Remote cerebellar hemorrhage: A case report

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Abstract

Remote Cerebellar Hemorrhage is a rare entity that manifests spontaneously after supratentorial craniotomy and spinal surgeries. We present a 53-year-old male who was admitted due to subdural hematoma along the left frontoparietotemporal convexity. After treatment of the subdural hematoma with craniotomy and evacuation, he developed remote cerebellar hemorrhage 1 week later. Brain computed tomography demonstrated the zebra sign. Follow-up imaging showed complete recovery without any neurologic symptoms or signs.

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Introduction

Remote Cerebellar Hemorrhage (RCH) is a rare entity that manifests spontaneously after supratentorial craniotomy and spinal surgeries. RCH is a self-limiting condition which may rarely evolve into a devastating complication, however, these complications are preventable if certain precautions are implemented.

Case Report

We present a case of a 53-year-old male with a history of left-sided head trauma two months prior. He presented with left-sided headaches of two weeks duration. There was no past medical history of hypertension, epilepsy, or liver disease. He was not on antiplatelets or anticoagulants therapy. Physical examination was negative for motor weakness and sensory loss. Cranial nerves were intact, no cerebellar signs, and no gait abnormalities. Laboratory workup was unremarkable; platelet count was normal range.

As standard workup, brain CT scan was done which demonstrated a large left frontoparietotemporal convexity subdural hematoma with associated mass effect, and an 11 mm left to right midline shift (Fig. 1). Subsequently, the subdural hematoma was treated with emergent left frontoparietal craniotomy and evacuation. On the first postoperative day, repeat brain CT showed what was thought to be an extra-axial hemorrhage. The discovery prompted re-exploration; however, chronic clot was found instead and removed (Fig. 2). One
week after the initial craniotomy, brain CT showed a new finding of bilateral cerebellar hematomas associated with partial effacement of the fourth ventricle (Fig. 3).

The leading diagnosis for a new finding of cerebellar hemorrhage, appearing on CT imaging as alternating hyperdense and hypodense areas (zebra sign) in a patient who recently underwent supratentorial surgery, is remote cerebellar hemorrhage (RCH). Other possible causes of cerebellar hemorrhage include cerebellar stroke.

Following the detection of cerebellar hemorrhage distant from the craniotomy site, remote cerebellar hemorrhage was established as the diagnosis. Since the patient didn’t display any signs of complications, such as increased intracranial pressure caused by further bleeding or hydrocephalus, treatment was conservative.

Follow-up brain CT, five months after the craniotomy, demonstrated complete resolution of the bilateral cerebellar parenchymal hematomas and interval development of small areas of encephalomalacia. No neurological abnormalities were identified (Fig. 4).

Discussion

Remote cerebellar hemorrhage is a rare complication that manifests after supratentorial and rarely spinal surgeries. It often appears on brain CT scan in a pattern known as the zebra sign [1,2].

The zebra sign is a unique pattern caused by subarachnoid bleeding into the cerebellar sulci facing the tentorium. It is characterized as areas of alternating hyperdense blood filled sulci and hypodense cerebellar gyri, a pattern comparable to zebra skin [2-8] (Fig. 3). The zebra sign, a common imaging feature of RCH, is found in 64% of cases. Less frequently, intracerebellar hemorrhage and mixed patterns have also been described in the literature [1].

RCH incidence, according to Papanastassiou et al. and König et al. ranges from 0.2% to 0.4% after surgeries requiring supratentorial craniotomies [5,6]. Supratentorial craniotomies are performed to treat intracranial aneurysms by clipping, tumors by debulking, epilepsy and vascular malformations by lobectomies, and hematomas by evacuation. Bilateral distribution of RCH is present in 55% of cases, slightly more prevalent than unilateral distribution [1].

Clinically, RCH may present with cerebellar signs, signs of increased intracranial pressure due to hydrocephalus or large hematoma, and brain stem signs due to herniation. However, in the literature, one-fourth of cases were identified incidentally, and of those who developed symptoms, 80% occurred within the first 24 hours after craniotomy [1]. In radiological literature Amini et al. [9] reported eight cases of remote cerebellar hemorrhage, three of which developed cerebellar symptoms, three were asymptomatic which are comparable to the
Fig. 2 – Axial brain CT scan subdural window. (A) and (B) performed after the first decompression of the subdural hematoma at presentation. (A) Demonstrating residual extra-axial hematoma on the left side of the brain (white arrow). (B) Cerebellum view showing no abnormalities. (C) and (D) axial brain CT scan after the second decompression surgery showing near complete resolution of hemorrhage with mild residual blood products and pneumocephalus but no abnormalities in the cerebellum.
Fig. 3 – Axial brain CT scan at the level of cerebellum, in subdural window performed 1 week after initial craniotomy. (A) Showing bilateral cerebellar hemorrhages (white arrow) which closely resemble the zebra sign in addition to elements of interstitial hemorrhage (mixed pattern). (B) The white arrow points toward a slight downward extension of the right cerebellar hemorrhage compared to the left side.

Fig. 4 – Axial brain CT scan subdural window. (A) Follow-up image prior to discharge showing bilateral cerebellar hemorrhages. (B) Five-month follow-up images after discharge showing complete spontaneous resolution of the bilateral cerebellar hemorrhages.
presented case, one developed nausea, and one has passed away due to the poor prognosis following head shot gun injury. All symptoms were self limited during follow up.

Many risk factors have been associated with RCH such as hypertension, epilepsy, antiplatelets/anticoagulants, compromised coagulation status such as in liver cirrhosis, excess CSF loss during and after surgery, and old age [3,6–8]. The mechanisms by which RCH develops are widely debated. However, there is a general consensus that bleeding origin is venous, most likely from bridging veins of the cerebellar tentorial surface [4,10]. CSF fluid loss and shifting of the cerebellum downward leading to stretching and tearing of veins, is the theory postulated by Yoshida et al [8]. In contrast, Konig et al [6] theorized that increased pressure gradients across veins generated after removal of space occupying lesions such as tumors is a possible mechanism by which RCH develops, a theory based on studies by Welch [11] and Courten et al [12].

Treatment of RCH is dependent on the clinical picture and imaging findings. Most cases resolve spontaneously, for which conservative management is adopted. Often complications may arise which warrant urgent intervention, some examples include: hydrocephalus which is treated by external ventricular evacuation, and compression of the infratentorial region due to bleeding which is treated by suboccipital decompression. As a preventive measure, avoidance of large CSF volume loss during craniotomy and spinal surgery is advised. If unavoidable, replacement with isotonic solution could be an effective measure [1,5].

In conclusion, RCH is a rare complication which may have a devastating outcome if left undiagnosed and untreated. Further knowledge of this entity helps with implementation of preventive measures and initiation of early interventions.

**Supplementary materials**

Supplementary material associated with this article can be found, in the online version, at doi:10.1016/j.radcr.2018.12.006.

**REFERENCES**

[1] Sturiale CL, Rossetto M, Ermani M, Volpin F, Baro V, Milanese L, et al. Remote cerebellar hemorrhage after supratentorial procedures (part 1): a systematic review. Neurosurg Rev 2016;39(4):565–73.
[2] Baeesa SS. Remote cerebellar hemorrhage in neurosurgery. Neurosciences 2012;17(4):305–8.
[3] Brockmann M, Groden C. Remote cerebellar hemorrhage: a review. Cerebellum 2006;5:64–8.
[4] Brockmann M, Nowak G, Reusche E, Russlies M, Petersen D. Zebra sign: cerebellar bleeding pattern characteristic of cerebrospinal fluid loss. J Neurosurg 2005;102:1159–62.
[5] Papanastassiou V, Kerr R, Adams C. Contralateral cerebellar hemorrhagic infarction after pterional craniotomy: report of five cases and review of the literature. Neurosurgery 1996;39(4):841–51.
[6] Konig A, Laas R, Herrmann HD. Cerebellar haemorrhage as a complication after supratentorial craniotomy. Acta Neurochir 1987;88(3–4):104–8.
[7] Huang C-Y, Lee P-H, Lin S-H, Chuang M-T, Sun Y-T, Hung Y-C, et al. Remote cerebellar hemorrhage following supratentorial craniotomy. Neurol Res 2012;34(5):422–9.
[8] Yoshida S, Yonekawa Y, Yamashita K, Ihara M, Morooka Y. Cerebellar hemorrhage after supratentorial craniotomy—report of three cases. Neurol Med Chir 1990;30:738–43.
[9] Amini A, Osbourn AG, McCall TD, Couldwell WT. Remote cerebellar hemorrhage. Am J Neuroradiol 2006;27(2):387–90.
[10] Ueyama T, Al-Mefty O, Tamaki N. Bridging Veins on the Tentorial Surface of the Cerebellum: A Microsurgical Anatomic Study and Operative Considerations. Neurosurgery 1998;43(5):1137–45.
[11] Welch K. The intracranial pressure in infants. J Neurosurg 1980;53:693–9.
[12] Courten G, Rabinowicz T. Intraventricular Hemorrhage in Premature Infants: Reappraisal and New Hypothesis. Devlp Med Child Neurol 1981;23:389–403.