A 2-year-old boy was presented to us with right-side hemiparesis after trivial fall. Diffusion-weighted magnetic resonance imaging showed an infarct in left basal ganglia and thalamic region. Secondary causes of stroke were ruled out. The child was managed conservatively. Basal ganglia infarct secondary to minor trauma is a rare event. Possible mechanisms leading to such an event and its management are discussed.

**Keywords:** Child, infarct, trivial trauma

**Introduction**

Although minor head injuries are common in childhood, ischemic injury following trivial trauma is uncommon. It forms less than 2% of ischemic strokes in children. Though rare, this condition usually remits in few weeks to months with conservative treatment. Usually, it is due to injury to “deep perforators” supplying the basal ganglia. We report the case of a 2-year-old male child who presented to us with hemiparesis within a few hours following minor head injury.

**Case Report**

A 2-year-old boy was presented to us with history of trivial fall while playing on level ground. There was no history of loss of consciousness; vomiting; seizure; or bleeding from ear, nose, or throat. After about 16 h of injury when his mother noticed weakness on right side of the body, she sought medical opinion. On examination, patient was conscious, oriented, pupils were bilaterally equal, and reacting to light. He had hemiparesis on right side with power Medical Research Council (MRC) 1/5 in upper limb and 0/5 in lower limb. He had no other deficits. Non-contrast computerized tomography scan (NCCT) of the head [Figure 1] showed calcification in bilateral basal ganglia region. Well-defined area of restricted diffusion was seen in left thalamus and adjoining basal ganglia on the MRI [Figure 2] suggestive of an infarct. MRI angiography [Figure 3] of the brain showed normal morphology and flow signal in bilateral internal carotid artery, anterior cerebral artery, and middle cerebral artery. Both vertebral arteries and basilar artery were normal. Angiography of neck vessels revealed no abnormality. MRI of cervical spine revealed no fracture, malalignment, or cord signal changes. Two-dimensional echo revealed no valvular vegetation or regional wall motion abnormality. Hematological workup for prothrombotic state was normal. He was managed conservatively and was administered aspirin (dose 3–5 mg/kg) and antiepileptic drug (levetiracetam). Limb physiotherapy was advised and he was discharged after 2 days of admission. At 3-month follow-up, power on right side improved to MRC 3/5 in both upper and lower limbs.

**Discussion**

Cerebral ischemic lesions are very rare in childhood with an incidence of 0.2–0.6/100,000 children per year. Less than 2% of these are attributable to minor trauma. Other secondary causes of cerebral ischemia in children are traumatic dissection of common or internal carotid arteries, arteries of circle of Willis, congenital heart disease causing embolism, or congenital thrombophilia. Biomechanical properties of a child’s brain and skull are different from those of an adult. Therefore,
consequences of pediatric head injury are also different. The anterior choroidal artery, posterior communicating artery, lateral lenticulostriate arteries from middle cerebral artery, medial lenticulostriate arteries, and recurrent artery of Heubner from anterior cerebral artery form part of deep perforators that arise from internal cerebral artery. Injury to these “deep perforators,” i.e., lenticulostriate, thalamoperforating, or choroidal vessels may cause basal ganglionic infarcts after head injury. In children, lateral lenticulostriate arteries make a more acute angle with the middle cerebral artery than medial lenticulostriate arteries, which makes them vulnerable to stretch even following trivial trauma. Moreover, lateral perforators are shorter in length in children, which get injured even on minimal trauma. Also, between the “fixed” intra- and extraparenchymal part of lenticulostriate vessels is a mobile segment of the vessel, which is prone to injury if stretched. In addition, the sphenoid bone in children is not well developed and does not cover the temporal lobes completely, which makes the brain more mobile as compared to the skull in the event of a head injury. Another hypothesis that has been proposed is that the friction between lenticulostriate arteries and the brain parenchyma can cause vasospasm in these vessels causing ischemia.

Some authors have demonstrated subcortical infarcts following trivial trauma in children having basal ganglia calcification on computed tomography (CT). According to them, this calcification represents “mineralizing angiopathy” of the lenticulostriate vessels, which makes them prone to damage even after trivial trauma. Thick hypercellular vessel walls with intramural and perivascular calcification are seen on pathology. Exact etiology of this “angiopathy” is not known.

A rare syndrome that occurs due to mutation in CACNA1A gene coding for a structural protein in

![Figure 1: NCCT of the head showing calcification (arrows) in bilateral basal ganglia region suggestive of “mineralizing angiopathy”](image1)

![Figure 2: (A) Diffusion-weighted MRI showing an area of restricted diffusion in left thalamic and basal ganglia region suggestive of an acute infarct. (B) Corresponding apparent diffusion co-efficient image](image2)
calcium channel can present with diffuse cerebral edema following minor trauma leading to acute ischemic stroke.[1-3] Certain viral infections especially of varicella zoster can cause secondary vascular insult, which may increase susceptibility to development of posttraumatic vasospasm or injury.[3] In any child presenting with neurological deficit after head injury, it is imperative to rule out an intracranial hematoma by performing a CT scan. In the absence of a hematoma, secondary causes of cerebral ischemia in children must be ruled out to establish a diagnosis of posttraumatic cerebral ischemic lesion. In these cases, an area of altered intensity may be seen on MRI, which is hypointense on T1, hyperintense on T2, FLAIR and diffusion sequences suggestive of an acute infarct. Magnetic resonance angiography must be performed to rule out any traumatic dissection of the vessels of circle of Willis or any of the major vessels of the neck. Any suspicious area may be further evaluated or confirmed on digital subtraction angiography. Echocardiogram must be performed to rule out any emboli from congenital heart disease. Patient must be worked up for thrombophilia including serum homocysteine levels, factor V Leiden, protein C levels, protein S levels, anti-cardiolipin antibody, and anti-phospholipid antibody. For treatment, most authors recommend use of oral antiplatelet drugs (aspirin) to prevent further progression of acute stroke as well as other secondary causes of stroke. Overall, clinical outcome in these cases is favorable with complete remission in most cases in few weeks to months as these lesions are generally small. Good prognosis in these children may also be attributed to neuronal plasticity in children.

**CONCLUSION**

Ischemic lesions of basal ganglia following minor trauma are rare in children. One can attribute them to anatomical peculiarities of “deep perforator vessels” of the brain in children. The presence of calcification in basal ganglia can point to a “mineralizing angiopathy” of lenticulostriate vessels. Secondary causes of stroke must be ruled out. Prognosis in these cases is good with complete remission in few weeks to months.

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**Conflicts of interest**

There are no conflicts of interest.

**REFERENCES**

1. Yang FH, Wang H, Zhang JM, Liang HY. Clinical features and risk factors of cerebral infarction after mild head trauma under 18 months of age. Pediatr Neurol 2013;48:220-6.
2. Shaffer L, Rich PM, Pohl KR, Ganesan V. Can mild head injury cause ischaemic stroke? Arch Dis Child 2003;88:267-9.
3. Kieslich M, Fiedler A, Heller C, Kreuz W, Jacobi G. Minor head injury as cause and co-factor in the aetiology of stroke in childhood: a report of eight cases. J Neurol Neurosurg Psychiatry 2002;73:13-6.
4. Landi A, Marotta N, Mancarella C, Marruzza D, Salvati M, Delfini R. Basal ganglia stroke due to mild head trauma in pediatric age—clinical and therapeutic management: a case report and 10 year literature review. Ital J Pediatr 2011;37:2.
5. Ivanov I, Zlatareva D, Pacheva I, Panova M. Does lenticulostriate vasculopathy predispose to ischemic brain infarct? A case report. J Clin Ultrasound 2012;40:607-10.
6. Dharker SR, Mittal RS, Bhargava N. Ischemic lesions in basal ganglia in children after minor head injury. Neurosurgery 1993;33:863-5.
7. Lingappa L, Varma RD, Siddaiahgari S, Konanki R. Mineralizing angiopathy with infantile basal ganglia stroke after minor trauma. Dev Med Child Neurol 2014;56:78-84.