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Case Report

Acute Ischemic Stroke as a First Manifestation of Severe Iron Deficiency Anemia in a Toddler: A Case Report

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Abstract

Iron deficiency anemia (IDA) remains the most prevalent micronutrient deficiency in children globally, often presenting with symptoms such as pale skin, reduced exercise tolerance, poor appetite, increased susceptibility to infections, and developmental delays. Among its numerous effects, the most concerning are those on the developing neurocognitive system. While its association with thromboembolic complications, particularly arterial ischemic stroke (AIS), is considered rare and often overlooked in pediatric populations, emerging evidence suggests a potential link. We present a rare case of a previously healthy toddler who was diagnosed with ischemic stroke due to a large vessels occlusion (Left Internal Carotid Artery) as the initial manifestation of severe IDA. This case highlights the importance of considering IDA as a possible underlying etiology in pediatric stroke presentations.

Keywords: Acute Ischemic Stroke; Severe Iron; Anemia; Toddler

Introduction

Iron deficiency anemia (IDA) is the most common micronutrient deficiency in children worldwide with multi-organ involvements if overlooked and one of the most serious effects lies in its impact on the neurocognitive system [1,2]. Arterial ischemic stroke is one of the most overlooked thromboembolic event in pediatrics. Also the incidence of thrombosis was 6-10 times greater in patients with iron deficiency anemia as compared to no-iron deficiency anemia [1-4]. Our case report is a rare case of ischemic stroke due to a large vessels occlusion (Left Internal Carotid Artery) as the initial presentation of profound IDA in a previously healthy toddler who presented to our pediatric tertiary care hospital in the United Arab Emirates.

A 2-year-old previously healthy boy presented to our emergency department with a prolonged right-sided focal seizure, followed by right-sided hemiparesis and inability to walk. His growth and developmental milestones were appropriate for age, with no family history of neurological or hematological disorders. There was no history of trauma or toxin exposure, although he had mild upper respiratory symptoms in the preceding week. He had no known past medical or surgical history, and no evidence of congenital or acquired hemophilia. On examination at admission, the child was drowsy but arousable with agitation in between (GCS 12/15: E4, V3, M5), and hemodynamically stable but tachycardic (HR 130 bpm). He had marked pallor (+++), periorbital puffiness, and no signs of shock or dehydration. Neurologically, he exhibited intermittent facial asymmetry while crying, with dense right-sided hemiparesis (power 2–3/5 in both upper and lower limbs) and brisk deep tendon reflexes on the right, along with an extensor plantar response. On the left side, motor power was normal with mildly increased reflexes and an equivocal plantar response. Sensory testing was limited due to age and irritability, but no asymmetry was appreciated. No additional cranial nerve deficits were

observed. Cardiovascular examination revealed a hyperdynamic precordium and a soft systolic flow murmur, likely reflecting the profound anemia (Hb 3.5g/dL). Other systemic examinations were unremarkable.

To quantify stroke severity at presentation, we retrospectively applied the Pediatric NIH Stroke Scale (PedNIHSS) based on the documented neurological findings. The patient scored 17 out of 42, indicating moderate-to-severe stroke. Scoring was based on impaired consciousness, absence of verbal responses, right-sided motor weakness (2-3/5), facial palsy, and signs of pyramidal tract involvement. Initial laboratory investigations are summarized in table 1.

Parameter	Patient Value	Reference Range (Units)
Hemoglobin	3.5 g/dL	11-14 g/dL
MCV	54 fL	75-87 fL
MCHC	28 g/dL	31-37 g/dL
RDW	20.1%	11.5-14.5%
Platelet Count	275 × 10^9/L	200-490 × 10^9/L
WBC Count	11 × 10^9/L	6-15 × 10^9/L
ESR	20 mm/hr	0-15 mm/hr
Ferritin	5.4 ng/mL	22-322 ng/mL
Serum Iron	4.3 µmol/L	9-21.2 µmol/L
TIBC	452 µg/dL	250-425 µg/dL
Transferrin Saturation	4.7%	20-50%
Vitamin B12	Normal	180-914 pg/mL
Folate	Normal	3-17 ng/mL
Haptoglobin	Normal	30-200 mg/dL
Protein C	52.1% >> 75	70-140%
Protein S	34.3% >> 67	65-140%
Lupus Anticoagulant	1.46 >> 1.1	<1.2

Table 1: Summarized lab results of our case along with normal reference lab values for comparison.

Computed Tomography scan of the Brain reveals hypodense area in the left fronto-parietal cortical and subcortical region and Large Hypodense area in the left parieto-occipital cortical and subcortical region (Figure 1A and 1B). A follow-up Magnetic Resonance Imaging confirmed acute infarction in the territory of the left internal carotid artery with restricted diffusion, while a contrast-enhanced Magnetic Resonance Angiography revealed The first segment of the left middle cerebral artery and the first segment of the left anterior cerebral artery appeared attenuated, likely due to reduced forward blood flow resulting from thrombosis in the proximal portion of the internal carotid artery (Figure 2A, 2B and 2C, respectively). No aneurysms, arteriovenous malformations, or hemorrhagic transformation were observed. A two-dimensional echocardiography was unremarkable except for a prominent coronary sinus and trivial aortic regurgitation. No cardiac source of embolism or septal defect was identified.

The child was managed with packed red blood cell transfusions, subcutaneous enoxaparin, levetiracetam, and oral iron supplementation. During his two-week inpatient stay, he underwent daily neurorehabilitation sessions. Although speech difficulties were not documented at initial presentation, mild expressive language delay became apparent during recovery, which gradually improved with therapy. His right-sided dense hemiparesis at admission showed gradual improvement; by discharge, he was ambulatory with mild right hemiparesis, able to walk independently with minimal support and perform basic daily activities. At the time of discharge, his neurological status was assessed using the modified Rankin Scale for children (mRS-Peds), with a score of 2, indicating slight disability but functional independence. He remained seizure-free on levetiracetam. Following discharge, the patient was lost to follow-up, with no further clinical encounters or readmissions documented in our institutional records.

Discussion

Acute ischemic stroke secondary to severe iron deficiency anemia (IDA) in children remains a rare but increasingly recog-

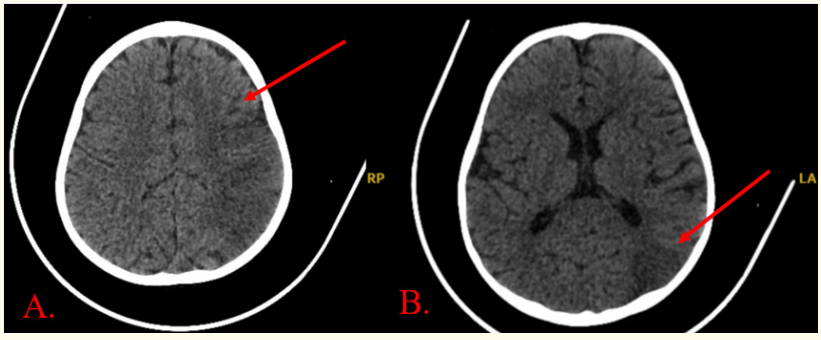

Figure 1: Computed Tomography scan of the Brain reveals A. Hypodense area in the left fronto-parietal cortical and subcortical region , B. Large Hypodense area in the left parieto-occipital cortical and subcortical region.

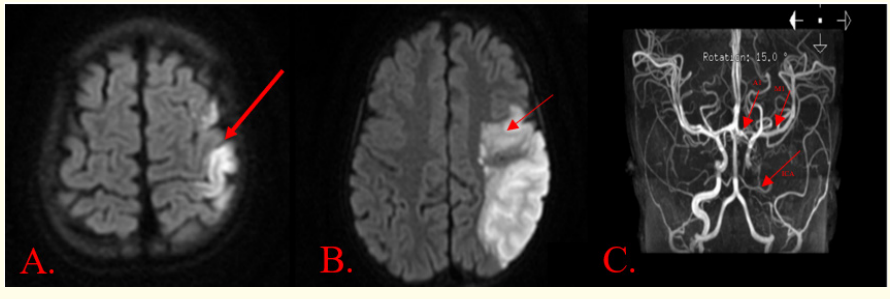

Figure 2: (A and B) Magnetic Resonance Imaging of the Brain: Axial Diffusion-weighted imaging (DWI) showing restricted diffusion in the left fronto-parieto-occipital regions suggestive of acute ischemia.

(C) Contrast-enhanced Magnetic Resonance Angiography showing occlusion due to possible thrombus in the Left Internal Carotid Artery from its origin to distal bifurcation (bottom arrow) and the first segment of the left middle cerebral artery and the first segment of the left anterior cerebral artery appeared attenuated, likely due to reduced forward blood flow resulting from thrombosis in the proximal portion of the internal carotid artery.

nized phenomenon. Early reports, such as those by Munot et al. and Hartfield., *et al*., described cases of profound anemia (hemoglobin levels as low as 2.5g/dL) associated with large arterial infarcts, suggesting that severe IDA alone may act as a significant prothrombotic trigger through non-traditional mechanisms [1-2]. More recently, Gonzalez et al. in 2024 described a pediatric case of ischemic stroke due to IDA, further enhanching this link [3]. These findings are consistent with data from Kalff., *et al*. in 2022, who explored the role of iron deficiency in promoting a prothrombotic state in children, highlighting mechanisms such as reactive thrombocytosis, increased blood viscosity from microcytosis, tissue hypoxia, endothelial dysfunction, and impaired red blood cell deformability [5]. Additionally, iron-deficiency related systemic inflammation may lead to upregulation of pro-inflammatory cytokines and platelet activation, contributing to a hypercoagulable state as seen in the study by Ciacciarelli in 2025 [6]. In our case, the hemoglobin level was critically low (3.5g/dL), pointing toward an anemic hypoxic mechanism as the likely contributor. Eventhough, there were transiently reduced protein C and protein S levels, as well as a mildly elevated lupus anticoagulant initially, these were not persistent and normalized with clinical improvement, consistent with what Kalff et al. described as a reactive lupus anticoagulant positivity in the setting of acute inflammation, rather than a true antiphospholipid syndrome [4].

All cases shared a common underlying mechanism of anemia-induced hypoxia contributing to stroke, while our case uniquely demonstrated a confirmed complete occlusion of the internal carotid artery (ICA) on imaging. The absence of any known stroke etiology in this case strengthens the hypothesis that profound anemia was a major contributing factor to cerebral hypoperfusion and subsequent infarction.

Conclusion

Our case highlights severe IDA as a potential primary reversible risk factor for acute ischemic event due to large vessels occlusion and stroke in children, even in the absence of classical risk factors. Clinicians should maintain a high index of suspicion for cerebrovascular complications in anemic children presenting with focal neurological signs or seizures. Early identification and treatment may significantly reduce morbidity. We recommend that iron studies be included in the stroke workup in all children, regardless of other risk factors.

Consent Statement

Written informed consent for publication of this case report, including clinical details and images, was obtained from the patient's parents.

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