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Case Report

Cerebral Venous Sinus Thrombosis Associated with Severe Iron Deficiency Anemia in a 15-Month-Old Child: A Case Report

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Abstract

Introduction: Cerebral venous sinus thrombosis (CVST) is a rare condition in children. Iron deficiency anemia (IDA), particularly when associated with thrombocytosis, has been reported as a potential risk factor for thrombosis. We report a case of CVST associated with severe IDA in a 15-month-old child.

Case Presentation: A 15-month-old girl presented with afebrile focal seizures and vomiting. Examination revealed pallor and right hemiparesis. Laboratory investigations showed severe microcytic hypochromic anemia (hemoglobin 3.5 g/dL), ferritin of 1 ng/mL, and thrombocytosis (770,000/mm^3). Brain MRI confirmed thrombosis of the superior sagittal sinus, the straight sinus, and the right transverse sinus. The patient was treated with low-molecular-weight heparin and oral iron supplementation, resulting in complete neurological recovery and radiological resolution.

Conclusion: This case highlights the possible association between severe iron deficiency anemia and cerebral venous thrombosis in children. Early recognition and treatment are essential to prevent serious complications.

Keywords: *Cerebral Venous Sinus Thrombosis; Iron Deficiency Anemia; Thrombocytosis; Child; Case Report*

Abbreviations

CVST: Cerebral Venous Sinus Thrombosis; IDA: Iron Deficiency Anemia; MRI: Magnetic Resonance Imaging; LMWH: Low-Molecular-Weight Heparin

Introduction

Cerebral venous sinus thrombosis (CVST) is a rare cerebrovascular disorder in children, with an estimated annual incidence of 0.67 cases per 100,000 children according to the Canadian Pediatric Ischemic Stroke Registry [1,2]. It is characterized by obstruction of cerebral venous drainage and potentially severe neurological complications. Its clinical presentation is often non-specific, which may delay diagnosis and management. Multiple risk factors have been described in pediatric CVST, including head and neck infections (otitis, mastoiditis, meningitis, and sinusitis), dehydration, trauma, congenital heart disease, nephrotic syndrome, inflammatory disorders, prothrombotic conditions, and hematological disorders such as iron deficiency anemia and thrombophilia [3,4]. Among these, iron deficiency anemia (IDA) has increasingly been reported as a potential contributing factor to thrombotic events, particularly when associated with thrombocytosis [3,4].

Iron deficiency anemia is the most common nutritional anemia in children worldwide and remains a significant public health issue, especially in developing countries [5,6]. Beyond its hematological and developmental consequences, IDA has also been associated with cerebrovascular complications, although the underlying mechanisms are not fully understood. We report the case of a 15-month-old girl with cerebral venous thrombosis associated with severe iron deficiency anemia and thrombocytosis.

Case Presentation

A 15-month-old female child presented to the pediatric emergency department after experiencing an afebrile focal seizure and non-bilious vomiting. The child had no prior personal or family history of cancer, genetic disorders, hematological diseases, vascular disorders, or other systemic diseases. Additionally, there was no history of recent cranial trauma or varicella infection. The child was the firstborn of a non-consanguineous couple and was exclusively formula-fed, with no introduction of solid foods.

On physical examination, the child appeared pale and asthenic without fever. Her Glasgow Coma Scale (GCS) score was 14 and her blood glucose level was normal. Respiratory and cardiovascular examinations were unremarkable, with an oxygen saturation of 97%, normal blood pressure, and a normal heart rate. Neurological examination revealed right hemiparesis, hyperreflexia, and a positive Babinski sign in the right lower limb.

Brain computed tomography (CT) was normal, and cerebrospinal fluid (CSF) analysis was also unremarkable, with a white blood cell count of 5/mm^3 and normal protein and glucose levels. The complete blood count (CBC) revealed severe microcytic hypochromic anemia, with a hemoglobin level of 3.5 g/dL, a mean corpuscular volume (MCV) of 53 fL, and a low mean corpuscular hemoglobin (MCH). Serum ferritin was 1 ng/mL, confirming iron deficiency. The platelet count was 770,000/mm^3, consistent with thrombocytosis, and antithrombin activity was 35% (reference range: 73 - 129%). Further laboratory investigations, including C-reactive protein (CRP), prothrombin time, fibrinogen, protein C, protein S, and factor V Leiden testing, were within normal limits. Brain magnetic resonance imaging (MRI) demonstrated thrombosis of the superior sagittal sinus, the straight sinus, and the right transverse sinus (Figure 1 and 2).

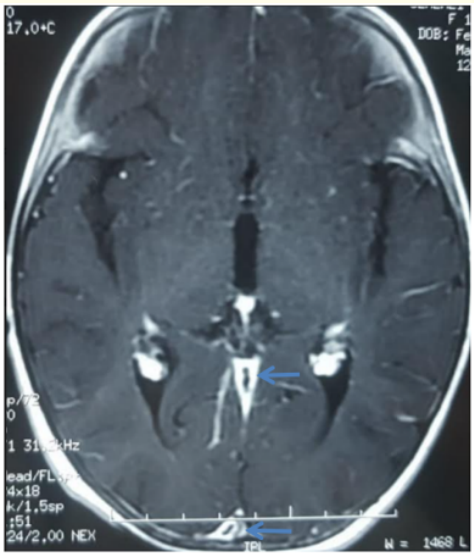

Figure 1: *Axial contrast-enhanced T1-weighted brain magnetic resonance imaging (MRI) demonstrating thrombosis of the superior sagittal sinus (Lower arrow) and the straight sinus (Upper arrow).*

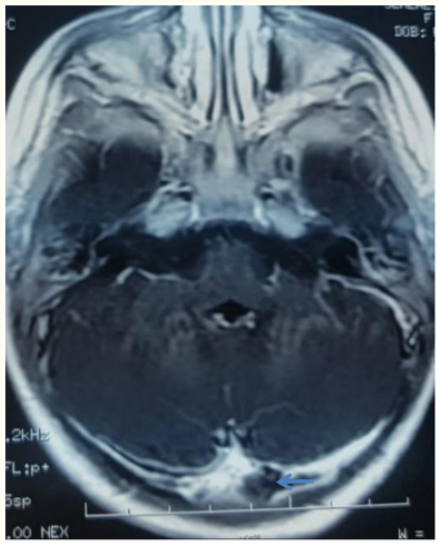

Figure 2: *Axial contrast-enhanced T1-weighted brain magnetic resonance imaging (MRI) demonstrating thrombosis of the right transverse sinus (Arrow).*

Written informed consent for publication was obtained from the patient's parents. Ethical approval was obtained from the Institutional Review Board (IRB) of Mohammed VI University Hospital, Oujda, Morocco.

The patient received therapeutic-dose low-molecular-weight heparin (enoxaparin 1 mg/kg twice daily) for 10 days, followed by oral anticoagulation with acenocoumarol (Sintrom®) for 2 months, together with oral iron protein succinylate (6 mg/kg/day). Despite severe anemia (hemoglobin 3.5 g/dL), no blood transfusion was administered because the anemia was clinically well tolerated, with stable hemodynamic status and no signs of cardiac failure or shock. Dietary diversification was reinforced, and the parents were advised to reduce excessive milk intake.

The patient's clinical course was favorable, with complete recovery of motor function. The patient was discharged on acenocoumarol (Sintrom®), oral iron supplementation, low-dose aspirin, and dietary counseling. Follow-up brain MRI performed 3 months after diagnosis demonstrated complete resolution of the cerebral venous sinus thrombosis. Oral iron supplementation was continued for 9 months, together with appropriate dietary diversification. Hemoglobin levels increased progressively by approximately 1 g/dL per month until normalization, with complete correction of iron deficiency.

Discussion

Cerebral venous sinus thrombosis (CVST) is a rare condition, occurring in approximately 0.6 cases per 100,000 children per year [7]. Its clinical presentation varies according to the location and extent of thrombosis, the patient's age, and the delay to diagnosis, which may contribute to underdiagnosis [8].

Brain magnetic resonance imaging (MRI) combined with magnetic resonance venography (MRV) is considered the imaging modality of choice for the diagnosis of cerebral venous sinus thrombosis. While MRI demonstrates the parenchymal consequences of venous occlusion, MRV accurately identifies the location and extent of sinus thrombosis, thereby facilitating early diagnosis and appropriate management [8].

Cerebral venous sinus thrombosis (CVST) in children is often multifactorial, with infections representing the most common underlying cause. These include otitis, mastoiditis, pansinusitis, meningitis, varicella, and respiratory or gastrointestinal infections. Systemic diseases such as lupus, congenital heart disease, and nephrotic syndrome may also predispose to thrombosis [3,4]. However, none of these risk factors were identified in our patient.

MRI combined with MR venography is the preferred imaging modality for confirming cerebral venous sinus thrombosis and for evaluating venous recanalization during follow-up. Follow-up imaging should be individualized according to the patient's clinical course and radiological evolution.

Anemia is an important risk factor for hypercoagulability in children with cerebral venous thrombosis, although the mechanisms involved are not fully understood. Iron deficiency anemia and microcytosis are the most frequently reported hematological abnormalities in these cases [9]. In the present case, the patient had severe microcytic anemia, a recognized risk factor for cerebral sinovenous thrombosis (CSVT).

Iron deficiency anemia and venous stroke have been reported in children, sometimes in association with thrombocytosis. Additionally, CSVT has been described in chronic anemias such as hemolytic anemia and Evans syndrome [10]. The diagnosis of iron deficiency may be challenging, particularly in the presence of hemoconcentration, dehydration, or elevated ferritin levels during the acute phase. Therefore, iron deficiency anemia should be actively investigated and treated, as it may represent a preventable risk factor for CSVT.

Thrombocytosis may have been a significant factor in the development of cerebral venous thrombosis in our patient, particularly given the presence of platelet aggregation. Given the progressive resolution of the thrombus following treatment and the absence of other identified underlying causes, we believe that the thrombocytosis observed in our patient was most likely secondary to iron deficiency [9,10]. Although antithrombin activity was decreased (35%), the remaining thrombophilia investigations were unremarkable, and severe iron deficiency anemia remained the principal identifiable risk factor. However, CSVT cannot be attributed solely to thrombocytosis, as hypercoagulability related to iron deficiency may also contribute to thrombus formation.

Among four children with stroke and iron deficiency anemia reported by Farias-Moeller., *et al*. [11], three had CSVT and one had an ischemic stroke. Two patients, similar to ours, had excessive cow's milk intake, a recognized risk factor for iron deficiency anemia. Likewise, our patient had a history of exclusive milk feeding without appropriate food diversification. Apart from the decreased antithrombin activity, no other constitutional or acquired prothrombotic disorder was identified.

Cases of venous and arterial thrombosis associated with anemia have been reported in the literature. Most reported cases involve autoimmune hemolytic anemia. Several mechanisms may explain the association between iron deficiency anemia and thrombosis, including reactive thrombocytosis, increased platelet activation, endothelial dysfunction, tissue hypoxia, and increased blood flow turbulence [11].

Early diagnosis and prompt anticoagulant therapy are essential to reduce the risk of long-term neurological sequelae, including persistent motor deficits, cognitive impairment, developmental delay, and epilepsy. Fortunately, our patient achieved complete neurological recovery without residual deficits, emphasizing the importance of early recognition and appropriate management [12].

Conclusion

In conclusion, this case supports the association between severe iron deficiency anemia and cerebral venous sinus thrombosis in children. Early recognition and treatment of iron deficiency may help prevent severe neurological complications. Further studies are needed to better understand the mechanisms linking iron deficiency anemia to thrombosis.

Human Subjects

Informed consent for publication was obtained from the patient's parents.

Conflicts of Interest

In compliance with the ICMJE uniform disclosure form, all authors declare the following: Payment/services info: All authors have declared that no financial support was received from any organization for the submitted work. Financial relationships: All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. Other relationships: All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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