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Pediatric cerebellar hemorrhages

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Abstract Cerebellar hemorrhage is a devastating condition with morbidity and mortality related not only to the etiology of the hemorrhage, but also to the timing of the intervention. Sixteen consecutive pediatric patients with acute cerebellar hemorrhages are presented: 6 had vascular abnormalities, 3 had tumors, and 2 had hemorrhages of unknown etiology. Thirteen of the 16 patients survived with only 1 of the 13 having persistent vegetative state as a neurologic outcome. Six of 8 patients presenting in a moribund condition had good outcomes, and 3 of 4 patients presenting with fixed and dilated pupils also had good outcomes. Thus, in contrast to adults, rapid evaluation by CT scanning, followed by the judicious use of ventricular drainage and prompt surgical treatment, have resulted in favorable outcomes in pediatric patients despite their poor clinical presentations. None of the neonates having cerebellar hemorrhages required surgical intervention; their courses could be followed clinically and with transfontanel ultrasound.

Key words Cerebellar hemorrhage · Pediatric arteriovenous malformations · Hemorrhagic pediatric tumors

Introduction

The widespread availability of neuroimaging facilities has made earlier diagnosis and treatment of cerebellar hemorrhages possible [2]; however, cerebellar hemorrhage is still a devastating disease with morbidity and mortality related not only to the etiology but also to the timing and success of neurosurgical intervention. Kobayashi et al. [6] have proposed treatment criteria for adult patients having hypertensive cerebellar hemorrhages, concluding that absent brain-stem reflexes and flaccid tetraplegia should preclude aggressive therapy. They stated that only patients having Glasgow Coma Scale scores of 13 or less with hemorrhages 4 cm or more in diameter should have surgical treatment.

These criteria have been challenged on the basis that they exclude potentially salvageable patients from surgical treatment [11]. In children cerebellar hemorrhages are usually associated with arteriovenous malformations [15], tumors [12, 16] in the posterior fossa, blood dyscrasias, and trauma; they are rarely caused by hypertensive vascular disease. The criteria for surgical intervention in pediatric patients with acute cerebellar hemorrhages are therefore quite different from those in adults. This report presents the etiologies, neurosurgical management, and outcomes in 16 consecutive pediatric patients presenting with acute cerebellar hemorrhages.

Patients and methods

Sixteen consecutive pediatric patients ranging in age from newborn to 18 years were treated for cerebellar hemorrhages. Nine were males and seven females. Six patients had vascular abnormalities; five of these were arteriovenous malformations and one was a vertebral artery aneurysm. Figure 1 shows the neuroimaging studies of a patient who had an arteriovenous malformation. Three patients had hemorrhages associated with tumors of the posterior fossa. The CT scan of a patient with a mixed oligodendroglioma/astrocytoma is shown in Fig. 2. Two patients had coagulopathies, one associated with extra-

Table 1 Etiology of cerebellar hemorrhages in 16 pediatric patients

Etiology	No. of patients
Arteriovenous malformation	5
Aneurysm	1
Posterior fossa tumor	3
Trauma	3
Coagulopathy	2
Unknown	2
Total	16

corporal membrane oxygenation and another with a factor VIII deficiency. Three patients sustained craniocerebellar trauma, and two had hemorrhages whose etiologies remain uncertain. Table 1 summarizes the etiologies of these cerebellar hemorrhages. Four patients presented with dilated and fixed pupils. Eight patients presented with impaired consciousness, seven of these having motor abnormalities and impaired brain-stem reflexes. The three patients sustaining craniocerebral trauma presented only with headache.

Ten patients underwent surgical intervention; two had placement of external ventricular drains alone, and eight underwent suboccipital craniotomies for removal of the cerebellar hematoma alone in two cases, removal of arteriovenous malformations in four cases, and removal of cerebellar tumors in two. All of the patients undergoing suboccipital craniotomy had placement of external ventricular drains as well. Five patients, two with coagulopathies, two with craniocerebellar trauma, and one with a hemorrhage of unknown origin, did not require surgical intervention.

Results

There were three deaths. A 14-year-old boy with a giant vertebral artery aneurysm died at home prior to receiving scheduled definitive neurosurgical treatment. His CT scan, made prior to rupture of the aneurysm, is shown in Fig. 3. A 13-year-old girl died following surgical evacuation of her hemorrhage. The etiology of her hemorrhage was not determined at surgery, and she had arrived for neurosurgical treatment with pinpoint pupils, posturing, without brain-stem reflexes, and in cardiopulmonary collapse. The third patient, a 14-year-old girl, also clinically brain-dead with a massive arterial hemorrhage, died despite placement of an external ventricular drain.

Of the 13 survivors, all but one had good outcomes. The exception was a patient with an arteriovenous malformation who had removal of his hematoma only. He remained in a persistent vegetative state and subsequent definitive treatment of his arteriovenous malformation therefore was withheld. Three of four patients who presented with dilated and fixed pupils made a good recovery. Six out of eight patients initially moribund ultimately had good outcomes, and two expired. Three patients with traumatic cerebellar hemorrhages did not require surgical evacuation of their clots. In two, the clot resolved spontaneously without producing obstructive hydrocephalus, while in the third patient increased intracranial pressure was ascertained by transcranial Doppler and serial neuroimaging studies. He required an external ventricular drain until his hemorrhage resolved. All three had good outcomes. Neither patient having a cerebellar hemorrhage from a coagulopathy required surgery. Table 2 lists the outcomes of all of the patients, and Table 3 relates outcomes to initial clinical presentation.

Table 2 Outcomes of all 16 patients treated for cerebellar hemorrhages

Outcome	No. of patients
Death	3
Vegetative state	1
Good outcome	12

Table 3 Outcomes of patients related to initial clinical presentations

Initial presentation	No. of patients	Outcome		
		Good outcome	Death	Vegetative state
Moribund	8	6	2	0
Fixed and dilated pupils	4	3	0	1

Discussion

In a study of the natural history of arteriovenous malformations of the brain, Fults and Kelly [4] concluded that the prognosis was poor for patients with posterior fossa arteriovenous malformations. The mortality rate in their series was 67% with the first hemorrhage. Further, recurrent posterior fossa hemorrhage was the rule among the unoperated survivors and most of the secondary hemorrhages were also fatal. They also determined that the prognosis for arteriovenous malformations in children was no different from that in adults. Certainly, in our five patients with arteriovenous malformations of the cerebellum, the patients presented with the characteristic rapid deterioration following the sudden onset of headache and rapid progression to a moribund condition with impaired brain-stem reflexes. Despite their initially terrible neurologic condition, rapid intervention resulted in good outcomes for all patients but one.

In adults, hemorrhage into metastatic brain tumors may account for 5% of all intracerebral hemorrhages [7], but hemorrhages into primary brain tumors are less frequent. The incidence of metastatic tumors to the posterior fossa of children would be expected to be very low. Vincent et

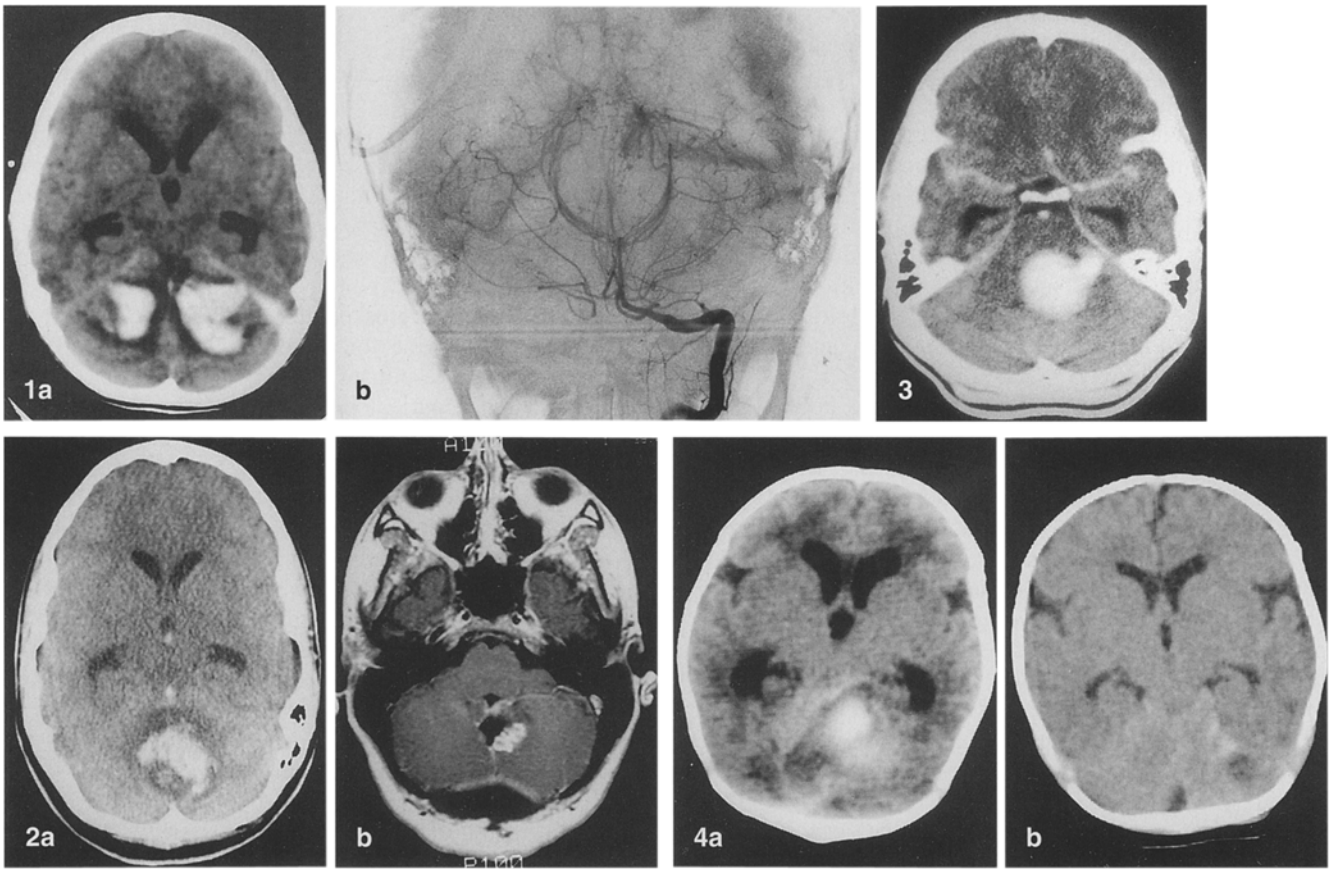

Fig. 1 **a** The CT scan of a patient with an acute cerebellar hemorrhage caused by rupture of an arteriovenous malformation shows bilobar hemorrhages. An external ventricular drain is in place, but the ventricles have not been collapsed. **b** The angiogram (anteroposterior view) of the same patient, made after evacuation of the cerebellar hemorrhage, shows the vascular malformation

Fig. 2 **a** The CT scan of a patient with a hemorrhage from her posterior fossa tumor shows the large clot and acute hydrocephalus. **b** An MR scan made following clot and partial tumor removal shows residual cerebellar tumor

Fig. 3 Axial view of a CT scan, contrast-enhanced, shows a large vertebral artery aneurysm. Its rupture was fatal

Fig. 4 **a** The CT scan of a newborn with a spontaneous cerebellar hemorrhage, unassociated with trauma or coagulopathy, shows the relatively large clot and ventriculomegaly. **b** Follow-up CT scan of the same patient made 6 weeks later shows resolution of the hemorrhage and marked decrease in the size of the ventricular system

al. [16] reported a cerebellar hemorrhage occurring in a child with a juvenile pilocytic astrocytoma. Prompt removal of the 4-cm hematoma and the associated midline tumor resulted in a good functional recovery, similar to our patient with a juvenile type of cerebellar astrocytoma. In Oldberg's series [10] of 832 patients with gliomas, hemorrhages occurred most frequently in oligodendrogliomas. Our second patient hemorrhaging from a posterior fossa brain tumor had a mixed oligodendroglioma/astrocytoma of the cerebellum totally resected, but required reoperation to complete the tumor resection. An MR scan 14 months later showed no evidence of disease. Poon and Solis [12] have reported a spontaneous cerebellar hemorrhage in a 4-year-old girl having an unsuspected ependymoma, another common posterior fossa tumor in children. She died within 5 h of admission without surgery.

One of our patients had a glomus jugulare tumor removed several years prior to its recurrence. Seven weeks after CT-documented gross total removal of the recurrent tumor from the posterior fossa, a massive posterior fossa hemorrhage occurred, presumably due to some compromise in vascular integrity. The child presented to an emergency room with a massive posterior fossa and intraventricular hemorrhage. External ventricular drainage alone did not improve her condition, and she subsequently died. The etiology of the posterior fossa hemorrhage was never proven; however, the patient may have been using drugs associated with hypertensive intracerebral bleeds. Whether or not rapid removal of the posterior fossa hematoma would have changed her outcome remains speculative.

Both patients having cerebellar hemorrhages associated with coagulopathies were neonates. One was undergoing extracorporal membrane oxygenation (ECMO). Frequent

monitoring with transfontanel ultrasonography showed no significant hydrocephalus, and the hematoma resolved spontaneously. Canady [1] has reported ultrasound abnormalities in 29 patients treated on ECMO. Ten, or 35%, developed evidence of intracranial hemorrhage; only one of these was in the cerebellum. None required surgical intervention. Our other patient with a coagulopathy had a factor VIII deficiency and was also followed with frequent cranial ultrasonography. Following correction of the coagulopathy the child had spontaneous resolution of the cerebellar hemorrhage, but later developed additional hemorrhages and communicating hydrocephalus, and ultimately required ventriculoperitoneal shunting.

In an autopsy study of 144 premature neonates conducted by Grunnet and Shields [5], 12 were found to have cerebellar hemorrhages. Eleven of the 12 also had germinal plate hemorrhages; the incidence sharply declined after 28 weeks' gestation, and was not seen in any premature infant above 32 weeks of gestational age. The authors concluded that prematurity rather than trauma was the most significant factor in the development of cerebellar hemorrhages in premature infants. Of note is a reference to a case report of Michael [8], who in 1932 described a 1-month-old infant who died of progressive hydrocephalus due to a hemispheric cerebellar hematoma. Certainly, with modern neuroimaging evaluations and surgical interventions, a favorable outcome would have been likely.

It is notable that none of the three newborns included in our series required surgical intervention. Initial moderate ventriculomegaly was not associated with progression (Fig. 4), clinical deterioration, or significant changes in resistive indices by transcranial Doppler ultrasound studies.

Two patients had unexplained cerebellar hemorrhages. One of these was a 13-year-old girl who underwent evacuation of the hematoma; at the time of surgery no evidence of a vascular malformation was seen, nor were any pathologic vessels seen on the scant amount of tissue sent for pathological diagnosis. Since this child died and no postmortem examination was allowed, the etiology remains unknown, although an occult arteriovenous malformation remains the most likely diagnosis. One newborn infant was noted incidentally to have a cerebellar hemorrhage on transfontanel ultrasonography. The child had no coagulopathy and there was no history of unusual trauma during delivery. Subsequent neuroimaging studies showed no evidence of a vascular malformation but did show some substance loss in the involved area of the cerebellar hemisphere.

Little attention has been given to traumatic pediatric cerebellar hemorrhages. Three of Pozzati and associates' [13] seven patients with isolated traumatic cerebellar hematomas were children. Two of these presented in coma but had good outcomes without surgery with hematomas less than 3 cm in diameter on CT scanning. The third patient, with a hematoma greater than 3 cm, had progressive neurologic deterioration on admission and required surgical treatment and later ventricular shunting. He was moderately disabled. Of the four adult patients, three died and one was moderately disabled. A more favorable outcome for children is at least suggested by that report [13], as with the three children with traumatic cerebellar hemorrhages in our series.

Brain-stem evoked potentials have been used in considering the outcomes of various types of head injury to patients [9]. Such studies were done in three patients with noteworthy findings. On one occasion, electrically absent pontine reflexes were associated with intact corneal reflexes; at the same time, the presence of the electrically determined evoked responses through the medulla did not correlate with the patient's absent gag reflex. Neuroimaging studies of this patient showed minor brain-stem signal changes, but there were no intraparenchymal hemorrhages, and the patient had a good clinical outcome. In one patient, who remains in a persistent vegetative state, and in another who is clinically doing well, there were serial improvements in brain-stem evoked responses over time. These findings clearly showed a degree of reversibility in some patients; therefore using the absence of brain-stem reflexes or evoked responses on an initial examination may be misleading in terms of the ultimate recovery in this group of patients. A systematic study of brain-stem evoked responses would be helpful in future series.

The fact that cerebellar hemorrhages produce pathology in the brain-stem by local compression generally indicates that, in the absence of a severe increase in intracranial pressure, supratentorial brain injury may be far less than suspected clinically. The role of external ventricular drainage in maintaining an adequate cerebral perfusion pressure is of critical importance, but carries with it the risk of so-called upward herniation.

The danger of precipitating upward transtentorial herniation by ventricular drainage has been emphasized by Cuneo and associates [3]. In 13 of 52 patients reviewed, upward herniation occurred in the context of ventricular drainage. Vaquero et al. [14] described two children who decompensated from hemorrhages within a few hours after institution of ventricular drainage for posterior fossa tumors. Both patients had pre- and posthemorrhage CT scans documenting the temporal relationship of the hemorrhage to placement of the external ventricular drains. Because of the possibility not only of upward transtentorial herniation, but also of increased bleeding subsequent to decreasing the intraventricular pressure, it is recommended that the external ventricular drainage system be maintained at a higher pressure than would be normally used. In our patients, a ventricular pressure of 15–20 mmHg was maintained.

In two patients in this series, cardiovascular collapse was concurrent with prolonged inadequate cerebral perfusion pressure, producing ischemia of the entire brain. When this occurred, one patient died and the other remained in a persistent vegetative state. Therefore it is imperative to maintain adequate cerebral perfusion pressure as rapidly

as possible, but without lowering the intraventricular pressure to a level that would predispose to rebleeding or upward transtentorial herniation.

Conclusion

Cerebellar hemorrhages in the pediatric age group are caused more often by arteriovenous malformations, tumors, and blood dyscrasias than in adults, whose hemorrhages are caused most often by hypertension. The outcomes of pediatric patients with cerebellar hemorrhages are potentially far better than those of adults; therefore rapid use of neuroimaging facilities and surgical treatment is indicated for pediatric patients having cerebellar hemorrhages even if they have dilated and fixed pupils and other impaired brain-stem reflexes at the time of their initial presentation. Neonates having cerebellar hemorrhages may not require surgical treatment.

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