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Case Report

Cerebral Venous Sinus Thrombosis in a Child with Iron Deficiency Anaemia

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Abstract

Children who have cerebral venous sinus thrombosis (CVST) are at risk for both emergency and long-term neurological effects. Non-specific symptoms like fever, altered mental status, and localized neurological abnormalities define it. Infection, trauma, prothrombotic diseases and, most critically, iron deficiency anaemia (IDA) are etiologic factors. IDA, which is frequently brought on by excessive cow milk consumption in newborns and toddlers, is a preventable risk factor for CVST. We discuss the case of a 32-month-old female who presented with lethargy and hemiparesis, and was diagnosed with CVST in the context of IDA.

Introduction

Cerebral venous sinus thrombosis (CVST) is a rare disease presenting with non-specific symptoms and signs.[1] The symptoms may not be localized and generally present with headache, vomiting, lethargy and neurological deficits.[1,2] Some may have hemiparesis, cranial nerve disturbance, and ataxia. The differential diagnosis for children who present with convulsions, stroke, headache, coma, or pseudotumor cerebri should include CVST.[1] In addition, a variety of factors, such as infections, trauma, recent intracranial surgery, prothrombotic diseases, and IDA, can cause CVST. A strong level of suspicion is needed to identify CVST early because symptoms might be obscure and inconsistent. We discuss the case of a 32-month-old female who presented with lethargy and hemiparesis, and was diagnosed with CVST in the context of IDA.

Case report

A 32-month-old girl presented with five days of fluctuating high fever, productive cough, and headache. On the day of admission, she experienced generalized convulsions lasting around five minutes. The child's medical and family history was normal. Her diet was predominantly milk only as she drank about 1000 ml of cow milk daily. She rarely ate meat and vegetables. She was diagnosed with febrile seizures and upper respiratory tract infection, and prescribed oral analgesics. A day after admission, she developed hemiparesis on her left side and appeared lethargic. Physical examination revealed a Glasgow Core Scale of 13 with no signs of raised intracranial pressure or focal neurological deficits.

The laboratory finding revealed moderate microcytic hypochromic anemia with Hb 8.3 gm / dL, MCV 64 fL, MCH 17.4 pg, WBC of 8920 cells / mm^3 with increased platelets 471,000 cells / mm^3. She had low ferritin 4.3 μg / L and serum iron 3.2 μmol

/ L. Her coagulation profile was normal, along with normal protein S, protein C and anti-thrombine III levels. Sickle cell screening was negative. She had normal CSF study reports. She tested negative for ANA and anti-ds DNA antibodies as well as antiphospholipid and anti-cardiolipin antibodies.

In view of headache as well as unspecified hemiparesis, neuroimaging – CT Scan with contrast was done. The result showed extensive thrombus in the superior sagittal sinus (Figure 1), straight sinus (Figure 2), great cerebral vein (vein of Galen) (Figure 2) and superficial cortical vein (Figure 3).

Figure 1. Postcontrast CT axial (right) and sagittal (left) shows filling defect in the superior sagittal sinus (white arrow)

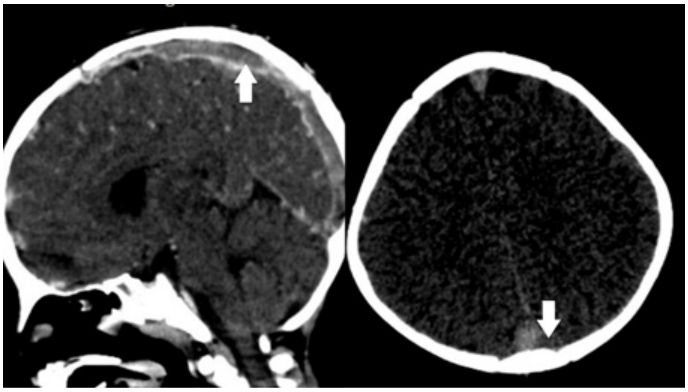


Figure 2. Postcontrast CT sagittal shows filling defect in the straight sinus (white arrow) and vein of Galen (red arrow).

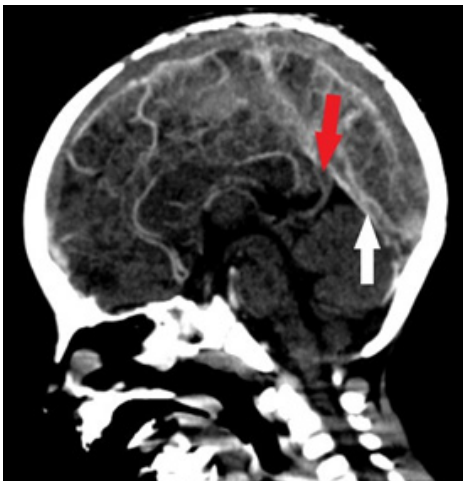
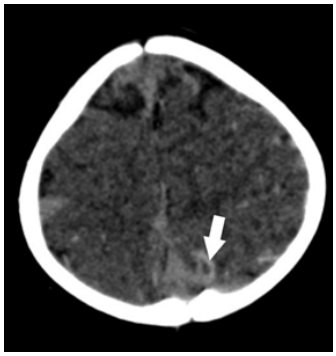


Figure 3. Postcontrast CT sagittal shows filling defect in the cortical veins (white arrow).



Low-molecular-weight heparin (LMWH) enoxaparin subcutaneous 1.5 mg / kg / dose twice daily was started, along with IV rehydration and supportive care. After 10 days of treatment, she remained hemodynamically and neurologically stable and discharged from hospital and is under regular follow up.

Discussion

CSVT is a rare and complex condition that obstructs blood flow in the brain's veins and sinuses.[3] Up to 40% of survivors experience neurologic sequelae, and mortality rates of less than 10% is observed.[4] It can cause headaches, vomiting, seizures and altered mental status. Common causes of CSVT include infections, trauma, and autoimmune diseases. Patients with congenital cardiac disease, nephrotic syndrome, and certain cancers may also be predisposed.[4] Prothrombotic conditions must also be taken into account because they raise the overall risk of thrombosis. CVST has been associated with lupus anticoagulant, and protein C and S deficiencies.[3,5]

The cerebral sinovenous system drains blood from the brain through superficial and deep sinuses and veins.[6] The absence of valves in this system means that it passively transports blood, and an obstructive venous thrombosis can cause pressure to be transferred to the nearby brain, leading to local cerebral edema and fluid extravasation. If forward flow is not restored and venous pressure exceeds arterial pressure, an ischemic infarct may occur.[6,7]

Anaemia, particularly IDA, has been linked to CVST in children.[8] Anaemia leads to reduced oxygen carrying capacity and increased cerebral blood flow, triggering the coagulation cascade and thrombus formation.[9] Iron plays a

role in controlling platelet levels, and its deficiency leads to thrombocytosis, which increases thrombosis. IDA has been linked to CVST in several publications.[6,9] Moreover, the majority of individuals had normal prothrombotic disease evaluations indicating IDA as a significant etiologic factor.[6] Interestingly, excessive cow milk consumption has been cited as a root cause of IDA.[6,9] Cow milk is naturally low in iron and can also cause occult stool blood loss, exacerbating anaemia. Casein and high calcium concentrations in cow's milk can also hinder iron absorption.[10]

CECT, MRI, and MRV imaging modalities are frequently used to diagnose CVST. Initial care comprises stabilization, symptomatic management, rehydration, and, if necessary, the use of antibiotics, anticonvulsants and anticoagulants, primarily LMWH. Follow-up imaging is necessary to assess thrombosis resolution and the duration of anticoagulant therapy.[4] Younger age, seizures, infarcts, or altered consciousness are poor prognostic markers. Anticoagulation is associated with improved cognitive outcomes and a lower risk of recurrence.[3,4]

Conclusions

CVST can present with non-specific symptoms such as fever, vomiting, altered mental status, and focal neurological abnormalities. IDA, infections, trauma and prothrombotic diseases can contribute to CVST. Diagnosis is aided by contrast-enhanced CT or MRI and MRV. Anticoagulation, iron replenishment, and dietary changes are important treatments for CVST.

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