Case Report

Mineralizing Vasculopathy Causing Motor Delay and Silent Strokes?

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Stroke in children is known to have varied causes and many newer ones continue to be identified. One such recently described entity is mineralizing vasculopathy of lenticulostriate vessels to basal ganglia. Although it is a well-known cause of infantile stroke following trivial head injury, this condition as an etiology of isolated motor delay without a prior history of stroke has not been described. We report a case of an infant with isolated unexplained motor delay who presented with hemidystonia and hemiparesis following a trivial fall. This case is unique because mineralizing vasculopathy as a cause of isolated motor delay prior to presenting as stroke has not been reported before. This case opens up the possibility of isolated motor delay following suspected silent strokes as a clinical presentation of mineralizing vasculopathy. Further studies are needed to determine whether this is a part of a spectrum including more severe clinical picture.

Keywords: Basal ganglia, mineralizing vasculopathy, motor delay, silent stroke, trivial head trauma

Case Report

An 8-month old female child with motor delay presented with a history of left-sided weakness following a fall from the walker at the ground level. There were no other signs of traumatic brain injury such as loss of consciousness, vomiting, convulsions etc. She was first born to a non consanguineously married couple with normal birth history. However, she had attained neck control at 5 months of age and was still not sitting with support and cognitive as well as language milestones were normal. At admission, she was conscious, oriented to mother and surroundings, had normal vitals and anthropometry with no external injury. She had a paucity of movements of left upper and lower limbs with variable tone and brisk deep tendon reflexes on the same side. She had intermittent dystonic posturing of the left lower limb. There was no cranial nerve palsy. There were no neurocutaneous markers and fundus was normal. A workup of pediatric stroke was conducted. Her complete hemogram, bleeding time, PT, APTT, INR, serum calcium, serum homocysteine, protein C, protein S, antithrombin III, HB electrophoresis, parathormone, lipid profile, ABG, lactate, ammonia, plasma tandem mass spectroscopy and urine gas chromatography and mass spectroscopy echocardiogram were normal and...
HIV, TORCH titres, lupus anticoagulant were negative. MRI of the brain [Figure 1] showed calcification of right basal ganglia and no evidence of infarct and MR angiogram of the brain was normal. CT of the brain [Figure 2] showed multiple linear calcifications in bilateral basal ganglia predominantly involving caudate nuclei. This was a rare case of mineralizing angiopathy with developmental delay and basal ganglia stroke. The child showed improvement in her movements during her hospital stay. She was discharged with physiotherapy and is being followed up in our out patient department. Presently, she is using both upper limbs equally, but the paucity of movement in the left lower limb is still present.

**DISCUSSION**

Basal ganglia stroke following trivial head trauma is known.\(^1\)\(^-\)\(^3\) Several hypotheses for this entity include transient arterial spasm, mechanical disruption of flow in the perforating arteries, thrombosis secondary to intimal trauma. Due to the compliance of the pediatric skull, the distorting forces on the vessels supplying basal ganglia is higher.\(^4\)

Basal ganglia calcification has been identified as a risk factor associated with stroke following trivial trauma.\(^5\) Recurrent strokes can cause mild disability.\(^6\)

Recently, a distinct clinico-radiological entity of mineralizing angiopathy has been identified. The classical phenotype is that of a 6-month to 2-yr old infant presenting with basal ganglia stroke with or without hemidystonia following trivial head trauma, linear mineralization along lenticulostriate arteries and is associated with good neurological outcome.\(^7\)

This is hypothesized to be a severe and persisting form of lenticulostriate vasculopathy. It is seen in 0.1% of infants and regresses with time. Neurosonogram is more sensitive than CT.

Our case had an underlying unexplained motor delay prior to presenting with basal ganglia stroke following trivial head trauma with calcified lenticulostriate vessels and a favorable short-term neurological outcome. Our patient’s clinico-radiological presentation is consistent with mineralizing vasculopathy and basal ganglia stroke. However, the patient had an underlying isolated motor delay which can be explained by past silent strokes possibly caused by the same entity. Hence, further studies are required to describe the clinical spectrum of mineralizing vasculopathy.

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**Conflicts of interest**

There are no conflicts of interest.

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