



## Posttraumatic Cerebellar Infarction in a 2-year-old Child

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### ABSTRACT

Posttraumatic cerebral infarction is a rare complication and is an indicator of poor clinical outcome of head-trauma; in spite of appropriate medical and surgical interventions. Cerebellar infarction following head trauma is also very rare and only a few reported cases are available in literatures. A 2-year-old child sustained head injury in a car accident and underwent surgery because of cerebral contusions and acute subdural hematoma. Cerebellar infarction was revealed in control imaging and she died on the third day after surgery.

**Keywords:** Cerebellum; Infarction; Trauma, Nervous System

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### Introduction

The cerebral infarction is a well-known complication of traumatic brain injury which occurs with a frequency ranging from 1.9% to 10.4% (1-3). Despite appropriate medical and surgical interventions, posttraumatic cerebral infarction is an indicator of poor clinical outcome and is associated with a high mortality rate. Several mechanisms have been mentioned for this complication, including direct vascular compression, cerebral vasospasm, vascular injury, embolization, and systemic hypoperfusion (1-7). Vascular compression

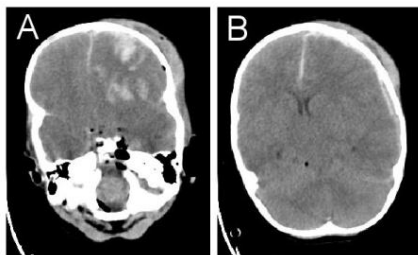
due to focal mass effect in which the major and/or small perforating intracranial vessels are frequently involved, is the common cause of posttraumatic cerebral infarction (4, 8). Other causes such as vessel dissection or thrombosis can lead to posttraumatic cerebral infarction without the effect of mass compression (4, 9-11).

Cerebellar infarction is uncommon and constitutes 1.5% of strokes, but may result in severe complications (12, 13). Cerebellar infarction has different heterogeneous causes. Nevertheless, arterial occlusion as a result of

intracranial vertebral artery dissection (40%), cardioembolism (due to patent foramen ovale, rheumatic valvular disease, etc.), hematologic disturbances and migraine, penetrating injuries of the vertebral artery, and forceful abrupt cervical hyperextension are the common causes (12-14). Cerebellar infarction following head trauma is very rare and only a few reported cases are available in literatures (15-19).

### Case Presentation

A 2-year-old girl sustained head injury in a road traffic car accident 30 minutes before admission. At presentation, she was comatose with the Glasgow Coma Score (GCS) of 8. She had mid-size and sluggish reacting pupils, with intact brainstem reflexes. Computerized tomography (CT) scanning of the head 1 hour after trauma, revealed left-sided frontal contusions, fronto-temporo-parietal and interhemispheric acute subdural hematoma (SDH), and considerable midline shift (Figures 1 A, 1B).

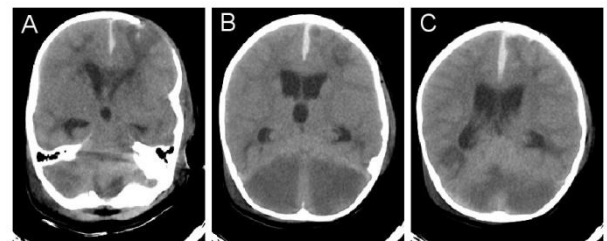


**Figure 1:** CT scanning of the head 1 hour after trauma, left-sided frontal contusions (A), fronto-temporo-parietal and interhemispheric acute SDH (B), and considerable midline shift.

Cervical spine X-ray (anteroposterior and lateral view) and CT scan were normal.

The patient underwent left frontal decompressive craniectomy emergently, with evacuation of acute subdural hematoma. After opening of dura, the contusive hemorrhagic–necrotic brain tissue was removed using gentle suction and bipolar diathermy and then duraplasty was performed. She was closely

monitored and maintained on ventilator in ICU. Medical interventions were targeted at controlling intracranial pressure (ICP), ensuring adequate blood flow and oxygen delivery, and minimizing cerebral edema. The patient's level of consciousness remained as 8-9 in GCS score until the second day after surgery. Thereafter; the patient showed neurological deterioration (GCS: 3, bilateral dilated nonreactive pupils) and circulatory collapse. Resuscitation performed, ventilation was continued and inotropic support instilled. Urgent brain CT scanning showed large bilateral cerebellar and occipital infarct in the territory of posterior cerebral arteries (PCAs) and superior cerebellar arteries (SCAs) with acute hydrocephalus (Figures 2A, B, C).



**Figure 2:** CT scanning reveals large bilateral cerebellar and occipital infarct in the territory of PCAs, SCAs and AICAs with acute hydrocephalus.

An emergent right frontal external ventricular drain (EVD) was placed. Following EVD placement, her vital signs slightly improved but she did not show any improvement neurologically. Regarding her poor general and neurologic status, decompressive surgery was deferred. Despite all aggressive measures she had another episode of cardio-respiratory arrest on the third postoperative day from which she could not be revived.

### Discussion

The incidence of posttraumatic cerebral infarction in patients with severe traumatic brain injury is higher after severe brain injury, resulting in poor clinical outcome and high

mortality rate and higher hospital length of stay (1-3). Presence of a blunt cerebral vascular injury, the need for craniotomy, or treatment with factor VII-a, poor admission GCS, low systolic blood pressure, brain herniation, and decompression craniotomy are risk factors for posttraumatic cerebral infarction (1, 2).

The overall mortality in craniocerebral trauma with complicating posttraumatic cerebral infarction is high, ranging from 21 to 75%, and reaches close to 100% if the territory of the MCA is involved (1, 3, 5, 8, 20).

Among the suspected mechanisms, mechanical shift of the brain and herniation across the falx leading to compression of the cerebral artery account for the majority of posttraumatic cerebral infarction cases (4, 5, 20). Common vascular territories involved in posttraumatic cerebral infarction are ipsilateral PCA territory secondary to transtentorial herniation, contralateral PCA territory due to compression of contralateral PCA against tentorial notch, and anterior cerebral artery (ACA) territory ischemia due to subfalcine herniation (5, 17). Cerebellar infarction in young patients has a wider etiologic spectrum than in older patients and trauma should be considered as a causative factor (13-19). Trauma to the vascular system (dissections or vertebrobasilar spasm), embolization, and systemic hypoperfusion (hemodynamic failure) are mechanisms whereby blood supply to the cerebellar tissue is compromised following trauma (16, 17, 19, 21). Taniura et al. reported a rare case in which severe local trauma with an instantaneous deformity of bone resulted in an injury to the cerebellar cortical artery, leading to the cerebellar infarction under the occipital bone (15). Blunt trauma to the head and neck could injure the vertebrobasilar system in the form of tearing of the intima, intramural and perivascular

hemorrhage leading to a partial narrowing of the injured vessel, progressive thrombosis and vascular occlusion (9, 22, 23). In this case, there wasn't enough time and the patient's condition didn't allow her involved vascular system to be evaluated by other methods. But the first above mentioned mechanism i.e. huge transtentorial herniation is supposed to be responsible for this vast infarction which is more extensive in the side of trauma and its resultant contusion. The latter may be due to direct compression of the left side posterior circulation arteries by herniation which is the side of contusion and indirect compression of the opposite side.

Larger cerebellar infarcts produce symptoms and signs localizing to the brainstem. Signs generally are of abrupt onset and may change suddenly with progressive cerebellar edema and obstructive hydrocephalus. Diminished level of consciousness, irregular respirations, extensor plantar responses, impaired oculocephalic responses, decreased or absent corneal responses, and impaired or absent pupillary responses are noteworthy in uncooperative or comatose patients (24, 25). However brain CT is the most commonly available initial imaging test that is used for cerebellar infarction, early-stage posterior fossa ischemia is rarely seen with brain CT. MRI provides early and accurate visualization of cerebellar infarction but CT scan is more useful in patients with head injury (24, 26). In this case the earliest CT scan couldn't illustrate the infarct and the subsequent CT scan in the third day revealed it.

Since neck vessel injury is probably an underdiagnosed complication of severe head or cervical spine trauma, doppler investigations can be recommended as a screening method to exclude neck vessel injuries; however interpretation of doppler findings may be difficult, particularly in the

vertebrobasilar system (27). Conventional digital subtraction angiography (DSA) is still the gold standard for the diagnosis of vertebral artery dissection, but invasiveness, patient discomfort and risk of complications are the main drawbacks of this technique. Computed tomography angiography (CTA) as a noninvasive tool providing high-resolution images of the arterial lumen and wall is a sensitive technique for the diagnosis of vertebral artery dissection. Being readily available and easy to perform, it can be used as a complement to unenhanced brain CT (28). Magnetic resonance imaging (MRI) and magnetic resonance angiography (MRA) can also help to demonstrate the vascular pathology in these cases (23, 29). But just as pointed before; the patient's condition and time limitation prevented to provide this evidence.

In addition to direct signs of cerebellar infarct, cerebellum tissue affected by ischemic infarction usually undergoes edematous swelling. It can provoke a mass effect on brain stem, fourth ventricle, cerebral aqueduct, and cisterna magna, if it is large enough. Fatal complications including obstructive hydrocephalous and brain stem compression can be potentially generated by this mass effect just as occurred in this patient (30).

There are a few evidence-based guidelines for the management of cerebellar infarction, and most available data are inferred from Class III studies (31). Without surgical intervention, about 80% of patients who have developed signs of brain stem compression will die, usually within hours to a few days (32). There are considerable literatures which propose that hydrocephalus resulting from fourth ventricle obstruction should be treated with surgical decompression rather than CSF diversion with external ventricular drainage (EVD). On the contrary, others argue that,

decompression of the brainstem follows only if the patient does not improve with relief of the hydrocephalus (31, 33-35). The indications for operative intervention in the management of cerebellar infarction are controversial. Some authors recommend surgery, regarding radiographic evidences of brainstem compression or cisternal effacement, in addition to the size of the infarct. Decisions for surgical intervention should not be made on the basis of imaging findings alone, and clinical considerations should complement radiographic appearance in the management of these cases. There are several technical considerations in the operative management of cerebellar infarction such as: size of the suboccipital bone removal, craniotomy versus craniectomy, and removal of the arch of the first cervical vertebra. Intraoperative judgment should determine the extent of bone removal necessary to achieve decompression, because the degree of mass effect is different in each patient. Therefore no rigid guidelines can be offered about a specified size threshold of bone removal (19, 30-38).

So the management of cerebellar infarction is yet in the realm of controversy and should be tailored to each patient individually.

## **Conclusion**

Cerebral and Cerebellar infarction following head trauma are very rare complication but with high mortality rate despite different medical and surgical interventions. So it must be considered in etiologically assessment of being aggravated the traumatic patient's condition.

## **Conflict of Interest**

No conflict of interest.

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