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Review Article

Cerebral Venous Thrombosis: An Update on Diagnosis and Management

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Abstract

Cerebral venous thrombosis (CVT) is an uncommon but significant cause of stroke, accounting for approximately 0.5–3% of all cerebrovascular events. It results from thrombosis within the cerebral venous system, leading to increased venous pressure, venous congestion, cerebral edema, and potentially venous infarction. This review summarizes current knowledge regarding the anatomy, pathophysiology, clinical presentation, diagnostic strategies, and therapeutic management of CVT. Diagnosis relies heavily on neuroimaging, with magnetic resonance imaging (MRI) combined with magnetic resonance venography (MRV) representing the gold standard. Anticoagulation remains the cornerstone of therapy, even in cases with intracerebral hemorrhage, with low-molecular-weight heparin preferred during the acute phase and transition to oral anticoagulants thereafter. Direct oral anticoagulants have emerged as safe and effective alternatives to vitamin K antagonists. Early recognition and treatment significantly improve prognosis, with 80–90% of patients achieving favorable neurological recovery. However, delays in diagnosis and inadequate management of risk factors can lead to severe complications or mortality. Ongoing research continues to refine treatment duration and preventive strategies, especially in high-risk populations such as pregnant women, cancer patients, and individuals with thrombophilia.

Keywords: *Cerebral venous thrombosis; Venous sinus thrombosis; Stroke; Anticoagulation; Diagnosis; MRI; Risk factors; Intracranial hypertension; Prognosis.*

1. Introduction and Anatomy

The cerebral venous system, composed of a set of veins and venous sinuses, is responsible for the brain's blood drainage.

Two venous systems can be distinguished according to their location and dependent structures: on the one hand, the superficial venous system, responsible for cortical drainage, consists of cortical veins that conduct blood into the dural venous sinuses, known as the superior sagittal sinus, transverse sinus, and sigmoid sinus, which ultimately drain into the internal jugular veins.

On the other hand, the deep venous system is responsible for draining subcortical structures. It is formed by the internal cerebral veins, which converge into the vein of Galen, from where blood flows into the straight sinus. This drains into the so-called confluens sinuum, the junction point between both venous systems, to finally drain collectively into the internal jugular veins.

Isolated thrombosis of a structure is most frequent in the superior sagittal sinus and the transverse sinus, though combined involvement of different sinuses is very common. The location of thrombosis has prognostic value, with cases being more severe if multiple venous structures are involved, particularly if the deep venous system is affected.

The presence of a clot in any of these cerebral venous structures is known as cerebral venous thrombosis. Interruption of blood flow at this level increases cerebral venous pressure, which, if elevated enough, can exceed the capacity of the collateral network, preventing drainage of the affected area and inducing venous congestion. This can evolve into cerebral edema and secondary venous infarction (1).

It is estimated to account for between 0.5–3% of all strokes, and is considered an uncommon cause of stroke (2). Its incidence in the general population is between 1.3 and 1.7 per 100,000 persons/year, but in young women it reaches its maximum, up to 2.8 per 100,000 persons/year (3).

This population subgroup is particularly susceptible to developing this condition due to the influence of hormonal factors that generate a hypercoagulable state, such as the use of contraceptives (especially those containing estrogen) (2) or physiological states such as pregnancy and puerperium, especially within the first six postpartum weeks (4).

This risk can be further potentiated if other risk factors coexist, such as obesity, polycystic ovary syndrome, diabetes, or hereditary thrombophilias such as antiphospholipid syndrome (5).

Cancer also stands out as an established risk factor due to the procoagulant state associated not only with the disease itself but also with certain oncological therapies (notably tamoxifen, cisplatin, l-asparaginase, and antiangiogenic treatments). It has been primarily described in the context of hematological malignancies, although also in primary brain tumors capable of invading cerebral venous structures by contiguity (1). In some cases, cerebral thrombosis may be the first manifestation of an occult cancer (6).

Autoimmune diseases such as systemic lupus erythematosus, Sjögren's syndrome, or inflammatory bowel disease also carry an increased risk of this pathology (2).

Other related risk factors include traumatic brain injury, recent neurosurgery, or transient states such as the use of certain drugs, especially corticosteroids, or infections localized in the head and neck (2).

2. Pathophysiology and Causes

2.1 Mechanism of Thrombus Formation

Vascular injury or initial endothelial dysfunction exposes collagen and tissue factor present in the subendothelium. Collagen promotes platelet adhesion and activation through GPVI receptors, while tissue factor triggers the extrinsic coagulation pathway, favoring thrombin generation and the conversion of fibrinogen into fibrin. In turn, thrombin activates PAR receptors on the platelet surface, inducing the release of secondary agonists such as ADP, serotonin, and thromboxane A_2. These mediators amplify the platelet response, recruiting and activating additional platelets and consolidating thrombus formation. Finally, a fibrin network stabilizes the platelet aggregate and incorporates erythrocytes and leukocytes, constituting the definitive thrombus, which can be released into the venous circulation and reach the cerebral circulation (7).

2.2 Pathophysiology

The pathophysiology of cerebral venous thrombosis (CVT) is not yet fully understood. This lack of clarity appears to be largely due to the significant anatomical variability among individuals and to the fact that much of the available research derives from experimental studies conducted in animal models (8,9).

Once the thrombus has formed and reaches the venous sinuses, a retrograde increase in venous pressure occurs, leading to brain injury through two main mechanisms. On one hand, the increase in venular and capillary pressure disrupts the blood–brain barrier, promoting the development of vasogenic edema. In addition, capillary rupture may lead to intraparenchymal hemorrhage, while the reduction in capillary perfusion pressure decreases cerebral blood flow. This hypoperfusion compromises neuronal metabolism, leads to cell death, and generates cytotoxic edema, a process known as venous infarction (8,9).

On the other hand, dural sinus thrombosis interferes with the normal absorption of cerebrospinal fluid (CSF), which is carried out mainly through arachnoid granulations and the glymphatic system. When drainage is obstructed, CSF pressure increases, culminating in intracranial hypertension. This complication is more frequently observed when the obstruction involves the dural sinuses, although it can also occur in cases of jugular vein or sigmoid sinus thrombosis (9,10).

2.3 Main Causes

Transient causes include infections of the central nervous system, ear, paranasal sinuses, oral cavity, face, or neck, as well as systemic infections. Pregnancy and puerperium, dehydration, mechanical factors such as head trauma, lumbar puncture, neurosurgery, or jugular catheter obstruction, and exposure to certain drugs (including oral contraceptives, hormone therapy, androgens, tamoxifen, and glucocorticoids) are also recognized causes (8,9).

Permanent causes include various inflammatory diseases such as systemic lupus erythematosus, Behçet's disease, sarcoidosis, and granulomatosis with polyangiitis. They also include central nervous system or hematological tumors, as well as structural abnormalities of the CNS such as dural fistula. An important group corresponds to thrombophilias, both hereditary and acquired, including protein C and S deficiencies, antithrombin deficiency, factor V Leiden mutation, G20210A prothrombin gene mutation, antiphospholipid syndrome, nephrotic syndrome, paroxysmal nocturnal hemoglobinuria, hyperhomocysteinemia, and anemia. Finally, congenital heart disease and thyroid disorders have been described as predisposing factors (9,10).

Classification of Causes of Cerebral Venous Sinus Thrombosis

Table 1. Classification of cerebral venous sinus thrombosis causes according to whether they represent permanent or transient risk factors.

CATEGORY	CAUSES
TRANSIENT	Infections: CNS, ear, paranasal sinuses, oral cavity, face, neck, systemic infections. Pregnancy and puerperium. Dehydration. Mechanical factors: traumatic brain injury, lumbar puncture, neurosurgeries, jugular catheter obstruction. Medications: Oral contraceptives, hormone therapy, androgens, tamoxifen, glucocorticoids. Inflammatory diseases: SLE, Behçet's disease, sarcoidosis, granulomatosis with polyangiitis, etc. Tumors: CNS, hematological. Dural fistula.
PERMANENT	Hereditary/acquired thrombophilias: protein C or S deficiency, antithrombin deficiency, factor V Leiden mutation, G20210A prothrombin mutation, antiphospholipid syndrome, nephrotic syndrome, paroxysmal nocturnal hemoglobinuria, hyperhomocysteinemia, anemia. Others: congenital heart disease, thyroid disorders.

3. Clinical Manifestations

3.1 Clinical Presentation

The signs and symptoms of cerebral venous thrombosis (CVT) are highly variable and often mimic those of other neurological disorders, making diagnosis challenging. This clinical heterogeneity is explained by the fact that manifestations depend on the location of the affected vein or venous sinus and, in some cases, may involve several sites simultaneously.

CVT presentations can be divided into:

- Isolated headache or increased intracranial pressure.
- Focal neurological deficits.
- Subacute encephalopathy.
- Cavernous/petrosal sinus syndrome with multiple cranial neuropathies.

There is often a significant diagnostic delay (3–16 days), especially in cases presenting with isolated headache, particularly in those with a history of headaches, which poses a considerable clinical challenge.

Headache is present in approximately 90% of patients with CVT. Venous drainage failure and the resulting increase in intracranial pressure may manifest as headache, nausea, vomiting, transient visual obscurations (13–27%), loss of visual acuity, papilledema, or diplopia (6–14%). In some cases, sixth cranial nerve palsy or other cranial neuropathies are observed (6–11%). When any combination of these features occurs without other neurological signs, the syndrome is known as isolated intracranial hypertension, estimated to occur in nearly one-third of patients with CVT. However, up to a quarter of patients may present with isolated headache without additional clinical signs of raised intracranial pressure.

The clinical characteristics of CVT-related headache are notoriously diverse. In patients with headache, a detailed neurological examination, assessment of features known to be associated with CVT, and consideration of secondary causes of headache in the differential diagnosis can help improve diagnostic accuracy (2,11,12).

Headache attributed to CVT does not have specific features; it requires that the headache develops in close temporal relation to CVT and that it worsens significantly in parallel with clinical or radiological signs of CVT extension, or improves significantly after CVT resolution. Key clinical features of CVT-associated headache include worsening in the supine position or with Valsalva maneuvers, subacute onset, and a more common diffuse than unilateral distribution. However, acute presentations mimicking migraine or thunderclap headache may also occur (13). The transverse sinus is a common CVT site in patients whose only manifestation is headache.

The relationship between venous recanalization and headache evolution remains unclear, suggesting that the underlying pathophysiological mechanisms are more complex than currently understood.

In addition to headache, many patients with CVT may present with focal neurological deficits or seizures. Symptoms depend on the affected brain area. Common focal symptoms include hemiparesis, aphasia, and visual loss. Unlike arterial strokes, deficits in CVT are usually progressive and sometimes bilateral, particularly when the superior sagittal sinus is affected.

Seizures, both generalized and focal, are much more common in CVT than in arterial ischemic strokes, occurring in nearly 40% of patients at initial presentation.

Finally, patients with deep venous system thrombosis may develop subacute encephalopathy with confusion and lethargy or experience rapid neurological deterioration progressing to coma due to edema of the bilateral thalami, basal ganglia, or other deep structures typically drained by these veins. Approximately 10% of patients with CVT have deep cerebral venous system thrombosis (2,11,12).

3.2 Atypical Presentations

Although most patients with CVT present with a subacute course and common clinical features, atypical forms of onset exist that may delay diagnosis or mimic other conditions.

The temporal evolution of CVT can be divided into:

- **Hyperacute (seconds to minutes):** exceptional and difficult to distinguish, for example presenting as thunderclap headache.
- **Acute (≤48 h):** challenging because focal neurological deficits can be mistaken for arterial ischemic stroke. It is estimated that 5–40% of patients with CVT may present acutely with focal deficits.
- **Subacute (>48 h–≤30 days):** the most frequent, with progressively worsening symptoms.
- **Chronic (≥1 month):** the clinical picture may resemble idiopathic intracranial hypertension. These patients often have prolonged headache, papilledema, and sometimes cranial nerve palsies, without other focal deficits.

One of the most relevant atypical forms is thunderclap headache, described in fewer than 5% of cases, which can mimic the typical presentation of subarachnoid hemorrhage.

Another unusual but documented manifestation is paralysis of one or several cranial nerves, rarely without other signs or symptoms. This may result from direct pressure exerted by the thrombus on the nerves or from venous stasis in the posterior fossa that drains into the venous sinuses, as well as edema in adjacent structures. Cavernous sinus thrombosis, in particular, presents with a characteristic clinical spectrum including headache, cranial nerve III, IV, V, or VI palsies, and ocular symptoms such as proptosis and conjunctival injection. Conversely, inferior petrosal sinus syndrome presents with headache and cranial nerve VII or VIII palsy, which may manifest with sensorineural hearing loss, vertigo, tinnitus, or peripheral facial paralysis, potentially being confused with other posterior fossa pathologies.

These atypical forms, although less common, highlight the importance of considering CVT in the differential diagnosis of unusual neurological presentations, especially when the clinical profile does not fully match other more common diseases (14,15).

3.3 Differences in Children, Adults, and Pregnant Women

Age and physiological status significantly influence the clinical presentation of CVT.

In the pediatric population, CVT is characterized by high clinical polymorphism. The most frequent manifestations are:

- **Seizures:** much more prevalent than in adults, often the initial symptom.
- **Fever:** often associated with local infections (otitis, sinusitis, mastoiditis).
- **Nonspecific symptoms in neonates:** may present as acute diffuse encephalopathy with lethargy, irritability, vomiting, feeding refusal, with or without seizures and focal neurological signs. These symptoms are easily confused with neonatal sepsis or perinatal hypoxic-ischemic encephalopathy.
- **Older children:** signs of intracranial hypertension (headache, vomiting, papilledema) predominate, along with seizures and focal deficits.

Children also have a higher risk of early ischemic or hemorrhagic stroke due to vessel fragility and immature regulatory mechanisms.

A high index of suspicion is necessary in the pediatric population, especially after head trauma or head and neck infections, since CVT etiology is usually provoked, in contrast to adults, where up to one-third of cases may be idiopathic.

In adults, as previously noted, the predominant symptom is headache, with or without signs of intracranial hypertension. Focal neurological deficits and seizures are also common, though less so than in children. In general, adults present a more stereotypical clinical course, facilitating recognition, except in atypical presentations.

Pregnancy and puerperium represent important risk factors, with up to 15–20% of CVT occurring in this context. Physiological changes during pregnancy increase the risk of thromboembolic events, and after delivery, additional predisposing factors such as dehydration, infections, cesarean section, or CSF hypotension from epidural anesthesia may also contribute.

In women, CVT often presents more acutely (13%), with headache as the main symptom. These differences disappear in older patients or in those without gender-related risk factors.

Early identification of CVT is crucial, as it can be mistaken for other more benign entities such as tension headache, migraine, or post-dural puncture headache, and is associated with increased maternal and fetal risk (16,17).

4. Diagnosis

The diagnosis of cerebral venous thrombosis requires a high level of clinical suspicion given the wide variability in clinical presentation, as previously explained.

4.1. Neuroimaging Studies

Non contrast head CT:

In approximately 25% of cases, neuroimaging studies reveal no significant abnormalities. Nevertheless, subtle findings may be identified, such as linear hyperdensities in cortical veins or dural venous sinuses (the "filled delta sign"), along with more evident features including cerebral edema, diffuse hypodensities, venous infarctions without a defined arterial distribution, and lobar or convexity subarachnoid hemorrhages[18].

Nonhemorrhagic infarcts are found on CT in approximately 10 percent, but up to 20 percent on MRI. Nonhemorrhagic lesions include focal areas of hypodensity caused by vasogenic edema or venous infarction, usually not respecting typical arterial boundaries, as well as diffuse brain edema[18].

Contrast head CT

Contrast-enhanced computed tomography (CECT) is the diagnostic modality of choice in the emergency setting when cerebral venous thrombosis (CVT) is suspected, with a reported sensitivity ranging from 75% to 100% depending on the affected venous sinus, when compared with magnetic resonance imaging (MRI). The absence of opacification of the superior sagittal sinus following contrast administration results in the characteristic "empty delta sign," a classic finding in this scenario. Its main limitation lies in its limited spatial resolution for evaluating cortical veins and the deep venous system[18,19].

In addition, several anatomic variants that may be seen on confirmatory vascular and MRI-based testing can mimic sinus thrombosis. These include: sinus atresia or hipoplasia, asymmetric sinus drainage, Arachnoid granulations and Intrasinus septal.

Brain magnetic resonance with and without our contrast

It is considered the gold standard for the diagnosis of cerebral venous thrombosis (CVT). T1-weighted sequences typically show hypo- or isointense thrombi, while T2-weighted sequences are useful for defining ischemic and hemorrhagic lesions. Post-contrast T1 imaging allows evaluation of superficial and deep venous sinuses and enables monitoring of recanalization to guide anticoagulation discontinuation[1819,20].

Parenchymal brain lesions associated with CVT include vasogenic edema, venous infarctions, and intracerebral or subarachnoid hemorrhages, each with characteristic signal patterns across T1, T2, T2*, diffusion, and SWI sequences[19,20].

Conventional MRI shows a pooled sensitivity of approximately 82% and specificity of 92%, though diagnostic performance may vary by location, with reduced sensitivity in the left lateral sinus, straight sinus, and cortical veins.

Magnetic resonance venography (MRV) is a valuable adjunct that provides direct visualization of thrombosed sinuses. Non-contrast techniques (time-of-flight and phase-contrast) demonstrate absence of flow, whereas gadolinium-enhanced MRV improves detection of thrombi in small-caliber sinuses and differentiates thrombosis from anatomical variants such as sinus hypoplasia. Combined with conventional MRI, MRV optimizes diagnostic yield. Meta-analyses report MRV sensitivity of 86% and specificity of 94%, comparable to CT venography, with superior differentiation of hypoplastic versus chronically thrombosed sinuses[19,20].

– Cerebral arteriography. Although not a routine test, it is useful in cases of nondiagnostic MRI and high suspicion or to assess complications such as dural fistula (in the case of unexplained worsening of HT)[18].

4. 2. Laboratory

Plasma D-dimer measurement is a valuable diagnostic tool in the evaluation of cerebral venous thrombosis (CVT). Elevated values (>500 μg/mL) demonstrate a sensitivity of 94.1% and specificity of 97.5%, particularly in acute and subacute presentations. However, a normal D-dimer level does not exclude CVT and may be misleading in patients with suggestive symptoms, identifiable predisposing factors, low thrombus burden (e.g., isolated cortical vein thrombosis), or subacute-to-chronic forms of the disease[21].

Routine laboratory investigations are recommended to identify underlying conditions that may predispose to CVT, including hypercoagulable states, infections, or inflammatory processes. These include complete blood count, basic metabolic panel, coagulation studies, pregnancy test in patients of childbearing potential, urinalysis, and iron studies[2,21].

A comprehensive thrombophilia workup should include measurements of antithrombin, protein C, and protein S levels, testing for the Factor V Leiden and prothrombin G20210A genetic variants, MTHFR gene variants, as well as antiphospholipid antibody testing (anticardiolipin antibodies, anti-β2 glycoprotein I antibodies, and lupus anticoagulant functional assay) and homocysteine levels. Hemoglobin electrophoresis should be performed if sickle cell disease is suspected[2,21,22].

In patients over 50 years of age with unprovoked venous thromboembolism, a malignancy workup is warranted. This may include serum tumor markers, computed tomography of the chest, abdomen, and pelvis, thyroid or testicular ultrasound depending on clinical context, and, in cases of high suspicion, whole-body PET-CT[23].

5. Treatment and Prognosis

Treatment is based on the early initiation of anticoagulation, management of intracranial hypertension, and other possible complications, as described below.

5.1 Anticoagulant Treatment

Unfractionated heparin (UFH) and low-molecular-weight heparin (LMWH):

The goal of anticoagulation therapy in cerebral venous sinus thrombosis is to prevent distal extension of the thrombus into other veins or sinuses, accelerate recanalization of the occluded vein or sinus, and treat the prothrombotic state to prevent recurrent cerebral or systemic venous thrombosis (2).

Clinical trials to date, such as that by Einhaupl et al. (n=20), demonstrated the benefit of UFH compared with placebo (24,25). Similarly, numerous observational studies, such as the prospective multicenter study by Hamish P. et al., support the use of LMWH or UFH, since nearly 80% of patients achieved complete recovery at 16 months (22,25).

Currently, both American (AHA/ASA) and European (ESO) guidelines recommend initial treatment with heparin as a bridge before starting oral anticoagulation (26,30), preferably with LMWH over UFH, based on studies—although not statistically significant—that suggest a better safety profile, fewer hemorrhagic complications, and improved outcomes with LMWH (12,22,26).

Intracranial hemorrhage (ICH) secondary to CVT is not a contraindication for treatment with LMWH or UFH. This is because ICH results from venous congestion due to thrombosis, and anticoagulation promotes recanalization, reperfusion, and venous outflow (27).

Although data are limited and randomized clinical trials are lacking, there is evidence and guideline-based recommendation for the use, efficacy, and safety of heparin in the pediatric population (28,29).

Oral anticoagulation with vitamin K antagonists (VKAs):

According to AHA/ASA and ESO guidelines, once the patient is stabilized and the acute phase is managed, treatment with vitamin K antagonists can be initiated. In addition to having similar efficacy, they offer the advantage of oral administration and better outpatient management compared with heparin (2,27).

Regarding treatment duration, major guidelines recommend between 3–12 months or even indefinite therapy depending on patient characteristics and recurrence risk:

- 3–6 months in patients with CVT provoked by a transient risk factor.
- 6–12 months for patients without a clear trigger or known temporary risk factor.
- Indefinite anticoagulation for patients with recurrent CVT or a permanent high prothrombotic risk.

Currently, the EXCOA-CVT randomized clinical trial (Extending oral antiCOAgulation treatment after acute Cerebral Vein Thrombosis) is underway, aiming to compare short- and long-term anticoagulation efficacy and safety in preventing new venous thrombotic events (30).

Direct oral anticoagulants (DOACs):

DOACs have demonstrated, both in clinical trials and real-world practice, a more favorable safety profile, pharmacokinetics, and administration compared to VKAs (26). This, along with several studies showing similar efficacy between VKAs and DOACs for venous thromboembolism treatment and prevention (24), has spurred interest in evaluating their use in CVT.

Notable trials include RESPECT-CVT, a randomized trial comparing dabigatran with warfarin after initial parenteral anticoagulation for 6 months. The study concluded that both drugs had similar efficacy and safety in preventing CVT recurrence, without significant differences in venous recanalization, though no statistical superiority was demonstrated due to study limitations (36). Similarly, the SECRET trial compared rivaroxaban with warfarin, reaching similar conclusions regarding efficacy, with low rates of thrombotic and hemorrhagic events, though limited by study size (32).

Other international observational studies, such as DOAC-CVT and ACTION-CVT, again concluded that DOACs are real and effective therapeutic options for CVT. The ACTION-CVT study in particular showed a lower rate of major hemorrhage (33,34).

These studies excluded patients with active cancer, antiphospholipid syndrome, or pregnant women. In the pediatric population, the EINSTEIN-Jr substudy compared rivaroxaban against VKAs or heparin, showing low recurrence and bleeding rates in both groups, with similar results regarding venous recanalization (35).

Management of Major Complications

Epilepsy:

Seizures occur in 35–50% of CVT patients during the acute phase (36). There is ongoing debate about seizure prophylaxis in CVT. Current guidelines recommend initiating antiepileptic treatment after the first seizure, given the structural cause and predisposition to recurrence, preferably intravenously (37). Due to the risk of late seizures more common in patients with acute seizures—treatment should be continued for at least the first year, with gradual tapering thereafter (36).

Intracranial hypertension (ICH):

Up to 40% of CVT patients present with ICH. This increase in intracranial pressure is mainly due to venous outflow obstruction and tissue congestion, aggravated by impaired CSF reabsorption leading to diffuse cerebral edema (2). The initial approach consists of adequate anticoagulation to promote reperfusion and venous outflow. Acetazolamide is a safe option, reducing CSF production (37). In contrast, corticosteroids are not useful in the acute treatment of ICH secondary to CVT; they not only fail to reduce intracranial pressure but may worsen existing vascular or structural lesions (38).

Although no specific clinical trials exist, guidelines also endorse other therapeutic options for managing this complication, such as hypertonic saline, mannitol, or decompressive craniectomy (2,37,39).

Hydrocephalus:

In CVT, CSF reabsorption through arachnoid granulations into the venous system may be impaired, leading to hydrocephalus. This is rare and usually associated with internal cerebral vein thrombosis or thalamic hemorrhages. Management is primarily surveillance and intracranial pressure monitoring in intensive care or neurocritical settings, with CSF drainage via ventriculostomy or ventriculoperitoneal shunt if needed (2).

Prognosis

Mortality:

CVT generally has a favorable prognosis, with mortality rates ranging from 1–6% during the acute phase, explained by improvements in treatment, better understanding of risk factors, and advances in diagnostic methods (26,39–41). This percentage may rise to 8–10% during follow-up (2,39–41).

The main causes of death in the acute phase include transtentorial herniation, while later deaths are often due to sudden hemorrhage or respiratory distress (42). Poor prognostic factors include concomitant CNS infection, malignancy, low consciousness, coma, motor deficit, deep venous system thrombosis, intracranial hemorrhage, older age, and male sex (41).

Neurological recovery:

Regarding neurological and functional recovery, 80–90% of patients achieve good outcomes without significant disability, defined in studies as a modified Rankin Scale (mRS) score <2 (43). Nevertheless, a considerable proportion of patients experience residual symptoms such as headache, fatigue, cognitive impairment, or mood disturbances, which may hinder return to normal life or work (43).

Risk of recurrence:

After a first CVT event, recurrence risk is low, ranging between 2–4% for another intracranial thrombotic event, while the risk of other extracranial thromboembolic events is higher (4–7%) (25). Risk factors for recurrence in adults include prior history of venous thromboembolism, evidence of acquired or hereditary prothrombotic states, female sex, or Black ethnicity (41).

Prevention in High-Risk Patients

Prevention strategies in high-risk patients or those who have already suffered CVT focus on identifying and managing risk factors. Preventive measures include (2,36):

- Correction of reversible factors that favor or worsen disease progression, such as dehydration, infections, anemia, withdrawal of prothrombotic drugs, etc.
- In women with personal or family history of thrombosis and additional risk factors (e.g., obesity, smoking), oral contraceptives—especially estrogen-containing formulations—and estrogen-based hormone therapy should be avoided.
- In situations of unavoidable temporary high risk (major surgery, pregnancy/puerperium, prolonged immobilization, active cancer, or severe thrombophilia), anticoagulant prophylaxis with LMWH may be initiated.
- Close clinical monitoring for patients with autoimmune diseases, malignancies, or congenital/chronic hematologic disorders.

In the absence of specific clinical trials or prevention guidelines, the main strategy remains the identification, classification, and, when possible, treatment of risk factors or triggers.

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