by guidewire-based lumbar drainage in a patient with ruptured intracranial aneurysms

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Abstract: We present a rare case of spinal subdural hematoma induced by guidewire-based lumbar drainage in a subarachnoid hemorrhage patient with a ruptured intracranial aneurysm. Decreased muscle strength and muscle tension of bilateral lower limbs were noted, and an MRI confirmed the spinal subdural hematoma from the sacral to the thoracic segments. The spinal subdural hematoma evacuation and spinal canal decompression were performed by laminectomy. However, the patient did not benefit from the surgery and developed lower limb muscle atrophy. The complication of the spinal subdural hematoma after lumbar drainage is extremely rare; only limited approaches are available for the treatment of spinal hematoma to improve the outcome and avoid severe consequences. Thus, the present case might suggest refraining from use of a guidewire during lumbar drainage for the prevention of spinal subdural hematoma and close observation of the related symptoms and signs for the early detection of spinal hematoma after the procedure. In addition, full decompression can be performed by complete hematoma evacuation and laminectomy of related segments for the treatment of spinal subdural hematoma induced by lumbar drainage.

1 Introduction

Lumbar puncture (LP) is a procedure commonly used to diagnose neurological dysfunction and therapeutically decrease cerebrospinal fluid (CSF) volume. The most frequently observed complications include post-LP headache and local structural injury. Severe complications such as spinal hematoma formation that could lead to myelopathy or cauda equina syndrome are rarely observed; such complications should be diagnosed and treated immediately to avoid irreversible paralysis or death. However, existing reports have not produced efficient solutions, and prevention of such complications is rarely mentioned [1].

2 Case report

A 59-year-old previously healthy male presented to the hospital with a severe headache at the onset, followed by disturbed consciousness and moderate coma for 6h. A subarachnoid hemorrhage (Hunt and Hess Grade IV) with intraventricular hemorrhage and obstructive hydrocephalus was observed on a head CT scan. External ventricular drainage (EVD) was performed at admission, which led to improvement of his clinical status to a mild coma. Digital subtraction angiography indicated multiple aneurysms, including an anterior communicating artery (Acom) aneurysm and a right anterior cerebral artery first segment (R-A1) aneurysm. Emergent surgery was suggested for clipping the aneurysm to avoid disease risks. Although his family members initially refused, aneurysm clipping was performed successfully at about 8 days after admission. Subsequently, the patient displayed spontaneous movements of the four limbs without paralysis of either side in a mild coma, and 10 days after admission the EVD was with-

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drawn to prevent infection. The CSF continued to exhibit old bloody hemorrhagic fluid, and hydrocephalus with enlarged bilateral ventricles was observed as assessed by a recheck head CT scan. Lumbar puncture followed using a guidewire-based drainage kit to provide continuous drainage. Obstructed drainage was found after 1 day, and another lumbar puncture revealed fresh hemorrhagic fluid. Decreased muscle strength and tension of the bilateral lower limbs were also observed. Magnetic resonance imaging (MRI) of thoracic and lumbar segments indicated spinal subdural hematoma from S1 (Figure 1A) extending to T7 (Figure 1B). Evacuation of the hematoma and spinal canal decompression with laminectomy performed within 12 h after MRI confirmed the hematoma. Laminectomy was performed from the lower segment (S1) upward to L4; it detected a tight dural stretch and subdural bluish staining (Figure 2A) that extended to L3 after subdural hematoma evacuation (Figure 2B) until CSF release was detected. No spinal vascular anomaly was identified during the surgery. However, after the second surgery, the patients did not show any improvement in muscle strength of the bilateral lower limbs, and his mental status remained mild coma. Thus, an extensive laminectomy and hematoma evacuation were considered; however, the family of the patient, despite pneumonia due to pulmonary infection and his weak physiological state after 2 open surgeries, refused further surgery. Rehabilitation, steroids, and neurotrophic medications were administered without obvious improve-

ment in the muscle strength of bilateral lower limbs after 1 month; the condition was also accompanied by decreased muscle tension and muscle atrophy. Coagulopathy was not detected before or after surgery during the patient's hospital stay. No antiplatelet or anticoagulation medication was administered before or after admission. The patient was discharged at 1.5 months after admission; drowsiness and bilateral lower limb paraplegia were observed without further follow-up at the outpatient clinic.

Ethical approval: The research related to human use has been complied with all the relevant national regulations, institutional policies and in accordance the tenets of the Helsinki Declaration, and has been approved by the authors' institutional review board or equivalent committee.

Informed consent: Informed consent has been obtained from the patient's guardian.

3 Discussion

A spinal hematoma may form at the epidural, subarachnoid, and subdural area as described in autopsies in 1682 [2]. Hematoma formation could be attributed to vascular anomalies, local neoplastic diseases, and anticoagulation

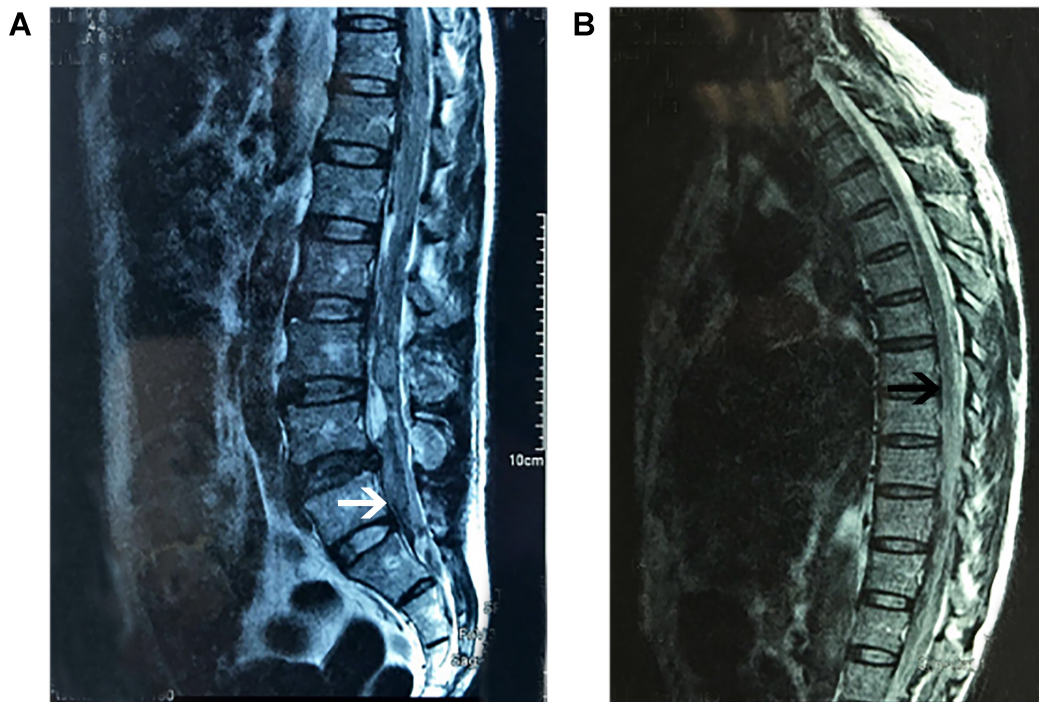


Figure 1: MRI T2WI indicated spinal subdural hematoma from S1 (A, white arrow) extending to T7 (B, black arrow).

or antiplatelet treatments. The injury to annulus fibrosis or venous plexus may occur while administering spinal anesthesia that leads to severe morbidity [3]. Edelson et al. described 8 patients with spinal subdural hematoma and thrombocytopenia: [4] most patients with spinal hematoma presented with flaccid paralysis and hyporeflexia because of direct compression of the spinal cord. A direct correlation between rapid deterioration of symptoms and recovery, as well as the time to definitive intervention, has been established [5]. Preexisting coagulopathy correlated significantly with a poor neurological outcome, irrespective of the intervention [1].

This patient did not present any previous history of coagulopathy, and the results of all the in-hospital coagulation tests were in the normal range. The formation of the spinal subdural hematoma might occur as a result of the use of a guidewire for lumbar drainage that could have injured the inside vessels, leading to extensive hemorrhage. Thus, we speculated that guidewire-induced lumbar drainage might not be a reliable choice for the procedure because the increased probability of the contact

between the wire and the vessels led to an increased risk of vascular injuries. Lumbar drainage was conducted 10 days after SAH instead of external ventricular drainage owing to old bloody CSF and hydrocephalus. Therefore, to prevent spinal hematoma, safe lumbar drainage or intermittent lumbar puncture for CSF drainage might be an option for conducting safe CSF drainage. Subsequently, close monitoring of the risks resulting from the procedure, including superficial sensation, muscle tension, muscle strength, and tendon reflexes of the lower limbs, is essential. In this patient with disturbance of consciousness, the muscle strength was initially found have deteriorated after the drainage, which suggested further examination such as lumbar MRI for diagnosis.

Furthermore, no significant correlation was established between the type of surgical intervention and good outcome, based on a previous report. However, the emergent neurosurgical intervention was suggested to manage complications such as cauda equina syndrome induced by intrathecal spinal hematoma [1]. In this patient, hematoma evacuation and spinal canal decompression were performed promptly after MRI confirmation. The evacuation was not extended to the T7 level because the patient's family refused surgery; thus the evacuation was only extended to the L3 level when the CSF release was observed. That might indicate the potential patency of the subarachnoid space, and the likely sufficiency of the decompression proved to be insufficient based on subsequent examinations. Therefore, the patient's outcome did not improve as significantly as was expected. We speculated that this phenomenon might correlate with the severity of the spinal cord and cauda equina compression by hematoma, and complete evacuation of the hematoma upward to T7 might contribute to an enhanced recovery of the patient.

Spinal subdural hematoma induced by lumbar drainage is extremely rare. Thus, avoiding the usage of a guidewire for lumbar drainage to prevent the potential spinal vascular injury, monitoring the related symptoms and signs for the early detection of spinal hematoma after the procedure, and managing the complication with hematoma evacuation and spinal canal decompression for functional improvement are crucial aspects in such cases.

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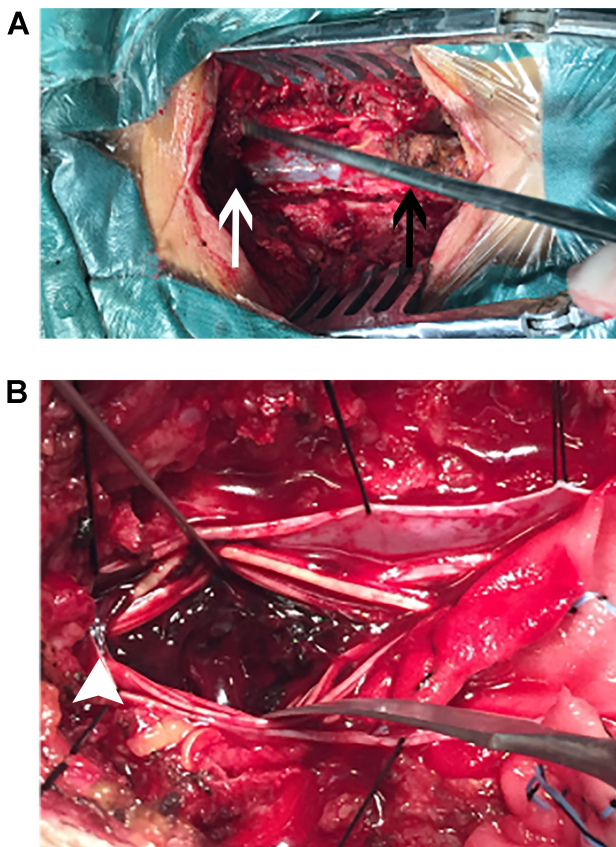


Figure 2: Laminectomy from the lower segment (S1, black arrow) upward to L4 (white arrow) with tight dural stretch and subdural bluish color (A) and extended to L3 (white arrow head) with subdural hematoma (B) (from left to right indicates the direction from the rostral to the caudal side).

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