Subarachnoid Hemorrhage Presenting with Seizure due to Cerebrospinal Fluid Leakage after Spinal Surgery

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Cerebrospinal fluid leakage may commonly occur during spinal surgeries and it may cause dural tears. These tears may result in hemorrhage in the entire compartments of the brain. Most common site of such hemorrhages are the veins in the cerebellar region. We report a case of hemorrhage, mimicking aneurysmal subarachnoid hemorrhage due to a cerebrospinal fluid leakage following lumbar spinal surgery and discuss the possible mechanisms of action.

Key Words: Cerebrospinal fluid leakage · Intracranial hemorrhage · Spinal surgery · Subarachnoid hemorrhage.

INTRODUCTION

Cerebrospinal fluid (CSF) leakage may commonly occur during spinal surgeries and it may cause dural tears\(^1\). These tears may result in hemorrhage in the entire compartments of the brain.

There are many risks related to this complication. One of the most gruelling complication is remote hemorrhages which can develop in any compartments of the brain\(^2\). Most of the reported hemorrhages are from the veins located in the cerebellar region\(^3\). Moreover, pneumocephalus and/or pneumorrhachis in the upper segments of the spine can rarely occur after CSF leakage.

We report a case of hemorrhage mimicking aneurysmal subarachnoid hemorrhage and concurrent pneumocephalus due to CSF leakage following lumbar spinal surgery. The possible mechanisms of action for this unusual complication is also discussed.

CASE REPORT

A 70 year-old woman with the history of controlled hypertension was admitted with the complaints of neurologic claudication and low back pain. Neurological examination was uneventful. Imaging studies revealed degenerative spondylolisthesis and spinal stenosis at L3–4 (Fig. 1A). The patients underwent decompressive laminectomy and an excessive CSF leakage was occurred after an accidental dural tear during posterior instrumentation (Fig. 1B, C). The tear was sutured primarily and a suction drain was inserted prior to the closure of the skin. The patient awoke in good neurological status however approximately 100 cc of hemorrhagic fluid was drained in the first 12 postoperative hours. Consecutively, the patient deteriorated and had focal seizures. Computed tomography of the brain showed sylvian-periinsular subarachnoid hemorrhage and pneumocephalus in the right cerebral hemisphere (Fig. 2A, B). Cerebral angiography was performed in order to exclude an underlying aneurysmal pathology, which did not reveal any vascular abnormality (Fig. 2C).

The patient was treated conservatively. Computed tomography angiography was repeated 4 weeks after the operation which demonstrated normal vascular findings. The patient was discharged in a good condition with full mobility and no neurologic deficit has been detected through the 4 year follow-up.

DISCUSSION

Cerebellum is the most commonly site of remote hemorrhage during supratentorial, infratentorial and spinal surgery\(^4\). Although the exact pathophysiology of these hemorrhages is
whereas only one case presented with seizure. Although previously reported intraparenchymal hemorrhages were in different sizes, shapes and locations, none of them were located in the right periinsular-sylvian cistern and mimicked aneurysmal subarachnoid hemorrhage. This "pseudo-subarachnoid hemorrhage" phenomenon has been reported in anoxic-ischemic encephalopathy with brain swelling, venous sinus thrombosis, subdural hematoma and intracranial hypotension. Engorgement of the superficial veins secondary to elevated intracranial pressure and severe brain edema manifesting as hypoattenuated parenchyma is the mechanism blamed for pseudo-subarachnoid hemorrhage. Brain sagging in conditions that causes intracranial hypotension narrows the subarachnoid spaces, displaces CSF, compresses the basal cistern and sylvian fissure and consequently results in brain edema. These neurological changes can be observed as hyperdensity of the anterior and middle
cerebral arteries as well as obliteration of the basal cisterns on computed tomography. CSF leakage during surgery may trigger the abovementioned mechanism, which may explain the pathophysiological process in our case.

On the other hand there are other reported mechanisms to explain remote intraparenchymal hemorrhage. One of the most widely accepted hypothesis is the rupture of blood vessels due to intracranial hypertension and consequent increase in transluminal venous pressure. Another possible mechanism is downward sagging of the cerebellum that causes stretching and occlusion of bridging cerebellar veins with subsequent hemorrhagic venous infarction. The previously reported cases suggested that, movement of the brain due to intracranial hypertension from massive CSF loss can cause acute occlusion of multiple infra- and supratentorial bridging veins. This would explain the presence of multiple foci of intraparenchymal hemorrhages seen in the entire brain tissue. It is reported that, alleviation in CSF pressure causes augmentation in the carotid flow velocity, especially in patients with normal pressure hydrocephalus.

In addition to the pathophysiology of pseudo-subarachnoid hemorrhage, these suggestions could also help to explain the mechanism of action. Otherwise, it is accepted that older people constitute the majority of patients who undergo spinal surgery and are much more vulnerable to the occurrence of intracranial hemorrhage after CSF leakage in association with a history of underlying chronic hypertension and age-related brain atrophy.

In the light of the relevant literature, we believe that the pattern of this confusing subarachnoid hemorrhage seems to be originating from both venous and arterial vasculature of the brain. Although our patient complained of headache, neurological deterioration of mental status and seizures, which can also be symptoms of hemorrhage in an aneurysmatic origin, cerebral angiography and control computed tomography showed no vascular pathology.

**CONCLUSION**

Increase of carotid blood flow velocity as a result of CSF leakage and brain edema related alterations in CSF pressure can cause pseudo-subarachnoid hemorrhage. Mimicking an aneurysmal subarachnoid hemorrhage, in clinical symptoms and radiologic findings makes this case interesting and extraordinary. This rare complication must be considered in conditions with unexplained neurological deterioration, after CSF leakage during spinal surgery, in older patients especially with chronic hypertension and brain atrophy.

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