Cerebellar Hemorrhage after Spinal Surgery: Report of Two Cases and Literature Review

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- OBJECTIVE AND IMPORTANCE: Cerebellar hemorrhage remote from the site of surgery may complicate neurosurgical procedures. We describe our experience with two cases of cerebellar hemorrhage after spinal surgery and review the three cases previously reported in the literature to determine whether these cases provide insight regarding the pathogenesis of remote cerebellar hemorrhage.
- CLINICAL PRESENTATION: One of our patients developed cerebellar hemorrhage in the vermis and right hemisphere after transpedicular removal of a partially intradural T9–T10 herniated disc with the patient in the prone position. The other patient developed cerebellar hemorrhage in the vermis and bilateral hemispheres after L3–S1 decompression and instrumentation with the patient in the prone position, during which the dura was inadvertently opened.
- INTERVENTION: The first patient was treated conservatively and had mild residual dysarthria and gait ataxia 2 months after surgery. The second patient underwent exploration and revision of the lumbar wound with primary dural repair. The cerebellar hemorrhage was treated conservatively, and the patient had mild dysarthria and ataxia 1 month after surgery.
- CONCLUSION: Cerebellar hemorrhage must be considered in patients with unexplained neurological deterioration after spinal surgery. Dural opening with loss of cerebrospinal fluid has occurred in every reported case of cerebellar hemorrhage complicating a spinal procedure, supporting the hypothesis that loss of cerebrospinal fluid is central to the pathogenesis of this condition. Because remote cerebellar hemorrhage can occur after procedures with the patient in the supine, sitting, and prone positions, patient positioning seems unlikely to play a causative role in its occurrence. (Neurosurgery 50:1361–1364, 2002)

Key words: Cerebellum, Cerebrospinal fluid, Intracranial hemorrhage, Remote hemorrhage, Spine

Cerebellar hemorrhage remote from the site of operation may complicate intracranial surgery. The occurrence of remote cerebellar hemorrhage (RCH) is most frequently associated with frontotemporal craniotomy with the patient in the supine position (1, 3, 5, 7, 10, 12). Patients who undergo repair of an unruptured aneurysm or a temporal lobectomy seem particularly predisposed to RCH (5).

Although the underlying pathophysiology of RCH has not been definitively elucidated, recent reports suggest that the condition is venous in origin (3, 5–7, 12). Cerebellar "sag" as a result of intraoperative cerebrospinal fluid (CSF) aspiration has been proposed to cause stretching and occlusion of bridging cerebellar veins coursing in the cephalad direction, leading to hemorrhagic venous infarction (5, 9, 12, 13). The relevance of patient positioning in the development of RCH is unclear.

If acute CSF hypovolemia does play a causative role in the pathophysiology of RCH, the phenomenon would be expected to occur after spinal procedures during which CSF was lost. We report our experience with two cases of cerebellar hemorrhage after spinal procedures and review the relevant literature describing this occurrence.

CASE REPORTS

Patient 1

A 43-year-old man presented with myelopathy and was found to have a herniated T9-T10 disc. This patient was briefly described previously (4). He had no history of arterial hypertension, trauma, hypercoagulable state, or recent infection. The patient underwent removal of the disc via a posterolateral, transpedicular approach in the prone position. The disc had eroded through the dura, and opening of the dura and sectioning of the dentate ligament on one side was necessary. The patient awoke from surgery neurologically intact. Neither intraoperative nor postoperative blood pressures were substantially elevated. At 12 hours after surgery, the patient developed some dysarthria, followed by progressive ataxia over the course of the next 24 hours. A computed tomographic (CT) scan was obtained 48 hours after surgery, revealing cerebellar hemorrhage in the vermis and right hemisphere (Fig. 1). Laboratory studies, including platelet count, prothrombin time, and partial thromboplastin time, were within normal ranges. Bilateral vertebral angiography was normal. The patient was treated conservatively, and his deficits did not progress. At 2 months after sur-



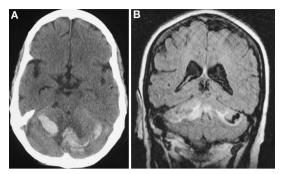
FIGURE 1. Patient 1. CT scan of the head without contrast obtained 2 days after surgery, demonstrating acute hemorrhage in the cerebellar vermis and superior aspect of the right cerebellar hemisphere.

gery, he had slight residual dysarthria and gait ataxia.

Patient 2

A 56-year-old woman underwent an L3-S1 decompressive laminectomy and pedicle screw fixation at an outside hospital for degenerative lumbar spondylolisthesis. The patient had no history of arterial hypertension, trauma, hypercoagulable state, or recent infection. The patient awoke from her initial surgery neurologically intact. Neither intraoperative nor postoperative blood pressures were noted to be elevated. On postoperative Day 2, the patient developed a severe occipital headache and vomiting, followed by the development of dysarthria and bilateral upper-extremity ataxia. A head CT scan revealed a bilateral hemorrhage in the superior folia of the cerebellar vermis and the cerebellar hemispheres (Fig. 2A). The patient was transferred to our institution.

On admission, the patient was alert and complained of a severe occipital headache. Neurological evaluation revealed dysarthria and bilateral ataxia. A repeat CT scan was unchanged. Laboratory studies, including platelet count, prothrombin time, and partial thromboplastin time, were within normal ranges. A drainage catheter connected to bulb suction remained in the subfascial space, and pink-tinged watery fluid was aspirated. Magnetic resonance imaging revealed early subacute hemorrhage within the cerebellum without ad-



ditional abnormalities (*Fig. 2B*). The patient's headache continued to worsen, but it was relieved by her lying supine. A repeat CT scan demonstrated no enlargement of the hemorrhage or hydrocephalus. More than 500 ml of watery fluid suggestive of CSF were drained from the wound via the suction catheter. The drain was removed on the evening of postoperative Day 3. The next morning, the patient's headache had resolved completely, even when she was in the upright position.

On postoperative Day 4, clear fluid began to leak from the lumbar incision. A CT myelogram was performed, which revealed a posterior pseudomeningocele with contrast extravasation at both the L3 and L5 levels. The patient was returned to the operating room, and a 3-mm dural tear at the caudal aspect of the previous laminectomy with copious CSF leakage was identified and repaired. The patient had no further headache but had residual mild dysarthria and ataxia at discharge 3 weeks after surgery.

DISCUSSION

Cerebellar hemorrhage after a spinal procedure has been reported previously and should be considered as a potential source of unexplained neurological decline after spinal surgery. The phenomenon was first described by Chadduck (2) in 1981, after a cervical laminectomy in which the dura was opened widely and the dentate ligaments were sectioned while the patient was in a sitting position. In 1994, Mikawa et al. (8) reported a cerebellar hemorrhage after durotomy and revision of a cervical fusion. Most recently, Morandi et al. (9) described a patient who developed cerebellar and supratentorial hemorrhage

FIGURE 2. Patient 2. *A*, noncontrast CT of the head demonstrating acute cerebellar hemorrhage within the superior vermis and both hemispheres; *B*, coronal fluidattenuated inversion recovery magnetic resonance scan demonstrating early subacute intraparenchymal cerebellar hemorrhage.

after resection of an intradural cervical schwannoma.

An important common feature among these three previously reported cases and our two cases is the intradural nature of the procedures, with consequent loss of CSF. In an analysis of RCH after supratentorial craniotomy at our own institution, almost all cases of RCH were associated with procedures that involved opening the CSF cisterns or the ventricular system (5). There is a growing consensus that hemorrhagic venous infarction underlies most cases of RCH (3, 5–7, 12). It is possible that downward cerebellar displacement, or "sag," resulting from intraoperative loss of CSF causes transient stretch occlusion of superior cerebellar veins draining in the cephalad direction into the deep venous system (5, 6, 12, 13). This may cause intracerebellar hemorrhage in patients with insufficient venous collaterals.

The pattern of cerebellar hemorrhage on the postoperative CT scans in our two patients after spinal surgery is similar to that seen after supratentorial operations (1, 3, 5, 12). On the basis of our two cases and the three previous reports, it would initially appear that cerebellar hemorrhage after spinal procedures is typically symptomatic. Postoperative imaging of the brain, however, would be distinctly uncommon in an asymptomatic patient after a spinal procedure. Thus, asymptomatic cerebellar hemorrhages are unlikely to be detected, and the true incidence of this occurrence may be higher than suggested by existing reports. In contrast, RCH after supratentorial surgery is frequently asymptomatic and detected incidentally on postoperative scanning.

Our patient with cerebellar hemorrhage after lumbar fusion with dural tear had a postural headache highly suggestive of intracranial hypotension, which resolved when suction drainage of the wound was discontinued. This case supports the association between low CSF pressure and cerebellar hemorrhage remote from the operative site. In patients with intracranial hypotension caused by spontaneous CSF leakage from spinal arachnoid diverticula, however, cerebellar hemorrhage has not been described. This probably relates to the chronicity of the disorder, during which time collateral venous drainage would be expected to develop. With the more abrupt loss of CSF that may occur during and immediately after spinal procedures, such collateral venous drainage would not have time to develop.

The relevance of patient positioning in the development of RCH after supratentorial surgery is unclear. Seoane and Rhoton (11) have suggested that jugular vein compression at the transverse process of C1 as a result of head positioning may be a causative factor in RCH. If RCH represents a hemorrhagic venous infarction, it is plausible that venous compression in the neck could cause or exacerbate the process. The occurrence of cerebellar hemorrhage after spinal procedures with the patient in both the prone and sitting positions, however, suggests that position is not relevant to the pathogenesis of this complication after spinal procedures and casts some doubt on its role in the development of RCH after supratentorial procedures.

CONCLUSIONS

Cerebellar hemorrhage must be considered in patients with unexplained neurological deterioration after spinal surgery. Dural opening is common to every reported case of a spinal procedure complicated by cerebellar hemorrhage, supporting the hypothesis that CSF loss is central to the pathogenesis of this condition. Because RCH can occur after procedures with the patient in the supine, sitting, and prone positions, patient positioning seems unlikely to play a causative role in its occurrence.

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COMMENTS

Friedman et al. report two cases of patients who experienced unexplained cerebellar hemorrhage after spinal surgery. In both patients, the spinal subarachnoid space was entered. In the second patient, cerebrospinal fluid (CSF) was actively aspirated from the wound by a bulb suction drain. We do not know whether such a drain was placed in the first patient. The resulting cerebellar hemorrhages are diffuse and relatively widespread. This is similar to the imaging picture seen with remote cerebellar hemorrhage after supratentorial surgery.

The authors hypothesize that the hemorrhages are the result of venous stasis. I am not convinced that these are venous infarctions. The hemorrhages span more than the territory of a single vein. The computed tomographic images and magnetic resonance imaging scan do not demonstrate the low density frequently seen surrounding a venous infarction. If the hemorrhage arose from diffuse cerebellar venous stasis, I would have expected to see diffuse cerebellar swelling and obstructive hydrocephalus.

Remote cerebellar hemorrhages remain a perplexing problem. I do not understand their pathophysiology or any avoidable predisposing factor.

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OCIETAS

Friedman et al. provide us with two interesting examples of cerebellar hemorrhage after spinal surgery. In both cases, the dura was opened and CSF was lost. Many readers are undoubtedly familiar with the phenomenon of remote cerebellar hemorrhage in the setting of intracranial surgery. As the authors point out, hemorrhagic venous infarction secondary to obstruction of draining veins caused by a "sagging cerebellum" has been the most popular explanation for this phenomenon. In the two examples that I have personally observed, I was relatively convinced that this was indeed the explanation. I believe that the two present cases and the previously reported cases in association with spinal surgery add credence to this theory. I disagree, however, with the authors' contention that position plays no role in the phenomenon. I think the authors would agree that although the overall position is not relevant, the head does need to be elevated above the site of the CSF leak for the sagging of the cerebellum to occur. One could argue that use of a very slight degree of "head-down" positioning when one contemplates opening the spinal dura might prevent this complication. This, of course, should be done only when intracranial hypertension is not an issue.

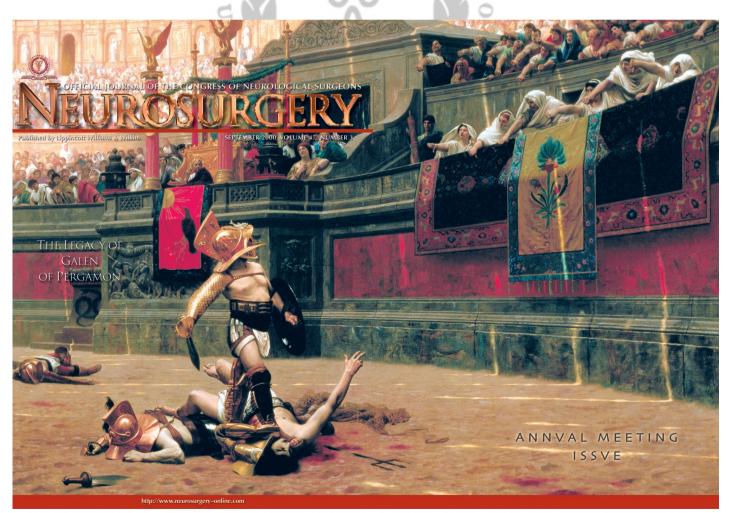
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The authors describe two cases of patients presenting with a cerebellar hematoma after spine surgery and perform a comprehensive literature review regarding this rare complication. In each patient, the dura was opened, intentionally or not, with an associated loss of CSF. In both patients, initial postoperative evaluation demonstrated no neurological deficits. Symptoms were noted as early as 12 hours after surgery in one patient. Each patient was managed conservatively regarding the cerebellar hemorrhage, without progression of symptoms. Both patients demonstrated mild residual deficits consisting of dysarthria and gait ataxia at the last follow-up evaluation. The authors' literature review showed that only three other cases of cerebellar hemorrhage after spine surgery have been reported. In all cases, the dura was opened, with loss of CSF. On the basis of this association and previous assumptions after intracranial surgery, the authors hypothesize that cerebellar "sag" as a result of intracranial hypovolemia is the primary event leading to RCH. The downward displacement of the cerebellum leads to stretch occlusion of the bridging veins and venous infarction.

This article provides a description of an extremely rare complication associated

with intradural spine surgery. As the authors correctly point out, the exact incidence of this complication is unknown, and it is likely to remain so, because cranial imaging after spine surgery is not routinely performed. Although definitive conclusions are lacking because of the small number of patients presenting with this complication, the authors are able to formulate several reasonable assumptions on the basis of the associations observed. Given the rarity of this complication, it is virtually impossible to formulate preventive measures; however, the possibility needs to be considered and aggressively evaluated should unexplained neurological deterioration occur after any intradural spine procedure.

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