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Case Report

Post-traumatic massive middle cerebral artery infarction in a child: A Case Report

Usman Daibu, Babagana Usman, Mohammed Babagana

Department of Surgery, University of Maiduguri Teaching Hospital, Maiduguri, Borno state, Nigeria.

Correspondences to: Usman Daibu, Department of Surgery, University of Maiduguri Teaching Hospital (UMTH), PMB 1414 Maiduguri, Borno state, Nigeria. **Email:** Usmandaibu561@gmail.com **Phone:** +2348061624082

Abstract

Background: Ischemic Stroke is extremely rare in children and usually occurs due to underlying medical conditions, which include congenital heart disease and hematological, metabolic, or immunological disorders. Traumatic causes are rare, with few cases reported in the literature. **Case report**: We report a case of a previously healthy two-year-old male toddler who presented to our facility with loss of consciousness following a motor vehicular accident, from which he partially improved about 3 minutes later with no other systemic injuries. On examination, he was irritable, with a Glasgow coma scale score (GCS) of 14/15 (E4V4M6) and no neurological deficit. A day after admission, he developed a progressive right hemiparesis with deterioration in his conscious level. GCS dropped to 8/15(E2V2M4) and transferred into the intensive care unit (ICU) where he was intubated and mechanically ventilated. Other nonoperative measures for the management of traumatic brain injury which included, nursing in 30 degrees head up, intravenous fluid, analgesic, and close monitoring of vital signs were instituted. Brain computed tomography done following neurologic deterioration revealed features of massive left middle cerebral artery infarction. All investigations done to rule out co-morbidities were normal. The patient died four days after admission. **Conclusion:** Post-traumatic Middle Cerebral Artery infarction among children is very rare and usually a diagnosis of exclusion. Initial computed tomography of the brain is usually unremarkable. However, features of infarction appear in subsequent imaging done due to neurologic deterioration. As such diagnosis is delayed leading to increased morbidity and possible mortality.

Keywords: *Middle Cerebral Artery, Children, Infarction, Trauma.*

Introduction

Ischaemic Stroke in children is extremely rare and linked to underlying conditions such as congenital heart diseases, haematological disorders, vascular or prothrombotic anomalies, and genetic or metabolic diseases.[1] Post-traumatic cause, especially following mild head injury, is rare. [1]However, in the absence of the known predisposing factors, minor head trauma was reported as the most frequent risk factor for lacunar strokes in childhood.[2] Here, we report a two-year-old boy with this rare diagnosis and review the literature.

Case presentation

A 2-year-old boy presented to the trauma centre of our facility with a loss of consciousness of about 3 minutes following a motor vehicular accident. No bleeding through craniofacial orifices, otorrhea, or rhinorrhea. The parents noticed no vomiting, convulsion, or differential limb weakness. No other systemic injuries. Not a known sickler and has no family history of sickle cell disease. There was no known premorbid illness. His post-resuscitation admitting Glasgow coma scale (GCS) was 14/15(V4), pupils were 3mm and reactive to light, and he moved all limbs equally. Other systemic examinations were essentially within normal. A day after admission, GCS dropped to 8/15 (E2V2M4) with anisocoria (left pupil 6mm and sluggish) and right hemiparesis. Computed tomography of the brain revealed an extensive hypodense area in the territory of the left middle cerebral artery (Fig 1). The patient's genotype was AA, and the complete blood count (CBC), clotting profile, and echocardiogram were within normal limits. The patient was transferred to the intensive care unit (ICU) and died four days after admission.

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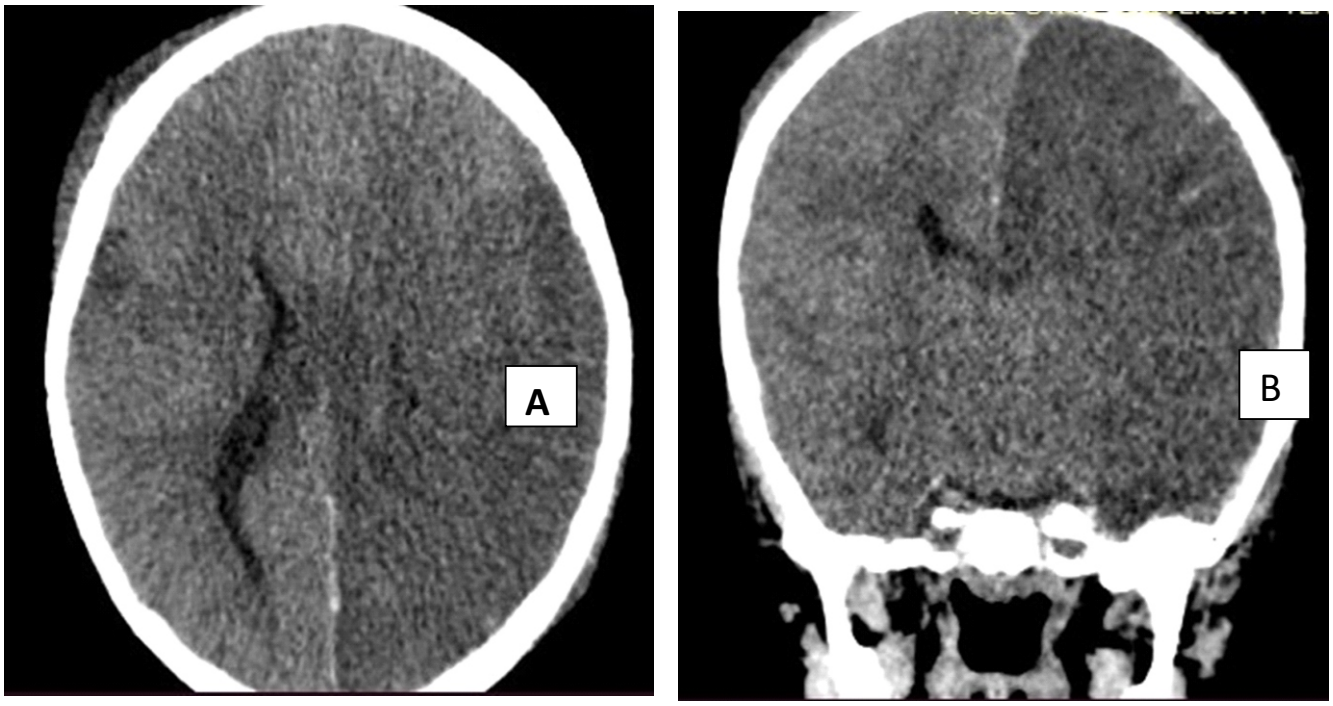

FIGURE 1: Computer tomography of the head**,** in axial (A) and coronal (B) views, Showing an extensive area of hypodensity in the region of the left middle cerebral artery in keeping with ischemic infarction.

Discussion

Traumatic brain injury (TBI) is defined as injury to the head and to the underlying brain caused by an external force, causing an impairment in brain function.[3] It affects the peadiatric population globally,[3] and is among the leading causes of acquired disability and deaths in infants and children.[4] Males are more affected and the outcome depends on the severity of the injury, [5,6] which can be assessed using the Glasgow coma scale (GCS) score or duration of loss of consciousness.[3] Patients with GCS scores of 13-15 are considered to have mild traumatic brain injury, 9-12 moderate traumatic brain injury, and 3- 8 severe traumatic brain injury. Based on the duration of loss of consciousness, 0-30 minutes of loss of consciousness is considered as mild traumatic brain injury, between 30 minutes and 24 hours as moderate traumatic brain injury, and more than 24 hours as severe traumatic brain injury.[3] Our patient's severity of the injury was assessed using the aforementioned. He presented with loss of consciousness of about 3 minutes and a post-resuscitation GCS score of 14/15(E4V5M6) which was within the mild traumatic brain injury category, but deteriorated a day after admission to the severe injury category, with a GCS score of 8/15(E2V2M4) and loss of consciousness of more than 24 hours.

Stroke in children is relatively uncommon,[7,] and ischaemic type usually occurs due to preexisting conditions such as congenital heart diseases, haematological disorders, vascular or prothrombotic anomalies, and genetic or metabolic diseases.[1] Traumatic aetiology, especially mild head injury, is very rare[1], and its diagnosis is that of exclusion. The middle meningeal artery is the most commonly affected in ischaemic Stroke due to its relatively straight orientation and wide calibre.[8,9] Stroke in children has a substantial case fatality rate of up to 4% despite the plasticity of the growing brain.[10] It has been proposed that previous infection with varicella zoster might increase the likelihood of an acute stroke in children following head trauma due to sensitization of small arteries to vasospasm.[11]

The pathophysiology of cerebral artery infarction is attributed to intimal trauma with thromboembolism and transient arterial spasm of the branches of the middle and anterior cerebral arteries in post-traumatic ischaemic basal ganglia stroke in children.[2, 12] The anatomical characteristics of the deep perforating branches of the middle cerebral artery in children, predispose them to vasospasm following mild head trauma.[2,13] Compared to adults, the arterial end branches that supply the caudate, putamen, and internal capsule arise at a more acute angle from the middle cerebral artery, following a recurrent course before penetrating the perforated substance.[14] So also, the incomplete development of the sphenoid bone in children facilitates brain movement during head trauma.[15,16] All these contribute to the disruption of blood flow or intimal trauma in the perforating branches following head trauma, resulting in occlusion or vasospasm.[1]

Genetic factors may increase the risk of a stroke after minor head trauma, but the mechanism is poorly understood.[15] The onset of symptoms varies, but neurological deficits occur after a lucid period of 3-60 hours post-injury.[17] The majority of traumatic ischemic strokes occur in children less than six years old and are very rare after that,[1] probably due to anatomic maturity of the perforating arteries. Our patient was a 2-year-old boy who presented with an altered level of consciousness (GCS-14/15) preceded by an initial loss of consciousness of 3 minutes following a blunt head injury from a motor vehicular accident. He deteriorated a day after admission with associated right hemiparesis. Our findings were similar to the pattern of clinical deterioration in post-traumatic ischemic Stroke in children reported by other authors.[18,19,20] The mechanism of trauma in our patient was a high-speed motor vehicular accident similar to that reported by Talvik et al.[20] This mechanism differs from the most typical low-speed injuries, such as falls from bed and mother's lap reported by other authors.[18,19] Most previous cases reported no initial loss of consciousness in these patients,[18,19,21] as the mechanism of injury was low-speed compared to a high-speed motor vehicular accident in our patient.

Initial computed tomography of the brain is usually unremarkable. However, subsequent brain CTs done following neurological deterioration will show features of infarction.[1,19] Even though we couldn't obtain a computed tomography of the brain of our patient on admission, an extensive area of hypodensity was seen in the left parieto-occipital lobe (left MCA territory) on brain CT done following deterioration in keeping with infarction (Fig. 1). Magnetic resonance imaging of the brain may show restricted diffusion in the corona radiata and lentiform nucleus which further support the diagnosis of MCA infarction.[18,22] Magnetic resonance angiography helps confirm the absence of vascular alterations such as dissecting aneurysms, vasospasms, and thrombosis.[18] None of these were done as our patient was quite unstable and could not withstand the long time needed to acquire magnetic resonance images. Ischemic Stroke in children is usually linked to underlying conditions such as congenital heart diseases, haematological disorders, vascular or prothrombotic anomalies, and genetic or metabolic diseases.[1] Our patient's genotype was AA, and complete blood count (CBC), clotting profile, and echocardiogram done to rule out secondary causes of ischemic stroke in children were within normal limits, similar to the findings of other authors.[1,18]

Owing to the paucity of cases, guidelines for the management of ischemic Stroke in children are not well established.[23] As such, current treatment options are often extrapolated from adult studies.[23] The main target of treatment includes protecting the developing brain by mitigating acute brain injury, and prevention of neurodevelopmental impairment and disability. Our patient was transferred into the intensive care unit (ICU) following neurologic deterioration with a drop in GCS score of 8/15 (E2V2M4) which necessitated intubation and mechanical ventilation.

Other non-operative measures for the management of traumatic brain injury which included, nursing in 30 degrees head up, intravenous fluid, analgesics, and close monitoring of vital signs were instituted. Persistent brain ischemia after head trauma may result in progressive cerebral oedema, and consequent raised intracranial pressure that may require additional measures like cerebrospinal fluid drainage or decompressive craniotomy. Our patient couldn't benefit from invasive intracranial pressure monitoring to assess for neurologic deterioration, as the facility for that was not available in our centre. Despite instituting the available measures, he deteriorated and died three days after admission.

The outcomes of a traumatic brain injury depend on the severity of the injury, and the extent of brain damage.[5,6] Our findings were similar to that of Calderon-Miranda *et al* who reported extensive areas of infarction involving the entire right hemisphere in a 22-month-old male toddler following a mild head injury secondary to a fall from the mother's lap.[19] The patient deteriorated similar to the index case and was managed conservatively in the intensive care unit, but deteriorated and died a few days later. Our findings were contrary to that of Garla *et al* and Tauseef *et al* who showed favourable outcomes in 19 and 9-month-old patients respectively, that survived middle cerebral artery infarction following mild head injury.[15,18] Even though, both patients developed new neurological symptoms a day after the mild head injury, their GCS score remained 15/15 and imaging showed less extensive brain involvement restricted to the basal ganglia, corona radiata, and internal capsule as opposed to almost the entire left cerebral hemisphere in the index case. The extensive brain ischemia, as well as, the lack of facility for invasive monitoring of intracranial pressure to detect early deterioration that will require further measures to avert mortality might have contributed to the mortality in our patient. Even though blunt mild head injury is a rare cause of ischemic Stroke in children, a high index of suspicion is needed to make an early diagnosis in any child with neurological deterioration following mild head injury.

Conclusion

Blunt mild head injury could result in ischaemic stroke in a child. Initial computed tomography of the brain is usually unremarkable. However, subsequent imaging following neurologic deterioration will show features of

infarction. As such, diagnosis is delayed resulting in increased morbidity and possible mortality. Post-traumatic ischemic stroke should be suspected in any child with a mild traumatic brain injury and subsequent neurologic deterioration, and so an extensive and timely medical workup is essential to rule out secondary causes of ischemic stroke that will require additional therapeutic measures.

Conflict of interest
The authors declare that they have no competing interests

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