

PROSPECTIVE OBSERVATIONAL STUDY OF CLINICAL OUTCOME IN CASES OF TRAUMATIC BASAL GANGLIA HAEMORRHAGE IN PAEDIATRIC POPULATION AT A TERTIARY CARE HOSPITAL OF BIHAR

Prasoon Saurabh¹, Rishi Kant Singh², Rajiv Ranjan², Rohit Kumar³

¹Senior Resident, Department of Neurosurgery, PMCH, Patna, Bihar, India

²Assistant Professor, Department of Neurosurgery, PMCH, Patna, Bihar, India

³Associate Professor, Department of Neurosurgery, PMCH, Patna, Bihar, India

Received : 09/10/2023
Received in revised form : 18/11/2023
Accepted : 01/12/2023

Keywords:

Clinical Outcome In Cases Of
Traumatic Basal Ganglia Hemorrhage
In Pediatric Population.

Corresponding Author:

Dr. Rajiv Ranjan,
Email: ranjan.drrajiv@gmail.com

DOI: 10.47009/jamp.2023.5.6.294

Source of Support: Nil,
Conflict of Interest: None declared

Int J Acad Med Pharm
2023; 5 (6); 1436-1439



Abstract

Background: Ischemic stroke is not a common disease in childhood, during which time children are undergoing physical and psychological development. What is the prognosis and anxiety level of children with basal ganglia ischemic stroke? To better diagnose and manage children with basal ganglia ischemic stroke, we analyzed and followed up with children with basal ganglia ischemic stroke where the etiology was confirmed to be trauma, at our hospital over a period of 1 year. **Materials and Methods:** This study reviewed the follow up cases of stroke in the basal ganglia and/or thalamus in children from September 2022 to August 2023 at the Department of Neurosurgery, PMCH, Patna Bihar. This study was approved by the ethics committee of the institute and consent was obtained from the parents of each child. Children where trauma was established cause of ischemic stroke in basal ganglia were included in the study. **Result:** A total of 8 children who came for follow-up during the study period were included in the study. Boys to girl's ratio were 5:3 among the study participants. The age of the children at the time of trauma and diagnosis ranged from 6 months to 14 years with a median age of 4.5 years. The follow-up time was 1–15 years, with a median time of 6.5 years. Major complaints by the parents were unilateral body weakness, focal seizures and facial paralysis. **Conclusion:** This study found that the overall prognosis of children with basal ganglia ischemic stroke is good, but it may cause fine motor disorders.

INTRODUCTION

Ischemic stroke is not a common disease in childhood, during which time children are undergoing physical and psychological development. The sequel of arterial ischemic stroke are serious; the incidence rate is high, at approximately 80%, and the mortality rate is approximately 5%.^[1,2] The impact is relatively large and persists for a long time, usually causing psychological and economic burdens on the family. The incidence rate of a long-lasting effect is approximately 1.2–8/100000, which is higher in males, blacks and infants.^[3,4] The aetiology of childhood stroke is essentially different from that of adults. There is almost no atherosclerosis in the aetiology of stroke in children.^[5,6] Basal ganglia ischemic stroke in children is a special type of stroke that occupies an important proportion of childhood stroke. For a long time, basal ganglia ischemic stroke was not well understood. Recently, an increasing number of studies have suggested that basal ganglia ischemic stroke can lead to psychological diseases

such as anxiety and depression, which seriously affect the development of children's physical and mental health as well as affect the long-term quality of life of the children.^[7,8] In addition, the basal ganglia area has a precise structure, and different brain regions play special functions. Most previous studies on basal ganglia stroke in children have not analyzed the differences among affected regions. The lenticular artery supplies blood to the head of the caudate nucleus, upper segment of the lenticular nucleus and forelimb of the internal capsule. The anterior choroidal artery supplies blood to the lower segment of the lenticular nucleus, caudate nucleus body, caudate nucleus tail, thalamus and posterior limb of the internal capsule.^[9] What is the prognosis and anxiety level of children with basal ganglia ischemic stroke? Are these factors related to different affected regions? These questions have not yet been answered. To better diagnose and manage children with basal ganglia ischemic stroke, we analyzed and followed up with children with basal ganglia

ischemic stroke where the etiology was confirmed to be trauma, at our hospital over a period of 1 year.

MATERIALS AND METHODS

This study reviewed the follow up cases of stroke in the basal ganglia and/or thalamus in children from September 2022 to August 2023 at the Department of Neurosurgery, PMCH, and Patna Bihar. This study was approved by the ethics committee of the institute and consent was obtained from the parents of each child. Children where trauma was established cause of ischemic stroke in basal ganglia were included in the study. Demographic data, clinical symptoms at the of presentation as well as at the time of diagnosis were noted and imaging manifestations of the patients in each group were retrospectively analyzed. Limb muscle strength, fine motor function, facial paralysis and convulsion were followed up. The modified Rankin scale score (mRS) was used to evaluate the overall prognosis.

To evaluate the outcome, disability was scored using the mRS for children based on information from the clinical examination by the neuropaediatricians. A score 0 indicated no symptoms at all. A score of 1 indicated nosignificant disabilities despite symptoms; the child exhibited behavior appropriate to his/her age and normal further development. A score of 2 indicated slight disability; the child was unable to carry out all previous activities, but had the same independence as other age and sex-matched children (no decrease in the gross motor function scale level). A score of 3 denoted moderate disability; the child required some help but was able to walk without assistance. In younger patients, this score also indicated adequate motor development despite mild functional impairment (reduction of 1 level on the gross motor function scale). A score of 4 indicated moderately severe disability; the child was unable to walk without assistance. In younger patients, this also entailed a reduction of at least 2

levels on the gross motor function scale. A score of 5 signified severe disability; the child was bedridden, requiring constant nursing care and attention. A score of 6 was indicated the child was dead.

RESULTS

A total of 8 children who came for follow-up during the study period were included in the study. Boys to girl's ratio were 5:3 among the study participants. The age of the children at the time of trauma and diagnosis ranged from 6 months to 14 years with a median age of 4.5 years. The follow-up time was 1–15 years, with a median time of 6.5 years. Major complaints by the parents were unilateral body weakness, focal seizures and facial paralysis. Other complaints were headache and vomiting. Out Of 8, 6 cases experienced different severities of weakness in the unilateral limbs when diagnosed, often reaching the peak level within 24 h after onset. All the patients had lower strength in their upper limbs compared to that in their legs (0-V level muscle strength). All the patients had history of head trauma prior to the onset of symptoms but the injuries were always very mild, such as that acquired by falling from the bed or tree. [Table 1]

Most of the children exhibited different degrees of fine motor disorder and dystonia on the affected side limbs. As time passed, the children's fine motor disorder and dystonia symptoms gradually eased, and the time required to recover their fine motor skills was approximately 2–3 years or longer. Fine motor disorders and dystonia may exist for a long time. One child had changed his handedness, manifesting as a child with a right-hand advantage changing to having a left-hand advantage. Regarding the child who changed his handedness, the lesion was in the caudate head group.

There was complete recovery from facial paralysis with the shortest recovery time being 1 week and the longest recovery time being 3 months.

Table 1: Demographic and clinical characteristics of the 8 cases at the time of presentation

Patient characteristics	Number of patients
Age at the time of trauma or diagnosis	
<1 year	1
1-3 years	2
4-6 years	4
>6 years	1
Subtype of lesion	
Caudate head	3
Thalamus	2
Lenticular nucleus	3
Symptom at the time of presentation	
Hemiplegia	8
Facial paralysis	4
Aphasia	2
Focal Seizure	2
Headache and vomiting	1

Children recovered quickly from focal convulsion, and those with abnormal EEG results were controlled within 1 week after treatment with oxcarbazepine or

levetiracetam. Patients without abnormal EEG results recovered within 1 week without targeted treatment. To evaluate the overall prognosis, the mRS score was calculated at the end of follow-up. The results

showed that the scores of all the cases were less than or equal to 2 points, with an average of 0.56 points.

DISCUSSION

Limb weakness was the main reason for treatment in children with basal ganglia stroke, followed by facial paralysis and focal seizures. Because limb weakness was not obvious in infants and early children, it was not easy to detect in the early stage of the disease; therefore, the time from onset to treatment was longer. The average time for treatment in this group of patients was 7 days, which was longer than the optimal time window when considering thrombolysis.

All 8 patients with limb weakness had lower power in their upper limbs than that in their legs (0–5 level muscle strength). This difference in limb strength occurs because there are more cortical motor projection nerve fibers in the upper limbs than in the lower limbs through the basal ganglia. Therefore, it is necessary to consider diseases other than basal ganglia ischemic stroke if the child's upper limb muscle strength is greater than his or her leg strength. In terms of aetiology, trauma was taken in consideration. All the cases had trauma within 12 h before the onset of the disease, indicating that trauma is the cause of ischemic stroke in the basal ganglia. The cause of ischemic stroke in the basal ganglia may be related to the anatomical characteristics of the lenticular artery (also known as the lateral central artery). Vincentelli et al. found that the lenticular artery did not directly enter the brain tissue after it exited the middle cerebral artery but went through a section in the lateral fissure—the extracerebral segment—and then turned upward to supply blood to the basal ganglia and internal capsule. In children, the extracerebral segment of the lenticular artery is short and straight, its exit angle is nearly a right angle, and its tension is high. In adults, the external segment of the lenticular artery is long and curved, with an acute angle, and relatively loose. Therefore, the extracerebral segment of the lenticular artery in children is more vulnerable to torsion or shear stress. In addition, the development of sphenoid bone in children is now complete; it cannot completely cover the temporal lobe, which makes the subarachnoid space relatively wide, consequently making the brain tissue and skull base more likely to undergo large horizontal displacement upon an acceleration or deceleration injury so that the lenticular artery is damaged.^[9] A study reported that a child had an abnormal lenticular artery at the age of 1 month, and several months later, an ischemic stroke was observed after mild trauma; at the same time, lenticulostriate artery mineralization was demonstrated.^[10] Ivanov et al reported trauma-associated ischemic stroke in an 8-month-old infant with pre-existing lenticulostriate vasculopathy.^[4] This indicates that the underlying lenticulostriate vasculopathy predisposed the infant to, or worsened,

vascular obstruction caused by head trauma. It has been suggested that the abnormal structure of the lenticulostriate artery could promote the occurrence of basal ganglia ischemic stroke.^[11] The relationship between basal ganglia calcification and ischemic stroke is still controversial. Some hold the opinion that basal ganglia calcification may be asymptomatic and idiopathic basal ganglia calcification; this inference was determined because even if a normal person carries undergoes CT examination, a certain proportion of bilateral basal ganglia calcification is evident.^[12]

Children with basal ganglia ischemic stroke recovered their muscle strength rapidly, and the muscle strength of their affected limbs recovered to grade V within 1 month. Prashant Jauhari et al. hypothesized that transient vasospasm secondary to trauma-induced stretching of lenticulostriate vessels may have led to a more favorable outcome in these children.^[13] Fine motor problems may remain in children with basal ganglia ischemic stroke. In this group of cases, fine motor problems and dystonia were not obvious at the follow-up 1 year after stroke in any of the groups based on lesion location, but a few children experienced changes in their fine motor skills that led to changes in handedness. The caudate head group had a higher incidence of a change in handedness, which may be related to the fact that the head of the caudate nucleus governs autonomous movement. It has been found that damage to the caudate nucleus of the cat leads to an abnormal posture of the contralateral limbs, abnormal precision and abnormal fine motion velocity.^[14] Facial paralysis, convulsion and aphasia in children with basal ganglia ischemic stroke was noted only in the acute stage. The above symptoms in this study were ameliorated within the first 3 months.

CONCLUSION

This study found that the overall prognosis of children with basal ganglia ischemic stroke is good, but it may cause fine motor disorders. When the caudate head affected, it is easy for the patient to develop fine motor disorders. For children with basal ganglia ischemic stroke, in addition to limb rehabilitation and motor function evaluation, emotional fluctuation should also be closely monitored.

REFERENCES

1. Medley TL, Miteff C, Andrews I, Ware T, Cheung M, Monagle P, et al. Australian clinical consensus guideline: the diagnosis and acute management of childhood stroke. *Int J Stroke*. 2019;14:94–106.
2. Kornfeld S, Studer M, Winkelbeiner S, Regényi M, Boltshauser E, Steinlin M, et al. Quality of life after paediatric ischaemic stroke. *Dev Med Child Neurol*. 2017;59(1):45–51.
3. Mallick AA, Ganesan V, Kirkham FJ, Fallon P, Hedderly T, McShane T, et al. Childhood arterial ischaemic stroke incidence, presenting features, and risk factors: a prospective population-based study. *Lancet Neurol*. 2014;13:35–43

4. Goraya JS, Berry S, Saggar K, Ahluwalia A. Stroke after minor head trauma in infants and young children with basal ganglia calcification: a lenticulostriate vasculopathy? *J Child Neurol.* 2018;33:146–52.
5. Bigi S, Fischer U, Wehrli E, Mattle HP, Boltshauser E, Bürki S, et al. Acute ischemic stroke in children versus young adults. *Ann Neurol.* 2011;70:245–54.
6. Toelle S, Avetisyan T, Kuyumjian N, Sukhudyen B, Boltshauser E, Hackenberg A. Infantile basal ganglia stroke after mild head trauma associated with mineralizing angiopathy of lenticulostriate arteries: an under recognized entity. *Neuropediatrics.* 2018. <https://doi.org/10.1055/s-0038-1649501>.
7. Ledochowski J, Desrocher M, Williams T, Dlamini N, Westmacott R. Mental health outcomes in children with acquired dystonia after basal ganglia stroke and associations with cognitive and motor outcomes. *Child Neuropsychol.* 2020;26:691–710.
8. Rafsten L, Danielsson A, Sunnerhagen KS. Anxiety after stroke: a systematic review and meta-analysis. *J Rehabil Med.* 2018;50:769–78.
9. Vincentelli F, Caruso G, Grisoli F, Rabehanta P, Andriamamonjy C, Gouaze A. Microsurgical anatomy of the cisternal course of the perforating branches of the posterior communicating artery. *Neurosurgery.* 1990;26:824–31.
10. Baby N, Vinayan KP, Roy AG. Mineralizing angiopathy of lenticulostriate arteries with infantile basal ganglia infarct following minor head trauma: a case series. *Ann Indian Acad Neurol.* 2019;22:316–9.
11. 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.* 2020;48:220–6.
12. Nobuharu Y, Takashi H. Asymptomatic familial basal ganglia calcification with autosomal dominant inheritance: a family report. *No to Hattatsu.* 2021;32(6):515–9.
13. Jauhari P, Sankhyan N, Khandelwal N, Singhi P. Childhood basal ganglia stroke and its association with trivial head trauma. *J Child Neurol.* 2022;31:738–42.
14. Villablanca JR. Why do we have a caudate nucleus? *Acta Neurobiol Exp.* 2023;70:95–105.