



Case Report

Critical cerebellar hemorrhage due to pilocytic astrocytoma in a child: A case report

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ABSTRACT

Background: Cerebellar hemorrhage is rare in children, and its cause is usually vascular disorders such as arteriovenous malformations or hematological disorders.

Case Description: A previously healthy 10-year-old girl presented with a loss of consciousness following sudden headache and vomiting. A non-contrast brain computed tomography (CT) scan revealed a massive cerebellar hemorrhage with obstructive hydrocephalus; however, subsequent CT angiography (CTA) showed no vascular abnormalities. An emergency craniotomy was performed to evacuate the hematoma, and histological analysis of the specimen obtained from the tissue surrounding the hematoma revealed a pilocytic astrocytoma (PA). Six months after the ictus, her recovery was scored at 2 on the modified Rankin Scale.

Conclusion: PA can be a cause of critical cerebellar hemorrhage. In this case of life-threatening massive hematoma, CTA was useful to exclude a major vascular pathology and to save time.

Keywords: Cerebellar hemorrhage, Computed tomography angiography, Intratumoral hemorrhage, Pediatric stroke, Pilocytic astrocytoma

INTRODUCTION

Hemorrhagic stroke in the cerebellum is rare in children, and the main causes are vascular lesions such as arteriovenous malformations (AVMs) and coagulopathy/thrombocytopenia related to hematological disorders.^[1,11,21] Six to nine percent of all pediatric intracranial hemorrhages (ICHs) are caused by brain tumors, which are usually malignant.^[11,21] We report a case of critical cerebellar hemorrhage due to pilocytic astrocytoma (PA).

CASE PRESENTATION

A previously healthy 10-year-old girl presented with a sudden loss of consciousness following sudden headache and vomiting. There were no antecedent symptoms, such as headache or ataxic gait, according to the parents. On admission, she was comatose, with a Glasgow coma scale (GCS) score of 6 and a blood pressure of 113/84 mmHg. Her pupils were isocoric, and the light reflex was maintained. She was intubated for respiratory assistance and sedated. Brain computed tomography (CT) revealed a cerebellar hemorrhage 5 cm in size with slight obstructive hydrocephalus [Figure 1a]. Subsequent CT angiography (CTA) did not show major vascular abnormalities, such

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as an AVM or a cerebral aneurysm [Figure 1b]. An emergent surgery was performed to evacuate the hematoma, and a piece of yellowish, soft tissue was obtained from the cavity wall during evacuation. The patient recovered consciousness 3 days after the operation with temporary (cerebellar) mutism. Pathological examination revealed a PA [Figure 2]. The patient recovered to a modified Rankin scale score of 2, 6 months after onset, and she was followed with MRI [Figures 3a and b] without additional treatment.

DISCUSSION

A rare case of massive cerebellar hemorrhage in a child due to PA was presented. CTA was useful to exclude a major

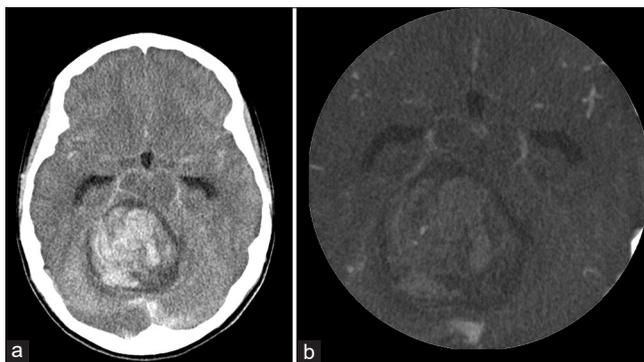


Figure 1: (a) Non-contrast computed tomography (CT) shows a massive cerebellar hematoma with slightly obstructive hydrocephalus. (b) CT angiography did not show apparent arteriovenous malformation or a cerebral aneurysm.

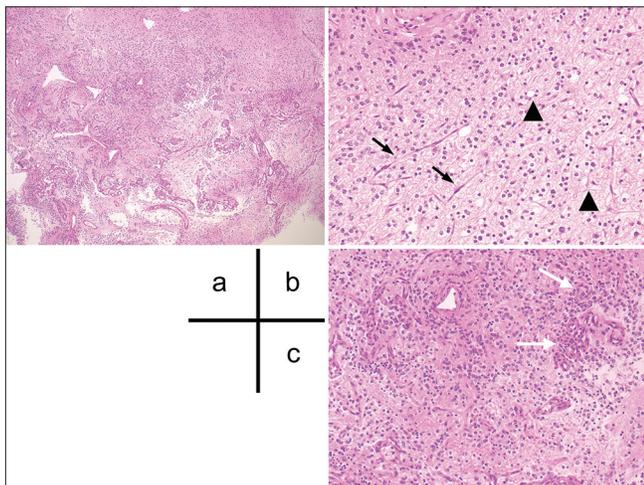


Figure 2: Histopathological examination of the specimen. (a) Hematoxylin-eosin (HE) staining shows a biphasic pattern with varying proportions of piloid areas alternating with spongy areas with high vascularity. (b) Bipolar neoplastic cells with elongated hair-like processes (black arrow) and oligodendrogloma-like cells with cytoplasmic vacuoles (arrowhead). (c) Glomeruloid-like vascular proliferation (white arrows) is visible.

vascular disorder and to save time in the treatment of this critical condition.

Hemorrhagic stroke accounts for approximately one-half of all cases of pediatric stroke, with an annual incidence of 1.2–13 cases/100,000;^[11,21] however, cerebellar hemorrhage is rare.^[1,16] Approximately 50% of all pediatric ICHs are due to vascular abnormalities such as AVMs, aneurysms, and cavernous malformations, while 20–23% are attributable to coagulopathy or thrombocytopenia related to hematological disorders, and only 6–9% are caused by brain tumors.^[11,21] Intratumoral hemorrhage usually occurs in malignant tumors, such as glioblastoma, oligodendroglioma, or metastatic brain tumors,^[26] whereas in benign tumors, such as low-grade astrocytoma, a hemorrhagic presentation is very rare.^[15] PA is the most common posterior fossa tumor in children.^[17] It is classified as Grade I by the WHO classification,^[12] and its clinical course is usually favorable in terms of survival rate and functional outcome. However, pathological studies of PA or pilomyxoid astrocytoma have revealed that intratumoral hemorrhage is not rare in PA and is found in 8–11.4% of cases.^[18,24] Sixteen cases of cerebellar hemorrhage from PA with various clinical presentations have been previously reported.^[2-5,7,9,10,13-15,18-20,22,23,25] [Table 1]. The etiology of intratumoral hemorrhage in PA is unclear, but in the present case, the tumor tissue had abundant microvasculature with a glomeruloid-like appearance [Figure 2]. The previous reports pointed out that other pathological features in hemorrhagic PA, such as endothelial proliferation, encased aneurysm rupture, and dysplastic capillary beds, are all considered to be potential causes for bleeding.^[6-8,19,24] These fragile microvasculatures can cause hemorrhages.

According to a previous report, initial GCS is the prognostic factor of cerebellar hemorrhage from PA.^[2] Massive cerebellar hemorrhage is life threatening and often requires emergent evacuation, whereas intraoperative hemorrhage from unknown vascular pathology can be lethal, especially in

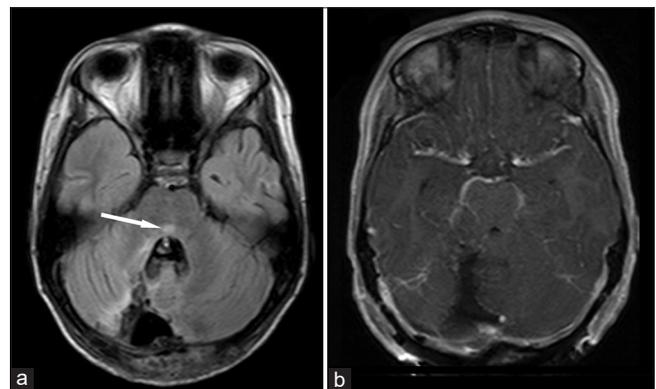


Figure 3: (a) Postoperative fluid-attenuated inversion recovery shows high intensity in the right middle cerebellar peduncle, cerebellar hemisphere, and dorsal midbrain (arrow). (b) Gadolinium-enhanced MRI shows no residual lesion.

Table 1: Previous reports on cerebellar hemorrhage from pilocytic astrocytoma.

Author	Age/sex	Initial onset	Aura before stroke	Timing of surgery	Outcome	Others
Mauersberger <i>et al.</i> ^[13]	10 y/M	Severe coma	Choreatic movements 2 days prior	Next day	Ataxia, dysarthria	EVD before operation
	10 y/F	Epilepsy	Headache and vomiting 10 months prior	Emergency	Died 2 weeks later as a result of infection	Rebleeding VPS was placed
Fogelson <i>et al.</i> ^[3]	9 y/F	Headache/vomiting	-	5 days later	Ataxia, dysarthria	-
Vincent <i>et al.</i> ^[23]	14 y/F	Headache/vomiting	-	Emergency	Good recovery	-
Specht <i>et al.</i> ^[20]	8 y/M	Severe coma	Vomiting 1 week prior	-	Died 12 days later	EVD was placed Diagnosed at autopsy
Meshiwala <i>et al.</i> ^[14]	13 y/M	Headache	Headache and balance difficulties 10 days prior	2 days later	Mild ataxia	-
Shibahara <i>et al.</i> ^[18]	8 y/M	Headache/vomiting	Headache and nausea 4 weeks prior	Done (NOS)	Survived (NOS)	Comorbidity with NF-1
Frassanito <i>et al.</i> ^[4]	7 y/F	Headache/vomiting	-	2 months later	Mild dysarthria	-
Lee <i>et al.</i> ^[10]	15 m/M	Vomiting	-	Emergency	Survived (NOS)	VPS was placed
Kumar <i>et al.</i> ^[9]	16 y/F	Vomiting	Gait disturbances 2 weeks prior	Done (NOS)	Survived (NOS)	-
Wilson <i>et al.</i> ^[25]	12 y/M	Severe coma	Headache 1 year prior	-	Died next day	EVD was placed Diagnosed at autopsy
	5 y/F	Moderate coma	-	Emergency	Died (NOS)	Diagnosed at autopsy
Ramdurg <i>et al.</i> ^[15]	9 m/NA	Drowsiness	-	Emergency	Survived (NOS)	Comorbidity with thrombopenia
Gaha <i>et al.</i> ^[5]	2 3/M	Headache	3 months	-	Good	
Sun <i>et al.</i> ^[22]	62 y/M	Gait disturbance/ headache	10 days	-	Good	
Donofrio <i>et al.</i> ^[2]	9 y/M	Headache/vomiting	2 weeks	Emergent	Good	
Present case	10 y/F	Severe coma	Sudden	Emergency	Slight ataxia	-

y: Years, M: Male; F: Female

a child. In such cases, CTA is much less time-consuming than other modalities, such as catheter angiography or MRI, to exclude the possibility of a difficult-to-treat AVM or cerebral aneurysm, although the possibility of an occult small AVM cannot be excluded.

In the operation, there was marked hemorrhage, presumably due to the fragile microvasculature of the tumor, while we could distinguish soft, yellowish tissue during evacuation of the hematoma, which could have been removed easily with suction. Although CTA showed no vascular anomaly, we suspected that there was a vascular origin of the hemorrhage, such as a small AVM or hemorrhagic tumor. Close inspection and biopsy are important not only after hemostasis but also during hematoma evacuation for definitive diagnosis. It is not usually easy to distinguish small pathology while controlling the hemorrhage and finding the origin, but meticulous control of the suction pressure is important to identify the

etiology of a hemorrhage that is difficult to explain. On the other hand, as the purpose of an emergent surgery is to save the patient's life and to minimize neurological deficits. As past reports have shown [Table 1], the prognosis for patients who presented with coma at the onset of the disease tends to be poor. Hence, focusing on early decompression of the hematoma and hemostasis is more important. In our case, as the pathology was PA and postoperative MRI showed no residual tumor, no additional surgery was indicated. However, if there is residual tumor after the operation, a planned second surgery with adequate preparation, such as a neuronavigation system, should contribute to achieving a better outcome.

CONCLUSION

A case of a patient with pediatric PA presenting with massive cerebellar hemorrhage was presented. CTA was useful to exclude the diagnosis of major vascular disorder and to save

time, and close inspection and biopsy of the hematoma cavity were important for establishing a definitive diagnosis.

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Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

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Conflicts of interest

There are no conflicts of interest.

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