

Posttraumatic Bilateral Basal Ganglia Infarct in Pediatric Age—Clinical and Therapeutic Management: A Rare Case Report and Review of Literature

Tarun Varshney¹ Vinod Sharma¹ Achal Sharma¹

¹Department of Neurosurgery, SMS Medical College and Hospitals, Jaipur, Rajasthan, India

Address for correspondence Tarun Varshney, MCh (Neurosurgery), Department of Neurosurgery, SMS Medical College and Hospitals, Jaipur 302004, Rajasthan, India (e-mail: tarunvarshney1984@gmail.com).

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Abstract

Keywords

- head trauma
- basal ganglia
- infarct

Ischemic stroke of basal ganglia after head trauma is rare in children younger than 18 months and accounts for less than 2% of all ischemic strokes in childhood. The clinical history of these lesions is particularly favorable because they are usually small and facial-brachial-crural hemiparesis is typical of this pathology that regresses over a period of time. The most effective therapeutic approach appears to be a conservative one, although the best treatment regimen is still not well defined. It is necessary to exclude conditions such as heart disease, coagulopathies, and acute traumatic arterial dissections. The authors present a rare case of bilateral basal ganglia infarct in an 18-month-old child following head trauma managed conservatively with good recovery.

Introduction

Ischemia of the basal ganglia following head trauma in children younger than 18 months is rare and accounts for less than 2% of all ischemic strokes in childhood.¹ It is caused by vasospasm of the lenticulostriate arteries. It manifests with nausea, vomiting, hemiparesis, and drowsiness, a clinical picture known as Juvenile Head Trauma Syndrome (JHTS).² We present a rare case of bilateral basal ganglia infarct in an 18-month-old child following head trauma managed conservatively with good recovery.

Case Report

An 18-month-old boy presented to the emergency department following head injury. He fell from a height of approximately 50 cm onto a thinly carpeted floor. The child did not lose consciousness and had no history of seizure. He developed a right hemiparesis after 8 hours. Glasgow coma scale (GCS) score at the time of presentation was 14, and the child was isochoric with isocyclic pupils, but there was a right brachio-crural hemiparesis with muscle strength of 2/5. A computed tomographic (CT) scan of the head was done, which did not show any abnormalities. A hematologic workup was performed to exclude the possibility of

a genetic predisposition to thrombosis that includes coagulation time, bleeding time, prothrombin time, and D-dimer levels. These investigations were normal. Antinuclear antibody and anti-Ds DNA were negative. We also evaluated thyroid hormone assays that were normal. A magnetic resonance imaging (MRI) of the brain was performed, and it documented the presence of a ischemic lesion of bilateral caudate nucleus and on left involving internal capsule (► Fig. 1a–c). Magnetic resonance angiography (MRA) was normal. We could not carry out genetic testing due to financial restrictions with family. Acetyl salicylic acid was started at a dose of 50 mg/day for a week. The child was discharged with improvement in motor power with strength of 3/5 in both the upper and the lower limbs, and is under regular

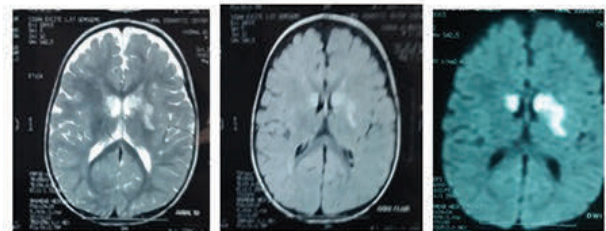


Fig. 1 MRI showing presence of a ischemic lesion of bilateral caudate nucleus and on left involving internal capsule.

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Table 1 Treatment, localization, and clinical outcomes in reported cases

Patient	Age (mo)/sex	First symptom	Lesion location (CT scan, MRI, or both)	Treatment	Clinical outcome (time of follow-up in months)
1	17/Male	Hemiparesis	Internal capsule	Conservative	Good/13
2	17/Male	Hemiparesis	Lentiform nucleus	Conservative	Good/13
3	12/Male	Left-sided hemiparesis	Left internal capsule and corona radiata	Conservative	Good/1
4	12/Female	Left-sided hemiparesis	Right lentiform nucleus and corona radiata	Conservative	Good/3
5	12/Male	Left-sided hemiparesis	Right lentiform nucleus and corona radiata	Conservative	Good/3
6	14/Female	Left-sided hemiparesis	Right basal ganglia and corona radiata	Conservative	Minimal pyramidal tract signs in her left side/1
7	11/Male	Left upper motor neuron 7th nerve palsy and left hemiparesis	Bilateral basal ganglia, internal capsule and periventricular white matter	Conservative	Mild dysarthria and bilateral extensor plantar/12
8	12/Male	Right hemiparesis	In left basal ganglia and internal capsule	Conservative	Good/3
9	18/Male	Right hemiparesis	Bilateral caudate nucleus and on left involving internal capsule	Conservative	Good/5

Abbreviations: CT, computed tomography; MRI, magnetic resonance imaging.

follow-up. At the last follow-up 1 month prior to this manuscript being written, the motor power was the same, but the child was able to use the upper limb for holding and playing with toys and could walk with support with slight limping. A review of the literature was conducted using PubMed. We searched the terms “head trauma,” “child,” and “basal ganglia ischemia.” We selected manuscripts reporting young patients aged 0 to 18 months presenting with lenticulocapsular ischemia due to head injury (→ **Table 1**). Most of the cases have unilateral lesion, and only one case has bilateral lesion. This makes our case a rare entity as the lesions are mirror image infarct.

Discussion

Cerebral infarction after a head injury is an unusual mechanism of stroke in children.¹ Secondary causes responsible for cerebral ischemic lesions in children younger than 18 months such as thrombophilia, arterial dissection, and embolic heart disease must be ruled out before labeling head injury as a cause of infarct.^{3,4} In cases similar to the one we describe here, it is necessary to exclude a possibility of surgical treatment of the lesions primarily responsible for symptoms such as bleeding, so the first investigation to be done is a CT scan. MRI of the brain with diffusion-weighted imaging is performed to look for infarct, and MRA is performed to look for anatomy of circle of Willis. The main etiology is due to the particular anatomical characteristics of the arteries and of the brain parenchyma at this age, especially the

lenticulostriate arteries supplying the basal ganglia. These vessels create an acute angle with the middle cerebral artery, which is more acute in childhood than in the adulthood.^{5,6} Stretching and distorting the angle of perforating branches during trauma damage the vessel and cause a decrease in the blood flow.⁷ The sphenoid bone in children does not fully cover the temporal lobes and facilitate stretching of the lenticulostriate arteries by traumatic forces causing vasospasm and thrombosis.⁸ Another cause may be the friction generated between the lenticulostriate arteries and the brain parenchyma that occurs during the separation of the gray and white matter following a brain trauma in children causing vasospasm and thrombosis. This leads to shutdown of the lenticulostriate flow causing ischemia in the internal capsule with a subsequent facial-brachial-crural hemiparesis.⁵ Most children have been reported to recover completely following conservative therapy.⁷ A complete remission of facial-brachial-crural hemiparesis is typical of this pathology even with persistence of the ischemic area on MRI. The remission is due to the neuronal plasticity of the central nervous system.⁹

Conclusion

Posttraumatic bilateral basal ganglia infarct is a rare entity closely related to the anatomic peculiarities of the brain and skull base in childhood. Young patients should be closely monitored and managed conservatively. Secondary causes must be investigated and ruled out. The prognosis is good in these cases.

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