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Case Report

Paediatrics Section

Ischaemic Stroke after Minor Head Injury in an Infant Presenting with Seizures and Hemiparesis: A Case Report

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ABSTRACT

Minor head trauma, though usually benign, can rarely precipitate cerebral infarction through vascular mechanisms such as vasospasm. This case report describes a 16-month-old girl child who presented with left-sided hemiparesis and left upper motor neuron facial nerve palsy, resulting from an acute non-haemorrhagic infarct in the right corona radiata and gangliocapsular region following a trivial trauma. Imaging studies confirmed the infarct, and the child was managed with supportive care and rehabilitation. Remarkably, the patient's motor function and facial nerve impairment showed significant improvement over the follow-up period. The occurrence of multiple seizures following trivial head trauma in a very young child, together with the radiological evidence of acute gangliocapsular infarction without major vascular abnormalities represents a rare clinical presentation. This report adds to the very limited literature on post-traumatic ischaemic stroke especially in infants and emphasises that persistent focal neurological deficit after seemingly insignificant head injury even in the absence of overt haemorrhage warrants urgent evaluation for cerebral infarction. Timely diagnosis facilitates optimisation of neurological outcomes.

Keywords: Arterial ischaemic stroke, Basal ganglia calcification, Magnetic resonance imaging, Motor deficit, Paediatric neurology, Physiotherapy

CASE REPORT

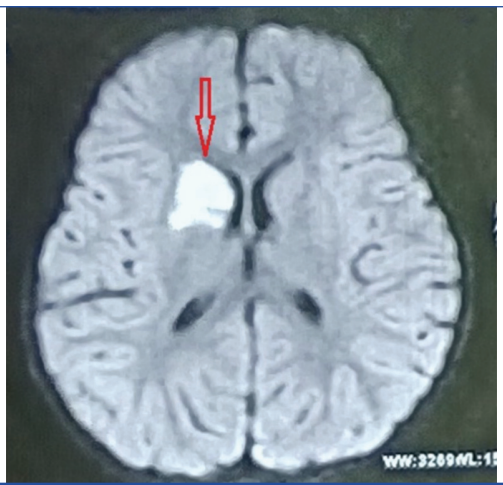
A 16-month-old girl child, the first born of a non-consanguineous marriage, was brought to the emergency room by her mother with complaints of five episodes of abnormal jerky movements involving both upper and lower limbs, each lasting less than one minute. According to the mother, the seizure episodes occurred at intervals of approximately 30 minutes. The child developed postictal drowsiness following each episode and regained consciousness completely between seizures. There was a history of falling from a height of one foot while walking one day prior following which the mother noticed decreased use of the left upper and lower limbs. There was no history of Ear, Nose and Throat (ENT) bleeding, vomiting or obvious external head injury. No history of recent viral exanthems, acute diarrhoeal diseases, recent vaccination, or prior similar illness. Antenatal, natal and postnatal period were uneventful. No history suggestive of any bleeding diathesis or blood transfusion in the past. There was no significant family history.

At presentation, the child was haemodynamically stable with a heart rate of 120 beats/min, respiratory rate of 34 breaths/min, blood pressure of 80/60 mmHg, oxygen saturation of 98% on room air and was afebrile. Capillary blood glucose measured 98 mg/dL. On examination, the child was comfortable with her mother, primarily used her right hand for activities, and showed paucity of movements in the left hand. She was alert, recognised parents, maintained eye contact, responded to name, obeyed simple commands. Notable findings included left upper motor neuron facial palsy, symmetrical muscle bulk bilaterally, and power of 4/5 in the left upper and lower limbs. Plantar reflexes were equivocal, deep tendon reflexes were exaggerated on the left side. Gait abnormality was also noted in the child. There were no signs of meningeal irritation. Cardiovascular examination was normal.

The complete blood count, coagulation profile, and liver and renal function tests were within normal limits. Antithrombin III, Protein C antigen, Protein S antigen, and serum homocysteine levels were normal. Factor V Leiden mutation by Real-time Polymerase Chain Reaction (RT-PCR) was not detected and Anti-double-stranded Deoxyribonucleic Acid (anti-ds DNA) by Enzyme-linked Immunosorbent Assay (ELISA) was negative. Electrocardiography and echocardiography findings were also unremarkable.

Computed Tomography (CT) scan of the brain revealed an ill-defined hypodense area in the right capsuloganglionic region with calcific foci in the bilateral basal ganglia. Magnetic Resonance Imaging (MRI) of the brain, along with Magnetic Resonance Angiography (MRA) and Magnetic Resonance Venography (MRV), confirmed an acute non-haemorrhagic infarct in the right corona radiata and gangliocapsular region [Table/Fig-1].

Differential diagnoses considered were Todd's paralysis following focal seizures, metabolic stroke mimics such as hypoglycaemia and electrolyte disturbances, cerebral venous sinus thrombosis, cardioembolic stroke, and inherited thrombophilic disorders. Todd's paralysis was considered unlikely because neuroimaging demonstrated a definite acute infarction in the right gangliocapsular region. Metabolic causes were excluded by normal blood glucose and biochemical investigations. Cerebral venous sinus thrombosis was considered less likely in view of the MRV findings, while electrocardiography, echocardiography, and thrombophilia



[Table/Fig-1]: MRI brain with MRA and MRV showing an acute non-haemorrhagic infarct in the right corona radiata and gangliocapsular region.

screening were unremarkable, making cardioembolic and inherited prothrombotic causes less likely.

The child was diagnosed with left-sided hemiparesis and left upper motor neuron facial nerve palsy secondary to the acute non-haemorrhagic infarct, likely due to post-traumatic vasospasm. She was started on oral aspirin at a dose of 3 mg/kg/day and oral levetiracetam at a dose of 40 mg/kg/day. Aspirin was discontinued after one week, while levetiracetam was advised to be continued for two years. The child was also supported with regular physiotherapy aimed at improving motor function of the affected limbs and gait training, along with general supportive care including adequate nutrition, hydration, and neurological monitoring. On follow-up after one year, the child remained seizure-free while on antiepileptic medications, with improved use of both limbs, supported by ongoing physiotherapy.

DISCUSSION

Stroke is described as a clinical syndrome involving a rapid onset of focal or global disturbances in brain function that last more than 24 hours or result in death, without any obvious non-vascular cause [1]. In children, key risk factors include cardiac issues such as cardiomyopathies, rheumatic heart disease, and endocarditis; haematological disorders like sickle cell anaemia and prothrombotic conditions related to protein C and S deficiencies, as well as leukaemia and lymphoma; post-infectious complications from varicella, Human Immunodeficiency Virus (HIV) infection and vascular abnormalities such as arteriovenous malformations and Moyamoya disease. Other contributors include drug use (like cocaine and ecstasy) and head or neck trauma that may lead to dissection of the carotid or vertebral arteries [2-4].

Ischaemic strokes resulting from minor head injuries are rare in children as such injuries usually lead to intracranial bleeding. Head injuries can trigger arterial strokes, while dehydration is associated with venous strokes. The occurrence of ischaemic lesions in the basal ganglia following minor head trauma is quite rare, representing less than 2% of all childhood ischaemic strokes [3]. In adults, most strokes are ischaemic (≈80%) and primarily due to atherosclerosis, whereas up to 45% of childhood strokes are haemorrhagic [4]. In 2021, approximately 2.7 million children were estimated to have stroke, of which 58.6% were ischaemic and 41.4% were haemorrhagic in nature [5].

While most reported cases involve older children, this case highlights a rare presentation of acute non-haemorrhagic infarction in a 16-month-old child, manifesting as left-sided hemiparesis and left upper motor neuron facial nerve palsy. The involvement of the right corona radiata and gangliocapsular region suggests disruption of the motor pathways, leading to the observed neurological deficits. Similar cases of acute ischaemic stroke following trivial head trauma have been reported in infants and young children, with lesions predominantly involving the basal ganglia and internal capsule [3,6,7].

The aetiology of the infarct in this child is likely related to post-traumatic vasospasm or injury to the lenticulostriate arteries, which may occur even in the absence of significant intracranial haemorrhage. Such vascular compromise can reduce cerebral blood flow and result in ischemia of vulnerable deep brain structures supplied by perforating end arteries [7,8].

Although several case reports and small case series have described arterial ischaemic stroke following minor head trauma in infants and young children, a comprehensive understanding of its pathophysiology, predisposing factors, and long-term outcomes remains limited because of the rarity of the condition [3,6-8]. Similar cases in recent literature have described infants developing ischaemic stroke with hemiparesis following trivial head trauma [6-8]. However, unlike most of these reports, the child presented with multiple generalised tonic-clonic seizures at onset in addition to hemiparesis. This difference highlights the variability in clinical presentation and emphasises the need for careful neurological assessment in infants after even trivial head injury.

Özcan AS et al., reported an infant who developed acute ischaemic stroke following minor head trauma, highlighting that significant cerebrovascular injury may occur even after apparently trivial falls. Similarly in the present case, the stroke occurred following minor trauma and neuroimaging demonstrated ischaemic infarction without evidence of major vascular abnormalities. However, unlike the case described by Özcan AS et al., this child presented with multiple generalised tonic-clonic seizures at onset in addition to hemiparesis and upper motor neuron facial palsy, thereby expanding the spectrum of clinical presentations associated with post-traumatic ischaemic stroke in infancy [6].

Another recent case highlighted recurrent basal ganglia infarction in an infant following trivial head trauma, where underlying mineralising lenticulostriate vasculopathy was identified as a contributory factor, indicating that subtle pre-existing vascular abnormalities may increase susceptibility to post-traumatic ischaemic events [8]. Previous case series have also shown that focal hemiparesis following minor head trauma in infants commonly corresponds to basal ganglia or internal capsule infarcts, highlighting the crucial role of early CT or MRI in establishing the diagnosis [7]. The history of minor head trauma raises questions about the threshold for vascular injury in paediatric populations. Early imaging, including MRI, is crucial for identifying ischaemic changes and guiding management.

Goraya JS et al., reported basal ganglia stroke after minor head trauma in infants with basal ganglia calcification which was like the present case except for the multiple generalised tonic-clonic seizures [9]. Ferriero DM et al., emphasised the importance of early recognition, neuroimaging, and multidisciplinary management in childhood arterial ischaemic stroke. Consistent with these recommendations, the present patient underwent prompt neuroimaging and received timely antiplatelet therapy and rehabilitation [10].

Mackay MT et al., through the International Paediatric Stroke Study, identified arteriopathies as the predominant risk factor for childhood arterial ischaemic stroke, with acute head and neck disorders, including trauma, also recognised as important contributors. The precipitating event in the present case was a minor trauma in the absence of vascular abnormalities [11]. Fullerton HJ et al., demonstrated that early diagnosis and appropriate management are important determinants of neurological outcome in children with arterial ischaemic stroke. In line with their findings, this child showed significant functional recovery following timely diagnosis and rehabilitation [12].

The management of this condition focused on supportive care and rehabilitation, which are essential for optimising recovery in paediatric stroke cases. Children often exhibit remarkable plasticity, which can facilitate recovery of lost motor functions. In the present case, timely intervention and a tailored rehabilitation program contributed to significant improvement in motor skills and facial function over time.

This case contributes to the growing body of literature on paediatric stroke and highlights the need for increased awareness of atypical presentations in young children. It emphasises the importance of a thorough assessment following head trauma, even in cases where the injury appears minor, to ensure timely diagnosis and intervention for potential vascular complications. Future studies should aim to elucidate the mechanisms underlying post-traumatic vasospasm in paediatric patients to enhance clinical understanding and management strategies.

CONCLUSION(S)

The present case emphasises the importance of recognising and investigating atypical neurological presentations in young children, particularly following head trauma. Early diagnosis through imaging

and appropriate management, including rehabilitation, can facilitate substantial recovery in paediatric patients. This case highlights the need for heightened awareness of vascular complications in children, advocating for a comprehensive evaluation of neurological symptoms following any head injury, regardless of its perceived severity.

REFERENCES

[1] Balachandran A, Kalyanshettar S, Patil S, Shegji V. Ischemic stroke in confederation with trivial head trauma. Case Rep Pediatr. 2016;2016:2572958. Doi: 10.1155/2016/2572958.

[2] Clara RJ, Shreedhara AK, Rashmi A, Raghavendra B. Trivial trauma as a cause of stroke in children. Open J Pediatr Child Health. 2020;5(1):01-02.

[3] Munjal S, Srivastava A, Kapoor K, Mehta VS. Basal ganglia infarct following trivial trauma in a child: Case report and review of literature. J Pediatr Neurosci. 2018;13(1):81-83. Doi: 10.4103/JPN.JPN_157_17.

[4] Ciccone S, Cappella M, Borgna-Pignatti C. Ischemic stroke in infants and children: Practical management in emergency. Stroke Res Treat. 2011;2011:736965.

[5] Yang Z, Duan H, Chen H, Hu H, Wang N, Luo T, et al. Global, regional, and national epidemiology of pediatric stroke, 1990-2021: A systematic analysis from the Global Burden of Disease Study. Pediatr Discov. 2024;4(1):e70031. Doi: 10.1002/pdi3.70031.

[6] Özcan AS, Aydın O, Yaradilmiş RM, Oncel EP, Akkaya B, Cinar HG, et al. Minor head trauma and ischemic stroke in an infant: A case report. Klin Padiatr. 2025 Jun 2. Doi: 10.1055/a-2597-0990.

[7] Mart ZO, Ulusoy E, Eser O, Er A, Sarioglu FC, Duman M. The significant danger of minor trauma in pediatric patients: Acute ischemic stroke. Childs Nerv Syst. 2025;41(1):369. Doi: 10.1007/s00381-025-07046-2.

[8] Singanamalla B, Koneti BB. Recurrent basal ganglia stroke due to mineralizing lenticulostriate vasculopathy. QJM. 2025;118(7):526-27. Doi: 10.1093/qjmed/hcaf009.

[9] 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(2):146-52. Doi: 10.1177/0883073817750501.

[10] Ferriero DM, Fullerton HJ, Bernard TJ, Billinghurst L, Daniels SR, DeBaun MR, et al. Management of stroke in neonates and children: A scientific statement from the American Heart Association/American Stroke Association. Stroke. 2019;50(3):e51-e96. Doi: 10.1161/STR.0000000000000183.

[11] Mackay MT, Wiznitzer M, Benedict SL, Lee KJ, Deveber GA, Ganesan V. Arterial ischemic stroke risk factors: The International Pediatric Stroke Study. Ann Neurol. 2011;69(1):130-40. Doi: 10.1002/ana.22224.

[12] Fullerton HJ, Wintermark M, Hills NK, Dowling MM, Tan M, Rafay MF, et al. Risk of recurrent arterial ischemic stroke in childhood: A prospective international study. Stroke. 2016;47(1):53-59. Doi: 10.1161/STROKEAHA.115.011173.

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