Cerebral Venous Thrombosis: A Narrative Review

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ABSTRACT
Background: Cerebral venous thrombosis (CVT) is an uncommon but potentially serious cerebrovascular disorder caused by partial or complete occlusion of the dural venous sinuses, cortical veins, or deep cerebral venous system. Although CVT accounts for approximately 0.5%–1% of all stroke cases, it has particular clinical importance because it frequently affects young adults and women of reproductive age. Its presentation is often heterogeneous and may range from isolated headache and features of intracranial hypertension to seizures, focal neurological deficits, venous infarction, intracerebral hemorrhage, or impaired consciousness.
Objective: This narrative review aimed to provide a comprehensive overview of cerebral venous thrombosis, with emphasis on cerebral venous anatomy, etiological and predisposing factors, pathophysiological mechanisms, clinical manifestations, diagnostic evaluation, prognostic predictors, and patient outcomes.
Keywords: Cerebral venous thrombosis, dural venous sinus thrombosis, cortical vein thrombosis, deep cerebral venous thrombosis, intracranial hypertension, venous infarction, magnetic resonance venography, computed tomography venography.
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INTRODUCTION

Cerebral venous thrombosis (CVT) is an uncommon but clinically important cerebrovascular disorder caused by partial or complete occlusion of the dural venous sinuses, cortical veins, or deep cerebral venous system. Although it represents only a small proportion of all stroke cases ranging from .05% to 3% of all stroke patients, CVT is associated with considerable diagnostic difficulty because of its variable and often nonspecific clinical presentation. Unlike arterial stroke, it frequently affects young adults and women of reproductive age, particularly in relation to pregnancy, puerperium, oral contraceptive use, and other prothrombotic conditions **(Ferro and Aguiar de Sousa, 2019).**

The clinical behavior of CVT is closely linked to the anatomical and physiological characteristics of the cerebral venous system. Cerebral venous drainage is maintained through a low-pressure, valveless, and highly interconnected network composed of superficial cortical veins, deep cerebral veins, and dural venous sinuses. This system drains blood from cortical and deep brain structures into the internal jugular veins and also contributes to cerebrospinal fluid absorption through arachnoid granulations, particularly along the superior sagittal sinus **(Liu et al., 2023, Shapiro et al., 2023)**

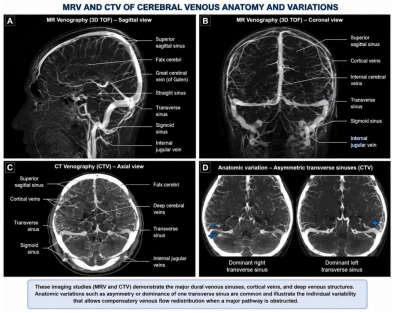

Figure 1: CT and MR Venographic Demonstration of Cerebral Venous Anatomy and Anatomical Variations

This venous architecture allows collateral redistribution of blood flow under normal conditions; however, it also explains the diverse consequences of venous obstruction.

Two distinct pathophysiologic mechanisms contribute to the clinical manifestations of CVST. First, thrombosis in the cerebral veins raises venous and capillary pressure, reducing cerebral perfusion. The resulting ischemia produces cytotoxic edema, which impairs energy-dependent membrane pumps and causes intracellular swelling. Disruption of the blood-brain barrier follows, leading to vasogenic edema and interstitial fluid accumulation. Elevated venous pressure can culminate in intraparenchymal hemorrhage. Second, obstruction of the

cerebral sinuses, especially when thrombi persist, interferes with cerebrospinal fluid absorption, causing elevated intracranial pressure (ICP), which in turn promotes both cytotoxic and vasogenic edema and may result in parenchymal hemorrhage. **(Bayot et al., 2023) (Aguiar de Sousa and Lucas Neto, 2024)**

he pathophysiology of CVT is mainly explained by the interaction between venous stasis, endothelial injury, and hypercoagulability.

Inherited thrombophilias, including Factor V Leiden mutation, prothrombin G20210A mutation, protein C deficiency, protein S deficiency, antithrombin deficiency, and hyperhomocysteinemia, may increase susceptibility to thrombosis.

Acquired thrombophilia such as nephrotic syndrome, or in those with antiphospholipid antibodies. Chronic inflammatory conditions, including systemic lupus erythematosus and inflammatory bowel disease, have also been associated with CVST, along with malignancy and vasculitides such as Wegener granulomatosis. Local infections like otitis media and mastoiditis may lead to thrombosis in the adjacent sigmoid and transverse sinuses. Additional risk arises following head trauma, certain neurosurgical interventions, direct injury to the venous sinuses or jugular veins (eg, catheterization), and even after lumbar puncture, dehydration, malignancy, COVID-19 infection. **(Ulivi et al., 2020) (Zhang et al., 2021).**

Clinically, CVT is characterized by a broad and heterogeneous spectrum. Headache is the most frequent manifestation and may occur alone or with signs of raised intracranial pressure such as vomiting, papilledema, diplopia, cranial neuropathy or visual disturbance. Other presentations include seizures, focal neurological deficits, cranial nerve palsies, altered mental status, encephalopathy, or coma. Severe manifestations are more likely in patients with deep venous system involvement, extensive sinus thrombosis, venous infarction, or hemorrhagic lesions **(Idiculla et al., 2020) (Müller et al., 2023)**

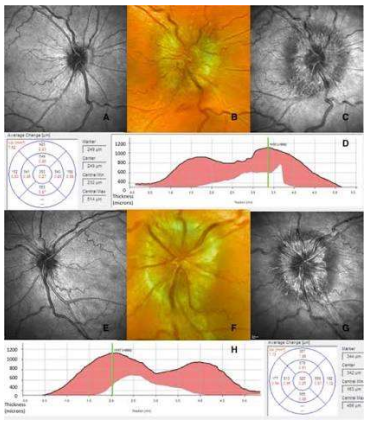

Figure 2: Ophthalmic Manifestations of Raised Intracranial Pressure in Cerebral Venous Thrombosis

Because clinical findings are often nonspecific, neuroimaging is essential for diagnosis. Direct signs of CVST on CT include the cord sign, a curvilinear hyperdensity within a cortical vein that can be seen up to 2 weeks after thrombus formation, and the dense triangle sign, a triangular hyperdensity in the superior sagittal sinus thrombosis. Intraparenchymal hemorrhages or infarcts may appear on noncontrast-enhanced CT and often cross vascular boundaries, with bilateral involvement in some cases.

MRI and MRV remain the gold standard for diagnosing CVT due to their superior sensitivity compared to CT. MRI is especially effective in evaluating parenchymal edema resulting from CVST. Imaging findings vary depending on the age of the thrombus, as signal characteristics evolve over time. Thrombi formed within the first 7 days are more challenging to visualize, but by the 2nd week, T1- and T2-weighted sequences typically demonstrate hyperintense signals. The combination of abnormal venous sinus signals on MRI and absent flow on MRV confirms CVT. A 2-dimensional time-of-flight (TOF) sequence revealing the absence of a flow void in the dural sinus is considered the most sensitive modality. Computed tomography venography and magnetic resonance venography are more accurate for confirming venous occlusion, identifying the involved sinus or vein, and assessing the extent of thrombosis. **(Pauls et al., 2020, Ota, 2024).**

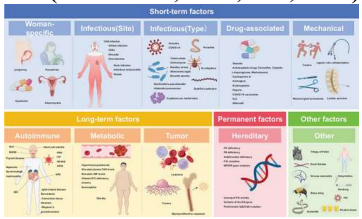

Figure 3: Risk factors for Cerebral Venous Thrombosis.

Laboratory evaluation is important for identifying underlying causes and guiding long-term management. Routine investigations may reveal anemia, thrombocytopenia, infection, inflammation, renal or hepatic dysfunction, or coagulation abnormalities. Etiological assessment should be individualized and may include thrombophilia screening, antiphospholipid antibodies, autoimmune markers, pregnancy testing, infection screening, and malignancy evaluation, especially in unexplained or recurrent cases. **(Jianu et al., 2021, Zöller et al., 2020).**

Management begins with identifying and treating life-threatening complications of CVST, such as increased ICP, seizures, and coma. Patients who present with seizures and have a hemorrhage or infarction on neuroimaging require specific anticonvulsant therapy along with seizure prophylaxis. Anticoagulation in CVST aims to prevent thrombus propagation, facilitate recanalization, and reduce the risk of recurrence. The approach has been debated due to concerns about hemorrhagic transformation of cerebral infarcts prior to anticoagulant administration. However, anticoagulation remains essential for limiting thrombus extension, reopening occluded cerebral veins. Therapy should begin as soon as the diagnosis is confirmed. Intravenous unfractionated heparin or subcutaneous low-molecular-weight heparin (LMWH) serves as a bridge to oral anticoagulation using a vitamin K antagonist (VKA), with a target international normalized ratio between 2.0 and 3.0. Recent evidence suggests that direct oral anticoagulants (DOACs) are a suitable alternative to VKAs for treating CVT. Treatment duration depends on the underlying cause. For provoked CVT, anticoagulation is generally continued for 3 to 6 months, while unprovoked CVT typically requires 6 to 12 months of therapy. Indefinite anticoagulation may be warranted in patients with recurrent CVT, or CVT in the context of severe thrombophilia.

Catheter-directed thrombolysis may be considered in settings where the prognosis is poor, for example, in cases involving extremely large cerebral venous thrombi and clinical deterioration despite anticoagulation therapy. Fibrinolytics, however, carry an increased risk of intracranial hemorrhage. In cases of large venous infarcts and hemorrhages causing a mass effect with a risk of herniation, decompressive surgery may improve clinical outcomes, particularly when performed early. EVT is recommended for patients who have progressive clinical deterioration or who have failed standard therapy or have contraindications to it.

The prognosis of CVT has improved with earlier diagnosis, modern neuroimaging, and appropriate anticoagulant therapy. Many patients achieve favorable functional recovery, but outcome remains variable. Overrall, 80% to 90% of patients with CVT achieve functional independence (modified Rankin Scale score of 0–2).However, several studies reported a high prevalence of residual symptoms related to cognition, mood, fatigue, and headache, which may impede return to previous level of activity.

Poor prognostic factors include coma at presentation, deep venous system thrombosis, intracerebral hemorrhage, malignancy, central nervous system infection, recurrent thrombosis, and persistent prothrombotic disorders. Long-term complications may include chronic headache, epilepsy, visual impairment, residual neurological deficits, recurrent venous thromboembolism, and persistent intracranial hypertension **(Ferro et al., 2004, Simaan et al., 2023)**

In Egypt and North Africa, CVT has particular clinical relevance because regional factors such as dehydration, high ambient temperature, infection-related thrombosis, delayed medical consultation, pregnancy-related risk, and limited access to early specialized neuroimaging may influence presentation and outcome. **(Ferro and Aguiar de Sousa, 2019)**

In our study, we predict that pregnancy, puerperium, oral contraceptive use, infection, malignancy, autoimmune disease, dehydration and systemic inflammatory conditions will be the most common risk factors for cerebral venous thrombosis in our patients as **(Hameed et al., 2021)** found.

Conclusion:

A Narrative review focusing on risk factors, clinical presentation, and diagnosis of cerebral venous thrombosis with concern on factors affecting prognosis and outcome.

Therefore, studying CVT in Minia University Hospital may help clarify local patterns of risk factors, clinical manifestations, radiological findings, complications, and prognostic predictors, supporting earlier diagnosis and improved patient care.

A comprehensive understanding of these elements is essential for improving clinical suspicion, diagnostic accuracy, risk stratification, and long-term outcomes

We predict the next era of research on cerebral venous thrombosis will focus on comparing warfarin versus new oral anticoagulant as a treatment for cerebral venous thrombosis and their effect on prognosis of patients with CVT.

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