

Paediatric Stroke: A Rare Presentation of Iron Deficiency Anemia in a Four Year Old Child

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INTRODUCTION

Stroke has emerged as an important cause of acquired brain injury in newborns and children. The ischemic varieties of arterial ischemic stroke and cerebral sinovenous thrombosis are more common than brain malignancy (incidence ~5/100,000/yr) and affect 1 in 2,000 newborns. The annual incidence rates of arterial ischemic stroke in infants and children range from 0.6 to 7.9/100,000 children per year.¹⁻³ Diagnosis is challenging and pathophysiology and risk factors are poorly understood. The frequent adverse neurologic outcomes suffered by most children who have strokes can be reduced by increasing pediatric physician awareness, facilitating early recognition, diagnosis, and specific treatment.

ABSTRACT

Paediatric stroke is an uncommon syndrome with even lesser annual incidence rates of arterial ischemic stroke in infants and children. In children the diagnosis of stroke is frequently delayed or missed. This is due to subtle and nonspecific clinical presentations, a complicated differential diagnosis and a lack of awareness by physicians and also delay in the seeking of medical attention as in our case.

We report you a rare case of a four year old child from remote Nepal who presented to our Out Patient Department after a long gap of around four months after the sudden onset of loss of consciousness and decreased movement of right limbs who after detailed history examination and lab investigations and imaging revealed ischemic stroke due to iron deficiency anemia.

KEY WORDS

Anemia, iron deficiency, pediatric stroke

CASE REPORTS

A four year old male child was brought to our Out Patient Department after four months of sudden onset right sided upper and lower limb weakness. It was associated with the loss of consciousness. The loss of consciousness persisted for about a week till the child fully gained consciousness and slowly the child started moving the limbs but the movement was restricted. There was no history of trauma bleeding from any sites, no seizure initially, but few days after the episodes the child had daily generalized tonic clonic seizures which lasted for few seconds and subsided on its own. There was no history of bluish discoloration or the swelling of limbs, fastbreathing, cough, yellowish discoloration of the eyes, rashes, fever, ear pain or discharge,

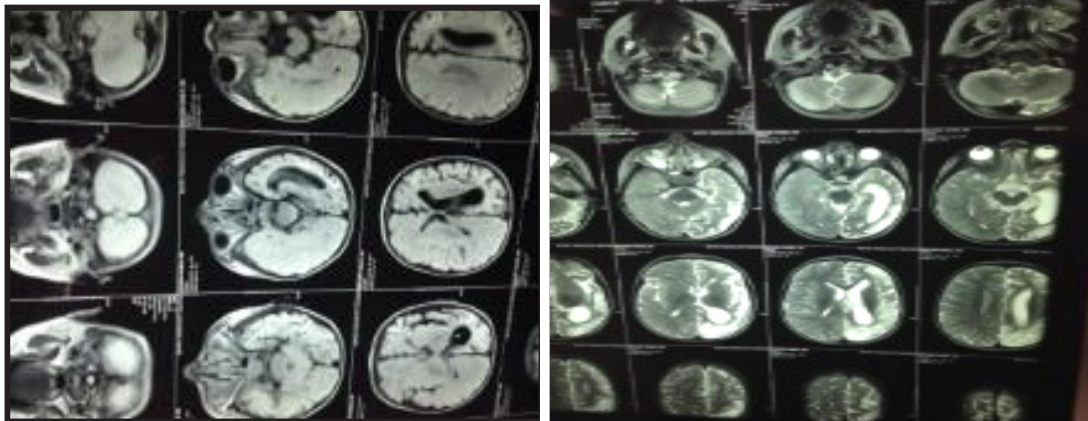


Figure 1. MRI: Showing atrophy of the left sided brain with decreased lateral ventricle size and volume loss in the left MCA territory.

difficulty in swallowing, drooling of saliva and deviation of angle of mouth after the child gained consciousness. He could comprehend and obey the command but the speech was absent. His developmental status was comparable with elder till the onset of stroke and he was exclusively breast fed till five months of age and was started on buffalo milk and other complementary food was started but calories intake was insufficient, family history was insignificant and the immunization status was unknown.

The child was conscious, afebrile, blood pressure taken on right arm in sitting position was 90/60 mmHg, pallor was present and neurocutaneous markers were absent. Anthropometric examination of weight for age and head circumference were -2.1 S.D. and -2.3 S.D. and height was -1.5 S.D. There was no abnormality detected on cardiac, chest and gastrointestinal system examination.

CNS examination: cranial nerves :were grossly intact, on motor examination there was increase tone in the right upper and lower limbs and increased deep tendon reflexes: biceps, supinator, knee jerks, ankle jerks were exaggerated (grade 4/5) in the right side as compared to the left and Babinskis sign was positive on the right side. Fundoscopic examination did not reveal papilledema.

The child was admitted in the ward and his investigations on 2016/1/16 showed Hb 7.2 g/dl TC -11,500, DC: polymorphs 39, lymphocytes 54, Eosinophils 7 platelets 5,65,000/mm³. PT 13 sec, INR 1, APTT 33 seconds, RBS 63 mg/dl, urea =22 mg/dl, creatinine 0.3 mg/dl, Na + 144 meq/L, K + 4.3 meq/L, P.C.V- 27.3 M.C.H -13.6 M.C.V- 51.5 M.C.H.C.- 26.4, SGPT -17 U/L. Peripheral blood smear showed anisopoikilocytosis with hypochromia, microcytes elliptocytes and occasional fragmented RBCs were seen abnormal cells and parasites were not seen. Coloric method of iron detection revealed iron as 26 micro gm/dl and TIBC as 490 micro gm/dl and serum ferritin 5.27 nanogm/dl .

MRI done showed heterogenous T2 high signal intensity in left MCA territory with volume loss and features suggestive of left MCA territory infarction

During the hospital stay the child had three episodes of seizures so the child was started on Valproate 20 mg/kg/day and iron with 5 mg/kg/day and aspirin 5 mg/kg/day an antithrombotic agent. During the hospital stay the recurrence of seizure decreased and the power of the right side of the limbs improved and were discharged.

DISCUSSION

Overall, 46 percent of Nepalese children ages 6-59 months are anemic and the most common causes in this age group is iron deficiency anemia. IDA seems to be an independent risk factor for stroke. Although IDA is thought to be an indolent disease with insidious long-term consequences, it may hold a 10-fold increased risk for acute stroke in well toddlers and may account for half of all strokes in otherwise well children of this age group.⁴ Healthy children who develop stroke may also be five times more likely to have thrombocytosis than children who do not develop stroke.

Three mechanisms to explain an association between IDA and childhood ischemic stroke have been suggested: a hypercoagulable state directly related to iron deficiency; thrombocytosis secondary to IDA; and anemic hypoxia, whereby a mismatch between oxygen supply and end-artery oxygen demand leads to ischemia and infarction.⁵ Data has shown an association between thrombocytosis and stroke in children. Severe iron-deficiency anemia with thrombocytosis may be a risk factor for carotid artery thrombus formation.⁶ Medical management with anticoagulation and antiplatelet therapy is a reasonable approach for these patients while the thrombus resolves.

Hence, there is utmost importance of early detection of iron-deficiency anemia in young children, especially in underdeveloped country like ours so that it can be managed before a life-threatening complication like stroke develops. This article may be beneficial in development of strategies aimed at the primary prevention and early detection of IDA in young children.

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