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Case Report

A Rare Complication of Spinal Surgery: Cerebelllar Hemorrhage

Spinal Cerrahinin Nadir Bir Komplikasyonu: Serebellar Hemoraji

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ABSTRACT

Remote cerebellar hemorrhage (RCH) after spinal surgery is encountered extremely rarely. A 64 year-old female patient developed symptoms of deteriorating consciousness and diplopia arising on the first postoperative day after recurrent spinal surgery. Cranial CT scan showed cerebral edema and evidence of a cerebellar hemorrhage. Urgent suboccipital decompressive craniectomy and expanded duraplasty were performed. Repeat CT at 24 h revealed hydrocephalus and an external ventricular drain (EVD) was inserted for 20 days. The patient's consciousness deteriorated after withdrawal of the EVD and a ventriculoperitoneal shunt was placed. The patient recovered completely except for gait ataxia and left foot drop. Although the exact cause is unknown, iatrogenic dural opening resulting in excessive cerebrospinal fluid (CSF) drainage and secondary development of venous infarction have been suggested to lead to RCH.

KEYWORDS: Cerebrospinal fluid, Drainage, Remote cerebellar hemorrhage, Spinal surgery

ÖZ

Spinal cerrahi sonrası uzak serebellar hemoraji çok nadiren ortaya çıkabilir. 64 yaşındaki bir bayan hastada, yinelenen spinal cerrahi operasyonundan 1 gün sonra bilincinde bozulma ve çift görme semptomları ortaya çıktı. Çekilen karanial BT'de serebellar hemoraji ve ödem bulguları izlendi. Hastaya acil olarak suboksipital dekompresif kraniektomi ve genişletilmiş duraplasti yapıldı. 24.saatte tekrarlanan BT'de hidrosefali görülmesi üzerine eksternal ventriküler drenaja (EVD) alınan hasta 20 gün süreyle takip edildi. EVD çıkarımını takiben hastanın bilincinin bozulması üzerine, ventrikülоperitoneal şant yerleştirildi. Hasta ataksik yürüme ve solda düşük ayak dışında tümüyle düzeldi. Kesin nedeni bilinmemesine rağmen, duranın iatrojenik olarak açılması sonucunda aşırı beyin omurilik sıvısı drenajının oluşması ve buna ikincil olarak gelişen venöz infarktın, uzak serebellar hemorajiye neden olduğu düşünülmektedir.

ANAHTAR SÖZCÜKLER: Beyin omurilik sıvısı, Drenaj, Uzak serebellar hemoraji, Spinal cerrahi

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INTRODUCTION

Supratentorial subdural hematoma is a rare but well-known complication after spinal surgery or spinal puncture (1). However, cerebellar bleeding after spinal surgery is extremely rare with few published case reports (1-15). It is a very serious clinical problem due to the location of the bleeding. Some authors suggest that remote cerebellar hemorrhage (RCH) or hemorrhagic infarction occurs due to venous infarction, but the pathophysiology and etiology of this condition are unknown (4,12,16,17). Intraoperative, or more likely postoperative, loss of cerebrospinal fluid (CSF) has been reported in most of these cases and therefore seems to contribute to the pathology of this complication (1,8). CSF loss might stretch and occlude the bridging cerebellar veins that course cephalad, leading to hemorrhagic venous infarction (4,8, 12,16,17). Peroperative patient positioning is another factor that is widely suspected to contribute to RCH, but its relevance is unclear (4,8,18).

In the current report, we describe the occurrence of RCH after lumbar spine surgery, summarize the purported mechanisms, and call attention to the early identification and treatment of this complication to avoid or minimize permanent neurological injury.

CASE REPORT

A 64-year-old woman presented with pain in her lower back and left leg. She was operated twice at the level of the L3-4 spinal vertebra with a six-month interval and left foot drop has developed with increasing left leg pain after the second operation. Investigation revealed herniated discs at L3-L4 and L4-L5, and stenosis of the lumbar spinal canal at same levels. The preoperative medical history was not remarkable. Anticoagulants had not been administered and no bleeding tendency was clinically observed before the surgery. She was operated in the prone position via a posterior approach. The surgery involved L3 and L4 laminectomies, L3-4 and L4-5 discectomies and screw fixation of the L3-L5 pedicles, and posterior interbody fusion with allograft. The dura mater was opened accidentally during the operation and was sutured in a watertight fashion. Approximately 100 ml of CSF escaped before the dura was closed. Two suction drains were placed in the epidural space. There were no additional neurologicL deficits in the early postoperative period. Six hours postoperatively the drains had drawn 300 ml serous fluid tinged with blood. At 24 h after surgery she complained of diplopia. A CT scan was obtained showing cerebral edema and evidence of a cerebellar hemorrhage (Figure 1). The result of laboratory investigations, including platelet count, and a clotting screen, were all in the normal range. The patient became increasingly drowsy over the first 36 h after surgery but was obeying commands and moving all limbs. A suboccipital decompressive craniectomy and expanded duraplasty was performed to reduce pressure within the posterior fossa. At 24 h after second surgery her neurological status deteriorated and a repeat CT showed hydrocephalus. An external ventricular drain (EVD) was placed and the patient's status improved during following 20 days. Her consciousness deteriorated after the withdrawal of the EVD and she was discharged from the hospital after the placement of a ventriculoperitenoal shunt. (Figure 2) The patient recovered completely except for gait ataxia and left foot drop. Diplopia was present on far lateral gaze bilaterally.

DISCUSSION

Several authors have stated that RCH is more likely to occur in patients who undergo surgery involving the risky processes of draining larger volumes CSF, such as aneurysm surgery or temporal lobectomy (1,4,19-24). Yakubian et al. (21) have suggested that brain dislocation because of intraoperative CSF loss as a possible mechanism for this phenomenon. Nevertheless, RCH has also occurred after spinal intervention with planned or occult opening of dura (1-9,11,12). Chadduck (3) first described RCH after a spinal procedure in 1981. Recently, Thomas et al. (12) documented a case in which supratentorial and infratentorial intraparenchymal hemorrhage developed after spinal surgery.

Incidence of RCH resulting from supratentorial craniotomy is 0.2-3.5% (1,12,20) but RCH is a more infrequent complication resulting from spinal surgery. After review of the literature we found only 15 cases of RCH complicating spinal surgery, without any information regarding incidence (1-15) but more than 50 cases of RCH complicating

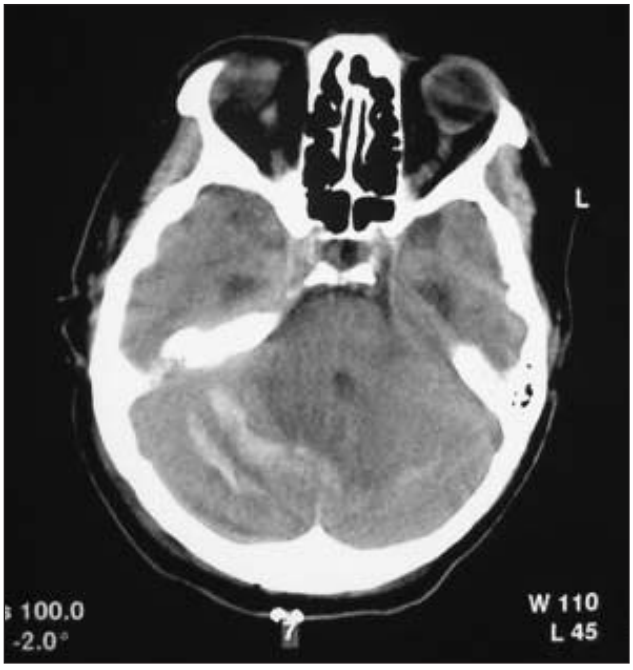

Figure 1: *Axial CT scan obtained 24 hours after spinal surgery, demonstrating acute hemorrhage in the superior aspect of the right cerebellar hemisphere.*

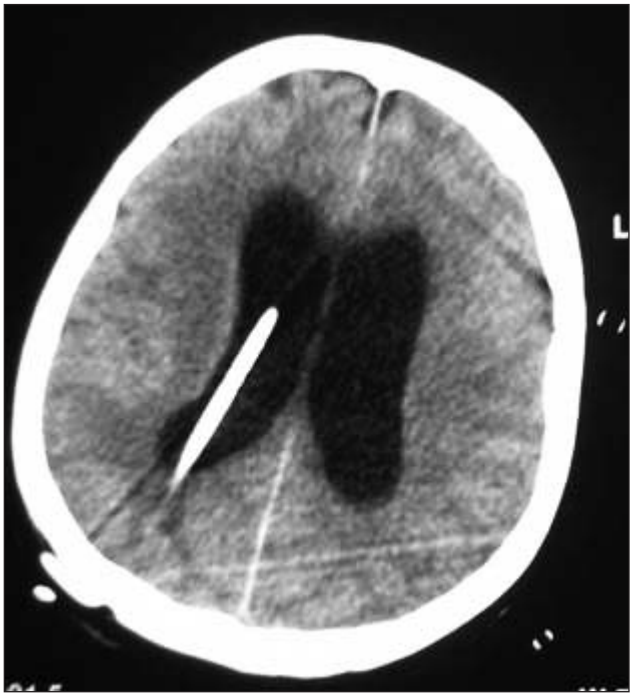

Figure 2: *The right ventricular catheter is seen on the CT before the patient is discharged from the hospital.*

supratentorial surgery have been reported (18,20,25-27). The classic associated bleeding pattern includes blood in the sulci of one or both cerebellar hemispheres and the vermis facing the tentorium. Additionally, intracerebellar hemorrhage, mainly in the upper parts of the cerebellum, is frequently observed (1,25).

Different authors have proposed several pathological mechanisms for RCH. Chadduck (3) postulated that elevated blood pressure might cause an increased gradient between intravascular pressure and CSF pressure, and thus induce hemorrhage into the cerebellar parenchyma. Andrews and Koci (2) speculated that their case of cerebellar infarction resulted from transient traction, kinking, or spasm of superior cerebellar arteries, and hemorrhage occurred upon reperfusion. Some researchers have theorized that a major factor associated with the development of RCH after spine surgery seems to be intracranial hypotension and a caudal sag of the cerebellum caused by intra- or postoperative excessive CSF drainage (1-5,7,8,10,12,16,17,19,21). Whether stretching leads to occlusion or rupture of the bridging cerebellar veins and causes hemorrhagic venous infarction or direct hemorrhage remains unknown (1,4,8, 12,16,17).

Most authors have agreed on two facts: (i) RCH is of venous origin, and (ii) RCH is a result of intra- and even more likely postoperative loss of CSF (1,5,7,8,10,21,25,28,29). Whether mechanical (shearing of cerebellar bridging veins) or hemodynamic (increase in venous blood pressure) causes, which may coexist, finally induce RCH remains unclear.

Brockmann et al. (1) stated that spinal and supratentorial loss of CSF result in a similar streaky subarachnoid bleeding pattern. The most probable pathomechanism accounting for both acute spinal or supratentorial loss of CSF seems to involve stretching of infratentorial cerebellar bridging veins during an upward and downward cerebellar herniation.

Other authors suggesting less-discussed theories belive that preexisting coagulopathies, arterial hypertension, or obstruction of the jugular vein from extreme head rotation might cause cerebellar hemorrhage (12,17,26,29,30). Karaeminogulları et al. (7) reported a case of a cerebellar arteriovenous malformation rupture requiring clot evacuation and decompression two days after lumbar surgery complicated bya dural tear. These factors seem less important but may aggravate or predispose RCH.

Our case did not have a known preexisting coagulopathy, history of arterial hypertension, or diabetes. In our case, drains were inserted, and RCH was assumed to be related to the, which would correlate with observations made by others who also found an increased amount of drained fluid within a few hours (1,5). Our case had a large CSF loss of about 100cc when it is recalled that the total amount of circulating CSF is about 150 cc. There was also a loss of a large amount of CSF from postoperative suction making up 300cc blood and CSF mixture in total. These factors may be responsible for development of RCH in our patient.

Avoiding any dural tearing, detailed preoperative search for a possible coagulopathy including a detailed history of bleeding and screening tests, and preoperative assessment of the blood pressure are among the measures to be taken to prevent RCH. Good surgical exposure and lighting of the surgical field, meticulous surgical handling, and working with diamond burrs and microcurettes when operating near the dura are the measures to be taken in order to prevent development of dural tears during surgery. The rate off iatrogenic dural tearing has been previously reported between 3.1-14% in different series (31-34). Tafazal's prospective study involving 1,549 cases in 14 different centers has stated that the rate of dural tearing was 3.5% for primary discectomy, 8.5% for spinal stenosis surgery and 13.2% for revision discectomy (33).

Other complications from a CSF leak occur because downward displacement of the brain exerts traction on cranial nerves, leading to diplopia (as observed in our case), visual field defects, vocal cord paralysis, and facial numbness (5,35).

Small cerebellar hemorrhages can be managed medically and followed-up with serial imaging; however, large hematomas that cause a significant mass effect in the posterior fossa may require surgical decompression (2-4,6,8,11,13). Compression of the fourth ventricle and resultant non-communicating hydrocephalus should be managed with CSF diversion procedures (1,2,6,8,23,26). Decompression is necessary if there are sign and symptoms of brainstem compression due to increased intracranial pressure and evacuation of the hematoma should be performed immediately in such cases. Expanded suboccipital craniectomy and duraplasty are the procedures used to decompress the posterior fossa (8). Abnormalities of coagulation parameters should always be ruled out as the underlying reason for RCH and at the very least need to be corrected to avoid aggravation of hemorrhage. Blood pressure should be monitored closely (25).

The outcome in patients with RCH varies significantly and seems primarily to depend on the extent of bleeding, its intracerebellar component, the underlying disease, the amount of time before action is taken and further complications (1). In cases with blood only in the sulci of the upper cerebellum, severe complications or serious permanent cerebellar defects are seldom observed. Nevertheless, the greater the extent of intracerebellar hemorrhage (as in our case), the greater the risk of acute obstructive hydrocephalus and associated complications. The outcome is good with only mild remaining neurological symptoms or complete recovery in more than 50% of all cases, while death occurs in ~10-15% (25). The exact pathomechanism leading to RCH is not yet fully understood, however, one can only assume that the kinetics and the extent of CSF loss are factors determining the amount of intracerebellar hemorrhage (1).

CONCLUSION

Cerebellar hemorrhage must be considered in patients with unexplained neurological deterioration after spinal surgery. Large-volume CSF loss intra- or postoperatively may be an etiological factor in RCH. Quickly pinpointing the correct diagnosis may prevent the aggravation of complications associated with RCH. Small cerebellar hematomas can be managed medically, but larger lesions that cause significant mass effect in the posterior fossa must be treated surgically.

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