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Clinical Article

Remote Cerebellar Hemorrhage Complicated after Supratentorial Surgery: Retrospective Study with Review of Articles

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Objective : Remote cerebellar hemorrhage (RCH) is one of the rare complications occurring after supratentorial surgery, and its pathomechanism is poorly understood. We report 10 cases of RCH from our institution and review 154 cases from a database in order to delineate incidence, common presentation, risk factors, and outcomes of this complication. In addition, the means of prevention are discussed.
Methods : We reviewed the medical records of 10 patients who experienced RCH after undergoing supratentorial surgery at our institution between 2001 and 2008. A database search in Medline revealed 154 cases of RCH in the English literature. Characteristic features were analyzed and compared.
Results : There were 10 cases of RCH among 3307 supratentorial surgery cases, indicating a 0.3% incidence. All patients had characteristic imaging features of RCH, namely a streaky bleeding pattern in the superior folia of the cerebellum. Seven patients had a history of preoperative hypertension. Four cases were related to cerebral aneurysms, and other four developed after the removal of brain tumors. Cerebrospinal fluid (CSF) drainage apparatuses were installed postoperatively in all cases. Outcomes according to modified Rankin scale (mRS) were good in 7 patients, with 1 fatal case.
Conclusion : RCH is a rare complication after supratentorial surgery, and the exact etiology still remains uncertain. Hypertension and perioperative loss of CSF seem positively correlated with RCH, but no single risk factor is totally responsible. Patients with RCH should be closely observed to improve their prognosis.

KEY WORDS : Remote cerebellar hemorrhage · Supratentorial surgery · CSF drainage.

INTRODUCTION

Most postoperative intracranial hemorrhages develop at the site of surgery, but sometimes they occur at sites other than the operative field in the form of epidural hemorrhage (EDH), subdural hemorrhage (SDH), subarachnoid hemorrhage (SAH), and intracerebral hemorrhage (ICH). Remote cerebellar hemorrhage (RCH) is an even rare complication after supratentorial surgery, with an incidence ranging from 0.08%[31] to 0.6%[12]. There have also been reports of RCH after spinal surgery[5,6,9,17,37]. In the literature,[10,12,19,39] various mechanisms and risk factors have been searched to explain the occurrence of RCH after supratentorial surgery, including hypertension, coagulopathy, jugular venous compression during surgery, and intraoperative or postoperative CSF over-drainage. However, the exact mechanism remains obscure.

We reviewed 10 cases of RCH that occurred at our institution over a period of 8 years, as well as 154 cases in the literature[1-3,7,10-16,18-27,29-39]. We evaluated the characteristics, potential risk factors, and possible mechanisms in each patient and discussed means of prevention.

MATERIALS AND METHODS

Between 2001 and 2008, 10 cases of RCH were observed after supratentorial surgery at our institution. In all cases, medical records, imaging studies, and laboratory findings were retrospectively reviewed to determine the general features of the patients, presentation of RCH, possible

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predisposing factors, treatment, and outcomes.

We also reviewed the Medline database using the keywords 'cerebellar', 'posterior fossa', 'infratentorial', 'supratentorial', 'hemorrhage', and 'craniotomy'. All the abstracts were reviewed, and relevant articles were collected. Further relevant articles were identified from the reference lists of collected papers. One-hundred-fifty-four cases of RCH were collected from 33 articles. All cases were reviewed using the same protocol from our own series.

RESULTS

General features

Nine of 2,612 supratentorial craniotomies performed at our institution during the most recent 8-year period were complicated by RCH, as was 1 of 695 burr hole trephinations performed during the same period. Hence, the overall incidence of RCH was 0.3%. Six patients were female, and four were male (Table 1). Ages ranged from 40 to 76 years, with a median age of 58. The primary pathology was a tumor in four cases (two meningiomas, one anaplastic oligoastrocytoma, and one glioblastoma), ruptured aneurysm in two, unruptured aneurysm in two, spontaneous ICH in one, and chronic SDH in one. Preoperative cerebral digital subtraction angiography (DSA) was performed in five cases, magnetic resonance imaging (MRI) was performed in seven, and computed tomographic (CT) angiography was performed in four. None of these studies demonstrated any abnormal findings in the posterior fossa.

Possible risk factors

Preoperative hypertension was present in 7 of 10 patients (70%) in this series. All patients were on medication and were maintained in the normal blood pressure range. Three patients were receiving antiplatelet therapy before surgery, two of them with aspirin alone and another with a combination of aspirin and clopidogrel. However, none of their preoperative coagulation profiles was abnormal. One patient exhibited thrombocytopenia (platelet count : 122,000) prior to surgery, due to liver cirrhosis. All patients were placed in the supine position for surgery, with head rotation ranging from 10° to 60°. Epidural drainage systems were installed for all craniotomy cases except for one with ICH in which the catheter was placed at the site of hemorrhage. A subdural drain was installed for the chronic SDH case. An additional lumbar drainage system was used in the patient with a convexity meningioma, and intraoperative extraventricular drainage (EVD) was used in patient with a glioblastoma. Intraoperative cisternal drainage was practiced in a case with anaplastic oligoastrocytoma and two cases of ruptured and two cases of unruptured aneurysms.

Clinical course

The events leading to the detection of RCH were : delayed awakening from anesthesia in three cases, deterioration of mental status in one case, and routine CT follow-up in six cases. In all cases, the CT scan demonstrated intracerebellar hemorrhage and SAH on the superior folia of the cerebellum-the typical zebra sign[5]. Hemorrhage was bilateral in four cases and unilateral in six cases. Among those with unilateral hematoma, four were on the same side as the primary operation, and two were on the opposite side. Nine patients were treated conservatively, and only one underwent extraventricular drainage and surgery for removal of the hematoma. Outcomes were analyzed according to the modified Rankin scale (mRS). Seven patients showed good outcomes (Gr 0 - three cases, Gr 1 - two cases, Gr 2 - two case), two poor outcomes (Gr 5), and one died from pneumonia.

Table 1. Characteristics of the RCH patients

Case	Age /Sex	Primary diagnosis	Procedure	CSF drainage	Presentation	Treatment	Outcome*	Underlying disease	Coagulopathy and medication
1	54/F	Meningioma	Craniotomy	LD + ED	Delayed awakening	Conserve	Gr 1	HTN DM	
2	68/F	Meningioma	Craniotomy	ED	Delayed awakening	EVD + DS	Gr 5	HTN	Aspirin®
3	40/F	Aneurysm, ruptured	Craniotomy	ED	Incidental	Conserve	Gr 0	HTN	
4	74/F	Chronic SDH	Burr-hole	SD	Incidental	Conserve	Gr 0	HTN	Aspirin® + Clopidogrel
5	55/M	Anaplastic oligoastrocytoma	Craniotomy	ED	Incidental	Conserve	Gr 1	HTN DM	
6	76/M	Hypertensive ICH	Craniotomy	EVD	Incidental	Conserve	Gr 6 (pneumonia)	LC	Thrombocytopenia
7	60/M	Glioblastoma	Craniotomy	ED	Delayed awakening	Conserve	Gr 5	HTN DM	
8	76/F	Aneurysm, ruptured	Craniotomy	ED	Incidental	Conserve	Gr 1	HTN	Aspirin®
9	51/M	Aneurysm, unruptured	Craniotomy	ED	Incidental	Conserve	Gr 0		
10	64/F	Aneurysm, unruptured	Craniotomy	ED	Alteration in mental status	Conserve	Gr 1		

*Assessed as modified Rankin Scale. DM : diabetes mellitus, DS : decompressive surgery, ED : epidural drainage, EVD : extraventricular drainage, HTN : hypertension, ICH : intracerebral hemorrhage, LC : liver cirrhosis, LD : lumbar drainage, SDH : subdural hemorrhage

Illustrative cases

Case 2

A 68-year-old female patient was transferred to our hospital for further evaluation of an extra-axial mass on her right frontal convexity that was discovered on CT scan. MR findings were suggestive of meningioma (Fig. 1A)- approximately 85 cm^3 in size by MR volumetry- and no abnormal findings were noted in her posterior fossa. She had been on antihypertensive drugs and prophylactic aspirin for six years at the time of admission. Preoperative blood and coagulation profiles were all within normal range. She underwent a right frontal craniotomy in the supine position. Her head was rotated 20° to the left, and her blood pressure (BP) was stable throughout the whole operation, which took 7 1/2 hours. At the end of the operation, a negative drainage apparatus was installed in the epidural space. Recovery from anesthesia was delayed in the intensive care unit (ICU), and her mental status remained drowsy with left hemiparesis. An emergency CT scan revealed EDH and ICH at the site of the operation and RCH on both cerebellar hemispheres (Fig. 1B). Both ICH and RCH were evacuated surgically, and an EVD was installed intraoperatively (Fig. 1C). On returning to the ICU, the patient was drowsy and had persistent grade 3 left hemiparesis. She later developed pneumonia, as well as ventriculitis and infection at the operative site, and consequently had to undergo another operation. The neurological deficit and mental status of the patient remained stationary for the next two months, after which she was transferred to another hospital for palliation in accordance with the wishes of the family.

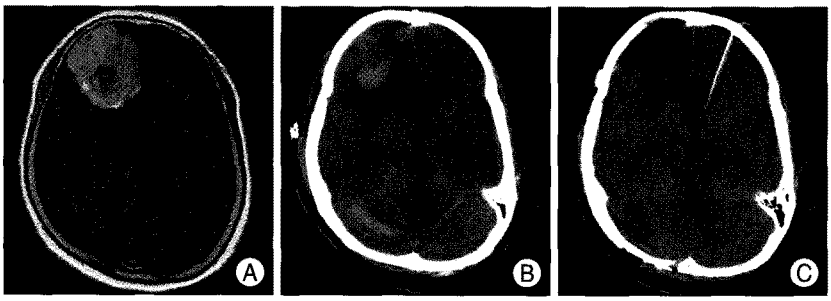

Fig. 1. A : Preoperative gadolinium-enhancement MRI demonstrating a well-enhancing extra-axial mass at the right frontal convexity. B : CT scan 6 hours after surgery revealing intracerebral hematoma at the operative site along with RCH in the right cerebellar hemisphere and both superior cerebellar folia. C : Encephalomalacia at the site of previous hemorrhage on the right cerebellum 1 month after surgery. RCH : remote cerebellar hemorrhage.

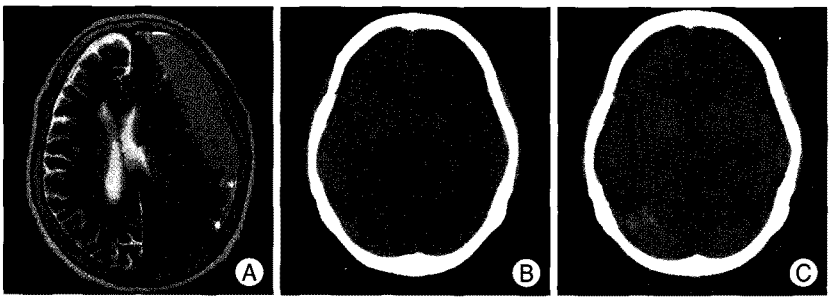

Fig. 2. A : Late, subacute phase, chronic subdural hemorrhage in the left frontoparietal area on T2-weighed MRI. B : CT on the second postoperative day demonstrating decreased subdural fluid collection and the presence of pneumocephalus, but no abnormal signal in the cerebellum C : Hemorrhage in the right cerebellum on the fifth postoperative day.

Case 4

A 74-year-old female patient presented with right hemiparesis and expressive dysphasia for three days. She had suffered a cerebral infarction 3 years before and was on clopidogrel at admission. Brain MRI revealed about 170 mL of chronic SDH (Fig. 2A). The patient also had a history of hypertension, and her BP was kept in normal range with medication. No abnormalities were found on preoperative laboratory examination. The same day, surgery was performed to drain the hematoma. The patient was placed in a supine position with her head rotated 45° to the contralateral side. Systolic BP was well preserved within normal range throughout the operation, and the whole procedure took less than an hour. On the day of surgery, a total of 100 mL of fluid was drained. On the second postoperative day, a follow-up CT scan showed that shifting of the mid-line was much eased, the brain was expanding well, and there were no abnormal findings in the pos-terior fossa (Fig. 2B). The drainage catheter was retained for 5 days, and less than 50 mL of fluid was drained each day. On the fifth postoperative day, the catheter was removed, and another follow-up CT scan revealed a new high signal density in the right cerebellar hemisphere suggestive of RCH (Fig. 2C). However, the patient was free of symptoms on detection, maybe due to small volume of RCH. She was later discharged without any neurological deficits.

Case 6

A 76-year-old male patient with sudden mental status deterioration was transferred to our emergency room. He was stuporous on admission, with a GCS score of 5. Brain CT revealed an approximately 130-mL intracerebral hematoma in his right basal ganglia,

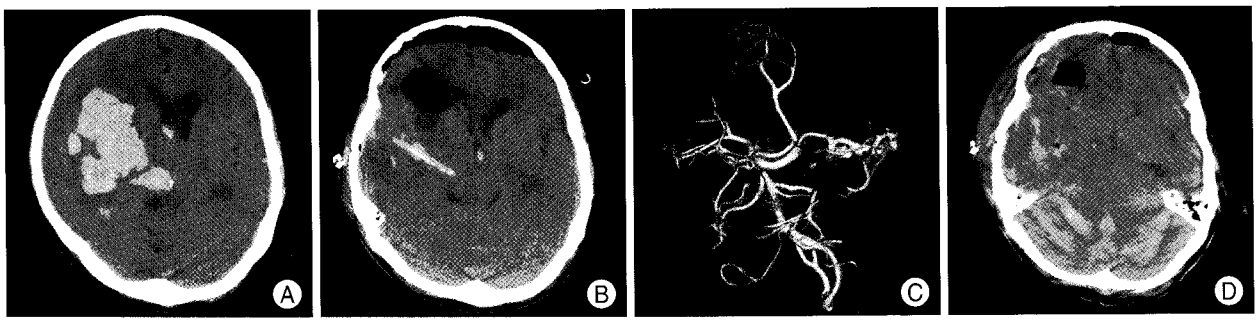

Fig. 3. A : Acute intracerebral hemorrhage in the right basal ganglia along with intraventricular hemorrhage on admission. B : CT on the first postoperative day; most of the hematoma has been removed, and pneumocephalus is present; there are no abnormal findings in the posterior fossa. C : No definite pathologic findings were detected on CT angiography D : RCH spreading over both cerebellar hemispheres and the vermis on CT, 3 days after surgery. RCH : remote cerebellar hemorrhage.

along with intraventricular hemorrhage (Fig. 3A). We learned that the patient was a heavy alcoholic and had been diagnosed with liver cirrhosis. Thrombocytopenia (platelet count : 122,000) was detected on preoperative laboratory evaluation, but prothrombin time and activated partial thromboplastin time were within normal range. Emergency surgery was performed with the patient in supine position with his head rotated 60° to the opposite side. A CT scan performed on the first postoperative day showed that the hematoma was evacuating properly (Fig. 3B). CT angiography was then performed to look for possible underlying pathology; it was reported as negative (Fig. 3C). On the third postoperative day, a follow-up CT revealed RCH in both cerebellar hemispheres and the vermis (Fig. 3D). The patient was treated non-surgically thereafter considering his poor prognosis. Forty-three days later he died of pneumonia.

Data from the literature

A total of 154 cases of RCH were reviewed from 33 articles (Table 2), and data were assessed to identify possible risk factors. Eighty-eight patients (57.1%) were male, 54 (35%) were female, and 6 were unknown sex. Their ages ranged from 10 to 83 years, with a median age of 51.

Aneurysm was the foremost initial

Table 2. Summary of reported cases in the literature

Year of publication	Authors	Number of cases	Diagnosis
1977	Yasagil MG et al.	2	Cerebral ischemia (2)
1982	Modesti LM et al.	1	Subdural hygroma
1983	Waga S et al.	1	Aneurysm
1985	Miyamoto Y et al.	1	Tumor
1987	Konig A et al.	4	Tumor (3), aneurysm (1)
1988	Ikakura K et al.	1	Aneurysm
1990	Yoshida S et al.	3	Aneurysm (3)
1993	van Calenbergh et al.	2	Aneurysm, tumor
1994	Kuroda R et al.	6	Tumor (3), AVM (1), ICA stenosis (1), Rathke's cyst (1)
1996	Brisman MH et al.	3	Tumor (2), aneurysm (1),
	Kang YG et al.	2	Aneurysm, tumor
	Lee JW et al.	3	Aneurysm (3)
	Papanastassiou V et al.	5	Aneurysm (4), Tumor (1)
	Toczek MT et al.	4	Temporal lobe epilepsy (4)
1997	Cloft HJ et al.	13	Aneurysm (12), tumor
1999	Kaplan SS et al.	1	Chronic SDH
	Koller M et al.	3	Aneurysm (1), chronic SDH (1), subdural hygromas (1)
	Park SJ et al.	4	Aneurysm (4)
	Tomii M et al.	3	Aneurysm (2), tumor (1)
	Yacubian EM et al.	3	Temporal lobe epilepsy (3)
	van Roost D et al.	2	Temporal lobe epilepsy (2)
			Aneurysm (16), temporal lobe epilepsy (15), tumor (7)
2001	Friedman JA et al.	42	CCF (1), CVM (1), arachnoid cyst (1),
			Meckel's cave Bx (1)
	Gelfenbeyn M et al.	1	Aneurysm (1)
2002	Honegger J et al.	10	Temporal lobe epilepsy (8), tumor (1) , aneurysm (1)
	Marquardt G et al.	9	Tumor (7), aneurysm (1), chronic SDH (1)
2003	Siu TLT et al.	3	Aneurysm, tumor, chronic SDH
2004	Maruyama T et al.	1	Aneurysm
2006	Amini A et al.	7	Aneurysm (3), tumor (2), arachnoid cyst (1), CVM (1)
	Jang JW et al.	6	Aneurysm (6)
	Vogels RLC et al.	2	Chronic SDH (2)
2007	Srikijvilaikul T et al.	2	Temporal lobe epilepsy (2)
	Tucker A et al.	3	Aneurysm (3)
2008	Bilginer B et al.	1	Temporal lobe epilepsy
Total	33	154	

AVM : arteriovenous malformation, CCF : carotid cavernous fistula, CVM : cavernous malformation, ICA : internal carotid artery, SDH : subdural hemorrhage

Table 3. Clinical characteristics of RCH in the literature

Characteristics	Number of cases (%)
Primary Diagnosis	
Aneurysm	68 (44.2)
Temporal lobe epilepsy	35 (22.7)
Tumor	32 (20.8)
Chronic SDH	6 (3.9)
Others	13 (8.4)
Clinical manifestation	
Alteration in mental Status	49 (31.8)
Asymptomatic	36 (23.4)
Cerebellar dysfunction	12 (7.8)
Headache	9 (5.8)
Delayed awakening	7 (4.5)
Focal neurological deficit	4 (2.5)
Seizure	4 (2.5)
Others	2 (1.2)
No description	34 (20.5)
Intervention	
Conservative	106 (68.9)
EVD/VPS	26 (16.9)
EVD/VPS + DS	10 (6.5)
DS	5 (3.2)
No description	7 (4.5)
Outcome*	
Good (Gr 0 - Gr 2)	118 (76.6)
Poor (Gr 3 - Gr 5)	21 (13.6)
Death	12 (7.8)
No Description	3 (2)

*Assessed as modified Rankin scale. DS : decompressive surgery, EVD : extraventricular drainage, RCH : remote cerebellar hemorrhage, SDH : subdural hemorrhage, VPS : ventriculoperitoneal shunt

indication for surgery (68 cases, 44.2%), followed by temporal lobe epilepsy (35 cases, 22.7%), tumor (32 cases, 20.8%) and chronic SDH (6 cases, 3.9%) (Table 3).

Perioperative hypertension was noted in 51 cases (33.1%); specifically, preoperative hypertension in 36 cases, intraoperative hypertension in 11 cases, and postoperative hypertension in 4 cases.

Coagulopathies detected on preoperative evaluation included factor VIII deficiency, pancytopenia from hepatic failure, and disseminated intravascular coagulation (DIC)[3,22,36]. Four cases (2.5%) of postoperative prophylactic heparin use[19] and 8 cases (5.1%) of aspirin use[10,29,38] were also identified.

RCH most commonly presented as an alteration in mental status; this was seen in 49 patients (31.8%). Thirty-six patients (23.4%) were asymptomatic, and 12 patients had cerebellar dysfunction (7.8%).

More than two-thirds of the RCH population (106 patients) was treated non-surgically (68.9%). Surgical intervention was used in 41 patients (26.3%). Twenty-six patients received either EVD or a ventriculoperitoneal shunt (VPS). Five had decompressive surgery (DS) alone,[12,15,18,21,33] and ten had EVD or VPS placement before DS[3,10,18,20,22,26]. The decision of whether to intervene was made based on clinical course and CT findings (i.e., extensive hemorrhage and associated hydrocephalus).

Twelve patients ultimately died, so the total case mortality rate was 7.8%. Outcomes were ranked using mRS : 118 (76.6%) patients had a good grade (Grade 0-2), and 21 (13.6%) had a poor grade (Grade 3-5).

Morbidity was difficult to determine because; 1) some of the morbidity occurred as a result of primary pathologies or other complications, 2) the follow-up period varied widely in each series, and 3) in some reports, no details were available concerning sequelae. However, if morbidity is defined as adverse signs or symptoms that can in any way be related to a cerebellar lesion on last follow-up, 13 patients (8.4%) suffered from some form of morbidity[12,19,26,27,29,31,34,35,38,39]. These ranged from mild nystagmus to severe cerebellar dysfunction with dysarthria, dysphasia, and ataxia.

DISCUSSION

Postoperative intracranial hematomas occurring at the site of surgery are not rare, and they mostly result from inadequate intraoperative hemostasis. There have been several reports concerning the development of intracranial hematomas at sites remote from the initial operation. However, remote intracerebral hematoma is still uncommon, and remote cerebellar hemorrhage is even rarer. So far, no pathomechanism has been found to properly explain its occurrence.

A blood coagulation disturbance was initially suggested as an underlying factor by König et al.[19]. In their series, all 4 patients received prophylactic heparin starting on the first postoperative day. Friedman and colleagues[10] also reported that recent preoperative usage of aspirin increased the risk of RCH. However, in reports published elsewhere, most patients had no abnormalities in their blood or coagulation profiles, nor were they using drugs that could hinder the normal coagulation cascade. Consequently, these factors are now considered to be minor contributors to RCH.

There are still some debates as to whether arterial hypertension should be considered an etiology of this complication, because hypertension itself is commonly associated with cerebellar hemorrhage and it exhibits many different characteristics-usually developing in the dentate nuclei and becoming much larger in volume[8]. RCH has been reported to develop even in the absence of arterial hypertension, and many researchers today agree that RCH is venous in origin[4,5,7,12,18,19,23,28,29,31,33,37,39]. However, we noted many

articles in which a large number of patients were hypertensive before they presented with RCH, including our series[14,18,21,22,26,29,33,38]. The overall incidence of hypertension was 33.1% among patients with RCH. A recent retrospective study also supported the observation that intraoperative hypertension is strongly associated with the development of postoperative cerebellar hemorrhage[10]. Siu and colleagues[29] suggested that arterial hypertension may not be the primary mechanism behind RCH, but it may aggravate bleeding by elevating the transmural pressure in ruptured veins and converting an initially innocuous venous hemorrhage to a clinically detectable complication.

Direct venous obstruction in relation to head rotation during surgery was advanced as another possible mechanism behind RCH. Many authors have suggested that the rotation and extension of the neck required in frontotemporal craniotomy could occlude the jugular veins and lead to venous hypertension and RCH[3,26,33]. Seoane and Rhoton[28], in their anatomical study of the internal jugular veins of 36 cadaveric specimens, reported that the vein was indented by the transverse process of the atlas in a significant proportion of the cadavers they studied (14 of 36, 39%). They believed this might further aggravate the obstruction and cause venous hypertension when coupled with head rotation and neck extension. However, many authors have produced evidence contrary to this theory. Cloft et al.[7] reported, in their radiological review, that the jugular veins and sigmoid sinuses were widely patent bilaterally in the imaging records from 11 of 12 RCH cases. They postulated that even if one vein was totally obstructed, the contralateral venous outflow could suffice. Yacubian and colleagues[35] argued that, if clinically significant venous obstruction occurred, cerebral edema would be apparent to the surgeon. In the report of Honegger et al.[12], no significant hemorrhage was ever detected on CT scans taken in the first postoperative hour, which demonstrates the characteristic delayed onset of this complication. We believe that RCH developing after spinal surgery serves as evidence against the theory, and we conclude that the head rotation employed in craniotomy is unlikely to contribute to RCH.

In reviewing the articles, we found that most of the RCH cases shared a handful of characteristic features : 1) hemorrhage occurred mostly in the cerebellar cortex facing the tentorium, which differed clearly from hypertensive cerebellar hemorrhage in which dentate nuclei are the most common source of hemorrhage; 2) most cases also had subarachnoid hemorrhage distributed over the superior surface of the cerebellum, resulting in a typical streaky bleeding pattern (zebra sign)[5]; 3) presentation was incidental in many cases, and even in cases with symptomatic deterioration, the patients recovered from anesthesia and then deteriorated, suggesting that the hemorrhage probably occurred after surgery; 4) a continuous drainage apparatus was used with negative pressure in most cases. Considering these features together, most authors agree on two facts : 1) RCH is venous in origin, and most likely the superior vermian vein is affected; 2) RCH is likely the result of intraoperative and, even more likely, postoperative loss of CSF.

Based on this assumption, a few hypotheses have been advanced concerning the mechanism by which venous channels remote from the site of operation can be damaged. Konig et al.[19] first suggested that the significant reduction in intracranial pressure occurring after the removal of supratentorial space-occupying lesions and drainage of CSF lead to a reciprocal rise in venous pressure and give rise to hemorrhage. Yoshida et al.[39] later speculated that, in the postoperative intracranial environment in which the brain is slack and the CSF space occupies a relatively large part of the intracranial cavity compared to the normal state, drainage of CSF may be accelerated by downward displacement of the cerebellum and resultant stretch of the superior vermian vein and its tributaries, leading to hemorrhage. Friedman and colleagues[10] agreed with the downward displacement hypothesis, but they believed that venous occlusion caused by stretching of the veins and hemorrhage was likely to be a manifestation of cerebellar venous infarction. However, Honegger and colleagues[12], in their recent report, rejected the above hypothesis, arguing that obstruction of cerebellar veins is unlikely to lead to RCH because deliberate obliteration of supracerebellar veins during infratentorial surgery would not produce cerebellar hemorrhage. Instead, they postulated that supratentorial CSF loss induces a transtentorial pressure gradient, and this gradient causes a suction effect influencing the infratentorial veins and the capillary beds of the cerebellum. However, this assumption is rebutted by the fact that RCH may occur after spinal loss of CSF[5,6,9,17,37], a situation in which cerebellar displacement occurs in the caudal direction. With all hypotheses considered, most authors consider removal of the supratentorial mass or massive CSF over-drainage causing caudal displacement of the cerebellum to play some role in RCH. This is supported by the fact that RCH most frequently occurs after aneurysm surgery, temporal lobectomy, or tumor surgery in which there is intraoperative cisternal drainage or removal of supratentorial contents. In our series, eight patients also primarily underwent surgery for either aneurysm or tumor.

There have even been rare cases of RCH after supratentorial surgeries other than craniotomy. These include five cases of burr hole drainage[18,25,36]. One case was actually

seen in our institution. Three patients received bilateral burr holes, and another patient had a past history of brain tumor resection. Consequently, the amount of drained fluid was suspected to be large. We believe that these cases lend credence to the pathomechanism mentioned above.

Although there have been some reports on mortality[10,18,19,21,22,25,29,38], the case mortality rate of RCH (7.8%) following supratentorial surgery derived from our review is much more favorable compared with that (20-75%) of spontaneous cerebellar hemorrhage reported in the literature[8]. This corresponds with the suggested benign nature of this condition, with venous origin and small hemorrhage volume. Therefore, conservative treatment is satisfactory in most patients, and surgical intervention is directed toward decompression in cases of acute obstructive hydrocephalus, either through indirect ventricular drainage or through direct posterior fossa craniectomy. Large hematomas necessitating emergent evacuation rarely occur. Furthermore, according to the report of Brockmann et al.[4], only hemorrhage severity and patient age are significant prognostic factors.

The morbidity associated with this complication was difficult to determine, as mentioned earlier, but we estimate it to be 8.4% based on our review[12,19,26,27,29,31,34,35,37,39]. In cases with long-term follow up, however, recovery from symptomatic cerebellar hemorrhage was quite good. Most patients had complete resolution on follow-up examination[1,10,12,14,20,27,30-37], and only a small numbers of patients were reported to suffer from severe morbidity[3,10,12,15,22]. The seven patients with good grades in our own study reinforce this finding.

Correction of preexisting risk factors such as coagulopathies, hypertension, and antiplatelet medication effect are recommended, as in any routine elective operation. Although minimization of intraoperative CSF loss or removal of intracranial contents might reduce postoperative RCH, it is unlikely this would outweigh the benefits of sufficient cerebral relaxation and exposure or adequate removal of the lesion. Consequently, the only actively adjustable risk factor is postoperative CSF drainage. Yoshida et al.[39] recommended the use of subgaleal drainage instead of epidural drainage and suggested opening drainage apparatuses in atmospheric pressure. However, literature[10] published elsewhere suggests that RCH may occur even when tubes are drained without negative pressure from the subgaleal space. Brockmann et al.[5] insisted on an infusion of Ringer solution to replace lost CSF in the setting of RCH, in order to prevent further aggravation. This, on the other hand, may worsen the situation when there is occlusive hydrocephalus, so extreme caution should attend its practice. The amount of drainage can be modulated, but there are no established guidelines yet, and further study is required.

CONCLUSION

RCH is a rare complication after supratentorial surgery. It usually occurs after surgeries entailing the removal of a large amount of CSF or supratentorial cerebral contents, such as aneurysmal surgery or tumor surgery. The exact etiology still remains uncertain, and among the suggested risk factors hypertension, intraoperative opening of the cistern, and postoperative CSF drainage seem to play some role in the development or aggravation of this entity. However, none of these is definitely responsible based on the rarity of this complication. Apparently, the only adjustable factor related to RCH is postoperative drainage of CSF. Therefore, future study should focus on defining the correlation between the amount of fluid loss and the severity of RCH. Once RCH has occurred, treatment should be administered in relation to the symptoms because surgical intervention is usually related with acute obstructive hydrocephalus. Therefore, close monitoring and appropriate follow-up imaging studies are essential, especially in elderly patents and those with massive hemorrhage. Apparently, outcomes in patients who survive this complication are generally good.

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