



RIGHT HEMIPLEGIA SECONDARY TO LEFT BASAL GANGLIA INFARCTION IN A TODDLER WITH FOLIC ACID DEFICIENCY: A CASE REPORT AND GLOBAL LITERATURE REVIEW

Neurology

Dr. Pazhaniyandi Pillai. K

Senior Resident, Department of Neurology, Tirunelveli Medical College, Tamil Nadu, India

ABSTRACT

Background: Pediatric arterial ischemic stroke (AIS) is rare but carries high morbidity. Basal ganglia infarction is often associated with minor trauma or vascular anomalies such as mineralizing angiopathy or Moyamoya disease. Nutritional deficiencies are rarely reported as isolated etiologies. **Case Presentation:** We describe a 3-year-old boy who presented with sudden-onset right hemiplegia following a minor fall. MRI revealed a left basal ganglia infarct. Stroke work-up was unremarkable for trauma-induced vascular injury, cardioembolic sources, or genetic predispositions. However, biochemical analysis revealed profound folate deficiency (2.2 ng/mL) with depleted iron stores. The child received supportive rehabilitation and nutritional supplementation, with gradual motor recovery. **Conclusion:** This is a rare case of pediatric basal ganglia infarction where folate deficiency and iron depletion were the only risk factors. This report adds to the expanding etiological spectrum of pediatric stroke and highlights the need for nutritional screening in pediatric AIS, especially in resource-limited regions.

KEYWORDS

Nutritional Cause, Folate Deficiency, Basal Ganglia Stroke, Pediatric Strokes.

INTRODUCTION

Pediatric AIS is a major cause of long-term neurological disability in children, with an estimated annual incidence of 1–2 per 100,000¹. The clinical presentation varies, but hemiparesis is the most common symptom, observed in up to 83% of children with AIS¹. The basal ganglia are disproportionately affected in pediatric strokes compared to adults, likely due to differences in vascular supply and developmental vulnerability².

The causes of basal ganglia stroke in children include congenital heart disease, sickle cell disease, vasculopathies, metabolic conditions, trauma, and infection³. In infants and toddlers, mineralizing angiopathy has been identified as a key risk factor for basal ganglia infarction following minor trauma^{2,4}.

Nutritional deficiencies, particularly iron deficiency anemia, have also been linked to increased risk of pediatric AIS⁵. However, isolated folate deficiency as a sole stroke risk factor is extremely rare. Folate deficiency can induce hyperhomocysteinemia, which promotes endothelial dysfunction, oxidative stress, and pro-thrombotic states^{6,7}. This case uniquely highlights folate deficiency and depleted iron stores as potential contributors to basal ganglia stroke in a toddler, with no other identifiable risk factor.

Case Presentation

A 3-year-old boy, second child of non-consanguineous parents, presented with sudden-onset right-sided weakness and frequent falls. The episode began shortly after a minor fall while playing with his brother. Within minutes, his parents noted facial asymmetry and inability to walk. There was no preceding fever, seizure, or head trauma of significant severity.

- Past medical history
- Born at term by normal delivery.
- Normal developmental milestones until presentation.
- No perinatal complications.
- Vaccinations were up to date.
- No family history of stroke, thrombophilia, or metabolic disorders.

Examination

On admission:

- Conscious, irritable but responsive.
- Right hemiplegia with hypotonia (upper > lower limb).
- Facial asymmetry with right lower motor neuron-type weakness.
- Deep tendon reflexes diminished on right side; plantar reflex extensor.
- No meningeal signs.

Investigations

- CBC: Hemoglobin 11.7 g/dL, normocytic normochromic picture.
- Biochemistry: Folate 2.2 ng/mL (low), iron saturation 7.9% (low), ferritin 10.2 ng/mL (low-normal), transferrin 280 mg/dL. Vitamin B12 was normal (358 pg/mL).

- Cardiac: Echocardiogram normal.
- Neuroimaging: MRI brain revealed left basal ganglia infarct (Figure 1). No calcifications were observed.
- Other tests: Stroke panel (ANA, antiphospholipid antibodies, coagulation profile) was normal.

Management & Outcome

The child was treated with physiotherapy, folate and iron supplementation, and close monitoring. Lumbar puncture was deferred as metabolic, infectious, and inflammatory etiologies were excluded clinically and biochemically. At 6-month follow-up, gradual improvement in motor function was noted with partial recovery of speech and facial weakness, although residual hypotonia persisted.

DISCUSSION

Stroke in Children

Childhood AIS differs fundamentally from adult stroke in etiology and risk factors. While atherosclerosis dominates in adults, pediatric stroke is multifactorial and often associated with systemic conditions³. The basal ganglia are a frequent site of infarction in young children, largely due to their end-artery supply and vulnerability to vascular insults².

Basal Ganglia Infarction After Minor Trauma

Multiple studies report basal ganglia infarction following trivial trauma in toddlers, especially with underlying mineralizing angiopathy^{2,4}. Recurrent episodes after repeated minor trauma have also been described⁵. However, in our patient, neuroimaging did not reveal calcifications, making this mechanism unlikely.

Nutritional Deficiencies and Stroke Risk

Iron deficiency anemia has been implicated as a common risk factor for pediatric stroke, present in up to 30% of cases⁵. Folate and vitamin B12 deficiency contribute through hyperhomocysteinemia, which damages vascular endothelium, increases oxidative stress, and enhances platelet aggregation⁶. Meta-analyses confirm that B-vitamin supplementation reduces homocysteine levels and may lower risk of recurrent strokes in adults⁷.

Global Review of Cases

Reports from Asia highlight iron deficiency as a stroke risk factor in malnourished children⁵.

Cases of Moyamoya disease presenting with basal ganglia infarction after minor trauma are described in East Asia and Europe⁸.

Psychiatric sequelae including ADHD and anxiety have been documented after basal ganglia infarction in childhood⁹.

However, isolated folate deficiency-related pediatric stroke is scarcely reported in recent global literature, making our case novel.

Novelty of Current Case

- No trauma-induced vascular injury, Moyamoya, or mineralizing angiopathy.
- Isolated folate deficiency with iron depletion was the only abnormality.
- Suggests nutritional deficiencies alone may predispose to pediatric basal ganglia infarction.
- Expands the etiological spectrum of pediatric AIS beyond classical causes.

CONCLUSION

This case highlights folate deficiency with iron depletion as a potential cause of basal ganglia infarction in children, independent of trauma or vascular anomaly. It emphasizes the need for nutritional screening in pediatric AIS, especially in developing countries where nutritional deficiencies are prevalent. Further research is warranted to establish causality and guide preventive strategies.

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