



Mineralizing Angiopathy: An Uncommon Cause of Pediatric Stroke with Good Outcome

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Acute ischemic stroke in young children following trivial head trauma is very rare. We report a toddler who presented with right hemiparesis and had an acute infarct in the left basal ganglia. The infarct occurred in association with mineralization of the corresponding lenticulostriate arteries (LSAs), visualized as punctate calcifications on computed tomography (CT) of the brain. This condition represents a benign cause of pediatric stroke, known as mineralizing angiopathy [1].

A previously healthy, 30-month-old male child presented with paucity of movements in the right upper and lower limbs, as well as deviation of the mouth angle towards the left side, noted after a time lag of 30 minutes following a fall from a bed. Examination revealed poor anti-gravity movements of the right upper and lower limbs with decreased tone and depressed muscle stretch reflexes on the right side, along with right-side upper motor neuron facial nerve palsy.

Brain magnetic resonance imaging (MRI) showed an acute infarct in the left lentiform nucleus and internal capsule with diffusion restriction and hyperintensity in the fluid attenuated inversion recovery sequence. An MRI angiogram did not reveal any occlusion of the vessels. Brain CT revealed calcifications in the bilateral basal ganglia in the distribution of the LSA (Fig. 1). A

detailed work-up for the etiology of stroke, including echocardiography, blood counts, hemoglobin electrophoresis, serum homocysteine levels, antiphospholipid antibody, protein C, protein S, antithrombin III, cytomegalovirus immunoglobulin G and M antibody titers, and factor V Leiden mutation analysis, did not reveal any significant findings. Iron deficiency anemia was noted with microcytic hypochromic anemia on a peripheral smear and low serum iron and ferritin levels. He was treated with oral aspirin (3 mg/kg/day), iron supplements, and physical rehabilitation. On follow-up after 1 month, he had intermittent dystonia, which was treated with trihexyphenidyl for a few days, following which he made a full recovery. Two years after the event, he has been free of symptoms with normal development.

Basal ganglia stroke can rarely occur following minor trauma and has been described in case reports and series [2,3]. Mineralizing angiopathy of the LSA in infancy was first described by Lingappa et al. [1] as a distinct clinicopathological syndrome with the characteristic radiological finding of linear mineralization along the LSA. This condition occurs in infants and toddlers between the ages of 6 and 24 months with rapid onset of neurological deficits after minor head trauma. Some children have episodes of hemidys-

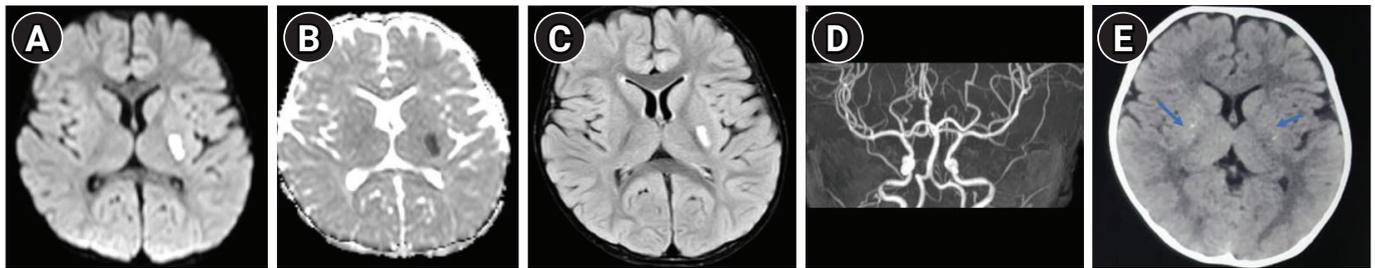


Fig. 1. (A) Axial diffusion-weighted image of the brain shows hyperintensity in the left putamen (B) with restriction in the corresponding apparent diffusion coefficient image. (C) An axial fluid attenuated inversion recovery image shows hyperintensity in the same region. (D) A magnetic resonance angiogram of the vessels of the circle of Willis is normal, and (E) punctate calcification is seen in the bilateral basal ganglia on an axial section of brain computed tomography. Arrows indicate punctate calcification noted in bilateral basal ganglia.

tonia, which is usually transient. This hemidystonia is often noticed early in the course of illness, usually 2 to 4 days after onset [1]. In a case series described by Gowda et al. [4], 28 of 38 children who had been diagnosed with mineralizing angiopathy had onset of hemidystonia within 48 hours, except for one case who had recurrent hemidystonia beginning after 48 hours. It is interesting to note that our patient did not have any dystonia during the first 3 weeks; instead, he developed recurrent episodes in the 4th week and responded to treatment with trihexyphenidyl. He became symptom-free after 1 week. This case highlights the point that hemidystonia can occur even later in the course of illness.

The shearing and distorting forces arising from trauma can lead to endothelial injury in the LSAs, which arise from the MCAs at acute angles in infants and toddlers. Existing mineralization in these vessels can further alter their contractility, leading to thrombus formation and resulting in ischemia. The etiology of calcification remains unknown, although some researchers have proposed that it may be associated with infections such as cytomegalovirus [5]. Recurrence of stroke with trauma has been noted in several case series [1,4].

Conservative treatment is administered for this condition. However, the role of antiplatelets is unclear and needs further research. Stroke in the pediatric age group has diverse etiologies and may lead to persistent disability. Mineralizing angiopathy represents an easily recognized cause of pediatric stroke, with characteristic features and good neurodevelopmental outcomes.

Written informed consent was obtained from the patient's parents.

Conflicts of interest

No potential conflict of interest relevant to this article was reported.

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Author contribution

Conceptualization: DN. Data curation: DN, SC, and GAV. Writing-original draft: DN. Writing-review & editing: SC, and GAV.

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