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ARTICLE TITLE	PRE-THERAPY MULTIMODAL NEUROIMAGING AS A TOOL FOR PERSONALIZED TREATMENT SELECTION IN CEREBRAL VENOUS THROMBOSIS: A RADIOLOGICAL PATTERN-BASED STRATIFICATION PERSPECTIVE
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PRE-THERAPY MULTIMODAL NEUROIMAGING AS A TOOL FOR PERSONALIZED TREATMENT SELECTION IN CEREBRAL VENOUS THROMBOSIS: A RADIOLOGICAL PATTERN-BASED STRATIFICATION PERSPECTIVE

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ABSTRACT

Background. Cerebral venous thrombosis (CVT) is a highly heterogeneous form of stroke that requires robust diagnostic workflows and precision therapeutic strategies. Pre-therapy multimodal neuroimaging—encompassing advanced CT venography (CTV), susceptibility-weighted imaging (SWI), perfusion mapping (CTP/MRP), and high-resolution black-blood MRI—enables a paradigm shift by functioning as a sophisticated marker to map specific radiological formes to targeted antithrombotic, neuroprotective, and endovascular therapies.

Aim. To establish a pattern-based stratification model driven by quantitative pre-therapy imaging variables to individualize patient selection, optimize direct oral anticoagulant (DOAC) management, and safely guide anticoagulation de-escalation timelines.

Material and Methods. This comprehensive review analyzes thrombus evolution, spatial occlusion architecture, and the complex perfusion dynamics of venous congestion, with a specific focus on cerebral blood volume/cerebral blood flow (CBV/CBF) dissociation. These quantitative imaging metrics were synthesized to map directly onto individual patient selection paradigms across major clinical trials, including RE-SPECT CVT, SECRET, TO-ACT, and the 24-month EXCOA-CVT trial results from ESOC 2026.

Results. Multimodal imaging patterns effectively demarcate venous congestion severity and guide patient selection for targeted therapies. This pattern-based stratification enables clinical scientists to safely navigate anticoagulation de-escalation windows (3 to 6 months) and optimize DOAC selection tailored to specific pathophysiological profiles.

Conclusions. Pre-therapy imaging-guided stratification provides a direct framework to optimize recanalization efficacy while minimizing iatrogenic hemorrhagic risks in patients with cerebral venous thrombosis.

KEYWORDS

Cerebral Venous Thrombosis, Pre-Therapy Neuroimaging, Radiological Patterns, Perfusion Mapping, Targeted Therapy

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Introduction:

Cerebral venous thrombosis (CVT), which accounts for approximately 0.5-1% of all acute cerebrovascular disorders, represents one of the most etiopathogenetically and clinically heterogeneous nosologies [1, 2]. The disease primarily affects young, working-age individuals and presents with a variable clinical presentation—ranging from isolated, benign cephalalgia to fulminant coma accompanied by multiple bilateral hemorrhagic complications [3, 4]. The first randomized controlled trials (RCTs) evaluating the safety of anticoagulants for CVT (Einhäupl et al., 1991; de Bruijn & Stam, 1999) demonstrated a reduction in the frequency of adverse outcomes with the use of heparin and its derivatives. However, the neuroimaging methods used at the time of those studies digital subtraction angiography, computed tomography, and magnetic resonance imaging with MR angiography allowed researchers to document only the fact that a large vessel was occluded, without providing detailed characteristics of the thrombus itself. Further development of neuroimaging techniques has enabled the medical community to shift its understanding of cerebral venous thrombosis: while it was previously recognized only as an established fact, modern methods now allow for the differentiation of various pathogenetic variants each with its own distinct neuroradiological patterns depending on the precise location and severity of the occlusion. As knowledge about the disease itself has accumulated, the question of revising treatment approaches for CVT has naturally arisen [5, 6]. Until recently, the management of CVT was based on intuitive approaches: systemic heparinization during the acute phase and fixed-duration courses (ranging from 6 to 12 months) of vitamin K antagonists for secondary prevention [5, 7, 9]. Long-term courses of heparin therapy and the use of vitamin K antagonists are risk factors for the development of various complications (bleeding, heparin-induced thrombocytopenia, teratogenic effects, and liver or kidney damage). A more nuanced approach to the treatment of CVT-categorizing the condition into

distinct types based on specific neuroradiological patterns has enabled a revision of treatment protocols. For certain types of CVT, this allows for a shorter duration of anticoagulant therapy, which should, in turn, reduce the incidence of treatment-related complications and improve clinical outcomes.

Multimodal imaging (Pre-therapy Functional and Structural Neuroimaging) is currently viewed not as a tool for detecting the occurrence of an occlusion, but rather as a source of precise markers [3, 18, 19]. The diversity of radiological patterns ranging from those reflecting the evolution of a thrombus and its localization within the affected sinuses to the perfusion characteristics of venous stasis allows for the stratification of patients into distinct groups. This facilitates the selection of specific oral anticoagulants and their respective dosing regimens, while also helping to shorten treatment duration and narrow the indications for endovascular intervention.

Materials and methods:

Search Strategy and Information Sources

To summarize current evidence on imaging-guided stratification and personalized management of cerebral venous thrombosis (CVT), a systematic and structured narrative literature search was conducted across MEDLINE/PubMed, Scopus, Web of Science, and Embase databases. The search encompassed landmark foundational studies, pivotal randomized controlled trials (RCTs), major clinical practice guidelines, and recent high-impact publications up to 2026.

The search matrix combined Medical Subject Headings (MeSH) and free-text terms including: *"cerebral venous thrombosis"*, *"dural sinus thrombosis"*, *"neuroimaging"*, *"CT venography"*, *"susceptibility-weighted imaging"*, *"perfusion imaging"*, *"anticoagulation"*, *"direct oral anticoagulants"*, *"endovascular therapy"*, and *"decompressive craniectomy"*.

Eligibility Criteria and Study Selection

Studies were selected based on their relevance to the diagnostic evaluation, pathophysiological characterization, or therapeutic management of CVT. The inclusion criteria prioritized:

1. **Clinical Practice Guidelines & Consensus Statements:** Statements from major neurological societies (e.g., AHA/ASA, Canadian Stroke Best Practice Recommendations, European Stroke Organization) [4, 5, 9].
2. **Randomized Controlled Trials & Feasibility Studies:** RCTs evaluating acute/secondary anticoagulation regimens, direct oral anticoagulants (DOACs), endovascular interventions, and duration of therapy (e.g., RE-SPECT CVT, SECRET, TO-ACT, EXCOA-CVT) [8, 10, 11, 14, 16, 17].
3. **Observational & Multi-Center Cohorts:** Large-scale prospective/retrospective registry studies assessing long-term prognosis and surgical decompression outcomes (e.g., ISCVT, DECOMPRESS2) [2, 13].
4. **Radiological & Pathophysiological Reviews:** Literature detailing advanced neuroimaging modalities, diagnostic mimics, thrombus evolution, and venous perfusion dynamics [1, 3, 18, 19].
5. **Updated Narrative & Systematic Reviews:** Comprehensive updates on emerging diagnostic paradigms and therapeutic advances [6, 12, 15, 20].

Articles non-pertinent to human cerebrovascular disease, non-English publications without accessible translation, and small single-case reports lacking novel radiological or therapeutic insights were excluded (**Figure 1**).

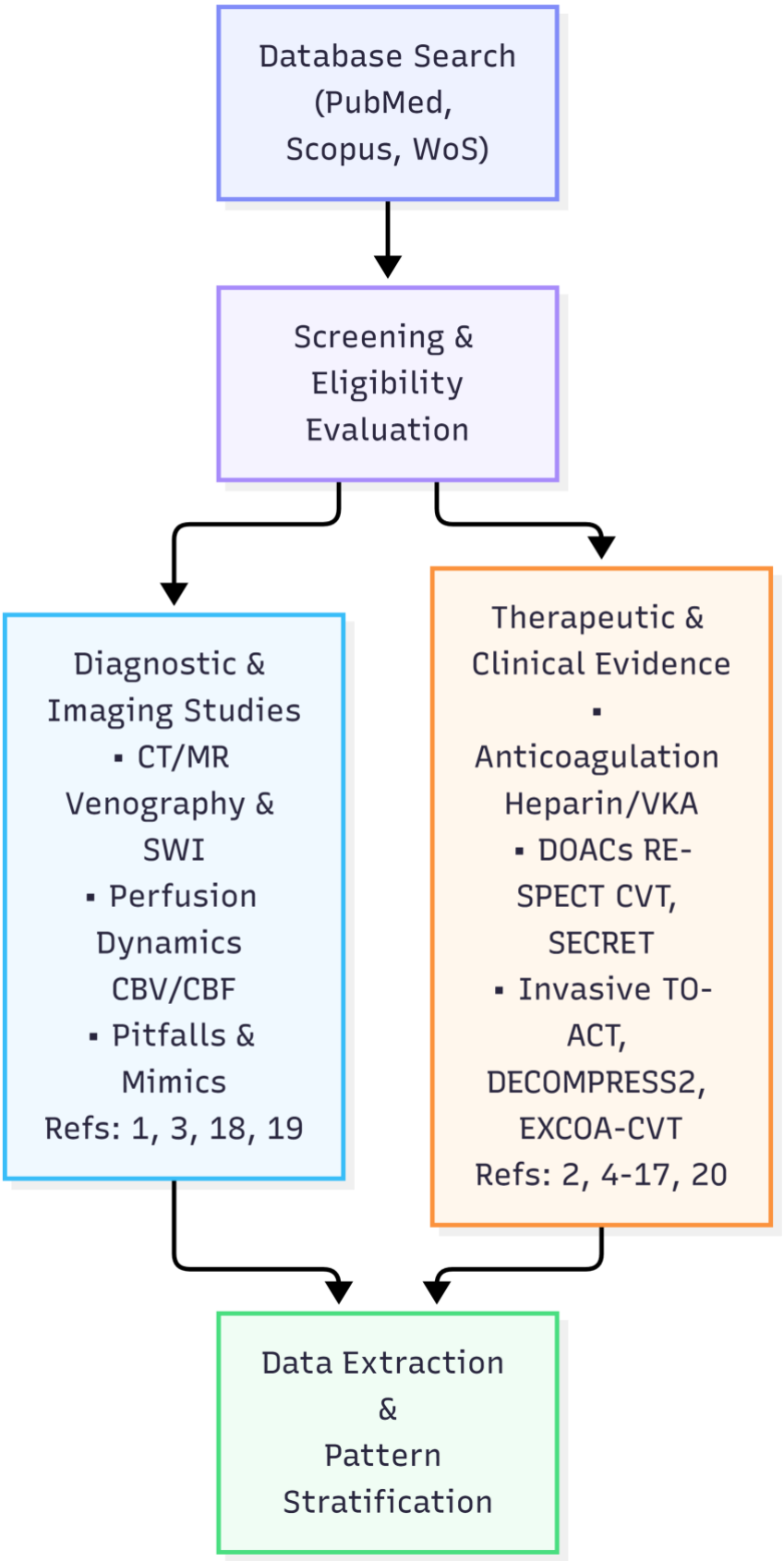


Fig. 1. *Methodological workflow illustrating database retrieval, evidence filtering, and pattern-based stratification for cerebral venous thrombosis*

Data Extraction and Categorization

Extracted data were categorized into two primary analytical domains:

- **Radiological and Pathophysiological Analysis:** Data on neuroimaging techniques—including CT venography (CTV), susceptibility-weighted imaging (SWI), magnetic resonance venography (MRV), perfusion mapping (CTP/MRP), and high-resolution vessel wall/black-blood MRI—were synthesized to characterize thrombus age, spatial sinus occlusion patterns, collateral venous drainage, and microvascular congestion dynamics [1, 3, 18, 19].
- **Therapeutic and Prognostic Outcomes:** Clinical efficacy, recanalization rates, recurrent thrombotic events, and hemorrhagic risks were extracted across treatment modalities:
- *Standard Anticoagulation:* Parenteral heparin derivatives and Vitamin K Antagonists (VKAs) [2, 7, 8].
- *DOAC Evaluation:* Dabigatran and Rivaroxaban trial data [10, 11].
- *Extended vs. Shortened Anticoagulation:* Trial outcomes evaluating duration of therapy (e.g., EXCOA-CVT 24-month outcomes) [16, 17].
- *Invasive & Rescue Interventions:* Endovascular thrombolysis/thrombectomy and decompressive surgery [13, 14].
- *Guideline Recommendations & Management Updates:* Contemporary management frameworks [4, 5, 6, 9, 12, 15, 20].

Summarizing and Pattern-Based Stratification

A qualitative synthesis was performed to map specific neuroradiological patterns directly onto targeted treatment workflows. By correlating pre-therapy imaging markers (such as clot burden, venous infarction risk, and CBV/CBF perfusion mismatch) with clinical trial selection criteria and guideline recommendations, a pattern-based stratification model was formulated to optimize DOAC selection, safely define anticoagulation de-escalation windows (3 to 6 months), and refine patient selection for invasive interventions.

Results:

Thrombus Evolution: MRI Techniques

The physicochemical evolution of a venous thrombus determines its radiological appearance on MRI, which is directly linked to the stages of hemoglobin degradation and changes in the magnetic properties of iron atoms [1, 18, 19]. Recognizing these patterns before starting treatment is critically important for predicting the efficacy of thrombolysis and the rate of spontaneous recanalization.

Acute stage: Deoxyhemoglobin Phase

During the acute phase (days 1-5), a thrombus consists primarily of intracellular deoxyhemoglobin, which possesses strong paramagnetic properties. On standard T1- and T2-weighted images, the signal from a thrombus remains isointense or moderately hypointense, perfectly mimicking the normal "flow void" phenomenon the loss of signal resulting from rapid blood flow [18]. This is the primary diagnostic pitfall that leads to false-negative conclusions.

To overcome this barrier, scanning in SWI (Susceptibility-Weighted Imaging) mode is used. Due to its high sensitivity to magnetic field inhomogeneities, SWI visualizes a thrombus as an area of marked signal loss (which does not correspond to the actual size of the thrombus) a phenomenon known as the "blooming effect" [3, 18, 19]. The extent and intensity of the "blooming" effect on SWI serve as a direct marker of thrombus density: fragmented, "fresh" thrombi-characterized by a moderate reduction in MR signal intensity show the greatest sensitivity to systemic anticoagulant therapy with heparins; conversely, extensive, dense conglomerates typically presenting with profound, complete signal loss and a significant expansion of the affected area beyond the anatomical vessel lumen are associated with a high risk of therapeutic refractoriness.

Subacute stage: Methemoglobin Phase

The transition to the subacute phase (days 6-15) is characterized by the transformation of deoxyhemoglobin into methemoglobin, which is accompanied by the appearance of a bright, hyperintense signal on both T1- and T2-weighted images (**Figure 2**) [1, 18]. During this period, three-dimensional "black-blood" sequences (3D black-blood MRI, such as T1-SPACE or CUBE) take on key importance [3, 19]. By completely suppressing the signal from flowing blood, "black-blood" sequences allow the radiologist to

evaluate in detail the internal structure of a thrombus, mural filling defects, and reactive changes in the vessel wall (venous phlebitis).

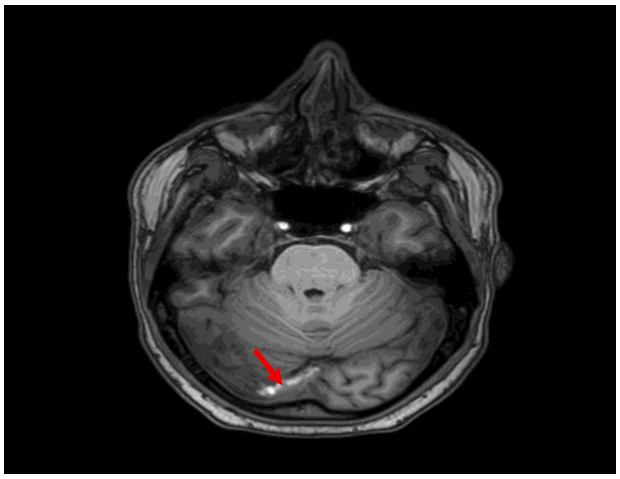


Fig. 2. *T1-weighted MRI demonstrating a hyperintense signal within the right transverse sinus (red arrow), characteristic of methemoglobin formation during the subacute phase of dural venous sinus thrombosis.*

Spatial Anatomy of Venous Occlusions and Targeted Interventions

The localization of the thrombotic process determines the clinical scenario and necessitates a differentiated therapeutic approach. Based on a multimodal analysis, three main spatial patterns have been identified:

1. Isolated thrombosis of the lateral or superior sagittal sinus (**Figures 3 and 4**). This pattern is characterized by the preservation of deep outflow pathways. In the absence of signs of parenchymal injury, this radiological phenotype dictates a conservative management approach, including early oral anticoagulation [5, 6, 10, 20].



Fig. 3. *Coronal contrast-enhanced CT scan demonstrating the classic "empty delta" sign within the superior sagittal sinus (red arrow), indicative of dural venous sinus thrombosis.*

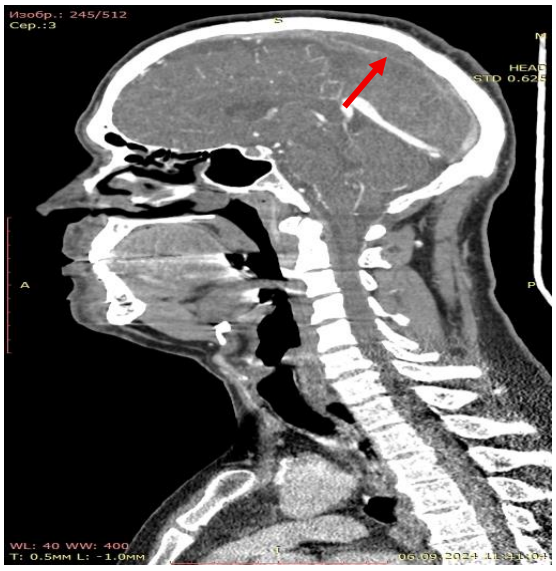


Fig. 4. *Sagittal contrast-enhanced CT scan revealing extensive thrombotic masses within the superior sagittal sinus (red arrow).*

2. Septic/infectious pattern of thrombosis (most commonly involving the cavernous or sigmoid sinuses). Radiologically, it manifests as concurrent thickening of the dura mater, signs of mastoiditis/sinusitis, and filling defects with peripheral contrast enhancement. This pattern requires the immediate initiation of etiotropic antibacterial therapy, concurrently with anticoagulants [4, 5].

3. Thrombosis of the deep venous system (the vein of Galen (**Figure 5**), straight sinus, and internal cerebral veins). This is the most prognostically unfavorable spatial pattern. It leads to bilateral involvement of the thalami and basal ganglia, rapid edema of the brainstem, and a depression of consciousness progressing to coma. This specific pattern is considered a key indicator for enhanced monitoring and potential selection for endovascular interventions [6, 14, 18].

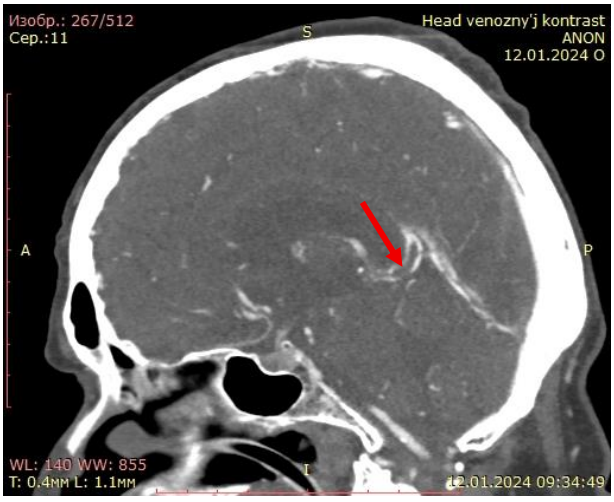


Fig. 5. *Sagittal non-contrast CT scan demonstrating acute thrombotic masses within the vein of Galen (red arrow).*

We also consider it necessary to highlight another spatial pattern: thrombosis of the cerebral veins (**Figure 6**). The neuroradiological pattern of cerebral venous thrombosis known as the "cord sign" - manifests as hyperdensity on a non-contrast CT scan, caused by the presence of thrombotic material; clinically, the patient's presentation is dominated by focal neurological symptoms rather than generalized cerebral symptoms. To confirm the presence of the "cord sign," it is recommended to supplement the non-contrast CT scan with a contrast-enhanced study, analyzing both the arterial and parenchymal phases of the post-contrast imaging.

Figure 6 presents a scan of the patient's head examination with a "cord sign" identified on a previous non-contrast study (**Figure 7**). No contrast enhancement of the vessel of interest is observed, confirming the neuroradiological pattern described above [18, 19].

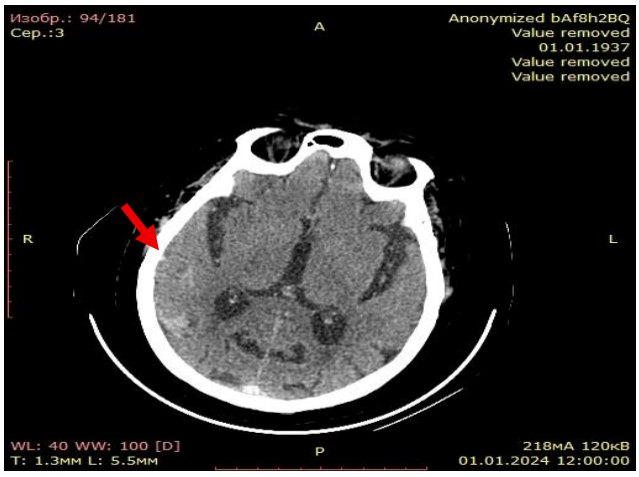


Fig. 6. *Non-contrast axial CT scan demonstrating the "cord sign" (red arrow)—a hyperdense linear structure representing acute thrombosis of a cortical vein against the background of ischemic changes.*

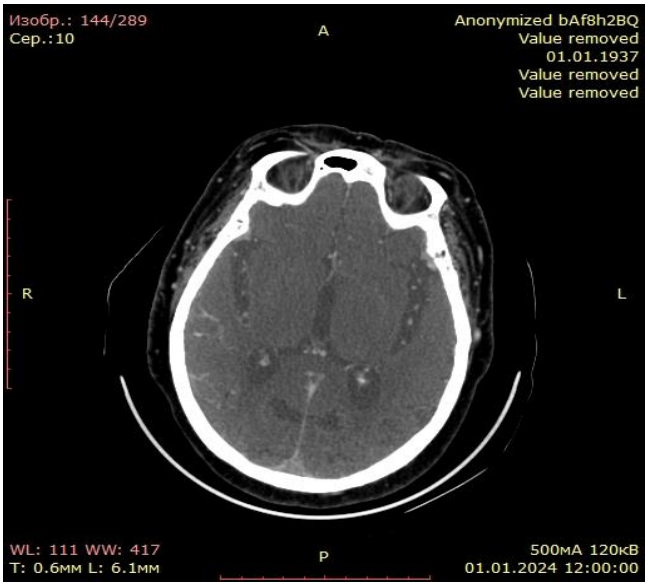


Fig. 7. *Axial contrast-enhanced CT scan (parenchymal phase) of the same patient showing no contrast opacification of the affected vessel (red arrow), confirming persistent cortical vein occlusion.*

Discussion:

Perfusion Paradoxes of Venous Congestion: CBV/CBF Dissociation as a Marker of Viability.

Perfusion mapping (CT perfusion or dynamic contrast-enhanced MR perfusion) provides unique functional markers that are fundamentally different from those seen in arterial stroke. In cases of arterial occlusion, a decrease in cerebral blood volume (CBV) correlates strongly with the formation of an irreversible necrotic infarct core [6, 18, 19]. In cases of CVT, a unique hemodynamic paradox is observed a dissociation between CBV and CBF parameters:

- A critical prolongation of blood transit time (a sharp increase in MTT and Tmax parameters to > 6-10 seconds), reflecting pronounced venous stasis and congestion.
- Preservation or compensatory increase in local cerebral blood volume (normal or elevated CBV) due to massive engorgement of the venular bed and the opening of collaterals.

This perfusion pattern indicates that the brain tissue is in a state of severe venous congestion and vasogenic edema, but it retains full metabolic viability (with no evidence of true cytotoxic injury). Elevated apparent diffusion coefficient (ADC) values on diffusion-weighted imaging (DWI) confirm the predominance of vasogenic edema over cytotoxic edema. The detection of this pattern provides a strong argument against overly aggressive surgical intervention: it proves that the parenchyma is completely salvageable and that the patient will respond to conservative pathogenesis-targeted anticoagulant therapy, despite a severe baseline neurological deficit [5, 18, 20].

Safety of Heparin Therapy in Hemorrhagic Patterns (de Bruijn et al.)

An early multicenter RCT by de Bruijn et al. [2] overturned the dogma regarding the absolute contraindication of anticoagulation in the presence of intracranial hemorrhage. It has been demonstrated that the hemorrhagic pattern of venous infarction (diapedetic hemorrhage resulting from venular rupture due to hydrostatic pressure) requires therapeutic heparinization. Decompressing the venous system by halting thrombus propagation eliminates the underlying source of bleeding.

Selection of DOACs Based on Recanalization Status (RE-SPECT CVT, SECRET)

The transition of patients from standard warfarin therapy to direct oral anticoagulants (DOACs) has become a precision process. The RE-SPECT CVT (2019) and SECRET (2023) RCTs demonstrated that in patients with a stable radiological pattern (defined as no progression of edema or hemorrhage on follow-up CT/MRI at 5-15 days), the use of dabigatran (150 mg twice daily) or rivaroxaban achieves an identical rate of complete sinus recanalization (approximately 80-90%) with a significantly lower risk of spontaneous major hemorrhage compared to warfarin [10, 11].

Endovascular Stalemate and "Criteria of Despair"(TO-ACT RCT)

The results of the TO-ACT randomized controlled trial (2020) demonstrated the futility of routine endovascular treatment (local thrombolysis or mechanical thrombectomy) in patients selected solely on the basis of clinical severity (coma, stupor). The rate of favorable outcomes at 90 days based on the modified Rankin Scale (mRS) did not differ significantly (67% in the endovascular treatment group versus 68% in the conservative therapy group).

Based on this study, a shift in the therapeutic approach has occurred: isolated deep cerebral venous thrombosis is no longer considered an automatic indication for surgery. Endovascular treatment is reserved exclusively as a "last-resort" therapy in the presence of a combination of two dynamic factors: progressive clinical deterioration and expansion of the cytotoxic necrosis zone on DWI/ADC mapping despite adequate administration of low-molecular-weight heparins (LMWH) [14].

A Revolution in Treatment Duration: Results of EXCOA-CVT (ESOC 2026)

The long-awaited EXCOA-CVT multicenter RCT, completed in 2026, has radically transformed approaches to the duration of secondary prevention. Patients were stratified based on a baseline assessment of thrombotic burden and the molecular genetic profile of thrombophilias. The following has been demonstrated [16, 17]:

- For cases involving transient risk factors and an imaging pattern of isolated sinus thrombosis with rapid recanalization within three months, prolonging anticoagulation up to 12 months yields no clinical benefit while doubling hemorrhagic risks.
- A short-term anticoagulation course (3-6 months) has been established as the new global standard of care for the majority of idiopathic CVT cases.
- Prolonged (up to lifelong) therapy is strictly indicated for patients with an ultra-high persistent molecular risk (such as antiphospholipid syndrome or antithrombin III deficiency).

Conclusions:

The transition from an intuitive approach to CVT toward precise algorithms based on radiological patterns represents a crucial step in improving the effectiveness of treatment for this nosology. Detailed assessment of thrombus properties using SWI and black-blood MRI, combined with perfusion mapping of the CBV/CBF dissociation phenomenon, provides the clinician with objective markers for accurately predicting the reversibility of brain parenchymal changes. The evidence base from recent multicenter studies (RE-SPECT CVT, SECRET, TO-ACT, EXCOA-CVT 2026) has established a clear link between radiological patterns and clinical outcomes. This has confirmed the safety of using direct oral anticoagulants (DOACs), narrowed the indications for surgical interventions to cases requiring "last-resort" treatment, and enabled a safe reduction in the duration of secondary prevention to 3-6 months for the majority of patients.

Future academic and practical horizons in this field lie in the integration of radiomics methods and deep learning neural network algorithms for the automated quantitative analysis of thrombus texture and perfusion maps, performed at the very moment of a patient's admission. Furthermore, a pressing objective remains the conduct of targeted, controlled trials for new classes of antithrombotic agents, such as selective inhibitors of coagulation factor XIa which are designed to ensure maximal recanalization of the sinuses while completely eliminating the risk of hemorrhagic complications.

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