

**MINERALIZING ANGIOPATHY CAUSING BASAL GANGLIA STROKE IN INFANT FOLLOWING MINOR TRAUMA – A CASE REPORT****Dr. Rajesh A. Kuber<sup>\*1</sup>, Dr. Kunaal Jain<sup>2</sup>, Dr. Arijit Ghosh<sup>2</sup>, Dr. Surbhi Chauhan<sup>2</sup> and Dr. Pooja Karanjule<sup>2</sup>**<sup>1</sup>Professor, Department of Radio-Diagnosis, Dr. D. Y. Patil Medical College and Research Center, Dr. D.Y. Patil Vidyapeeth, Pimpri, Pune.<sup>2</sup>Resident, Department of Radio-Diagnosis, Dr. D. Y. Patil Medical College and Research Center, Dr. D.Y. Patil Vidyapeeth, Pimpri, Pune.**\*Corresponding Author: Prof. Dr. Rajesh A. Kuber**

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**ABSTRACT**

Basal ganglia stroke following minor trauma in infants is a distinct clinico-radiological entity termed mineralizing angiopathy. Healthy infants with no previously known systemic illness presents with hemiparesis and facial paresis following a trivial injury. Neuroimaging reveals non-hemorrhagic focal infarct in basal ganglia. We report a case of a 1-year old male child presenting with left-sided hemiparesis following trauma. The neurological outcome is usually good in most children.

**KEYWORDS:** Mineralizing Angiopathy, Basal Ganglia, Infantile Stroke, Minor Trauma, Sonographic Lenticulostriate Vasculopathy.**INTRODUCTION**

A cerebral vascular accident (CVA) or stroke occurring in children is rare with combined incidence of ischemic and hemorrhagic pediatric CVA ranges from 1.2 to 13 cases per 100,000 children below 18 years of age.<sup>[1]</sup> Pediatric stroke has a mortality rate of 0.6 per 100,000 deaths per year.<sup>[2]</sup> Mortality is high with half of the surviving patients developing neurological and cognitive impairment and more than quarter develops epilepsy.<sup>[3]</sup>

Basal ganglia stroke is considered rare, occurring in less than 2% of all pediatric CVA. Its occurrence in infants following trivial trauma is known and previously reported; however, its co-existence with mineralizing angiopathy has been recently established.<sup>[4,5,6]</sup>

Infants affected presents with hemiparesis with facial paresis and show small non-hemorrhagic infarct in basal ganglia on CT and/or MRI. It has characteristic presentation and evolution of symptoms – previously healthy infants of age 6-24 months are usually affected, sudden onset with rapid progression of neurological deficit following a trivial trauma, affected side shows dystonia between 2 to 4 days which subsides within 24 hours, linear mineralization along course of bilateral lenticulostriate arteries are seen on CT images, subsequent minor trauma can again cause recurrent stroke and short and long-term neurodevelopment is good with good prognosis except in infarct occurring in recurrent stroke.<sup>[7]</sup>

**CASE REPORT**

A 1-year old male child presented with sudden onset weakness of left upper and lower limbs following fall 1 day back. No history of loss of consciousness, seizures, vomiting and fever. Birth history was normal. No history of any congenital heart disease. Milestones achieved were normal for age. Parents gave a history of non-consanguineous marriage.

CNS examination revealed, left-sided hemiparesis, increased tone in left upper and lower limbs with brisk deep tendon reflexes and left plantar reflex was extensor. Sensory examination and examination of cranial nerves were normal. All laboratory investigations were within normal limits.

He was referred to the department of radiology for non-enhanced computed tomography (CT) and magnetic resonance imaging (MRI) of the brain.

Multidetector CT (MDCT) scan of the brain (Figures 1 and 2) revealed hypodense lesion (CT value – 20 to 22HU) in dorsal portion of lentiform nucleus and adjoining posterior limb of internal capsule extending to right corona radiata and body of caudate nucleus. Multiple linear hyperdense calcific densities (CT value – 70 to 90HU) were noted in lentiform nuclei bilaterally. On sagittal and coronal reformatted images, these linear calcifications were noted along the expected course of lenticulostriate arteries.

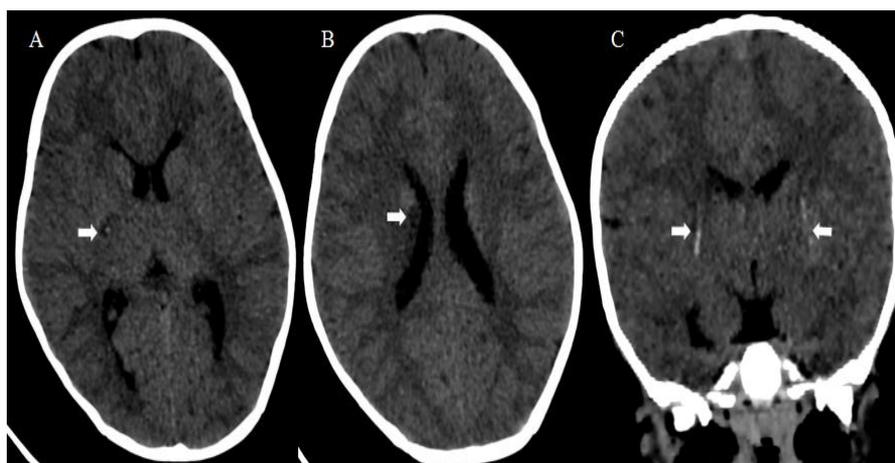
MRI brain (Figure 3) revealed focal hyperintense lesion in dorsal portion of the right lentiform nucleus and adjacent posterior limb of internal capsule seen extending to right corona radiata and body of caudate nucleus on T2-weighted images, appearing hypointense on T1-weighted images and showing restricted diffusion on DWI with corresponding low values on ADC and no blooming on GRE. These findings were suggestive of the acute non-hemorrhagic infarct. MR angiography was normal.

Stroke workup was done. 2D echocardiography was normal.

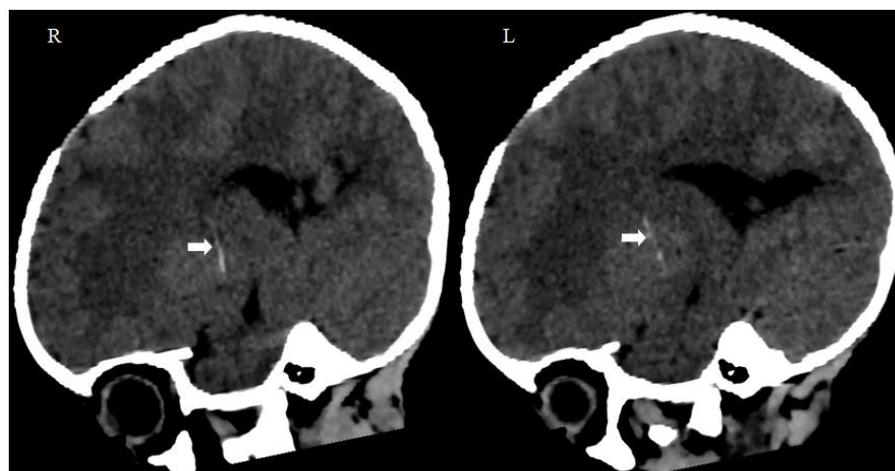
The Antiphospholipid antibody, homocystenine and protein C and S levels were within normal limits.

The clinic-radiological presentation was consistent with mineralizing angiopathy causing acute basal ganglia stroke.

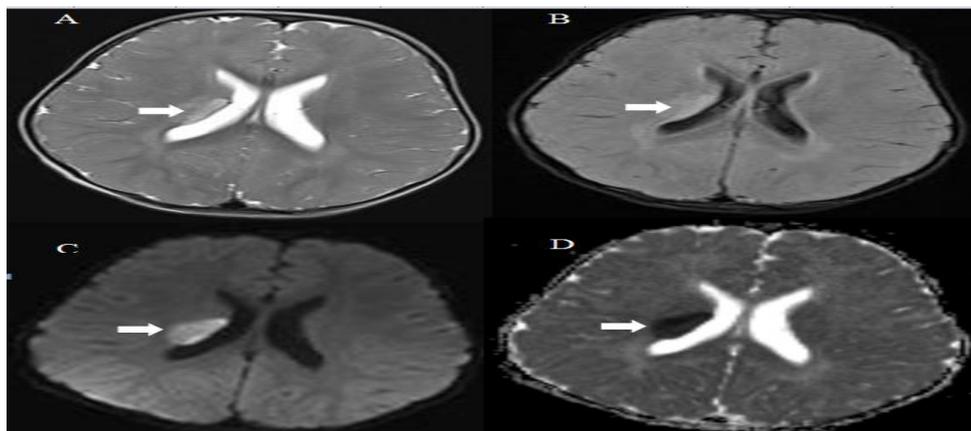
The patient was put on 5mg/kg/day of aspirin and physical rehabilitation. Following 20 days of treatment, improvement in power was noted with an ability to grasp an object with the left hand.



**Figure 1: Axial and coronal non-enhanced CT images showing hypodense lesion in dorsal portion of lentiform nucleus and adjoining posterior limb of internal capsule extending to right corona radiata and body of caudate nucleus with linear hyperdense calcific densities in lentiform nuclei bilaterally (marked by white arrows).**



**Figure 2: Sagittal non-enhanced CT images showing linear hyperdense calcific densities along the course of on right (R) and left (L) lenticulostriate arteries (marked by arrows).**



**Figure 3: Axial T2-weighted (A) and FLAIR (B) images showing focal hyperintense lesion in dorsal portion of right lentiform nucleus and adjacent posterior limb of internal capsule seen extending to right corona radiata and body of caudate nucleus showing restricted diffusion on DWI (C) with corresponding low values on ADC (D).**

### DISCUSSION

Basal ganglia stroke following minor trauma is considered rare, occurring in less than 2% of all pediatric ischemic stroke below the age of 18 months. Clinical manifestations, stroke mechanism and its radiologic features still remain unclear.<sup>[4]</sup> On imaging, hyperdense foci were seen within the area of infarct which was diagnosed as thrombus or tiny foci of hemorrhage.<sup>[8]</sup>

Yang et al showed basal ganglia calcifications in patients with stroke following minor trauma, in 10 out of 16 infants. These were likely to represent linear vascular mineralization not documented in this series. However, with MDCT it is possible to demonstrate linear vascular mineralization along lenticulostriate arteries in coronal and sagittal reformatted images. Etiology of this vascular mineralization however, remains unknown. Association between basal ganglia infarction following minor trauma and positive serological test for cytomegalovirus (CMV), echovirus, Epstein-Barr virus (EBV) and mycoplasma were documented by Yang et al.<sup>[6]</sup> Only CMV seropositivity was co-existent with basal ganglia mineralization. Echovirus, EBV and mycoplasma infections were seen in conjunction with CMV and not associated independently with basal ganglia mineralization. Further research is required as it is not clear whether acute CMV infection causes mineralization.

Sonographic lenticulostriate vasculopathy (SLV) is seen in approximately 0.4% of all live-born neonates and 1.9% to 5.8% of all ill neonates.<sup>[9]</sup> It is similar to linear vascular mineralization in basal ganglia region. SLV is seen inconjunct to a variety of congenital and acquired disorders which resolves over time. Pathologically it is characterized by thickened hypercellular vessel wall showing intramural and perivascular mineralization. These findings are easily demonstrated on neonatal neurosonography, however not visualized in CT/MRI images.<sup>[9,10]</sup>

Mineralizing angiopathy most probably represents sequelae to SLV which is extensive enough to be demonstrated on CT images. Ivano et al reported a case of basal ganglia infarction in an 8-month-old infant with preexisting SLV.<sup>[11]</sup> Hence, the association between SLV and mineralizing angiopathy may be possible. The possibility of underlying lenticulostriate vasculopathy a risk factor for vascular obstruction following head trauma needs to be considered. Ligappa et al postulated that mineralized lenticulostriate arteries under stress following minor trauma may be a predisposing factor for thrombosis.<sup>[7]</sup> The mineralized vessels although obstructed after minor trauma, remains asymptomatic at ages beyond 2-3 years, even though mineralization may persist.

CT is considered as the primary imaging modality in children presenting with focal deficit following head trauma according to 2007 National Institute of Health and Clinical Excellence guidelines for assessment and management.<sup>[12]</sup> MRI although preferred for stroke imaging, it fails to demonstrate mineralization on both gradient recalled echo (GRE) sequence and MR angiography (MRA). MDCT with thin sections due to its multiplanar construction (MPR) capabilities can easily demonstrate vascular mineralization, not possible on conventional CT with 5mm thin axial sections.<sup>[13]</sup>

Caudate nucleus, globus pallidus, putamen and internal capsule are supplied by lenticulostriate arteries and terminal branches of anterior communicating arteries create an acute angle with middle cerebral artery (MCA). It is more pronounced in childhood as compared to adults, a peculiar anatomic characteristic of these arteries which is a predisposing factor for pediatric stroke following minor trauma.<sup>[14]</sup> Between intraparenchymal and extra-parenchymal segments of lenticulostriate arteries which are relatively fixed, a small mobile segment undergoes stretching during trauma causing vasospasm and/or thrombosis resulting in ischemia in the involved vascular territories supplied by

this artery. Sphenoid bone is not fully developed in children, thus allowing brain greater mobility which facilitates stretching of lenticulostriate arteries by traumatic force.<sup>[15]</sup>

### CONCLUSION

Mineralizing angiopathy is a distinct clinic-radiological entity causing basal ganglia stroke in infants after minor trauma. It likely represents sequelae to persistent lenticulostriate vasculopathy. MDCT with MPR capabilities are mandatory to demonstrate linear vascular calcifications along the course of lenticulostriate arteries. Infants with unilateral stroke have a better prognosis than those with bilateral/recurrent stroke and may lead to significant long-term disability.

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