REVIEW / REVISIÓN DE TEMA

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# Update on the Acute Management of Cerebral Venous Thrombosis

## Actualización sobre el manejo agudo de la trombosis venosa cerebral

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## SUMMARY

Cerebral venous thrombosis (CVT) is a rare but increasingly recognized cause of stroke with highly variable clinical presentations. Early diagnosis relies on CT or MR venography, as symptoms often range from headache to seizures, focal deficits, or coma. Anticoagulation remains the cornerstone of therapy, even in patients with intracranial hemorrhage. Low-molecular-weight heparin is preferred in the acute phase, while unfractionated heparin is reserved for patients requiring imminent neurosurgical intervention. Transition to oral anticoagulation is traditionally achieved with vitamin K antagonists, although recent randomized and observational studies suggest that direct oral anticoagulants provide similar efficacy with fewer major bleeding complications. Neurocritical care is vital for patients with severe CVT, focusing on seizure management, intracranial hypertension control, and systemic stabilization. Decompressive craniectomy is a life-saving option in cases of malignant cerebral edema. Endovascular therapy has not demonstrated superiority over standard anticoagulation and is currently reserved for refractory cases. Overall, timely diagnosis, tailored anticoagulation, and multidisciplinary critical care remain the key determinants of outcome in patients with CVT.

KEYWORDS: Intracranial sinus thrombosis, Anticoagulants, Direct-Acting Oral Anticoagulants, Critical Care, interventional radiology.

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## RESUMEN

La trombosis venosa cerebral (TVC) es una causa rara pero cada vez más reconocida de enfermedad cerebrovascular, con presentaciones clínicas altamente variables. El diagnóstico temprano se basa en la venografía por tomografía computarizada o resonancia magnética, ya que los síntomas suelen variar desde cefalea hasta crisis epilépticas, déficit focal o coma. La anticoagulación sigue siendo la piedra angular del tratamiento, incluso en pacientes con hemorragia intracraneal. En la fase aguda se prefiere la heparina de bajo peso molecular, mientras que la heparina no fraccionada se reserva para pacientes que requieren una intervención neuroquirúrgica inminente. La transición a la anticoagulación oral se logra tradicionalmente con antagonistas de la vitamina K, aunque estudios recientes, tanto aleatorizados como observacionales, sugieren que los anticoagulantes orales directos ofrecen una eficacia similar con menos complicaciones hemorrágicas mayores. El cuidado neurocrítico es fundamental en pacientes con TVC grave, enfocándose en el manejo de las crisis epilépticas, el control de la hipertensión intracraneal y la estabilización sistémica. La craniectomía descompresiva es una opción que salva vidas en casos de edema cerebral maligno. La terapia endovascular no ha demostrado superioridad sobre la anticoagulación estándar y actualmente se reserva para casos refractarios. En conjunto, el diagnóstico oportuno, la anticoagulación personalizada y la atención crítica multidisciplinaria siguen siendo los principales determinantes del pronóstico en pacientes con TVC.


PALABRAS CLAVE: embolia y trombosis intracraneal, anticoagulantes, inhibidores del factor Xa, cuidados críticos, radiología intervencionista.

## INTRODUCTION

Cerebral venous thrombosis (CVT) is an uncommon subtype of cerebrovascular disease (CVD), with an estimated incidence of 1.2 to 1.6 cases per 100,000 person-years [1]. Epidemiological data remain highly variable across different regions, particularly in low- and middle-income countries, reflecting both sociodemographic factors and limitations in diagnostic resources [2]. Unlike other forms of CVD, CVT is characterized by remarkably heterogeneous clinical presentations, creating substantial challenges for clinicians. [3]

Recent studies indicate that mortality associated with CVT has declined to below 15% in most series. Functional limitations, as assessed by the modified Rankin Scale, are similarly low in many patients [4]. Nevertheless, many individuals experience chronic sequelae—such as epilepsy, headache, neurocognitive impairment, and fatigue—that, although not severely disabling, significantly affect quality of life [5,6]. These findings underscore the need for further research on long-term follow-up and secondary prevention strategies.

The improved survival and functional outcomes observed in recent years are largely attributable to earlier recognition and optimized acute management. A significant proportion of patients require intensive care admission and may develop acute complications that threaten prognosis [7]. Timely initiation of anticoagulation, combined with neurocritical care, is central to improving outcomes. More recently, additional therapeutic approaches have been proposed, including the use of direct oral anticoagulants (DOACs) and rescue measures such as surgical or endovascular interventions for patients with malignant or refractory disease courses [8]. The objective of this review was to provide an updated overview of the acute medical, surgical, and interventional management of CVT. A narrative literature review was conducted using the major databases: Scopus, WoS, Medline, and SciELO. Articles with no available access, duplicate articles, and articles without an available abstract were removed from the review. All articles in Spanish and English were included.

## MEDICAL AND NEUROCRITICAL MANAGEMENT

### Anticoagulation

Anticoagulation remains the cornerstone of therapy in cerebral venous thrombosis (CVT). Evidence

consistently supports the use of unfractionated heparin (UFH) or low-molecular-weight heparin (LMWH) in the acute phase, even in the presence of venous hemorrhage, as restoration of venous outflow reduces the risk of thrombus propagation and further bleeding [9,10]. Current guidelines recommend LMWH over UFH in patients not requiring imminent neurosurgical procedures, owing to more predictable pharmacokinetics and lower risk of heparin-induced thrombocytopenia [11,12]. The choice between unfractionated heparin and low-molecular-weight heparin is primarily determined by the need for imminent neurosurgical procedures or lumbar puncture in the setting of intracranial hypertension. Since such scenarios are relatively uncommon, most patients can be safely managed with LMWH as first-line therapy.

Transition to oral anticoagulation is indicated once the patient is stabilized. Vitamin K antagonists (VKAs) remain the most established long-term agents, though increasing data suggest that DOACs are a safe and effective alternative in selected patients [13-16]. Four clinical studies directly compared DOACs with VKAs in cerebral venous thrombosis (CVT). Table 1 summarizes the findings of these recent clinical studies directly comparing vitamin K antagonists with direct oral anticoagulants in the treatment of cerebral venous thrombosis. The RE-SPECT CVT trial in 2019 randomized 120 adults stabilized after heparin to dabigatran or warfarin for 24 weeks [17]. No recurrent venous events occurred in either arm, recanalization rates were similar, and bleeding complications were rare, suggesting comparable safety and efficacy. The ACTION-CVT study, the largest cohort to date with 845 patients, showed that DOACs had similar efficacy to warfarin in preventing recurrent thrombosis, death, and promoting recanalization, but were associated with a significantly lower risk of major bleeding [13]. The SECRET trial, a phase II feasibility randomized controlled trial (RCT), randomized ~55 patients to rivaroxaban or standard-of-care anticoagulation. Although underpowered for efficacy, it confirmed the feasibility of conducting DOAC trials in CVT and showed no excess bleeding with rivaroxaban [18]. Finally, the CHOICE-CVT trial randomized 89 Chinese patients after LMWH to dabigatran or warfarin for 180 days. Dabigatran was associated with fewer bleeding events (2.3% vs 20%) and comparable recanalization, though there was a non-significant trend toward higher recurrence compared with warfarin. [15]

**Table 1.** Clinical trials of DOACs for CVT.

| Study (year) | Design; n | Population & Timing | Intervention vs. Comparator | Outcomes reported | Headline finding |
|---|---|---|---|---|---|
| RE-SPECT CVT (2019) [17] | RCT, PROBE; n = 120 | Adults with acute CVT, stabilized after 5–15 d heparin; 24 wks treatment | Dabigatran 150 mg BID vs warfarin (INR 2–3) | Recurrent VTE/ major bleeding; recanalization | No recurrent VTE; major bleeding 1 vs 2; recanalization similar → both safe/effective |
| ACTION-CVT (2022) [13] | Multicenter retrospective; n = 845 | Adults with CVT on oral anticoagulation; median follow-up ~345 d | DOACs vs warfarin | Recurrent VTE, major bleeding, death, recanalization | Similar efficacy; DOACs had less major bleeding (aHR 0.35, p=0.02) |
| SECRET (2023) [18] | Phase II randomized feasibility; n ≈ 55 | Symptomatic CVT; randomized within ~14 d; 180 d follow-up | Rivaroxaban vs warfarin/LMWH | Composite safety; efficacy & feasibility | Feasible; small sample, no definitive efficacy; safety broadly comparable |
| CHOICE-CVT (2024) [15] | Single-center open-label RCT; n = 89 | Adults 18–80; oral AC after 10–15 d LMWH; 180 d | Dabigatran vs warfarin | Recurrent CVT/ DVT, bleeding, recanalization, papilledema | Less bleeding with dabigatran; similar recanalization; trend toward more recurrence (underpowered) |

AC: Anticoagulation; aHR: Adjusted Hazard Ratio; BID: Bis in die (twice a day); CVT: Cerebral Venous Thrombosis; DOACs: Direct Oral Anticoagulants; DVT: Deep Vein Thrombosis; INR: International Normalized Ratio; LMWH: Low Molecular Weight Heparin; PROBE: Prospective, Randomized, Open-label, Blinded End-point; RCT: Randomized Controlled Trial; VTE: Venous Thromboembolism; wks: weeks; d: days.

Collectively, these studies indicate that DOACs achieve similar efficacy to VKAs with a more favorable bleeding profile. While current evidence supports their use as a reasonable alternative in adults with CVT, limitations include small RCT sample sizes and the observational design of the largest study. VKAs remain the preferred option in specific settings such as antiphospholipid syndrome, but in most other patients, DOACs represent a practical and increasingly supported choice for long-term secondary prevention.

Duration of anticoagulation depends on etiology. For CVT with a reversible provoking factor, treatment for 3–6 months is recommended; for unprovoked cases, 6–12 months; and for recurrent CVT, severe thrombophilia, or persistent risk factors, indefinite anticoagulation should be considered. [12,19]

**General medical and supportive care**

Supportive measures are crucial. Adequate hydration and analgesia for headaches are standard, while opioids should be avoided when possible. Corticosteroids are not recommended, as they have not shown benefit in reducing mortality or disability [12]. Similarly, acetazolamide is not routinely indicated for reducing intracranial pressure in CVT; however, it may be considered in selected patients with papilledema or visual symptoms, according to recent consensus guidelines [19]. In refractory cases with vision-threatening intracranial hypertension, surgical options such as shunting or optic nerve sheath fenestration can be considered. [19]

**Neurocritical Care Unit (NICU) management**

Patients with severe CVT require admission to the NICU. Neurological monitoring should focus on mental status, focal deficits, and seizure activity, while systemic monitoring includes hemodynamics, electrolytes, and renal/hepatic function to guide anticoagulation.

Severe cerebral venous thrombosis (CVT) is characterized by clinical or radiological features indicating high risk of deterioration or death, and such patients require admission to a dedicated NICU. Although definitions vary, the following criteria are commonly considered markers of severity:

- Altered level of consciousness, particularly when associated with deep venous system thrombosis.
- Status epilepticus or recurrent uncontrolled seizures.
- Intracranial hypertension with impending or established signs of herniation (e.g., rapid neurological decline, malignant cerebral edema, or severe papilledema).
- Extensive focal neurological deficits or rapid progression of deficits due to large venous infarcts or bilateral involvement.
- Radiological evidence of deep cerebral venous thrombosis, extensive sinus occlusion, or large hemorrhagic lesions.
- Clinical instability requiring airway protection, hemodynamic support, or close metabolic monitoring in the context of anticoagulation.

Seizures occur in 30–40% of patients. Antiepileptic drugs (AEDs) are indicated if seizures occur, but routine prophylaxis is not recommended. Guidelines advise consideration of AEDs in patients with early seizures and large supratentorial lesions, where secondary prevention of early recurrence may be beneficial [11]. Clinicians must also be mindful of interactions between AEDs and anticoagulants.

Intracranial hypertension (ICP elevation) is a key determinant of prognosis. General measures include head elevation, optimization of sedation and analgesia, and normoventilation. Osmotherapy with mannitol or hypertonic saline is recommended as a temporizing strategy. Corticosteroids and acetazolamide have not shown clear benefit in this context [12]. In refractory cases, escalation to mechanical ventilation and barbiturate coma may be attempted, though evidence is limited.

Decompressive craniectomy is a lifesaving measure for patients with malignant cerebral edema or impending herniation and is supported by both European and North American guidelines.

**Management in the presence of hemorrhage**

Unlike arterial stroke, the presence of venous hemorrhage is not a contraindication to anticoagulation. Studies and guidelines consistently support continuing anticoagulation in CVT with hemorrhagic infarction, as the underlying mechanism is venous congestion rather than primary arterial rupture [8,11,16,19]. Anticoagulation may need to be individualized in massive hemorrhage or after neurosurgical intervention, but in most cases, LMWH should be continued with careful monitoring.

### Special considerations

Pregnancy and the postpartum period represent frequent settings for CVT. LMWH is the treatment of choice during pregnancy and for at least six weeks postpartum. VKAs and DOACs are contraindicated during pregnancy. Estrogen-containing contraceptives should be avoided after CVT, and risk-factor counseling—including smoking cessation and discontinuation of hormonal therapies—is essential. [8, 11]

### Prognosis and long-term care

With timely anticoagulation and appropriate ICU care, most patients achieve functional independence. Nonetheless, mortality or dependency remains around 10–15% in contemporary cohorts [5]. Recanalization occurs in the majority within the first year, though a subset of patients may develop residual venous occlusion. Long-term management includes counseling on recurrence risk, careful consideration of anticoagulation duration, and lifestyle modification. Table 2 summarizes the main recommendations from selected scientific societies regarding key aspects of the medical management of cerebral venous thrombosis.

**Table 2.** Summary of recommendations of selected clinical guidelines.

| Domain | ESO 2017 [8] | AHA/ASA 2024 [11] | German S2k 2024 [16] | Thrombosis Canada 2023 [19] |
|---|---|---|---|---|
| Acute anticoagulation (heparin choice) | Recommends parenteral anticoagulation; prefers LMWH over UFH. | LMWH preferred in the acute phase (more predictable, less HIT). | LMWH preferred in acute phase. | Initiate UFH or LMWH; LMWH favored if no imminent neurosurgery/ intervention. |
| Anticoagulation in presence of intracranial hemorrhage | Recommends anticoagulation in acute CVT (does not explicitly stratify hemorrhage). | Venous hemorrhage is not a contraindication to anticoagulation. | — | Anticoagulation is recommended even with intracranial blood (except in massive/rapid expansion or surgical emergency). |
| Oral anticoagulation (VKA vs DOACs) | No DOACs (2017); transition to VKA. | DOACs reasonable in selected patients; VKA established option. | DOACs preferred over VKA for 3–12 months after acute phase. | VKA standard; DOACs acceptable in selected adults (based on RESPECT-CVT, ACTION-CVT). Avoid DOACs in APS. |
| Duration of anticoagulation | No firm recommendation (evidence limited). | 3–12 months depending on provoking factor; indefinite in recurrence/chronic risk. | 3–12 months (individualized). | Provoked: 3–6 m; unprovoked: ≥6–12 m; extended if recurrence/ persistent risk. |
| Seizures | Start AEDs if early seizure plus supratentorial lesion; no routine prophylaxis. | — (not explicitly addressed in summary). | — | Treat seizures if they occur; no evidence for prophylactic AEDs in seizure-free patients. |
| Intracranial hypertension – medical therapy | Do not recommend corticosteroids or acetazolamide for mortality/disability. | — | — | General measures (head elevation, ophthalmology for papilledema); acetazolamide may be considered with visual symptoms, case by case. |

AEDs: Anti-Epileptic Drugs; AHA/ASA: American Heart Association / American Stroke Association; APS: Antiphospholipid Syndrome; CVT: Cerebral Venous Thrombosis; DOACs: Direct Oral Anticoagulants; ESO: European Stroke Organisation; HIT: Heparin-Induced Thrombocytopenia; LMWH: Low Molecular Weight Heparin; m: months; UFH: Unfractionated Heparin; VKA: Vitamin K Antagonist.

**Table 2.** (*Continuation*).

| Domain | ESO 2017 [8] | AHA/ASA 2024 [11] | German S2k 2024 [16] | Thrombosis Canada 2023 [19] |
|---|---|---|---|---|
| Intracranial hypertension – surgery | Decompressive craniectomy recommended to prevent herniation (life-saving). | Offer craniectomy for malignant edema/ impending herniation. | Decompressive surgery indicated if parenchymal lesion with herniation risk. | Neurosurgery consult; hemicraniectomy in patients with mass effect and impending herniation. |
| Pregnancy/ contraception | Avoid estrogen-containing contraceptives; consider LMWH prophylaxis during pregnancy/ postpartum. | — | LMWH prophylaxis during pregnancy and ≥6 weeks postpartum; avoid combined hormonal contraceptives. | LMWH prophylaxis ante- and postpartum reasonable in women with prior CVT. |

AEDs: Anti-Epileptic Drugs; AHA/ASA: American Heart Association / American Stroke Association; APS: Antiphospholipid Syndrome; CVT: Cerebral Venous Thrombosis; DOACs: Direct Oral Anticoagulants; ESO: European Stroke Organisation; HIT: Heparin-Induced Thrombocytopenia; LMWH: Low Molecular Weight Heparin; m: months; UFH: Unfractionated Heparin; VKA: Vitamin K Antagonist.

## SURGICAL MANAGEMENT

Although cerebral venous thrombosis (CVT) carries a favorable prognosis in more than two-thirds of patients, a subset presents with severe manifestations from the outset. The primary mechanisms underlying the pathophysiology of CVT include reduced cerebrospinal fluid absorption secondary to venous outflow obstruction, leading to increased intracranial pressure [20]. Impaired venous drainage also results in elevated venous and capillary pressures, disruption of the blood–brain barrier, and ultimately the development of vasogenic edema, venous infarction, and hemorrhagic lesions. These mechanisms align with the study by Canhão et al. [21], which identified transtentorial herniation from mass effect due to parenchymal lesions as the leading cause of early mortality, occurring in approximately 4% of cases.

To rapidly lower intracranial pressure in severe presentations, including those with transtentorial herniation, several groups have explored decompressive craniectomy. This approach was initially motivated by evidence of benefit in malignant middle cerebral artery infarction, the similarity of the underlying pathophysiology, and the observation of better recovery potential in CVT compared with arterial stroke. Most groups advocate for a large craniectomy, like that used in malignant arterial infarction (≥15 cm, extending to the temporal base), with wide duraplasty to maximize decompression, bilaterally if required. In general, evacuation of infarcted tissue (internal decompression) is not recommended.

The first reports of this procedure date back to the late 1990s. Given the rarity of the disease, no randomized trials exist to date, and most evidence on decompressive craniectomy in CVT derives from case series, retrospective studies, and prospective registries. In 2009, Coutinho et al. [22] reported three institutional cases and conducted a literature review, identifying six publications with a total of 13 patients. Eleven had favorable outcomes (modified Rankin Scale [mRS] ≤3). The authors noted that their management strategy had shifted in favor of surgery after observing little benefit from endovascular thrombolysis in this subgroup.

Similarly, in 2010, Théaudin et al. [23] described 12 patients with "malignant" CVT, defined as supratentorial lesions due to superficial venous thrombosis, with or without sinus involvement, and clinical or radiological signs of transtentorial herniation either at presentation or despite ongoing heparin therapy. Eight patients underwent decompressive craniectomy, including five with fixed pupils; seven survived, and six achieved excellent functional outcomes (mRS 0–1) after a median follow-up of two years. By contrast, all four patients who did not undergo surgery died within five days.

As part of the ISCVT initiative, Ferro et al. [24] published in 2011 a multicenter prospective registry and systematic review. They reported 69 patients treated with decompressive craniectomy (65%), hematoma evacuation (10%), or both (25%). Patients ranged from 15 to 68 years of age, and nearly 70% had a Glasgow Coma Scale (GCS) score <9 before surgery. Most had unilateral lesions, 90% of which were hemorrhagic. At one-year follow-up, 56% achieved mRS 0–2, while overall mortality was 16%. Surgery was typically performed within 48 hours. Comatose patients and those with bilateral lesions were more likely to have poor outcomes; however, complete recovery occurred in roughly one-third of cases.

A more recent systematic review by Misra et al. [25] analyzed 51 studies including 483 patients undergoing decompressive surgery. Most were women (67%), 27% presented with GCS <9, and 83% had midline shift. A meta-analysis of four studies with control groups demonstrated that surgical patients had a significantly lower probability of unfavorable outcomes (odds ratio [OR] 0.03; 95% CI 0–0.22). Surgery within 48 hours was strongly associated with reduced mortality (OR 0.26; 95% CI 0.10–0.69).

In Latin America, Arauz et al. [26] conducted a retrospective analysis of 471 patients from two Mexican centers, of whom 26 required decompressive craniectomy. The average age was 35 years, and 76% were women. Most had GCS <9 (80%), parenchymal lesions >6 cm (96.2%), and bilateral hemorrhages (65.4%). In-hospital mortality was 42.3%, higher than that reported in other cohorts; eight of the 11 deaths were directly attributable to CVT. At long-term follow-up (mean 52 months), nine patients achieved favorable outcomes (mRS 0–2), five had mRS 3–5, and no recurrences were reported.

During the COVID-19 pandemic, the first cases of CVT associated with viral vector vaccines were described. These patients typically exhibited thrombocytopenia, low fibrinogen, and elevated D-dimer levels, a syndrome termed vaccine-induced immune thrombotic thrombocytopenia (VITT) [27]. In 2023, Krzywicka et al. [28] published an international registry of 128 VITT-related CVT cases from 84 centers in 20 countries. Thirty-four underwent surgery; the mean age was 45 years, and 76% were female. Symptoms developed around eight days post-vaccination. Compared with non-surgical patients, surgical cases had more focal deficits, higher D-dimer, lower fibrinogen, and more hemorrhagic lesions. In-hospital mortality was 65%, markedly higher than in non-VITT CVT. The authors attributed this to greater severity at baseline and higher rates of postoperative intracerebral hemorrhage. At discharge, 94% of surgical patients were dependent or deceased, though among survivors, 80% ultimately regained functional independence on follow-up. These findings suggest that early decompressive craniectomy may still be considered in VITT-associated CVT.

In 2024, the DECOMPRESS2 study reported on 110 patients from 15 centers across Europe, Asia, and the Americas [29]. The cohort was predominantly young women with severe presentations; 31% were comatose, and 18% had pupillary abnormalities. Nearly 90% had parenchymal lesions, with an average size of 6.5 cm. More than two-thirds deteriorated neurologically before surgery, which was performed on average one day after admission. Decompressive craniectomy was performed in 98% of cases, with hematoma evacuation in nearly one-third. At discharge, 44% had poor outcomes (mRS 5–6), and mortality was 24%. Only 8% achieved mRS 0–2 at discharge, but functional outcomes improved over time: at one year, 16% achieved mRS 0–1 and 34% reached mRS 0–2. Mortality in one year was 34%. Prognostic factors for poor outcome included coma (GCS <9) and pupillary abnormalities. Older age (>37 years), concomitant hematoma evacuation, and left-sided hemicraniectomy were associated with worse recovery. Importantly, decompressive surgery was rated as valuable by 80% of patients and caregivers, with satisfaction improving during follow-up.

Currently, decompressive craniectomy is recommended by the 2017 ESO guideline (low-quality evidence; strong recommendation), the 2024 AHA Scientific Statement, and the Thrombosis Canada Clinical Guide. All three documents concur that decompressive craniectomy should be offered as a life-saving intervention in patients with acute CVT, parenchymal lesions, and imminent herniation. In Figure 1, we present a flowchart algorithm for decompressive craniectomy in patients with CVT.

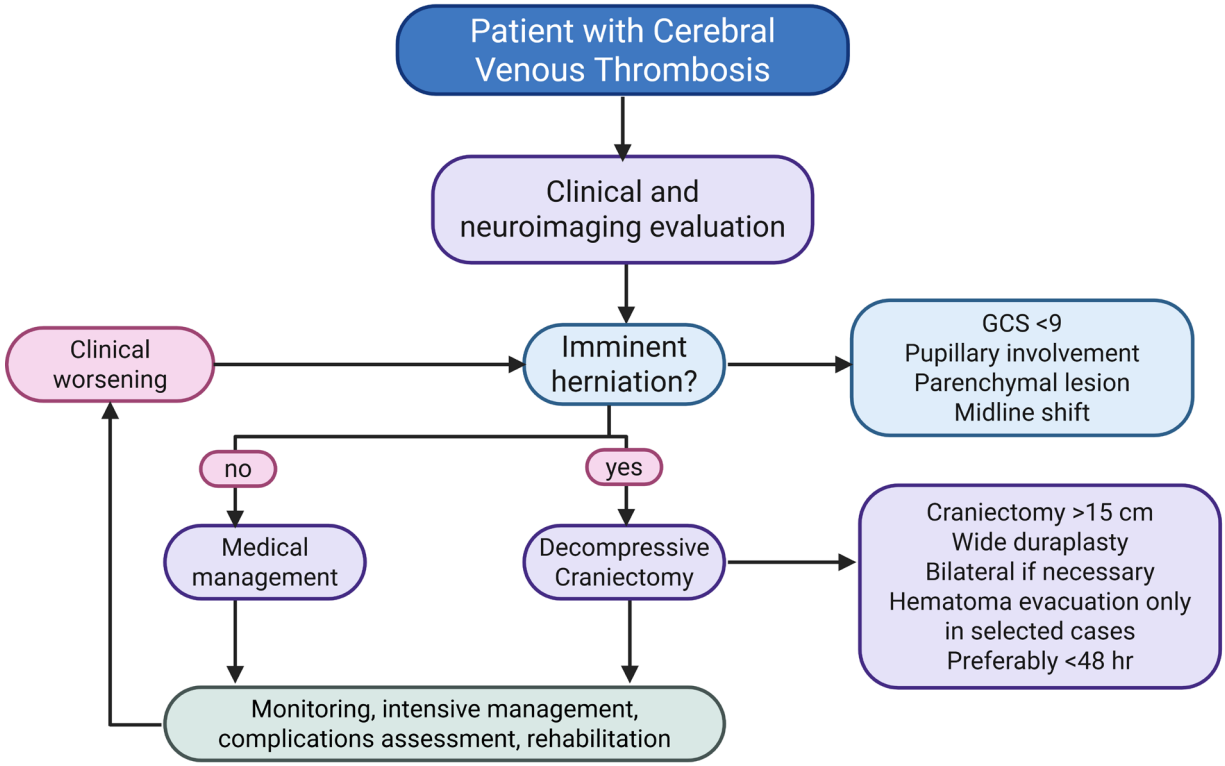



**Figure 1.** Algorithm for Decompressive Craniectomy in Patients with Cerebral Venous Thrombosis.

## ENDOVASCULAR MANAGEMENT

Cerebral venous thrombosis (CVT) generally carries a favorable prognosis when promptly diagnosed and treated. However, some patients experience clinical deterioration, often due to a high thrombus burden or the limited ability of heparin to dissolve the clot, and up to 13% may die or remain severely disabled despite appropriate medical therapy [14]. Endovascular therapy has therefore been proposed as a strategy to achieve rapid recanalization and improve outcomes in selected patients.

Several interventional approaches have been investigated, beginning with direct local intrasinus thrombolysis and later including mechanical techniques such as rheolytic thrombectomy, direct aspiration thrombectomy, balloon-assisted thrombectomy or angioplasty, and stent retriever thrombectomy. At present, no evidence supports the superiority of one technique over another. [30]

The only randomized controlled trial comparing endovascular therapy with standard medical management is the TO-ACT trial (Thrombolysis or Anticoagulation for Cerebral Venous Thrombosis) [31]. This multicenter, endpoint-blinded study enrolled 67 patients (33 assigned to endovascular therapy and 34 to standard care). Interventions included mechanical thrombectomy, local thrombolysis with alteplase or urokinase, or both. Patients were eligible if they were considered at high risk of poor outcomes, defined by altered mental status, coma (GCS < 9), intracranial hemorrhage, or deep venous thrombosis. Exclusion criteria included diagnosis more than 10 days earlier, pregnancy, thrombocytopenia, or signs of transtentorial herniation. No significant difference in functional outcomes was observed (mRS 0–1 at 12 months: 67% vs. 68%; RR 0.99, 95% CI 0.71–1.38). The study was limited by its small sample size, the inclusion of patients with severe baseline profiles, and non-standardized use of devices and techniques. No subgroups were identified that clearly benefited from intervention.

A 2022 meta-analysis likewise found no benefit of endovascular therapy and even suggested higher mortality [32]. Because the analysis was based on retrospective individual patient data, treatment decisions were highly heterogeneous, reducing generalizability. Other retrospective studies similarly failed to demonstrate benefit [32–34], though questions remain regarding which patients might be most appropriate for intervention and the optimal timing of therapy.

Current clinical guidelines recommend considering endovascular therapy for patients who deteriorate despite anticoagulation or who have contraindications

to standard medical treatment [11]. Typical scenarios include coma, depressed consciousness, or intracranial hypertension refractory to anticoagulation.

More recently, a 2024 meta-analysis by Ghozy et al. [35] synthesized 38 studies including 682 patients. Functional independence (mRS 0–2) was achieved in 82.6%, new or expanded intracranial hemorrhage occurred in 5.1%, and mortality was 6.7%. The study did not analyze outcomes by specific technique but concluded that endovascular therapy appears safe and is associated with high rates of favorable outcomes.

Taken together, current evidence does not support the routine use of endovascular therapy in CVT. In patients with clinical deterioration or those meeting TO-ACT trial inclusion criteria, it may represent a reasonable adjunct to medical management. Endovascular therapy appears safe and may improve functional outcomes in carefully selected cases, but optimal patient selection and preferred techniques remain to be defined.

## CONCLUSIONS

Cerebral venous thrombosis remains a rare but treatable form of stroke in adults. Early and accurate diagnosis with CT or MR venography is crucial, given its protean clinical presentations. Anticoagulation, preferably with low-molecular-weight heparin acutely, followed by oral therapy, is the cornerstone of treatment and should not be withheld in the presence of hemorrhagic lesions. Emerging evidence supports direct oral anticoagulants as an alternative to vitamin K antagonists for long-term secondary prevention, though caution is required in selected subgroups such as antiphospholipid syndrome. Neurocritical care is critical in severe cases, addressing seizures, intracranial hypertension, and systemic instability. At the same time, decompressive craniectomy provides a life-saving intervention in patients with malignant cerebral edema or impending herniation. Endovascular therapy has not shown benefit in randomized trials but may be considered in patients who deteriorate despite adequate anticoagulation. Together, these strategies underscore the importance of timely recognition, tailored anticoagulation, and multidisciplinary management to optimize outcomes in CVT.

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