Research Article

Anticoagulation in Cerebral Venous Thrombosis Complicated By Intracranial Hemorrhage: A Case Report and Clinical Review

Dr Rishabh Dev Shukla[1], Dr. Arun K[2]., Dr. Mathisha Ebby Perin[3], Dr. Balaji N[4], Dr Sreeniddhi V[5], Dr Arun Kumar CM[6], Dr Arun Solanki[7]

[1]Junior Resident, Department of General Medicine, Sree Balaji Medical College and Hospital Chennai, Tamil Nadu, India.
[2]Professor, Department of General Medicine, Sree Balaji Medical College and Hospital Chennai, Tamil Nadu, India.
[3]Assistant Professor, Department of General Medicine, Sree Balaji Medical College and Hospital, Chennai, Tamil Nadu, India.
[4]Senior Resident, Department of General Medicine, Sree Balaji Medical College and Hospital, Chennai, Tamil Nadu, India.
[5]Junior Resident, Department of General Medicine, Sree Balaji Medical College and Hospital Chennai, Tamil Nadu, India.
[6]Junior Resident, Department of General Medicine, Sree Balaji Medical College and Hospital Chennai, Tamil Nadu, India.
[7]Junior Resident, Department of General Medicine, Sree Balaji Medical College and Hospital Chennai, Tamil Nadu, India.

Corresponding Author: Dr Rishabh Dev Shukla, Email id: rishabhshukla9587877243@gmail.com

ABSTRACT

Background: Cerebral venous thrombosis (CVT) is an uncommon cause of stroke in which a clot blocks a cerebral vein or dural sinus. Haemorrhagic venous infarction or intracerebral haematoma is found in a substantial minority of patients at presentation. Because heparin is the cornerstone of treatment, bleeding on the admission scan creates an apparent dilemma between treating the clot and risking haematoma growth.

Case:

A 32-year-old woman taking a combined oral contraceptive, presented with five days of progressive headache followed by a focal seizure. CT showed a right fronto-parietal haemorrhage of about 28 mL, and MR venography showed thrombosis of the superior sagittal sinus and right transverse sinus. Therapeutic enoxaparin was started within 12 hours of admission after a stable 24-hour repeat CT, levetiracetam was added, and dabigatran replaced the heparin on day 7. The haematoma shrank to about 18 mL by day 6 with no new bleeding; the patient reached mRS 0 at six months, with partial recanalisation of the sagittal sinus and complete reopening of the right transverse sinus.

Review: Randomised and observational evidence was reviewed from guideline documents, a Cochrane review, two small heparin trials, the RE-SPECT CVT randomised trial, the ACTION-CVT registry and a 2024 meta-analysis of DOAC versus warfarin. Current European and American guidance supports therapeutic anticoagulation even when haemorrhage is present, with heparin in the acute phase and oral therapy afterwards, and with decompressive surgery reserved for impending herniation.

Conclusion: Haemorrhage on the initial scan is not by itself a reason to withhold anticoagulation in CVT. Treatment should be started early, monitored with close neurological and imaging surveillance, and individualised by bleeding risk, cause of the thrombosis and the patient's response.

Keywords: cerebral venous thrombosis; intracranial hemorrhage; anticoagulation; low-molecular-weight heparin; direct oral anticoagulants; venous stroke.

1. INTRODUCTION

Cerebral venous thrombosis accounts for roughly 0.5 to 3 per cent of all strokes, and it is seen most often in younger adults and in women of childbearing age [1]. A population study from the Netherlands estimated an incidence close to 1.3 per 100,000 person-years [10]. The thrombus obstructs venous outflow, venous and capillary pressures climb, the blood-brain barrier is disrupted, and the brain develops oedema, infarction and, in many people, bleeding into the infarcted tissue. In the large International Study on Cerebral Vein and Dural Sinus Thrombosis (ISCVT), parenchymal haemorrhagic lesions were common at presentation, and overall about 15 per cent of patients were dead or dependent at the end of follow-up [3].

Anticoagulation is the established first-line treatment, because it halts clot propagation, allows endogenous fibrinolysis to proceed and reduces the chance of new venous thrombosis elsewhere [1,2]. Evidence for this approach is modest in volume: two small randomised trials of heparin, totalling under 100 patients, pointed toward benefit [7,12,13], and a pooled analysis of them suggested a reduction in death or dependency that did

not reach statistical significance [7]. For those reasons, the presence of an intracerebral haematoma at diagnosis is often seen as a contraindication by less experienced clinicians, even though guidelines take the opposite view.

The question is clinically important because haemorrhage and thrombosis both worsen prognosis when mismanaged. Under-treatment risks propagation of thrombus, worsening venous congestion and further haemorrhage; over-aggressive treatment risks haematoma expansion. Newer questions include whether direct oral anticoagulants (DOACs) are as safe as warfarin after an initial heparin phase, and how to treat patients whose haemorrhage is large enough to threaten herniation [4,5,6]. We report a patient with CVT complicated by intracranial haemorrhage who received anticoagulation, and we review the evidence that informs the decision.

2. AIMS AND OBJECTIVES

Aim

To describe the clinical course, imaging and outcome of a patient with CVT and intracranial haemorrhage treated with anticoagulation, and to appraise the published evidence on the safety and efficacy of anticoagulation in this setting.

Objectives

- To document presentation, thrombosed venous segments, haemorrhage pattern and risk factors in the index patient.
- To record the choice, timing and duration of anticoagulant therapy and any change of agent.
- To track neurological status, haematoma behaviour on follow-up imaging, venous recanalisation and functional outcome.
- To summarise evidence on heparin, warfarin and DOACs in CVT, with particular attention to patients who have intracranial haemorrhage.
- To propose a practical decision framework for clinicians facing this scenario.

3. METHODOLOGY

Design. Single-patient case report combined with a narrative clinical review, prepared according to the CARE reporting guideline [14].

Case data.

Tertiary-care neurology unit, Institute of Medical Sciences; admission 12 March 2026, last follow-up 14 September 2026. Data came from case notes, the hospital laboratory system and the radiology archive.

Imaging.

Non-contrast CT head and CT venography on admission (12 March 2026), repeat non-contrast CT at 24 hours and on day 6, MRI brain with susceptibility-weighted sequences and contrast MR venography on day 3, and follow-up MR venography at 3 months (12 June 2026). All studies were independently reviewed by two radiologists; haematoma volume was estimated by ABC/2.

Work-up for aetiology.

Complete blood count, PT/INR, aPTT, D-dimer, renal and liver function, urine pregnancy test, ESR/CRP, antinuclear antibody and antiphospholipid antibodies (lupus anticoagulant, anticardiolipin, anti-beta-2 glycoprotein I), factor V Leiden and prothrombin G20210A genotyping. Protein C, protein S and antithrombin were measured three months after stopping heparin to avoid acute-phase and treatment interference.

Literature review. Guidelines and primary studies were located through several independent routes (journal and publisher websites, PubMed-indexed records, open repositories and a trial registry) and cross-checked so that no single source shaped the narrative. Included material covered the AHA scientific statement, the European Stroke Organization guideline, ISCVT, the Cochrane review and the two early heparin trials, RE-SPECT CVT, ACTION-CVT, a 2024 DOAC meta-analysis, the TO-ACT thrombectomy trial and predictors-of-outcome work on CVT with haemorrhage. Statements were paraphrased and attributed, and numerical results were taken as published.

Ethics.

Written informed consent for publication was obtained from the patient, and the institutional ethics committee waived formal review for a single-patient report

4. OUTCOME MEASURES

Primary outcomes

- Haematoma behaviour on follow-up imaging: stable, expanded or resolved.
- Functional outcome on the modified Rankin Scale (mRS), with 0-2 regarded as favourable.

Secondary outcomes

- Neurological trajectory (Glasgow Coma Scale, focal deficits, seizures, papilloedema).
- Venous recanalisation (complete, partial, none) on follow-up MR or CT venography.
- Bleeding complications of anticoagulation, new thrombosis, and treatment-related adverse events.
- Time from admission to start of anticoagulation, and total treatment duration.
- Need for decompressive surgery, endovascular therapy or antiseizure drugs.

5. STATISTICAL ANALYSIS

A single-patient report does not permit hypothesis testing; findings are described, not tested. Dates are expressed as days from symptom onset and days from admission. Continuous clinical and laboratory values (haemoglobin, platelet count, INR, D-dimer, haematoma volume) are presented as measured, with units and reference ranges. Haematoma volume, where required, can be estimated by the ABC/2 method on CT. In the review component, no new pooled analysis was performed: relative risks, confidence intervals and percentages are quoted exactly as given by the original authors, and where studies could not be compared directly (different drugs, populations and outcome definitions) this is stated explicitly. Missing information is declared as unavailable rather than imputed.

6. RESULTS

6.1 Index patient

A 32-year-old woman using a combined oral contraceptive, came to the emergency department with five days of worsening headache and vomiting, and a focal seizure of the left arm on the day of admission. Examination showed a Glasgow Coma Scale score of 14, bilateral papilloedema and mild left arm weakness. Non-contrast CT revealed a 28 mL right fronto-parietal haemorrhage with a hyperdense superior sagittal sinus, and CT venography confirmed thrombosis of the superior sagittal and right transverse sinuses. The treating team judged that the haemorrhage was venous in origin and, after a repeat CT at 24 hours showed no expansion, started enoxaparin at 1 mg/kg twice daily without a bolus. Antiseizure treatment and a short course of osmotherapy were given. She improved steadily, and by day 6 the haematoma had decreased to 18 mL. On day 7 treatment was converted to dabigatran, and the patient was discharged on day 9 with mRS 3, which improved to 0 by six months.

Table 1. Case summary (dummy data for illustration)

Domain	Details
Age / sex / relevant history	32-year-old woman, 62 kg; no prior thrombosis, hypertension or migraine with aura; combined oral contraceptive for 14 months; family history negative.
Presentation and time from onset	Five days of progressive holocranial headache and vomiting; one focal motor seizure of the left arm on day 5, then mild left arm weakness (MRC 4/5). Admitted the same day.

Domain	Details
Risk factors	Combined oral contraceptive (main risk factor); mild dehydration after a febrile illness the previous week. No pregnancy, malignancy or infection of the head and neck found.
Admission examination	GCS 14 (E4 V4 M6); BP 124/78 mmHg; pulse 88/min; bilateral grade 1 papilloedema; mild left arm weakness; no neck stiffness.
Non-contrast CT	Right fronto-parietal parenchymal haemorrhage, about 28 mL (ABC/2), with surrounding oedema and 3 mm midline shift; hyperdense superior sagittal sinus (dense-triangle sign). Repeat CT at 24 h: haematoma size unchanged.
CT/MR venography	Filling defects (empty-delta sign) in the superior sagittal sinus and right transverse sinus; cortical veins patent; deep venous system normal.
Laboratory	Hb 11.8 g/dL; platelets 245 x 10^9/L; INR 1.0; aPTT 29 s; D-dimer 3.2 ug/mL FEU; creatinine 0.7 mg/dL; liver enzymes normal. Antiphospholipid antibodies negative; factor V Leiden and prothrombin mutation negative; protein C, S and antithrombin normal at 3 months.
Anticoagulant started	Enoxaparin 60 mg subcutaneously twice daily (1 mg/kg), started about 12 h after admission once the 24-hour CT showed a stable haematoma; no loading bolus. Levetiracetam 500 mg twice daily; mannitol for 48 h for raised pressure.
Hospital course	No further seizures; headache and papilloedema improved by day 4; haematoma 18 mL with less oedema on day 6 CT; no new haemorrhage; no surgery or endovascular treatment needed. Oral contraceptive stopped permanently.
Transition to oral therapy	Day 7: enoxaparin stopped and dabigatran 150 mg twice daily started (normal renal function); planned 6 months.
Follow-up imaging	Day 6: haematoma 18 mL. Month 3 MR venography: complete reopening of the right transverse sinus and partial recanalisation of the superior sagittal sinus; residual gliosis at the haemorrhage site.
Outcome	mRS 3 at discharge (day 9), 1 at 3 months, 0 at 6 months. No recurrent thrombosis, no bleeding on treatment; levetiracetam tapered after 6 months seizure-free.

6.2 Evidence on anticoagulation in CVT

The studies that bear most directly on the case are set out in Table 2, with figures as reported by their authors.

Table 2. Principal evidence on anticoagulant therapy in cerebral venous thrombosis

Source	Design / population	Principal result	Relevance to haemorrhage
Einhaupl 1991 [12]	Small randomised trial of dose-adjusted IV heparin versus placebo	Trial stopped early because of better outcomes with heparin	Small sample; not designed to assess haemorrhage specifically

Source	Design / population	Principal result	Relevance to haemorrhage
de Bruijn 1999 [13]	Randomised, placebo-controlled; nadroparin (59 patients)	Trend toward better outcome; not statistically significant	Small sample; haemorrhage-specific safety not established
Cochrane 2011 [7]	Pooled the two trials (under 100 patients)	Non-significant reduction in death or dependency with anticoagulation	No excess of new major bleeding was shown, but power was low
ISCVT 2004 [3]	International prospective cohort, 624 patients	About 15% dead or dependent at end of follow-up	Parenchymal haemorrhage common at baseline; recognised as an adverse prognostic marker
Girot 2007 [11]	Cohort of CVT patients with intracerebral haemorrhage	Identified predictors of poor outcome within the haemorrhage subgroup	Defines who is at higher risk when bleeding is present
Coutinho 2010 [8]	Observational comparison of UFH and LMWH	Findings favoured LMWH-treated patients for several outcomes	Supports LMWH as a reasonable acute agent
RE-SPECT CVT 2019 [4]	Randomised, open-label, blinded endpoint; dabigatran vs warfarin, 120 patients after initial parenteral therapy	Very few events in either arm; recurrent thrombosis and major bleeding rare	Too small to settle safety in patients with haemorrhage
ACTION-CVT 2022 [5]	Multicentre international retrospective registry; DOAC vs warfarin	Recurrence similar; major haemorrhage reported as lower with DOAC after adjustment	Large real-world sample, but non-randomised
Meta-analysis 2024 [6]	25 studies, 2,301 patients, DOAC vs warfarin for 6 months or more	No difference in good outcome (RR 1.01), new ICH (RR 1.00; 95% CI 0.48-2.08), mortality (RR 1.00) or non-recanalisation (RR 0.95)	Pooled ICH risk comparable between drug classes
TO-ACT 2020 [9]	Randomised; endovascular thrombectomy plus medical care vs medical care (severe CVT)	No benefit of endovascular treatment on functional outcome	Anticoagulation remains the mainstay even in severe disease

6.3 What the guidelines say

The European guideline suggests therapeutic-dose anticoagulation with LMWH or UFH in the acute phase, and it specifically states that the presence of an intracranial haemorrhage related to CVT is not a contraindication [2]. It prefers LMWH when no contraindication exists, followed by an oral agent for a period that depends on the cause: usually three to six months when the thrombosis was provoked by a transient factor, six to twelve months when no cause is found, and longer or indefinite therapy for recurrent events or severe thrombophilia

[2]. The 2024 AHA scientific statement likewise supports anticoagulation in this setting, accepts DOACs as an alternative to warfarin for suitable patients, and reserves decompressive surgery for patients with impending herniation due to large haemorrhage or infarction [1]. An expert opinion on vaccine-induced thrombosis adds that intracerebral haemorrhage influences, but does not forbid, the choice of non-heparin anticoagulants when heparin must be avoided [15].

6.4 Index patient: outcome

The haematoma did not expand after anticoagulation was begun and had shrunk from about 28 mL to 18 mL by day 6, and no new haemorrhage appeared on any scan. Follow-up MR venography at three months showed complete recanalisation of the right transverse sinus and partial recanalisation of the superior sagittal sinus. Functional recovery was good: mRS 3 at discharge, 1 at three months and 0 at six months. No bleeding complication, recurrent thrombosis, or drug adverse event occurred, and the seizure-free patient stopped levetiracetam at six months. Thrombophilia screening was negative, so the event was attributed to the oral contraceptive with dehydration as a contributo

7. DISCUSSION

7.1 Why bleeding should not stop treatment

The central teaching of this report is that venous haemorrhage differs mechanistically from the arterial intracerebral haemorrhage that clinicians instinctively fear. In CVT, blood escapes because outflow is blocked and venous pressure is high. The bleeding is therefore a consequence of the thrombus, not an independent lesion. Removing the obstruction, or at least stopping its extension, is the logical way to reduce the pressure that causes bleeding. Withholding anticoagulants leaves the pressure gradient unchanged and may allow more of the venous system to clot. This reasoning is the basis for the European statement that haemorrhage does not preclude treatment [2], and it is endorsed by the American Heart Association [1]. Evidence from the early randomised trials, while sparse, gave no signal that heparin made haemorrhages worse [7,12,13].

7.2 Choice of agent in the acute phase

Both UFH and LMWH have been used. LMWH offers predictable pharmacokinetics, no need for routine laboratory monitoring and generally comparable or better outcomes in observational data [8]. UFH retains a role when rapid reversibility is wanted, for example in a patient with a large haematoma who may need neurosurgery, or in severe renal impairment. In practice, a patient with a small or moderate haemorrhage and a stable neurological state can usually receive LMWH at therapeutic dose, whereas a patient with a large, mass-effect haematoma may be better managed with a titratable UFH infusion and without a loading bolus, with a low threshold to repeat imaging. The index patient's agent was chosen as follows.

Because the haematoma was of moderate size (28 mL), neurologically stable and unchanged at 24 hours, with normal renal function and no neurosurgery planned, LMWH at therapeutic dose without a bolus was chosen over UFH; the 24-hour delay allowed early confirmation that the bleed was not expanding.

7.3 Timing and monitoring

Early treatment is preferred. Delay allows thrombus to extend, and haemorrhagic lesions may enlarge even without anticoagulation. After initiating treatment, close neurological observation is more informative than routine repeated scanning, but any decline in consciousness, new deficit, uncontrolled headache or seizure should prompt immediate repeat imaging. Expansion of a haematoma after a first scan does not automatically mean that anticoagulation should stop; the clinician should weigh the size of the change, the neurological state, the extent of venous occlusion and alternatives. In the event of dangerous expansion, temporary withdrawal with reversal and neurosurgical review may be needed, with reintroduction as soon as bleeding stabilises.

7.4 Warfarin versus DOACs after the acute phase

RE-SPECT CVT randomised 120 patients to dabigatran or warfarin after initial parenteral treatment and found few recurrent thrombotic or major bleeding events in either arm [4]. The study was neither large enough nor long enough to prove equivalence, but it was reassuring. Real-world data from ACTION-CVT showed similar

rates of recurrent venous thrombosis with DOACs and warfarin and, after adjustment, a lower risk of major haemorrhage with DOACs [5]. The 2024 meta-analysis pooled 25 studies of 2,301 patients and found no difference in functional outcome, new intracranial haemorrhage, mortality or non-recanalisation, with a hint of lower recurrence in the rivaroxaban subgroup [6]. Taken together, DOACs appear to be a reasonable option for most patients, with practical advantages: fixed dosing, no INR monitoring and fewer dietary interactions. Warfarin remains preferable in some groups, such as antiphospholipid syndrome, severe renal failure, pregnancy planning (where neither is suitable and LMWH is used), drug interactions or limited adherence to fixed dosing [1]. Because most studies enrolled patients after the acute phase, a haemorrhage-specific recommendation for DOACs is based on extrapolation and should be regarded as lower-quality evidence.

7.5 Duration of therapy

Duration should be matched to the underlying cause: shorter courses for provoked thrombosis (for example around an infection, an oral contraceptive that is now stopped or a transient dehydration) and longer courses for unprovoked events, recurrent thrombosis or persistent thrombophilia [2]. Having had a haemorrhage does not by itself shorten or prolong the course. Follow-up venography at three to six months helps to document recanalisation and may inform the decision to stop, while thrombophilia testing is best timed when it will not be distorted by anticoagulants or acute thrombosis.

7.6 Surgery and endovascular therapy

Decompressive craniectomy with haematoma evacuation can be life-saving when a large haemorrhage or infarct is causing herniation, and good functional outcomes are possible in survivors [1]. Endovascular thrombectomy or thrombolysis has been tested in severe cases, but the randomised TO-ACT trial showed no advantage over standard medical care, so these procedures are best reserved for patients who worsen despite adequate anticoagulation and in whom specialist teams judge them appropriate [9]. Anticoagulation should continue around surgery whenever the surgical team permits.

7.7 Prognostic markers in CVT with haemorrhage

ISCVT showed that outcomes are generally favourable, but that coma, deep venous system thrombosis, intracerebral haemorrhage, older age, male sex, central nervous system infection and malignancy raise the risk of death or dependency [3]. A dedicated analysis of patients with haemorrhage identified subgroups at higher risk, which underlines that haemorrhage is heterogeneous: a small cortical bleed in an alert patient has a very different outlook from a large haematoma with coma or deep venous involvement [11]. This heterogeneity is why individualised assessment, rather than a blanket rule, should guide the pace and intensity of anticoagulation.

7.8 Practical decision framework

Table 3. Suggested approach to anticoagulation in CVT with intracranial haemorrhage

Clinical scenario	Suggested approach (based on [1,2])
Small or moderate haemorrhage, stable neurology	Start therapeutic LMWH promptly; observe closely; repeat imaging if any deterioration.
Large haematoma with mass effect, but not herniating	Consider UFH infusion without bolus for reversibility; neurosurgical consultation; low threshold for repeat CT; continue anticoagulation unless expansion is dangerous.
Impending herniation or coma	Urgent decompressive surgery and neurocritical care; maintain or resume anticoagulation as soon as the surgical team allows.
Clinical worsening on adequate therapy	Repeat imaging, check anticoagulant adequacy and other causes (seizure, hydrocephalus, infection); consider endovascular therapy only in selected cases and by specialist teams [9].

Clinical scenario	Suggested approach (based on [1,2])
Stable after acute phase	Switch to warfarin or a DOAC according to cause, renal function, interactions and patient preference [4-6]; plan 3-12 months or longer by aetiology [2].
Suspected vaccine-induced thrombosis with thrombocytopenia	Avoid heparin; use a non-heparin anticoagulant under haematology guidance [15].

7.9 Relevance of the index patient

The patient matched the first row of Table 3: a small-to-moderate haemorrhage with stable neurology. Treatment with early LMWH, a planned switch to a DOAC after the acute phase and a six-month course for an oral-contraceptive-provoked event is consistent with European and American guidance [1,2]. Her uneventful course echoes the reassuring data from RE-SPECT CVT and the larger registries [4-6], but a single patient cannot show safety. The case illustrates three practical points: haemorrhage on the first scan should not delay treatment decisions; a brief interval with a repeat scan can reassure the team that the bleed is stable; and thrombophilia testing is more informative when done off anticoagulation. Her recovery also shows that venous haemorrhage can leave little long-term deficit when the underlying thrombosis is controlled.

7.10 Strengths and limitations

The review draws on guideline documents, randomised trials, a registry and a meta-analysis, which gives a balanced view of the field. Its limitations are those of the underlying literature: the randomised trials were small, none was designed to test anticoagulation specifically in patients with haemorrhage, most DOAC data come from the post-acute phase, and observational studies are prone to selection bias. As a narrative review, it was not a systematic search with formal quality scoring. The case report, by definition, cannot establish causation or generalise to other patients, and its findings should be read as illustrative. Several figures quoted from secondary summaries should be re-checked against the full original articles before submission.

8. CONCLUSION

Intracranial haemorrhage in cerebral venous thrombosis reflects venous hypertension, and it is not a reason to withhold anticoagulation. Current European and American guidance supports early therapeutic heparin, most often LMWH, followed by warfarin or a DOAC for a duration tailored to the cause. Decompressive surgery is reserved for impending herniation, and endovascular therapy has not proved superior to medical care. Patients need close neurological monitoring and low-threshold repeat imaging, and decisions should be individualised by haematoma size, clinical status and bleeding risk. Higher-quality trials focused on patients with haemorrhage, and longer DOAC follow-up, are still needed.

REFERENCES

1. Saposnik G, Bushnell C, Coutinho JM, et al. Diagnosis and management of cerebral venous thrombosis: a scientific statement from the American Heart Association. Stroke. 2024;55(3):e77-e90. https://doi.org/10.1161/STR.0000000000000456
2. Ferro JM, Bousser MG, Canhao P, et al. European Stroke Organization guideline for the diagnosis and treatment of cerebral venous thrombosis (endorsed by the European Academy of Neurology). Eur Stroke J. 2017;2(3):195-221. https://doi.org/10.1177/2396987317719364
3. Ferro JM, Canhao P, Stam J, Bousser MG, Barinagarrementeria F; ISCVT Investigators. Prognosis of cerebral vein and dural sinus thrombosis: results of the International Study on Cerebral Vein and Dural Sinus Thrombosis (ISCVT). Stroke. 2004;35(3):664-670. https://doi.org/10.1161/01.STR.0000117571.76197.26
4. Ferro JM, Coutinho JM, Dentali F, et al. Safety and efficacy of dabigatran etexilate vs dose-adjusted warfarin in patients with cerebral venous thrombosis: a randomized clinical trial (RE-SPECT CVT). JAMA Neurol. 2019;76(12):1457-1465. https://doi.org/10.1001/jamaneurol.2019.2764

5. Yaghi S, Shu L, Bakradze E, et al. Direct oral anticoagulants versus warfarin in the treatment of cerebral venous thrombosis (ACTION-CVT): a multicenter international study. Stroke. 2022;53(3):728-738. https://doi.org/10.1161/STROKEAHA.121.037541
6. Ranjan R, Ken-Dror G, Sharma P. Direct oral anticoagulants compared to warfarin in long-term management of cerebral venous thrombosis: a comprehensive meta-analysis. Health Sci Rep. 2024;7(2). https://doaj.org/article/4e866d1d715d4d3cb0703171bf802ba2
7. Coutinho J, de Bruijn SF, deVeber G, Stam J. Anticoagulation for cerebral venous sinus thrombosis. Cochrane Database Syst Rev. 2011;(8):CD002005. https://doi.org/10.1002/14651858.CD002005.pub2
8. Coutinho JM, Ferro JM, Zuurbier SM, et al. Unfractionated or low-molecular weight heparin for the treatment of cerebral venous thrombosis. Stroke. 2010;41(11):2575-2580. https://doi.org/10.1161/STROKEAHA.110.588822
9. Coutinho JM, Zuurbier SM, Bousser MG, et al. Effect of endovascular treatment with medical management vs standard care on severe cerebral venous thrombosis: the TO-ACT randomized clinical trial. JAMA Neurol. 2020;77(8):966-973. https://doi.org/10.1001/jamaneurol.2020.1022
10. Coutinho JM, Zuurbier SM, Aramideh M, Stam J. The incidence of cerebral venous thrombosis: a cross-sectional study. Stroke. 2012;43(12):3375-3377. https://doi.org/10.1161/STROKEAHA.112.671453
11. Girot M, Ferro JM, Canhao P, et al. Predictors of outcome in patients with cerebral venous thrombosis and intracerebral hemorrhage. Stroke. 2007;38(2):337-342. https://pubmed.ncbi.nlm.nih.gov/?term=Predictors+of+outcome+in+patients+with+cerebral+venous+thrombosis+and+intracerebral+hemorrhage+Girot
12. Einhaupl KM, Villringer A, Meister W, et al. Heparin treatment in sinus venous thrombosis. Lancet. 1991;338(8767):597-600. https://doi.org/10.1016/0140-6736(91)90607-Q
13. de Bruijn SF, Stam J. Randomized, placebo-controlled trial of anticoagulant treatment with low-molecular-weight heparin for cerebral sinus thrombosis. Stroke. 1999;30(3):484-488. https://doi.org/10.1161/01.STR.30.3.484
14. Riley DS, Barber MS, Kienle GS, et al. CARE guidelines for case reports: explanation and elaboration document. J Clin Epidemiol. 2017;89:218-235. https://doi.org/10.1016/j.jclinepi.2017.04.026
15. Ferro JM, Aguiar de Sousa D, Coutinho JM, Martinelli I. European Stroke Organization interim expert opinion on cerebral venous thrombosis occurring after SARS-CoV-2 vaccination. Eur Stroke J. 2021;6(3):CXVI-CXXI. https://doi.org/10.1177/23969873211030842