



(CASE REPORT)



EXTENSIVE CEREBRAL VENOUS SINUS THROMBOSIS IN A 9-MONTH-OLD INFANT WITH SEVERE DEHYDRATION AND SUSPECTED SEPSIS: A CASE REPORT

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World Journal of Advanced Research and Reviews, 2026, 31(03), 1800–1804

Publication history: Received on 18 August 2026; revised on 24 September 2026; accepted on 26 September 2026

Article DOI: <https://doi.org/10.30574/wjarr.2026.31.3.2485>

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ABSTRACT

Cerebral venous sinus thrombosis (CVST) is an uncommon but potentially severe pediatric cerebrovascular disorder. We report a previously healthy 9-month-old boy admitted with severe dehydration and hypovolemic shock after five days of acute gastroenteritis associated with fever and suspected sepsis. On admission, he was obtunded, hypotonic, poorly reactive, and markedly dehydrated with peripheral hypoperfusion. Laboratory investigations showed severe microcytic anemia (hemoglobin 7.1 g/dL; mean corpuscular volume 59.5 fL), thrombocytopenia, leukocytosis, and elevated inflammatory markers. During hospitalization, he developed status epilepticus. Contrast-enhanced brain computed tomography demonstrated extensive thrombosis of the superior sagittal sinus with extension into the left transverse sinus. Therapeutic anticoagulation with enoxaparin was initiated, followed by oral acenocoumarol. A thrombophilia workup did not reveal a documented congenital prothrombotic disorder. Early follow-up computed tomography showed partial regression but persistent superior sagittal sinus thrombosis. On pediatric hematology recommendation, anticoagulation was continued until July 2026, for approximately 14 months. Later magnetic resonance venography showed favorable evolution without evidence of active thrombosis. This case highlights the multifactorial nature of pediatric CVST and the importance of urgent neuroimaging when neurological deterioration occurs during severe dehydration or systemic inflammatory illness.

Keywords: Cerebral venous sinus thrombosis; Infant; Dehydration; Gastroenteritis; Anticoagulation; Pediatric stroke

1 INTRODUCTION

Cerebral venous sinus thrombosis (CVST), also termed cerebral sinovenous thrombosis, is an uncommon but serious cause of pediatric stroke. The Canadian Pediatric Ischemic Stroke Registry estimated an incidence of approximately 0.67 cases per 100,000 children per year, with the highest frequency in neonates [1]. Pediatric CVST is usually multifactorial, and acute systemic illness, infection, dehydration, anemia, and prothrombotic states are among the most frequently reported associations [2-4]. Contemporary guidance also recognizes dehydration, infection or sepsis, anemia, and thrombophilic conditions as clinically relevant predisposing factors [5].

Clinical manifestations vary with age and may include seizures, altered consciousness, vomiting, focal neurological deficits, encephalopathy, or coma. In infants, these manifestations may initially be attributed to dehydration, shock, or infection, delaying diagnosis [2-4]. We describe a 9-month-old infant who developed extensive superior sagittal and left transverse sinus thrombosis in the setting of severe dehydration, hypovolemic shock, suspected sepsis, and severe microcytic anemia.

2 MATERIALS AND METHODS

2.1 Case report design

This single-patient case report was prepared from the clinical record, laboratory investigations, neuroimaging reports, treatment records, and follow-up data of a 9-month-old infant managed for cerebral venous sinus thrombosis. Clinical and laboratory values are reported as documented in the medical record. No experimental intervention was undertaken for the purpose of this report.

2.2 Ethical statement

Institutional ethics committee approval was not required for this single-patient case report in accordance with institutional policy. Written informed consent for publication, including publication of anonymized clinical images, was obtained from the patient's parent or legal guardian.

3 RESULTS

3.1 Case presentation

A previously healthy 9-month-old boy (weight 7 kg, length 68 cm, head circumference 45 cm) was admitted with severe dehydration and circulatory shock. He had been born at 39 weeks of gestation after an uncomplicated vaginal delivery, had normal psychomotor development, and was appropriately vaccinated. There was no known personal or family history of thrombosis and no parental consanguinity.

Five days before admission, he developed acute gastroenteritis with approximately five watery stools per day and repeated vomiting. He initially received symptomatic treatment and oral antibiotics as an outpatient. Because the gastrointestinal losses persisted and fever developed with progressive clinical deterioration, he was referred to our hospital.

At admission, the infant was obtunded, hypotonic, and poorly reactive. Marked dehydration was present, with decreased skin turgor and sunken eyes. Peripheral hypoperfusion included tachycardia at 180 beats/min, cold extremities, and mottled skin. The respiratory rate was 67 breaths/min with intercostal and subcostal retractions and nasal flaring, although pulmonary auscultation was unremarkable. Cardiac examination revealed normal heart sounds without murmurs. The abdomen was soft without hepatosplenomegaly or palpable masses, and deep tendon reflexes were preserved and symmetrical.

Initial laboratory investigations showed severe microcytic anemia with hemoglobin 7.1 g/dL and mean corpuscular volume 59.5 fL. The white blood cell count was 24,200/mm³, including 15,500 neutrophils/mm³, and the platelet count was 50,300/mm³. Serum sodium was initially 138 mmol/L and decreased to 128 mmol/L during the first day; potassium was 5.8 mmol/L. Procalcitonin was 21 ng/mL and C-reactive protein was 14 mg/L.

Chest radiography and cerebrospinal fluid analysis were unremarkable. Blood culture, stool culture, and cerebrospinal fluid culture were negative. Urinalysis showed leukocyturia with pseudohyphal yeast forms, and urine culture was reported as suggestive of a possible carbapenemase-producing *Escherichia coli*. Serologic testing for cytomegalovirus, Epstein-Barr virus, and human immunodeficiency virus was negative, and quantitative immunoglobulin levels were within normal limits.

The infant underwent fluid resuscitation and received intravenous ceftriaxone and gentamicin. Red blood cell transfusion was performed for severe anemia. By May 21, 2025, hemoglobin had increased to 12.4 g/dL and the platelet count to 139,000/mm³.

During hospitalization, the patient developed acute status epilepticus. Electroencephalography performed subsequently was normal. Contrast-enhanced brain computed tomography demonstrated extensive cerebral venous sinus thrombosis involving the superior sagittal sinus with extension into the left transverse sinus (Figure 1A-B).

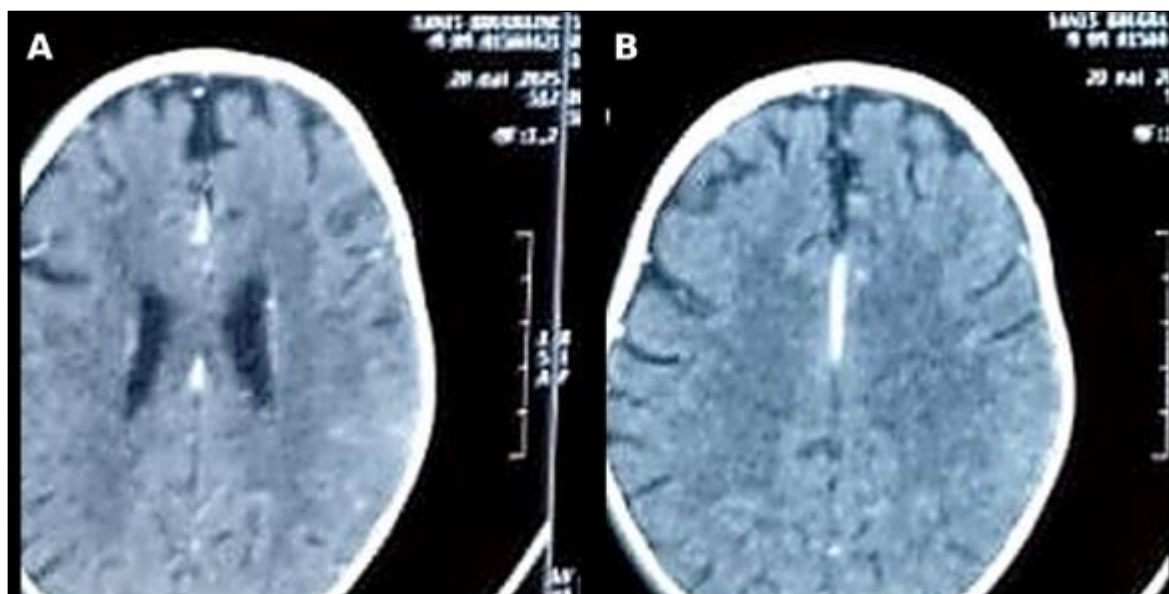


Figure 1: Initial contrast-enhanced brain computed tomography. Selected axial images from the initial examination showing findings consistent with superior sagittal sinus thrombosis and extension into the left transverse sinus.

Therapeutic anticoagulation with subcutaneous enoxaparin was initiated on May 17, 2025 after assessment of coagulation parameters. The formulation used was enoxaparin 2,000 anti-Xa IU/0.2 mL (20 mg/0.2 mL). Oral acenocoumarol was introduced on May 28, 2025 with an initial overlap period with low-molecular-weight heparin and serial coagulation monitoring.

A thrombophilia evaluation included antithrombin activity, proteins C and S, factor VII, homocysteine, and activated protein C resistance. No documented congenital thrombophilic disorder was identified in the available evaluation.

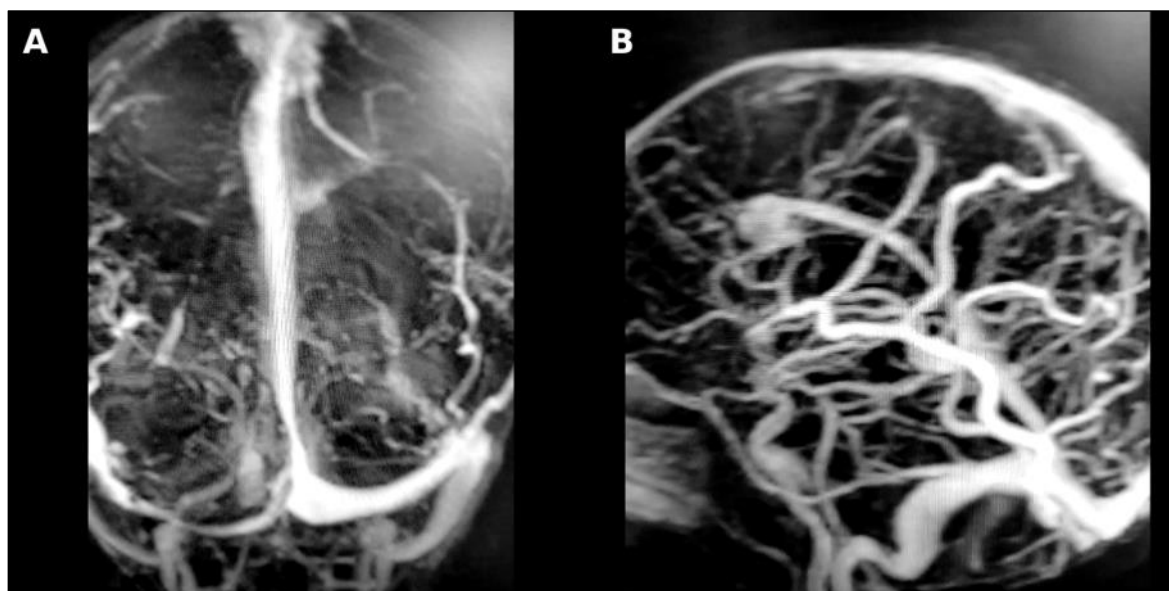


Figure 2: Follow-up magnetic resonance venography. MR venographic reconstructions show good opacification of the superior sagittal sinus without visible residual endoluminal thrombus or evidence of active cerebral venous thrombosis. A hypoplastic right transverse sinus is also noted.

The clinical course was favorable, with resolution of fever and no recurrent seizures. Follow-up brain computed tomography performed on June 19, 2025 showed partial radiological regression, although thrombosis of the superior sagittal sinus persisted. On the recommendation of the pediatric hematology team, anticoagulation was continued until July 2026, for a total duration of approximately 14 months.

Subsequent follow-up magnetic resonance venography showed good opacification of the superior sagittal sinus without a visible residual endoluminal thrombus or evidence of active cerebral venous thrombosis. A hypoplastic right transverse sinus was also noted (Figure 2A-B). At the latest follow-up, the child remained clinically well without recurrent seizures or new thrombotic events.

4 DISCUSSION

Pediatric CVST is uncommon and usually occurs in the setting of one or more acquired or inherited risk factors. The disorder is most often multifactorial rather than attributable to a single cause [1-4]. The present patient had several plausible transient acquired risk factors, particularly profound dehydration and hypovolemic shock secondary to gastroenteritis. Dehydration can promote thrombosis through intravascular volume depletion, reduced cerebral venous flow, and venous stasis, while systemic inflammation may enhance endothelial activation and coagulation [3-5].

The infectious component should be interpreted cautiously. The infant had fever, leukocytosis, markedly elevated procalcitonin, and circulatory shock, but blood culture was negative and no bloodstream pathogen was identified. Accordingly, suspected sepsis is more appropriate than microbiologically confirmed sepsis. Infection-related pediatric cerebral venous thrombosis is well recognized [6].

Severe microcytic anemia was another potential contributor. Anemia and microcytosis have been reported in pediatric CVST cohorts [2], and severe iron-deficiency anemia has been described in young children with cerebral sinovenous thrombosis [7]. A recent systematic review also supports an association between iron-deficiency anemia and stroke or cerebral venous thrombosis, although the evidence remains predominantly observational [8]. In our patient, iron deficiency was not definitively established; therefore, severe microcytic anemia is the more rigorous description.

The development of status epilepticus was an important diagnostic warning sign. Seizures are common in pediatric CVST, whereas nonspecific neurological manifestations in infants may obscure the diagnosis [1-4].

Anticoagulation is the mainstay of treatment for most pediatric cerebral sinovenous thrombosis. Updated ASH/ISTH guidance suggests anticoagulation rather than no anticoagulation for pediatric cerebral sinovenous thrombosis, while acknowledging the low certainty of pediatric evidence [9]. Observational pediatric studies also support this approach, showing greater thrombus propagation among untreated children and favorable neurological outcomes among anticoagulated patients [10,11].

Treatment duration should be individualized. Earlier European pediatric guidance recommended tailoring anticoagulation to etiology, persistence of thrombogenic factors, and speed of sinus recanalization [12]. More recent Kids-DOTT data indicate that selected patients with provoked pediatric cerebral sinovenous thrombosis may have favorable outcomes with shorter treatment, although persistent complete veno-occlusion was an exclusion from shortened therapy [13]. Therefore, the approximately 14-month course in this patient should not be interpreted as a universal standard; it reflected an individualized pediatric hematology decision in the setting of persistent thrombosis on early follow-up imaging.

Radiological follow-up is useful for documenting thrombus evolution, and pediatric cohorts have reported partial or complete recanalization during anticoagulation [14]. Our patient similarly showed persistent thrombosis on early computed tomography followed by favorable later magnetic resonance venographic evolution.

This report has limitations. It describes a single patient; systemic sepsis was not microbiologically confirmed; iron deficiency was not definitively demonstrated despite severe microcytosis; and serial vascular imaging was not available at standardized intervals. The precise frequency of the initial enoxaparin administration was not consistently documented in the available record and therefore is not reported.

5 CONCLUSION

Cerebral venous sinus thrombosis should be considered in infants with severe dehydration or systemic inflammatory illness who develop seizures, altered consciousness, or unexpected neurological deterioration. In this patient, severe dehydration and hypovolemic shock after acute gastroenteritis, together with suspected systemic infection and severe microcytic anemia, likely acted as transient acquired prothrombotic factors. Prompt neuroimaging identified extensive superior sagittal and left transverse sinus thrombosis. Anticoagulation, correction of precipitating factors, and prolonged hematology and radiological follow-up were associated with favorable clinical and imaging outcomes.

STATEMENTS ON COMPLIANCE WITH ETHICAL STANDARDS

Acknowledgments

The authors thank the clinical, radiology, and pediatric hematology teams involved in the patient's care.

Disclosure of conflict of interest

The authors declare no financial or non-financial conflicts of interest related to this case report.

Statement of ethical approval

Institutional ethics committee approval was not required for this single-patient case report in accordance with institutional policy.

Statement of informed consent

Written informed consent for publication, including publication of anonymized clinical images, was obtained from the patient's parent or legal guardian.

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