VILNIUS UNIVERSITY
FACULTY OF MEDICINE
Medicine

Institute of Biomedical Sciences, Department of Radiology, Nuclear Medicine and Medical Physics

Tamem Khalaf 2025, group 5

INTEGRATED STUDY MASTERS THESIS

Radiological Diagnosis of Cerebral Venous Infarction. Case Presentation

Supervisor
Dr. doc. Jūratė Dementavičienė
Associate professor

Head of the department
Dr. Prof. Algirdas Edvardas Tamošiūnas
Professor

Vilnius, 2025

tamem.khalaf@mf.stud.vu.lt

1. Abbreviations

- Cerebral venous thrombosis (CVT)
- Cerebrospinal fluid (CSF)
- Central nervous system (CNS)
- Superior sagittal sinus (SSS)
- Inferior petrosal sinus (IPS)
- Superior petrosal sinus (SPS)
- Transverse sinus (TS)
- Dural sinus thrombosis (DST)
- Platelet factor 4 (PF-4)
- Vaccination-Induced immunological Thrombotic Thrombocytopenia (VITT)
- Paroxysmal nocturnal hemoglobinuria (PNH)
- Granulomatosis with polyangiitis (GPA)
- Cerebral venous sinus thrombosis (CVST)
- The modified Rankin Scale (mRS)
- Glasgow Coma Scale (GCS)
- Computed tomography (CT)
- Magnetic resonance imaging (MRI)
- International Study of Cerebral Venous Thrombosis (ISCVT)
- Middle cerebral artery (MCA)
- MR-venography (MRV)
- Grading of Recommendations, Assessment, Development, and Evaluation (GRADE)
- European Stroke Organization (ESO)
- C-reactive protein (CRP)
- Dual oral anticoagulation (DOAC)
- Low molecular weight heparin (LMWH)
- Unfractionated heparin (UFH)
- The National Institutes of Health Stroke Scale (NIHSS)
- Intracranial haemorrhage (ICH)
- Vitamin K antagonists (VKA)

- Magnetic resonance black-blood thrombus imaging (MRBTI)
- The cerebral venous extent score (CVES)
- The cerebral venous occlusion score (CVOS)
- Spectral Presaturation with Inversion Recovery (SPIR)
- Diffusion weighted imaging (DWI)
- Dural arteriovenous fistula (dAVF)

2. Keywords

- Cerebral venous thrombosis, dural sinus thrombosis, intracerebral haemorrhage, venous infarction, cerebral blood flow.

3. The objective of this document

This document provides an overview of cerebral venous thrombosis (CVT), emphasising its clinical presentation, diagnostic methods, pathogenesis, and treatment approaches. The objective is to connect recent advancements with a clinical case, covering some aspects of prognosis assessment.

4. Abstract

Cerebral venous thrombosis (CVT) is characterised as an uncommon type of stroke resulting from thrombosis in the venous circulation of the brain, which causes obstruction of one or more cerebral veins and/or the dural venous sinus.

Blood clots, physiologically develop at the location of an injury to inhibit blood loss. Thrombosis, on the other hand, refers to the pathological development of a blood clot without any associated injury. The body's circulation includes veins that remove blood that is low in oxygen and high in carbon dioxide and metabolites from the organs and tissues, and arteries that transport blood that is rich in oxygen and nutrients to the organs and tissues. Arterial thrombosis refers to the thrombosis of an artery, while vein thrombosis refers to the thrombosis of a vein.

Cerebral venous thrombosis (CVT) presents notable diagnostic and therapeutic challenges, especially in young and postpartum individuals, due to its varied and occasionally misleading clinical manifestations. This document aims to provide an overview of cerebral venous thrombosis (CVT), emphasising its clinical presentation, diagnostic methods, pathogenesis, and treatment approaches. The objective is to present the recent advancements together with a case study. [1]

5. Epidemiology

The incidence of CVT has increased in various regions over recent decades. Population-based studies indicate an average incidence of 12 cases per million annually; however, significant heterogeneity exists among the studies. Certain low- to middle-income countries has shown evidence of a potentially elevated prevalence of CVT. [17]

CVT occurs in women three times more frequently than in men, particularly during their reproductive years, with the median age at the thrombotic event being in the fourth decade of life. [18]

Studies from India and the United States show a different pattern of this known. Namely, increased incidence in the elderly population and males. Data from the United States indicate racial disparities, showing a higher incidence rate among black individuals compared to other racial groups. [18,19]

6. Anatomy

The cerebral veins empty into significant venous formations known as dural venous sinuses (Figure 1). The dural venous sinuses are responsible for draining cerebrospinal fluid (CSF), which is a clear fluid that surrounds the brain and spinal cord. Cerebrospinal fluid (CSF) provides protection to the brain and plays a crucial role in maintaining the equilibrium of physical and chemical environments. [2]

Individuals diagnosed with cerebral venous thrombosis (CVT) exhibit the presence of blood clots within one or multiple dural venous sinuses, cerebral veins, or both anatomical structures. [3]

6.1 Venous Drainage of the Central Nervous System

The brain, spinal cord, brainstem, and cerebellum make up the central nervous system. The CNS drainage system is complex and, unlike other organ systems, does not correspond to the arterial systems.

The dural venous sinuses receive blood from the brainstem, cerebellum, and cerebrum via a network of veins. Both the internal and external vertebral plexuses receive blood from the spinal veins, which drains the spinal cord.

6.2 Venous Dural Sinuses

The dura mater's periosteal and meningeal layers are separated by the dural venous sinuses. The best way to conceptualize them is as blood pools that drain the central nervous system. The internal jugular vein is where all dural venous sinuses eventually empty. The dural venous sinuses lack valves, in contrast to the majority of the body's veins.

The total number of venous sinuses is eleven. The dura mater's falx cerebri contains the superior, inferior, and straight sagittal sinuses. They meet at the sinus confluence, which is located above the internal occipital protuberance. The inferior sagittal sinus and the great cerebral vein continue into the straight sinus. [5]

Transverse sinus continues bilaterally and curves into sigmoid sinus to meet internal jugular vein opening from confluence.

Ophthalmic veins feed into the cavernous sinus on either side of the Sella turcica. Blood returns to the internal jugular vein via the superior or inferior petrosal sinuses. [4,61]

6.2.1 Dural Sinuses

These are the venous channels located between the endosteal and meningeal layers of the dura mater.

6.2.2 UNPAIRED SINUSES

- The superior sagittal sinus (SSS) is the largest dural venous sinus and lies in the midline between the inner table of the skull (superiorly) and the falx cerebri (laterally).
- The inferior sagittal sinus lies in the inferior free margin of the falx cerebri.
- The straight sinus (SS).
- The occipital sinus, the smallest dural venous sinus, is important to identify during posterior fossa craniotomy.
- The inter-cavernous sinus.

6.2.3 PAIRED SINUSES

- The transverse sinus (TS).
- Sigmoid sinuses – inferior continuation of the TS, which continue inferiorly into the jugular bulb at the skull base.
- The superior petrosal sinus (SPS).
- The inferior petrosal sinus (IPS).
- The sphenoparietal sinus. [5]

6.3 Veins of The Cerebrum

Cerebral veins transport blood from brain tissue and deposit it in the dural venous sinuses. They are classified into superficial and deep that surround the brain's gyri and sulci. After entering the brain parenchyma, the veins travel through the subarachnoid space and pierce the meninges before draining into the dural venous sinuses.

6.3.1 Superficial System

The superficial system of veins is largely responsible for draining the cerebral cortex:

- Superior cerebral veins: Drain the superior surface, carrying blood to the superior sagittal sinus.
- Superficial middle cerebral vein: Drains the lateral surface of each hemisphere, carrying blood to the cavernous or sphenopalatine sinuses.
- Inferior cerebral veins: Drain the inferior aspect of each cerebral hemisphere, depositing blood into cavernous and transverse sinuses.
- Superior anastomotic vein (Trolard): Connects the superficial middle cerebral vein to the superior sagittal sinus.
- Inferior anastomotic vein (Labbé): Connects the superficial middle cerebral vein to the transverse sinus.

6.3.2 Deep System

Thrombotic occlusion of deep cerebral veins usually both internal cerebral veins, vein of Galen , straight sinus often with widespread dural sinus thrombosis (DST).

- Subependymal veins – There are numerous subependymal veins, which will not be described here in detail. These receive blood from the medullary veins and carry it to the dural venous sinuses. The great cerebral vein (vein of Galen) is worthy of a mention; it is formed by the union of two of the deep veins and drains into the straight sinus.
- Medullary veins: Originate 1-2cm below the cortical grey matter and drain into subependymal veins. These drain the deep areas of the brain. [5]

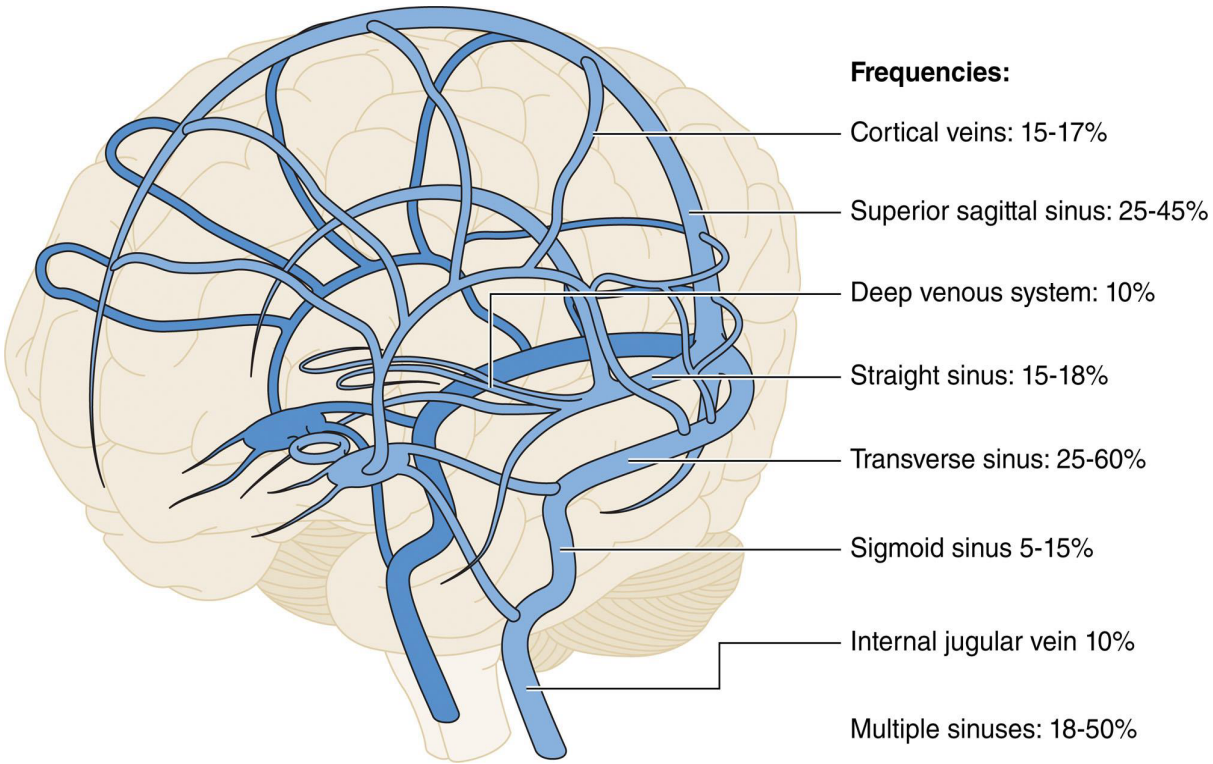


Figure (1) Anatomy of the cerebral venous system and distribution of CVT. [70]

7. Impact of Impaired Brain Drainage:

Blood clot formation obstructs a dural venous sinus, this may impair the drainage of both venous blood and CSF from the intracranial space.
Impaired drainage of CSF leads to increased intracranial pressure. This may be accompanied by several symptoms such as headache, nausea, vomiting, visual problems owing to papilledema (swelling of the optic nerve disc), and diplopia (double vision) due to compression of the abducens nerve. [26]

The obstruction of venous blood flow increases pressure within the venous system and capillaries. This may cause vasogenic oedema which refers to the swelling resulting from the rupture of the blood-brain barrier and the consequent leakage of fluid from capillaries into the surrounding brain tissue. If the decreased blood flow is significant enough to cause metabolic disturbances in the brain cells, this may lead to cytotoxic oedema which is the accumulation of fluid in the brain tissue due to swelling of damaged brain cells caused by lack of oxygen or glucose. Ultimately, the increased pressure in the veins and capillaries may also cause these blood vessels to rupture, causing cerebral haemorrhage. Damage to the brain tissue may cause epileptic seizures or neurologic deficits, such as paralysis or sensory deficits. In the most severe cases, extensive brain lesions may cause decreased consciousness and ultimately death due to brain herniation (Compression of the brain within the skull). [6]
Some people with CVT have merely symptoms and evidence of elevated intracranial pressure; others may have symptoms and signs of brain oedema or haemorrhage. [26]

8. Prevalence and risk factors

Cerebral venous thrombosis (CVT) is an uncommon condition. In a study done in the Netherlands, CVT was shown to affect an estimated 14 to 20 individuals per million adults annually, which translates to approximately 250 to 350 cases per year in the Netherlands. It constitutes merely approximately 1% of all strokes in adults. In contrast, arterial thrombosis of the brain, also known as ischaemic stroke, accounts for around 87% of all strokes, while the remainder strokes result from various forms of cerebral haemorrhage, including intracerebral or subarachnoid haemorrhage. [7]
CVT differs from other types of strokes in several aspects. Patients with cerebral venous thrombosis (CVT) are frequently diagnosed at a young age, with a median age of 49 years and

an interquartile range (IQR) of 40 to 61.4 years. Women are disproportionately impacted, comprising approximately two-thirds of the patient population. [12]

The risk factors for cerebral venous thrombosis (CVT) differ from those associated with ischaemic and haemorrhagic strokes, however they are similar to those for venous thrombosis in other anatomical locations. Namely, the use of hormonal contraceptives, inherited or acquired thrombophilia, pregnancy or recent childbirth, infections (head or neck), malignancies, trauma (head), and inflammatory conditions. [6,62]

Risk factors for CVT range from transient situations such as pregnancy and infection to more permanent causes such as inherited thrombophilia and acquired prothrombotic conditions. In recent decades, the catalogue of recognized risk factors has expanded. Similar to anaemia and obesity, especially in women utilising oral contraceptives.

A genetic study identified a correlation between the ABO blood group and CVT. [11]

The COVID-19 pandemic introduced further correlations, both with the disease itself and the adenovirus vector SARS-CoV-2

vaccines, while the exact nature of these connections has not been completely clarified. CVT and thrombocytopenia have manifested as an exceedingly unusual consequence following the administration of ChAdOx1 nCov-19. COV2.S vaccinations were associated with a higher incidence in younger demographics, exhibiting much more severe manifestations and fatality rates reaching 50%. 9.10 Interestingly, both heparin-induced thrombocytopenia and vaccine-induced thrombotic thrombocytopenia include PF-4 (platelet factor 4). [8]

Antibodies are proteins produced by platelets, which are cells that play a role in blood coagulation. PF4 is involved in the regulation of coagulation and immunological responses. [10]

Cerebral venous thrombosis is infrequently observed in heparin-induced thrombocytopenia, whereas nearly fifty percent of vaccine-induced thrombotic thrombocytopenia cases exhibit cerebral venous thrombosis. The immunological response elicited by the vaccination, characterised by PF4 antibodies, is predominantly linked to a rare syndrome known as vaccination-Induced immunological Thrombotic Thrombocytopenia (VITT). [9]

8.1 Infectious diseases as a risk factor

Otitis and mastoiditis may lead to thrombosis of the nearby structures such as sigmoid and transverse sinuses. Hypoplasia of the contralateral transverse sinus, a common anatomical variant, leads to impaired absorption of cerebrospinal fluid, which in turn leads to intracranial

hypertension accompanied by papilledema. It was previously referred to as "otitic hydrocephalus," a term that is a misnomer, as the ventricles typically do not exhibit enlargement in instances of sinus thrombosis.

The incidence of infectious sinus thrombosis has decreased, ranging from 6 to 12 percent in extensive studies of adults with sinus thrombosis. 1.5 Children exhibit increased frequencies of systemic infections, such as neonatal sepsis, as well as local infections, including otitis.

Thrombosis of the cavernous sinuses represents a specific condition, typically resulting from infections in the paranasal sinuses (ethmoid and sphenoid), the orbit, or facial regions. [15]

Table 1. Causes of and Risk Factors Associated with Cerebral Venous Sinus Thrombosis.

Genetic prothrombotic conditions
Antithrombin deficiency[5]
Protein C and protein S deficiency[6-8]
Factor V Leiden mutation[9-11]
Prothrombin mutation (the substitution of A for G at position 20210)[9,11,12]
Homocysteinemia caused by gene mutations in methylenetetrahydrofolate reductase[13,14]
Acquired prothrombotic states
Nephrotic syndrome
Antiphospholipid antibodies[7,15]
Homocysteinemia[14]
Pregnancy[16,17]
Puerperium[17]
Infections
Otitis, mastoiditis, sinusitis[6]
Meningitis
Systemic infectious disease[6]
Inflammatory disease
Systemic lupus erythematosus[18]
Wegener's granulomatosis[8]
Sarcoidosis
Inflammatory bowel disease
Behçet's syndrome[19,20]
Hematologic conditions
Polycythemia, primary and secondary
Thrombocythemia
Leukemia[21]
Anemia, including paroxysmal nocturnal hemoglobinuria[22]
Drugs
Oral contraceptives[9,23]
Asparaginase[6,21]
Mechanical causes, trauma
Head injury[24]
Injury to sinuses or jugular vein, jugular catheterization
Neurosurgical procedures
Lumbar puncture[25]
Miscellaneous
Dehydration, especially in children[6]
Cancer[1,6]

Table (1) Causes and Risk Factors Associated with Cerebral Venous Sinus Thrombosis.

8.2 Other rare risk factors or comorbidities:

- Disseminated intravascular coagulation.
- Paroxysmal nocturnal hemoglobinuria (PNH).
- Heparin-induced thrombocytopenia.
- Vaccine-induced prothrombotic immunogenic thrombocytopenia

(VITT).

- Severe hyperhomocysteinemia.
- Dysfibrinogenemia.
- Malignancies: carcinomas, lymphomas, carcinoid,leukemias.
- Sickle cell anemia, hypochromic or hemolytic anemia.
- Collagenosis: lupus erythematosus, Sojgren's syndrome.
- Vasculitides: Behçet's disease, granulomatosis with polyangiitis (GPA; formerly: Wegener's granulomatosis).
- Sarcoidosis. [14,33]

9. Risk factors and outcome

Taking into consideration the variety of risk factors that could contribute to the formation of CVT it is of importance to discuss whether multiple risk factors could influence the outcome. In a study done by J. Kalita et. Al. in 2018 to examine the burden of risk factors in cerebral venous sinus thrombosis (CVST) and their association with clinical severity, the extent of MRI and MRV abnormalities, and patient outcomes.

Using the modified Rankin Scale (mRS) to assess the outcome severity, one hundred twenty-eight consecutive patients with CVST were included in the study. Demographic data, clinical presentation, and neurological findings were documented, and risk factors for CVST were assessed.

Patients were categorised according to risk factors into prothrombotic conditions only in 46 (35.9%), prothrombotic with other risk factors in 36 (28.1%), and non-prothrombotic risk factors in 20 (15.6%), as well as no risk factors in 26 (20.3%). Thirty-three individuals (25.8%) exhibited more than two risk factors. Of the 22 patients, 16 (72.7%) with female gender-specific risk factors also presented with additional prothrombotic conditions. In the MRV analysis, over two sinuses were affected in 35 patients (27.3%), while 94 patients (73.4%) exhibited parenchymal lesions on MRI.

Thirty-one patients (24.2%) exhibited poor outcomes (mRS > 2) at discharge, while 25 out of 122 patients (20.5%) demonstrated similar outcomes at the three-month follow-up. The quantity of risk factors showed no correlation with clinical severity, or the extent of abnormalities observed in MRI or MRV. In the multivariate analysis, age, GCS score, and mechanical ventilation were identified as predictors of the outcome at 3 months. [13]

10. Pathogenesis

The pathophysiology of CVT is better explained by defining the site of the thrombus. Therefore, to understand the symptoms and signs of CVT, distinguishing between the mechanisms mentioned below is crucial. Thrombosis of the cerebral veins, which results in local effects due to venous obstruction, and thrombosis of the major sinuses, which leads to intracranial hypertension. In most patients, these two processes occur concurrently. Occlusion of the cerebral veins can lead to localized brain oedema and venous infarction. Pathological examination reveals enlarged, swollen veins, oedema, ischaemic neuronal damage, and petechial haemorrhages. The latter may develop into large haematomas, which exhibit a distinctive appearance on computed tomographic (CT) scans (Fig.2 A). Two distinct types of cerebral oedema may occur. Cytotoxic oedema arises from ischaemia, which impairs energy-dependent cellular membrane pumps, resulting in intracellular swelling and vasogenic oedema which results from a breach in the blood–brain barrier, leading to the extravasation of blood plasma into the interstitial space. Vasogenic oedema can be reversed with successful treatment of the underlying condition. Magnetic resonance imaging (MRI) has demonstrated the presence of both cytotoxic and vasogenic oedema in cases of cerebral vein thrombosis. [16,63]

The second mechanism involves the development of intracranial hypertension due to occlusion of major venous sinuses which in turn impairs the normal physiological draining mechanism. Normally, The cerebrospinal fluid is transported from the cerebral ventricles through the subarachnoid spaces at the brain's base and surface to the arachnoid villi, where it is absorbed and drained into the superior sagittal sinus. Therefore, thrombosis of the sinuses results in elevated venous pressure, reduced absorption of cerebrospinal fluid, and, as a result, increased intracranial pressure. [16] The blockage in cerebrospinal fluid drainage occurs at the terminus of its transport pathway, resulting in the absence of a pressure gradient between the subarachnoid spaces on the brain's surface and the ventricles. Consequently, the ventricles remain undilated, and hydrocephalus typically does not occur as a complication of sinus thrombosis. Approximately 20% of patients with sinus thrombosis present with intracranial hypertension alone, lacking evidence of cortical vein thrombosis. [15]

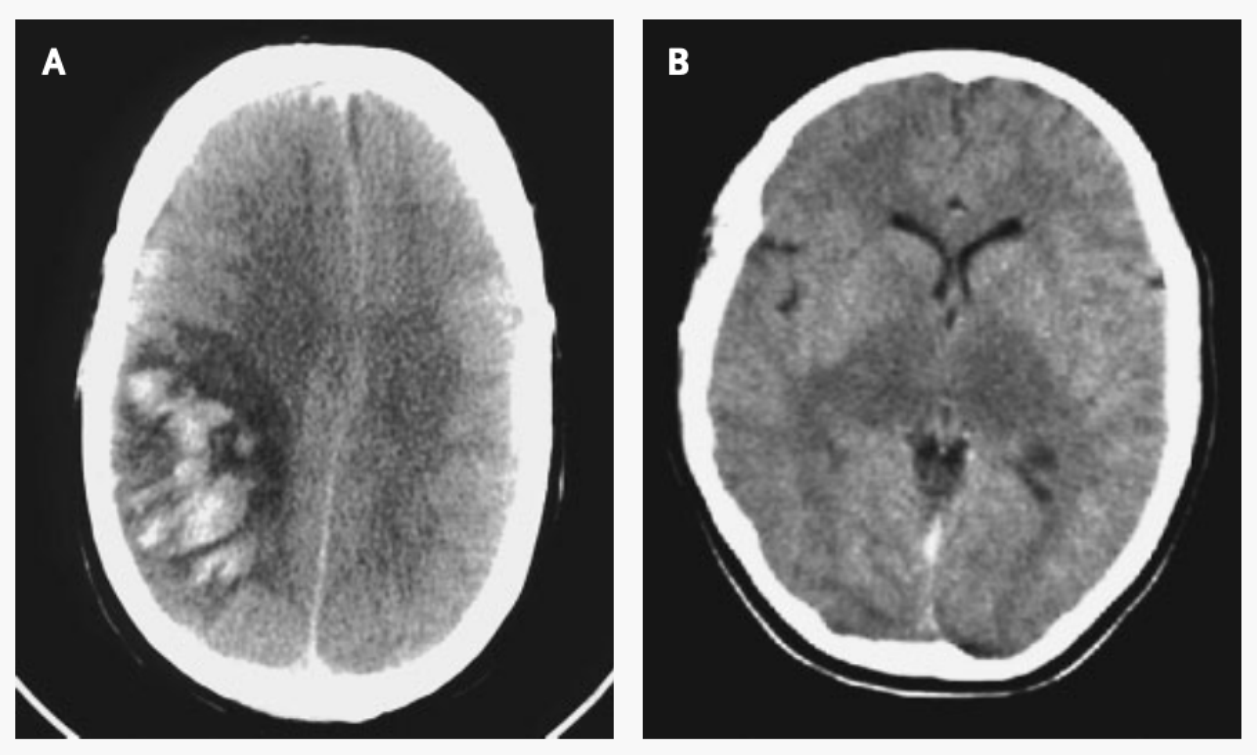


Figure (2) CT Imaging of Sinus Thrombosis.

A CT scan (Panel A) shows a large infarct in the right parietal lobe (at left in the image) in a patient with sinus thrombosis. The infarct is haemorrhagic (white patches). The falx cerebri is displaced to the left as a result of compression by the infarct. In the CT scan in Panel B, bilateral thalamic edema (dark area in center) has been caused by thrombosis of the straight sinus. [15]

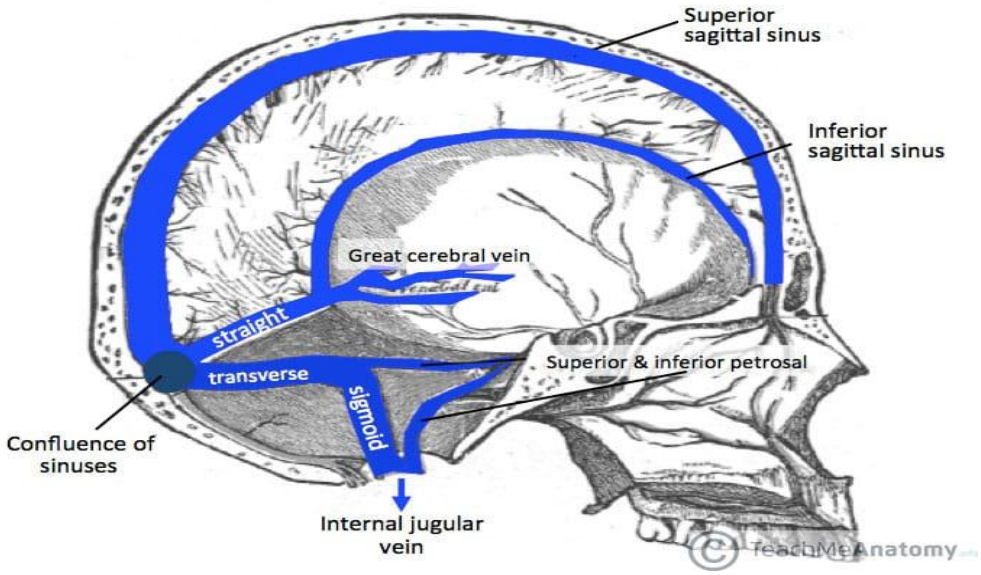


Figure.3 Sagittal section showing the dural venous sinuses and the great cerebral vein.

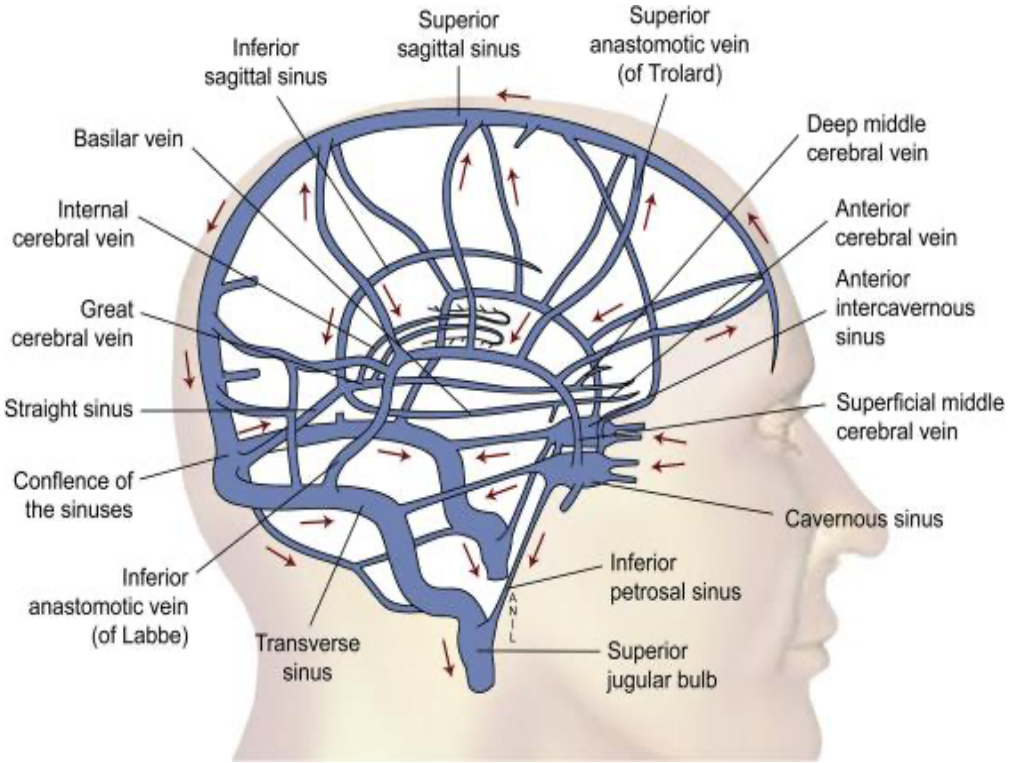


Figure (4) Venous anatomy. [71]

11. Clinical Presentations

90% of patients experience an acute or subacute headache as the main symptom of dural sinus thrombosis, often without any neurologic examination abnormalities. Patients with cerebral venous thrombosis (CVT) might present with a wide range of symptoms, with the most common and non-specific symptom being a severe headache. Other common symptoms include diplopia (14%), aphasia (19%), paresis or paralysis (37%), and epileptic seizures (20–40%). When they first arrive at the emergency room, about 5% of patients are in a vegetative state. [64]

The onset of cerebral venous thrombosis (CVT) can range from acute to subacute and chronic presentations. Symptoms are more diverse than those of other stroke types and are often nonspecific, potentially leading to underdiagnosis. Symptoms and signs of cerebral venous thrombosis (CVT) can be categorised into presenting syndromes, with the most prevalent

being isolated intracranial hypertension syndrome, focal syndrome, and encephalopathy. Approximately 90% of patients with cerebral venous thrombosis (CVT) report headaches. [21]

The initial clinical presentation may indicate symptoms resulting from elevated intracranial pressure, the location of the clot, and the burden of thrombus. Typical manifestations encompass headache, seizures, focal neurological deficits, and altered levels of consciousness. Patients typically present sub-acutely within 48 hours to 2 weeks following symptom onset; approximately one-third to one-half may present more acutely with stroke-like symptoms or a thunderclap headache. The clinical presentation in the two largest prospective series of cerebral venous thrombosis, namely the International Study of Cerebral Venous Thrombosis (ISCVT) and the Turkish VENOST study, is summarised in Table 2. [20, 21]

A sub-study of the ISCVT indicated that elderly patients exhibited a higher likelihood of presenting with depressed levels of consciousness and a lower likelihood of isolated symptoms of intracranial hypertension. [22]

A prospective international study involving 1,281 individuals done by Lindgren E. et al. in 2020. Showed a 20-40% incidence of acute symptomatic seizures, with 6% of cases progressing to status epilepticus. Neuroradiological findings linked to acute seizures. [23]

Symptoms and clinical presentation could vary depending on many factors and, most importantly, like stroke, on the site of occlusion. Namely, CVST is often accompanied by infection resulting in ocular symptoms due to CN III, IV, V,VI palsies. [24]

Presenting findings of cerebral venous thrombosis

	ISCVT (2004)	VENOST (2017)
N	624	1144
Age	39 (mean)	18–36 y (47%)
	37 (median), range 16–86	37–50 (33%)
		51 + (20%)
% Female	75	68
Headache	89%	87%
Visual loss	13%	27%
Cranial neuropathy/diplopia	14% (diplopia only)	11%
Depressed level of consciousness/encephalopathy	22%	18%
Seizure	40%	24%
Focal deficits	Motor 40%	18%
	Sensory 5%	
	Other 3%	

Table (2) Venous Thrombosis (ISCVT) and the Turkish VENOST study, presenting findings.

12. Distinctions between cerebral venous thrombosis (CVT) and stroke:

Stroke can be categorised into ischaemic and haemorrhagic based on the aetiology, ranking as the second greatest cause of mortality. [26]

Stroke is induced not alone by arterial thrombosis but also by cerebral venous thrombosis. Arterial stroke is presently the predominant subtype of stroke, and research in this area has progressively advanced. Venous thrombosis, a specific variant, constitutes 0.5–1% of all strokes. [28]

The incomplete comprehension of venous thrombosis, together with its varied clinical presentations and neuroimaging characteristics, frequently results in delayed admissions and a propensity for misdiagnosis. [27]

This part aims to summarize the key differences between CVT and arterial stroke, hoping to enhance the understanding of the pathogenesis of intravenous stroke as well as the diagnosis and treatment that will be discussed later.

When comparing CVST to stroke, it is essential to have a solid understanding of how each condition originates. CVST is a clot formation in the sinuses of the brain, which disturbs the flow of blood and causes an increase in the intracranial pressure. On the other hand, stroke occurs when an artery is blocked, eventually causing ischaemia. [29]

When it comes to the clinical presentation, a range of differences can be noticed. For instance, ischaemic stroke could lead to a variety of symptoms depending on the site of the occluded artery. An example is when the middle cerebral artery MCA is occluded, giving rise to symptoms like hemianopia, ataxia, nystagmus, hemiplegia, and brain stem cranial palsy. [29]

When comparing the symptoms of a stroke to those of a thrombosis, the CVT manifestations include severe headaches, hazy vision, and convulsions that are mainly caused by elevated intracranial pressure. [30]

Young individuals are typically affected by CVT, which is more prevalent in females and is frequently associated with birth control or pregnancy. Strokes are most commonly experienced by elderly individuals who have high blood pressure, deterioration of the arteries, and diabetes. Although, studies in the US and India show a new trend of CVT being increased among the elderly. [18,19]

In a review study done by Yifan Z. et al. in 2022. Show that CVT, unlike stroke, has a favourable outcome, mostly affecting young and middle-aged patients. It even shows that 13% of all patients die or are severely handicapped.

13. Diagnosis and Neuroimaging

Due to the infrequency of CVT, the significant diversity in clinical manifestations, and often non-specific beginning of symptoms, the identification of the condition is quite challenging. Many individuals without CVT get brain scans to exclude the diagnosis, resulting in unnecessary radiation exposure and expenses. On the other hand, numerous individuals with CVT encounter diagnosis delays, which may impact outcomes negatively. The diagnosis of CVT is typically established using head CT-venography, MRI, or MR-venography. The contrast-enhanced venous system reveals a filling defect at the location of the thrombus (Figure, right panel). Imaging reveals a parenchymal brain lesion in approximately 50% of patients: focal oedema and intracerebral haemorrhage in about 32% (Figure 5, left panel) and focal oedema without haemorrhage in around 18%. Sulcal subarachnoid haemorrhage occurs

in 10% of cases, while subdural haematoma is observed in 3% of instances. [31,23]

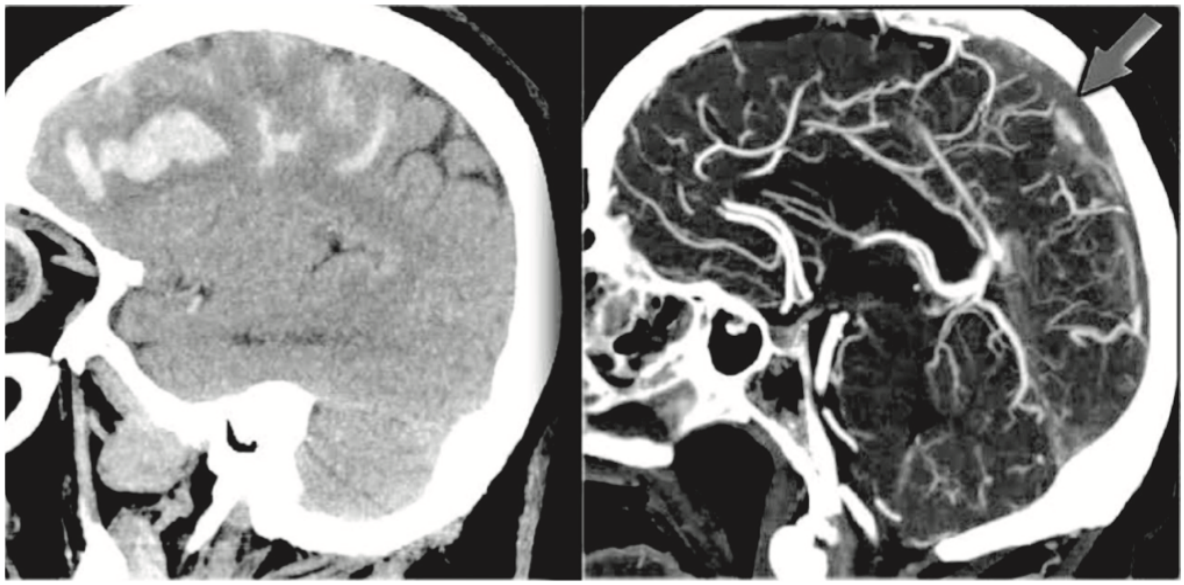


Figure (5) On the left reconstructed sagittal CT-images in a patient with bilateral parasagittal hemorrhage due to thrombosis of the superior sagittal sinus.
The red arrow on the contrast enhanced image indicates the filling defect caused by the thrombus.

If CVT is suspected clinically, imaging is the most important test for diagnosis. Clinical suspension normally falls into symptoms of increased intracranial pressure or focal neurological symptoms due to injury. Although, most patients present with a combination of both. The next question will be about the appropriate modality to use as a first-line method. Computed tomography is widely used in most health institutes in Europe as first-line imaging modality in neuroimaging. Indications vary depending on the institute and include headache, focal neurological symptoms and acute onset personality change. CT without contrast is often with findings suggestive of CVT in only 30% of CVT cases. [37,38,40]
In a plane CT without contrast, only one third of cases present a homogeneous hyperdense cortical veins or cortical sinus as the initial sign. [39]
The filled or dense delta sign is seen in the thrombosis of the superior sagittal sinus.

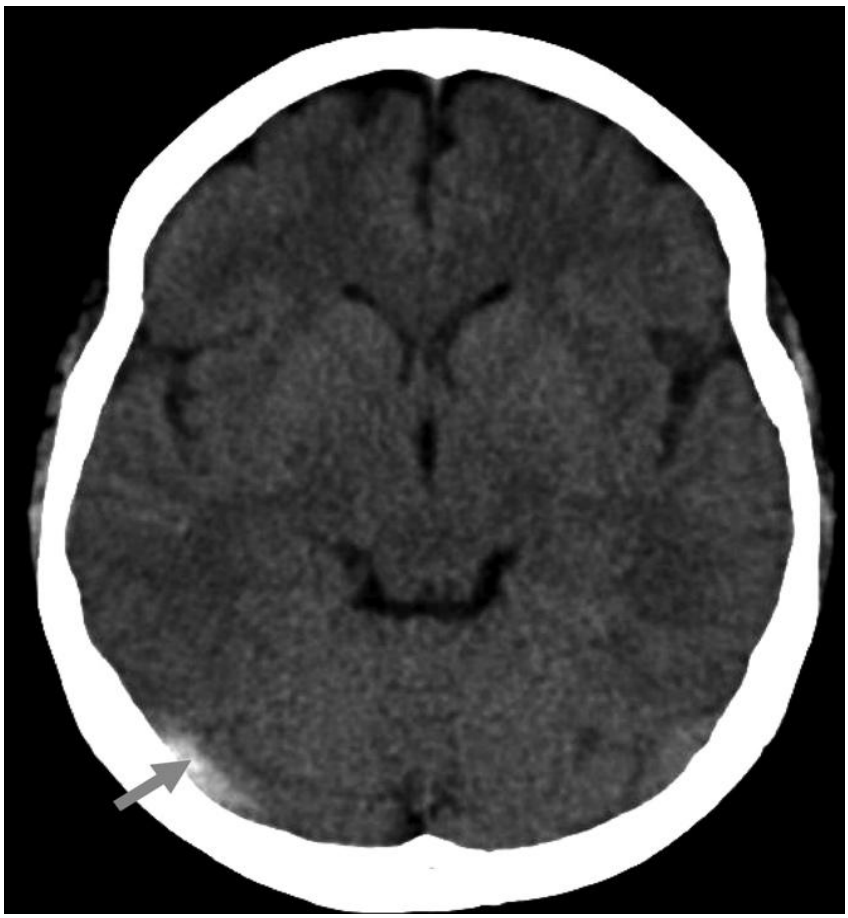


Figure (6) Noncontrast computed tomography head scan showed spontaneous hyperdensity of right transverse sinus. [70]

MRI is known to be more sensitive than CT in CVT detection. Signs like hyperintense vein sign is one of the findings. The intensity of the thrombi might vary and is time dependent. The older the more intense. During the first week, thrombi appear isointense to the surrounding brain tissue on T1-weighted imaging and hypointense on T2-weighted imaging. Thereafter, the thrombi will appear more hyperintense on both T1 and T2-weighted imaging. [28]

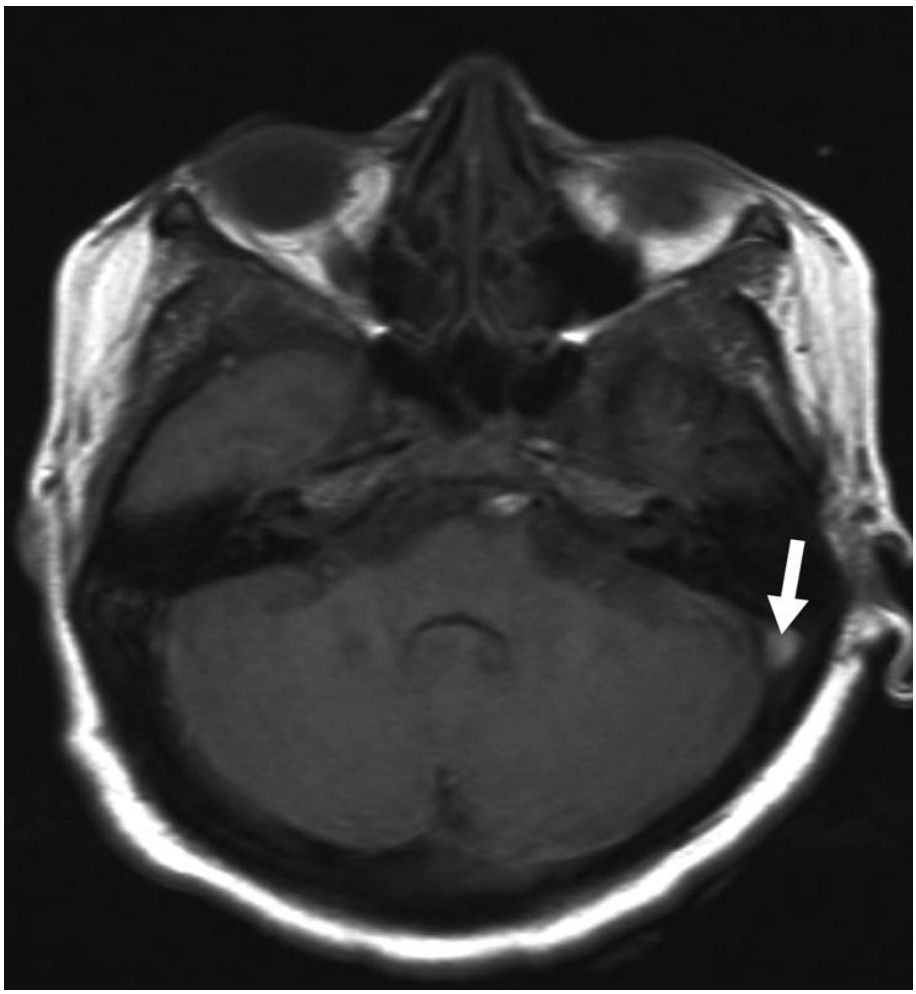


Figure (7) Flair magnetic resonance image showing hypersensitivity signal at left sigmoid sinus (arrow). [70]

In a study aimed to update the 2017 Grading of Recommendations, Assessment, Development, and Evaluation (GRADE) questions published by the European Stroke Organization (ESO) guideline done in 2024, computed tomography and magnetic resonance imaging, both with venous angiography were considered equivalent. MRI is considered superior to CT due to the lack of radiation exposure; MRI shall be preferred in younger patients during pregnancy. [32]

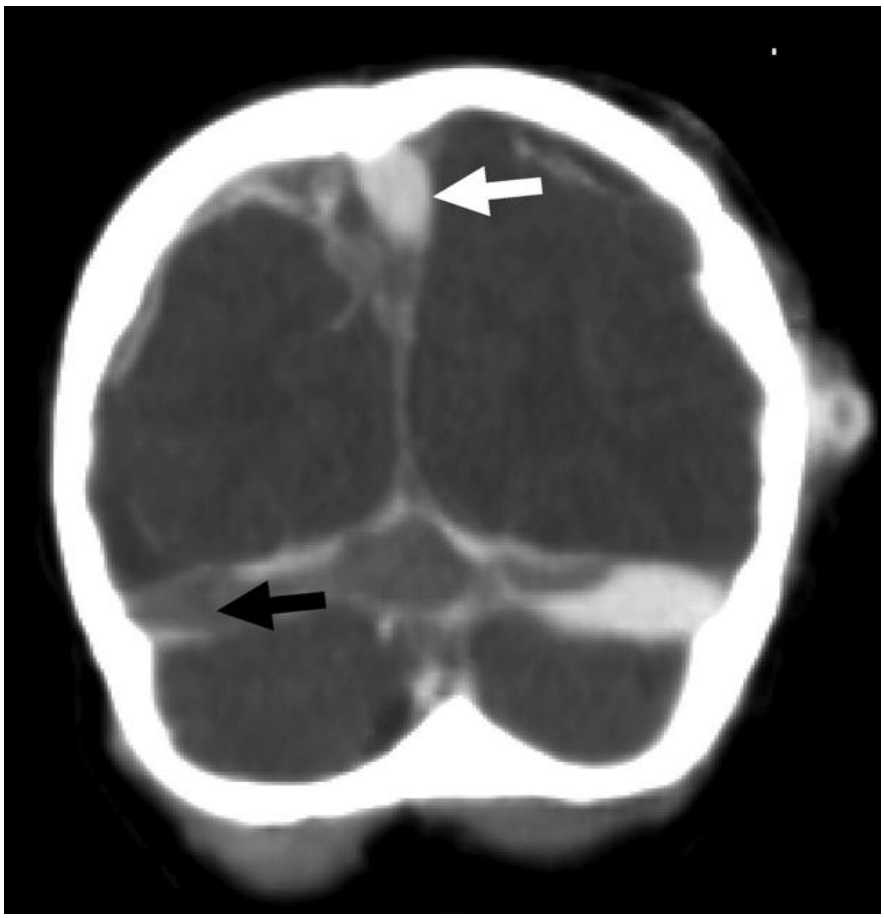


Figure (8) Computed tomographic venogram showing mixed density within venous sinuses (high-density contrast in patent segments (white arrow) and low density (black arrow) in nonperfusing thrombosed segments). [70]

Invasive diagnostic measures, namely, cerebral angiography less commonly used to establish the diagnosis. Findings vary depending on the site of occlusion. Failure of appearance of the whole or part of the sinus due to occlusion, enlarged veins due to collateral drainage and reversed flow are signs of CVT. Anatomic variants such as atresia or hypoplasia of the cerebral veins along with artifacts might influence the accuracy on MRV and CTA and could be better visualized with invasive methods. [42]

Injecting contrast directly into the cerebral veins or sinuses using a microcatheter, direct cerebral venography, facilitates interventions and the visualization of the intraluminal thrombi as a filling defect or as a complete occlusion. The latter is seen as a cupping appearance within the sinus. [43]

It is no longer recommended to measure D-dimer to rule out CVT. It could on the other hand be used only in selected cases with low clinical probability with isolated headache, lack of neurological findings, and symptoms duration of less than 30 days; negative d-dimer has a significantly reliable negative predictive value. [32]

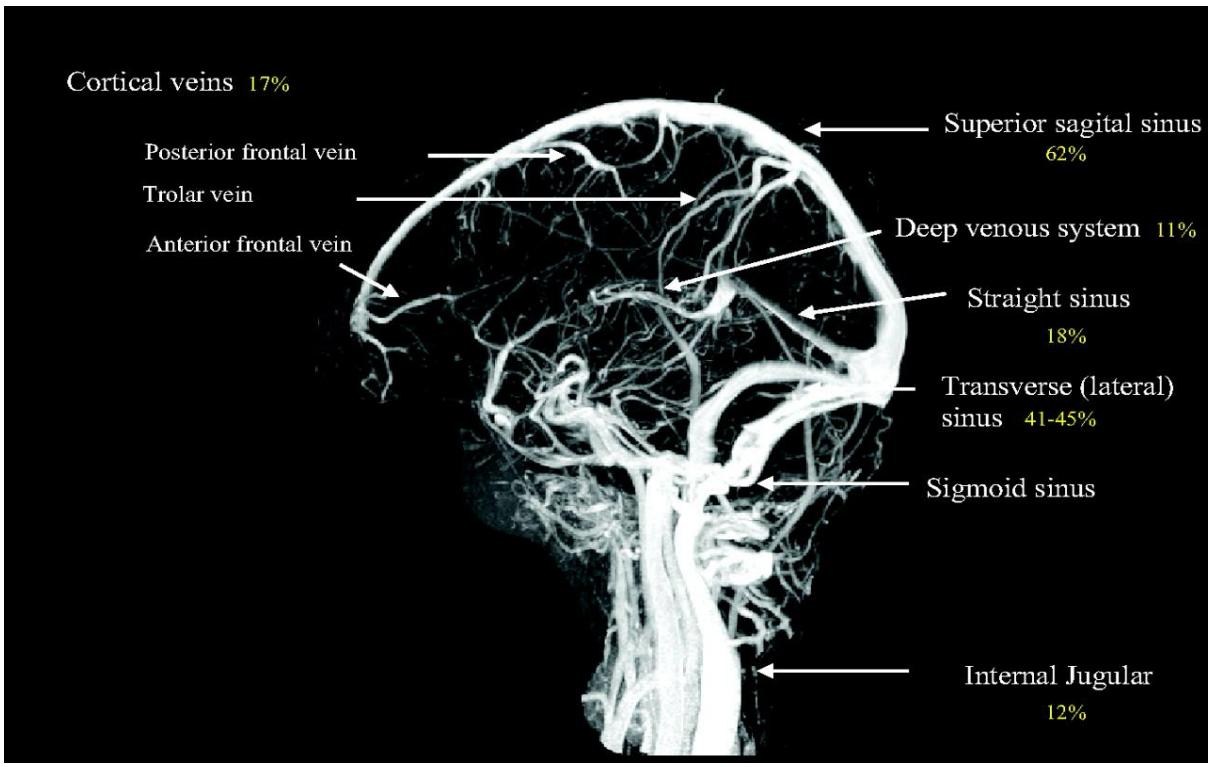


Figure (9) Magnetic resonance venogram showing the cerebral venous system and most frequent (%) location of cerebral venous and sinus thrombosis, as reported in the International Study on Cerebral Venous and Dural Sinuses Thrombosis (n=624). [70]

Laboratory tests are included in the initial assessment strategy of patients with headache or patients visiting the emergency department. Depending on the institute's recommendation, different blood test panels will be drawn. That might include a complete blood count, CRP, and chemistry panel. According to the latest recommendations (Class I, Level of Evidence C) from the American Heart Association and American Stroke Association CBC, chemistry panel, prothrombin time and activated partial thromboplastin time should be performed. The latter-mentioned associations recommend screening for potential prothrombotic conditions (Class I, Level of Evidence C).

Considering headache is the most frequent symptom [19], clinicians might suggest a lumbar puncture to rule out other conditions like subarachnoid haemorrhage, since 30% to 40% OF CVT present with ICH. [35]

Whereas Subarachnoid haemorrhage was found in only 0.5% to 0.8% of patients with CVT. [41].

A typical finding is the presence of erythrocytes if done within the right time frame after the onset of symptoms. [25]

13.1 Confirming the diagnosis

CT venography and MR venography are the definitive examinations to confirm the diagnosis of cerebral venous thrombosis. [34]

Digital subtraction angiography is generally the modality of choice only when invasive interventions are indicated. CT venography facilitates the precise visualisation of both the superficial and deep cerebral venous systems. Thrombi manifest as filling deficiencies, known as the "empty delta sign" in the superior sagittal sinus, and can typically be easily differentiated from arachnoid granulations. Numerous small to medium-sized studies have shown a high sensitivity and specificity of CTV in comparison to digital subtraction angiography or a consensus evaluation of alternative imaging modalities. In comparison to MRI, CTV exhibits less sensitivity for cortical vein thrombosis.

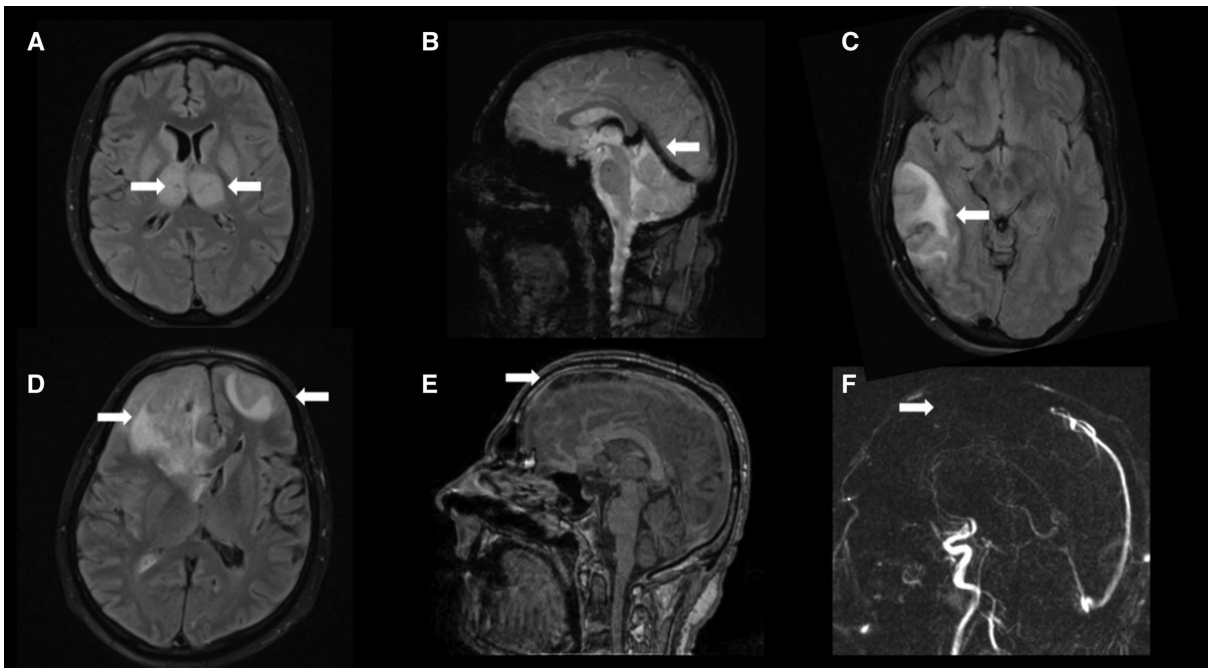


Figure (10) Typical findings of cerebral venous thrombosis on magnetic resonance imaging. A, Bilateral thalamic hyperintensity (arrows) on fluid-attenuated inversion recovery (FLAIR) in a patient with deep cerebral vein thrombosis. B, Susceptibility-weighted imaging shows hypo-intensity of the straight sinus (arrow), vein of Galen, and internal cerebral veins. C, Venous infarction due to transverse sinus thrombosis with heterogeneous FLAIR hyperintensity (arrow). D, Bilateral FLAIR hyperintensities (arrows) with mass effect in a patient with superior sagittal sinus thrombosis (arrow), shown in E on a contrast-enhanced T1 sequence and in F absent venous filling defect (arrow) with phase-contrast magnetic resonance venography.

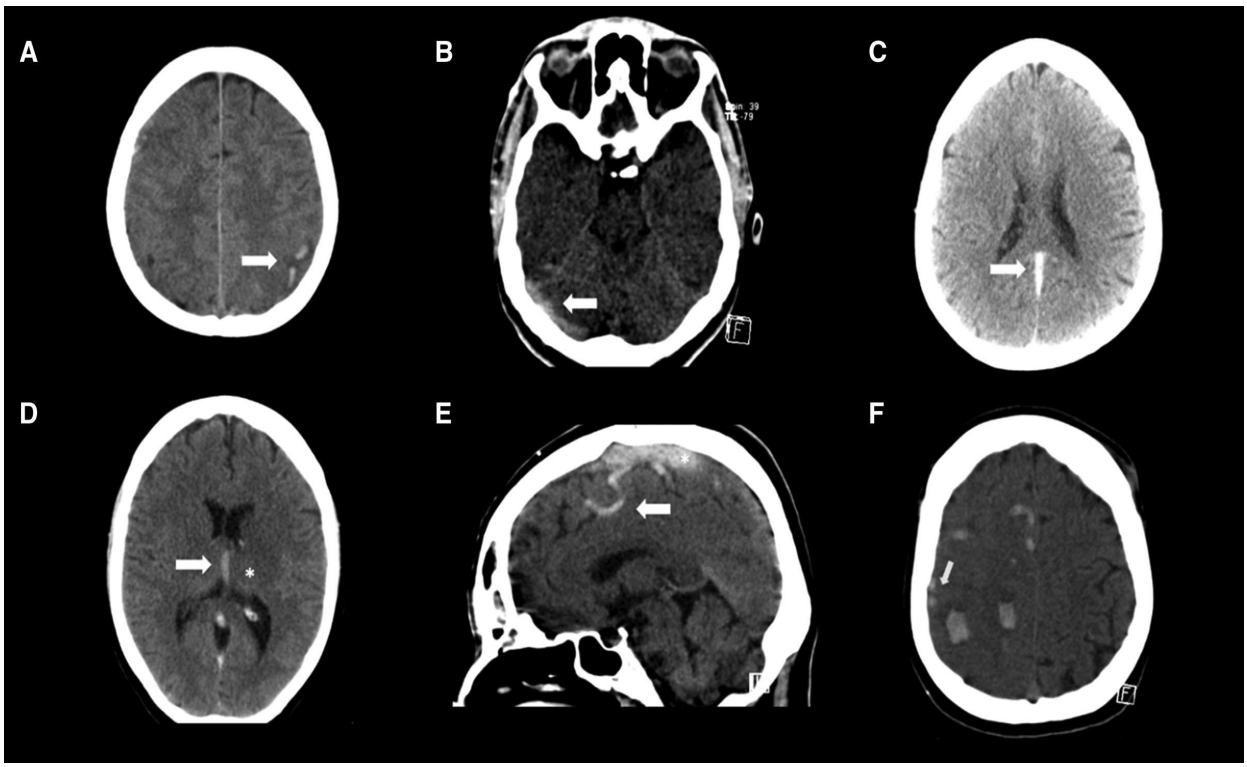


Figure (11) Typical findings of cerebral venous thrombosis on noncontrast computed tomography. A, Left-sided juxtacortical C-shaped haemorrhages. B, Transverse sinus thrombosis. C, Straight sinus thrombosis. D, Internal cerebral vein thrombosis (arrow) and left thalamic hypodensity (*). E, Cord sign (arrow) and hyperdense sagittal sinus thrombosis (*). F, Multiple small haemorrhages in same patient as in E. Arrows indicate cord sign.

14. Treatment

As discussed earlier, CVT is an uncommon but potentially life-threatening cause of stroke. therefore, admitting patients to the stroke unit if available is advised as it reduces mortality and morbidity. [45]

Transferring the patient to a stroke centre is recommended in case of an unavailable stroke unit and/or or worsening symptoms. According to the Stroke Unit Trialists Collaboration, the most important components in a stroke unit are physiotherapy, occupational therapy, and nursing care directed to stroke care. Upon admission, in addition to the above-mentioned components, a neurology consult is recommended to assess the severity and potentially identify the sequel. Other specialties are often involved in optimizing the risk factors and other healthcare issues. [66]

Treatment aims to prevent thrombus formation, restore drainage, and prevent damage to brain tissue. In the acute phase, despite the concomitant ICH, anticoagulants remain the first-line treatment. [48]

Anticoagulant therapy is divided into 3 phases. The initial phase is usually initiated with parenteral anticoagulation. Thereafter, the maintenance phase with oral anticoagulants followed by the last phase, where anticoagulation is either discontinued or in special cases where the risk of recurrence is increased, continued as prophylaxis. [47]

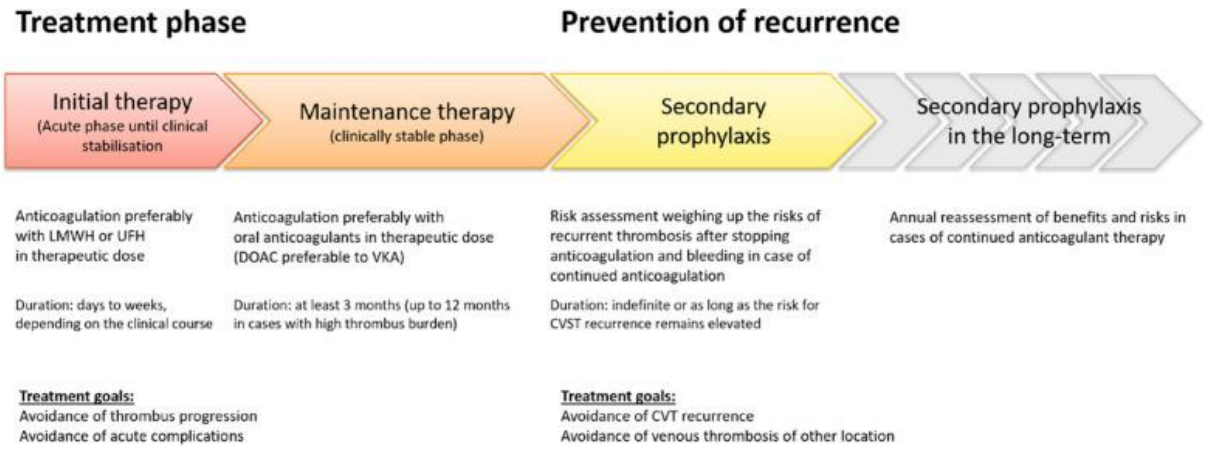


Figure (12) Treatment Phases [49]

Based on the meta-analysis, European guidelines present weak recommendation for UFH over LMWH since there is no significant trend to improve outcomes and mortality without a change in bleeding rates using UFH. [67,50].

The acute phase of CVT usually end after the observation and clinical stabilization. Thereafter, the focus shifts towards switching to oral anticoagulants, namely DOAC and vitamin K antagonists (VKA). Several studies show that DOACs are superior to VKA. The risk ratio (RR) that was calculated for any VTE complication was 0.17 (95% CI 0.02-1.71), and for the recurrence of CVT, it was 0.37 (0.03-4.3), favouring DOACs. [52]

The duration of anticoagulation should not be less than 3 months and should not exceed the maintenance phase of 3 to 12 months with the aim of achieving further recanalization or an improvement in the clinical outcome of the index event. It is also of importance to mention in case of recurrent CVT or extra cerebral VTE, anticoagulation should be continued as a secondary prophylaxis. [1,46]

In the (TO-ACT) randomized trial in patients with one of the following risk factors: qualitative or quantitative impairment of consciousness, parenchymal haemorrhage, or deep cerebral vein thrombosis that were considered as high-risk patients, no evidence of endovascular thrombolysis with or without thrombectomy versus therapeutic anticoagulants were shown. [51]

Decompressive craniectomy for CVT should be administered to patients with acute severe cerebral venous thrombosis and parenchymal lesions at risk of herniation as a critical therapeutic intervention. Factors linked to adverse outcomes included age greater than 50 years, midline shift exceeding 10 mm, and complete effacement of basal cisterns. [53, 45]

15. Prognosis

The Modified Rankin Scale (mRS) is used to assess the degree of disability or dependence in daily activities.

1. Score 0: No symptoms at all.
2. Score 1: No significant disability despite symptoms; able to carry out all usual duties.
3. Score 2: Slight disability; unable to carry out all previous activities but can manage own affairs.
4. Score 3: Moderate disability; requires some help but can walk without assistance.
5. Score 4: Moderately severe disability; unable to walk or attend to bodily needs without assistance.
6. Score 5: Severe disability; bedridden, incontinent, and requires constant care.
7. Score 6: Death. [55]

The VENOST study done on 1,144 patients with CVST shows a favourable prognosis. Using the Modified Rankin Scale (mRS) to assess functional outcomes, 78.4% show an mRS of 0/1, 11.7% have an mRS of 2, and 10% have an mRS of 3/5. Another study, namely, the ISCVST study, shows that 78.1% recovered completely, 8% had partial recovery, and 14% had functional disability or died. [20]

Other scores, like the National Institutes of Health Stroke Scale (NIHSS) and Glasgow Coma Scale (GCS), are frequently used to assess the severity of the neurological deficit. However, they are not applicable for various clinical situations and not specific for CVT. In a study done by Min Li et al. in October 2023 on the development and validation of a clinical-based severity scale for patients with CVT. A CVT severity scale was successfully established after recruiting CVT patients confirmed by MRV, CTV, and magnetic resonance black/blood imaging and digital subtraction angiography from 26 tertiary hospitals in China Mainland from the period of January 2021 to May 2022. Items 1–11 were directly inherited from NIHSS. Items 12–18 were new items.

1A	Level of Consciousness	0 = Alert; keenly responsive. 1 = Not alert, but arousable by minor stimulation to obey, answer or respond. 2 = Not alert, requires repeated stimulation to attend, or is obtunded and requires strong or painful stimulation to make movements. 3 = Responds only with reflex motor or autonomic effects or totally unresponsive, flaccid, areflexic.
1B	Level of Consciousness Questions	0 = Answer both questions correctly. 1 = Answer one question correctly. 2 = Answer neither question correctly.
1C	Level of Consciousness Commands	0 = Perform both tasks correctly. 1 = Perform one task correctly. 2 = Perform neither task correctly.
2	Best Gaze	0 = Normal. 1 = Partial gaze palsy. 2 = Forced deviation, total gaze paresis not overcome by the oculocephalic manoeuvre.
3	Visual Field	0 = No visual loss. 1 = Partial hemianopia. 2 = Complete hemianopia. 3 = Bilateral hemianopia, blindness.
4	Face Palsy	0 = Normal symmetrical movement. 1 = minor paralysis; flattened nasolabial fold, asymmetry on smiling. 2 = partial paralysis; total or near total paralysis of lower face. 3 = complete paralysis of one or both sides; absence of facial movement in the upper and lower face.
5A	Motor Left Arm	0 = No drift; limb holds 90 (or 45) degrees for full 10 seconds. 1 = Drift; limb holds 90 (or 45) degrees, but drifts down before full 10 seconds; does not hit bed or other support. 2 = Some effort against gravity; limb cannot get to or maintain 90 (or 45) degrees, drifts down to bed, but has some effort against gravity.

		3 = No effort against gravity; limb falls. 4 = No movement.
5B	Motor Right Arm	0 = No drift; limb holds 90 (or 45) degrees for full 10 seconds. 1 = Drift; limb holds 90 (or 45) degrees, but drifts down before full 10 seconds; does not hit bed or other support. 2 = Some effort against gravity; limb cannot get to or maintain 90 (or 45) degrees, drifts down to bed, but has some effort against gravity. 3 = No effort against gravity; limb falls. 4 = No movement.
6A	Motor Left Leg	0 = No drift; leg holds 30 degrees for full 5 seconds. 1 = Drift; leg falls by the end of the 5 second period, but does not hit bed. 2 = Some effort against gravity; leg falls to bed by 5 seconds, but has some effort against gravity. 3 = No effort against gravity; leg falls to bed immediately. 4 = No movement.
6B	Motor Right Leg	0 = No drift; leg holds 30 degrees for full 5 seconds. 1 = Drift; leg falls by the end of the 5 second period, but does not hit bed. 2 = Some effort against gravity; leg falls to bed by 5 seconds, but has some effort against gravity. 3 = No effort against gravity; leg falls to bed immediately. 4 = No movement.
7	Limb Ataxia	0 = Absent. 1 = Present in one limb. 2 = Present in two limbs.
8	Sensory	0 = Normal; no sensory loss. 1 = Mild to moderate sensory loss; patient feels pinprick is less sharp or dull on the affected side, or there is a loss of superficial pain with pinprick but patient is aware of being touched. 2 = Severe or total sensory loss; patient is not aware of being touched in the face, arm and leg.

9	Best Language	0 = Normal; no aphasia. 1 = Mild to moderate aphasia; some obvious loss of fluency or facility of comprehension without significant limitation on ideas expressed or form of expression, reduction of speech and/or comprehension makes conversation about provided material difficult or impossible. 2 = Severe aphasia; all communication is through fragmentary expression, great need for inference, questioning and guessing by the listener, range of information that can be exchanged is limited, listeners carries burden of communication, examiner cannot identify materials provided from patient response. 3 = Mute or global aphasia; no usable speech or auditory comprehension.
10	Dysarthria	0 = Normal; no dysarthria. 1 = Mild to moderate dysarthria; patient slurs at least some words and at worst can be understood with some difficulty. 2 = Severe dysarthria; patient's speech is so blurred as to be unintelligible in the absence of or out of proportion to any dysphasia or is mute.
11	Extinction and Inattention	0 = Normal. 1 = Visual, tactile, auditory, spatial or personal inattention or extinction to bilateral simultaneous stimulation in one of the sensory modalities. 2 = Profound hemi-inattention or hemi-inattention to more than one modality, does not recognize own hand or orients to only one side of space.
12	Epilepsy	0 = Normal. 1 = Focal epilepsy. 2 = Generalized epilepsy. 3 = Status epilepticus.
13	Headache	0 = Normal. 1 = Mild; intermittent, bearable. 2 = Moderate headache; persistent, bearable, concomitant with nausea and/or vomiting.

		3 = Severe headache; persistent, unbearable, concomitant with nausea and/or projectile vomiting.
14	Tinnitus	0 = Normal. 1 = Mild tinnitus; intermittent. 2 = Moderate tinnitus; persistent, can only be heard in quiet environment. 3 = Severe tinnitus; persistent, can be heard in noisy environment, concomitant with vertigo or hearing loss.
15	Papilledema	0 = Normal. 1 = C-shaped halo with a temporal gap. 2 = Circumferential halo. 3 = Loss of major vessels on the optic disc. 4 = Obscuration of all vessels on the optic disc.
16	Ophthalmoplegia	0 = Normal. 1 = Unilateral ophthalmoplegia. 2 = Bilateral ophthalmoplegia.
17	Mental disorders	0 = Normal. 1 = mild; can be persuaded. 2 = severe; cannot be persuaded.
18	Neck discomfort	0 = Normal. 1 = Pain, but no stiff neck. 2 = Stiff neck.

[56]

In another study by Zhongao W. et al. to estimate the thrombus burden and predict the severity of the intracranial hypertension in patients with CVST, a total of 87 CVST confirmed patients by contrast MRV or CTV. Thereafter, using magnetic resonance black-blood thrombus imaging (MRBTI), the cerebral venous sinuses were divided into 15 segments. The superior sagittal sinus was divided into 3 segments, while the unilateral transverse sinus and unilateral sigmoid sinus were divided into 2 segments. Other sinuses and veins were just continued as one segment, namely, the straight sinus, the torcular herophili, and the unilateral internal jugular vein. [57]

The scoring rules are identified as follows:

- no thrombus was counted as zero points
- thrombus filling ≤50% of the segment as one point

- thrombus filling >50% but <100% of the segment as two points
- thrombus filling 100% of the segment as three points.

Calculated for each segment. Meanwhile, the calculation of the weight drainage of each side of the common unilateral dominance of the transverse sinus was done manually. Calculation formulas were used to calculate the weighted drainage of the left and right transverse sinuses, the weighted score on the left side of cerebral venous sinuses, followed by the same for the other side, and finally the total thrombosis score. Scores of 0 to 30 were assigned to the minimum and maximus, respectively, where the higher the score, the higher the burden. The novel CVST-score was assessed then against the two most frequently used methods in clinical research. The first method, the cerebral venous extent score (CVES), is composed of the number and location of the involved sinuses. The sinuses were divided as above and received 1 point for a thrombus and 0 point if no thrombus was present. The second method, the cerebral venous occlusion score (CVOS), was based on whether the sinuses were fully occluded. [56] [58] [59]

The imaging parameters were described by Yang Q, Duan J, Fan Z, Qu X, Xie Y, Nguyen C et al. in 2016 and were obtained by using a 3.0 T MRI system (MAGNETOM Verio, Siemens Healthcare, Erlangen, Germany) with a standard 32-channel head coil for signal reception. [60]

Based on the study structure above, 119 patients were included individually, and after initial assessment, 32 patients were excluded due to reasons like failure to get CSF opening pressure, lumbar puncture, or contraindication to lumbar puncture due to reasons like brain herniation. Other patients had non-thrombotic CVSS due to giant arachnoid granulations, and one patient had giant meningioma affecting the ICP. The patients were grouped based on their ICP. The final analysis was done to investigate the ability of the novel CVST score to non-invasively predict the ICP. It was shown that there is a potential correlation between CVST-Score and ICP as well as better diagnostic performance for intracranial hypertension than other methods. [61]

16. Case Presentation

A male patient born 1959 with arthrosis in the hip joint (surgical intervention is planned), He underwent surgical evacuation of a subdural haemorrhage in 2013 in Republican Hospital of Lithuania, but no documentation could be found. The patient mentioned that there was no paralysis involved and the reason he was hospitalized was due to lethargy, drowsiness and

impaired consciousness. Arrives in the department of neurology at the Vilnius University Santaros Clinic. Complaining of mild left sided headache. On the 3rd of august 2014, he noticed some vision problems but did not consult a doctor. He mentioned that while driving, his wife noticed that he was deviating to the right and even had a small accident. Around the 3 rd. of august, he began to have difficulty walking, but did not understand the essence of the illness. When he did not improve, he was referred for treatment by his family doctor.

On status, the patient is oriented to all parameters, alert and communicative. Tongue tilted to the right. Pronounced homonymous hemianopsia, left sided. No sensory deficits are observed. No paresis. No gait disturbances. Blood pressure 130/80 mmHg. Heart rate 60 bpm. Regular rhythm.

After assessing the patient initially and due to the focal neurological deficit, sudden onset, not improving on its own, acute brain circulatory disorder, left hemianopsia, damage to the cranial nerves VII, XII on the right diagnosis was made. Suspension of haemorrhage after hospitalisation in 2013. Further investigation with CT scan of the brain was ordered urgently to differentiate ischemia or haemorrhage. Stroke work up including lipid profile, glucose, electrolytes, ECG and Ultrasound of the Carotids were also obtained.

On the 11th of august 2014 a head CT without contrast was conclusive of the following findings:

- High-density inhomogeneous masses with oedema and haemorrhage (~ 16x12x40mm, 150ml) are observed on the right occipital lobe. The occipital horn of the right lateral ventricle is displaced. Subarachnoid spaces are compressed.
- The middle structures are displaced to the left (~0.5cm). Frontal, sphenoidal sinuses, mastoid cells are normal.
- Condition after trephination.
- Ultrasound of the Carotids shows Initial atherosclerotic changes in the carotid arteries. Blood flow in the vertebral arteries is in the normal direction, blood flow is sufficient.

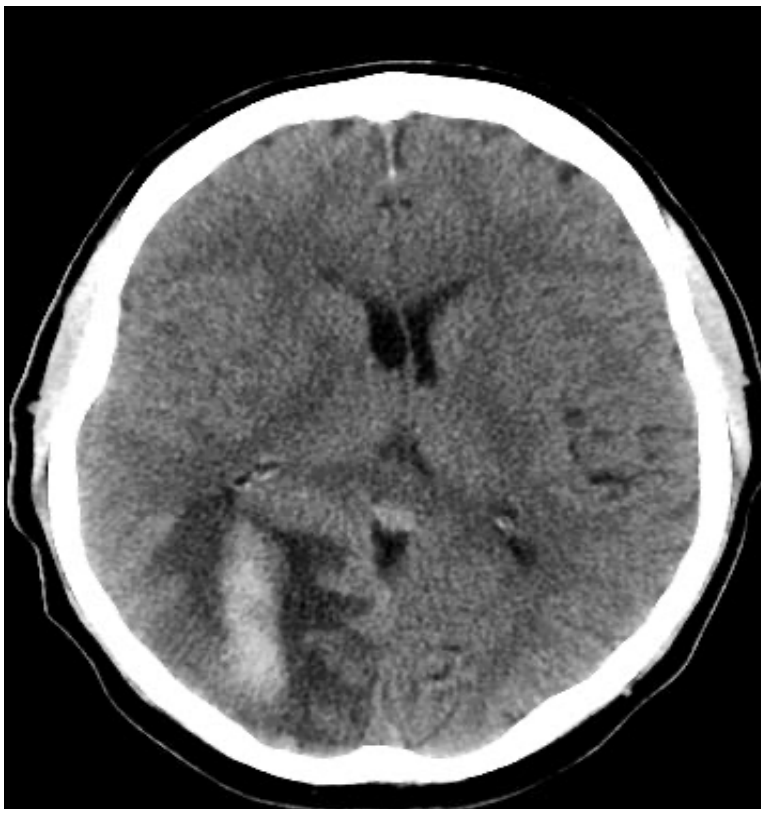


Figure (13) Head CT showing Intracerebral haemorrhage with pronounced perifocal oedema in the right lobe.

Due to pathological changes in the brain CT, an MRI scan was performed on the 13th of august, in T1, T2, SPIR, DWI sequences in the axial, sagittal and coronary planes showing:

- Intracerebral resorbing haemorrhage in the right occipital lobe, local perifocal oedema.
- Signs of mild vascular encephalopathy. Intracranial arteries within normal limits.
- Venous sinus thrombosis - a picture of chronic and acute thrombosis with signs of revascularization.
- Congenital anatomical variant of the venous sinuses - hypoplasia on the left.

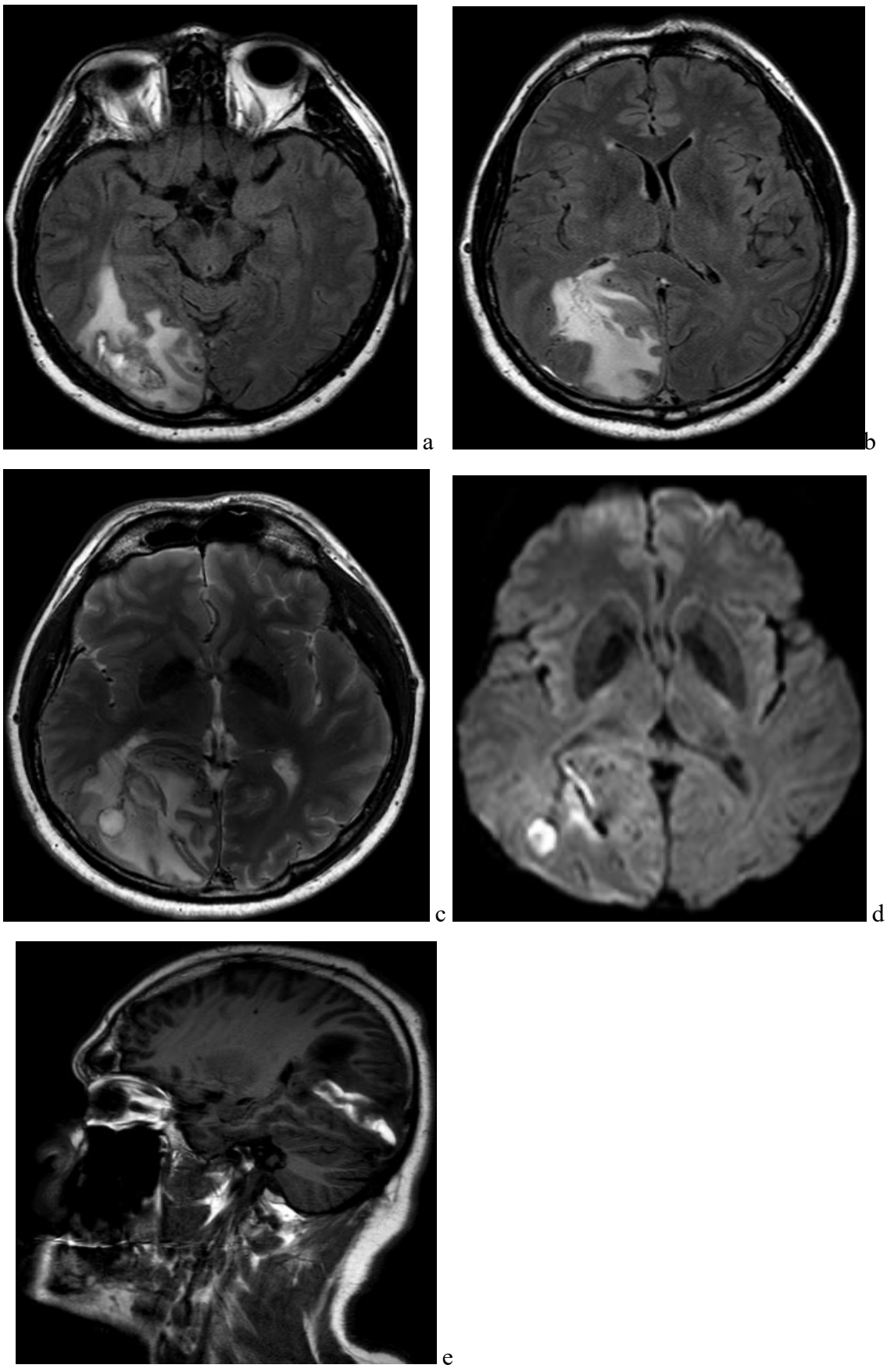
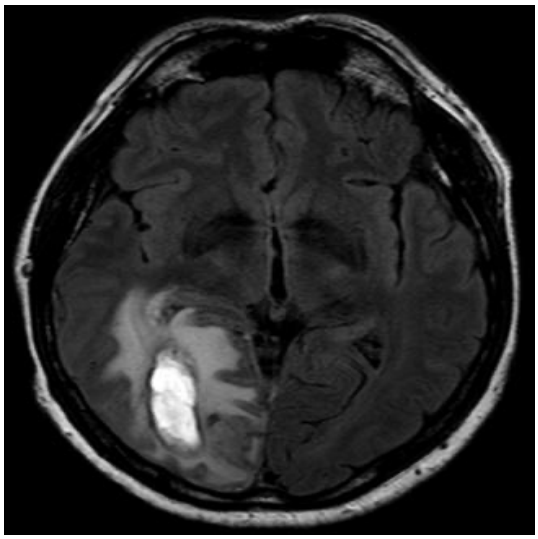
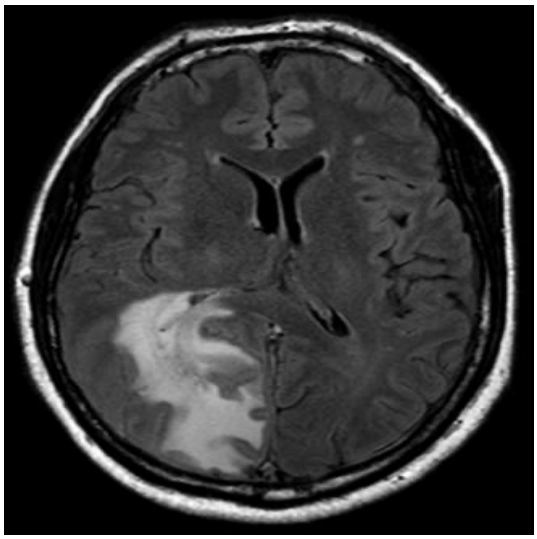


Figure (14) MRI of the Brain. Axial plane (a,b,c,d), Sagittal plane (e)

On the 22nd of august a follow up MRI scan of the brain was performed showing:

- Intracerebral haemorrhage of the right occipital lobe, resorption phase, pronounced perifocal oedema.
- Partial thrombosis of the venous sinuses, especially pronounced in the right transverse and spinous antrum.
- A mass in the lower part of the left cerebellopontine angle, possibly of venous origin - cavernoma, varicose veins?


a

b

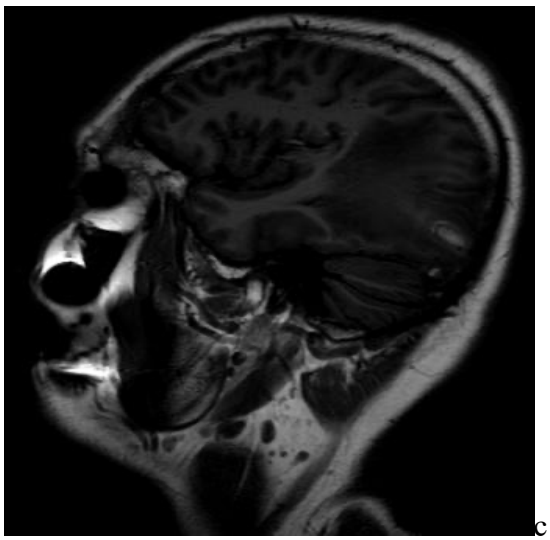

c

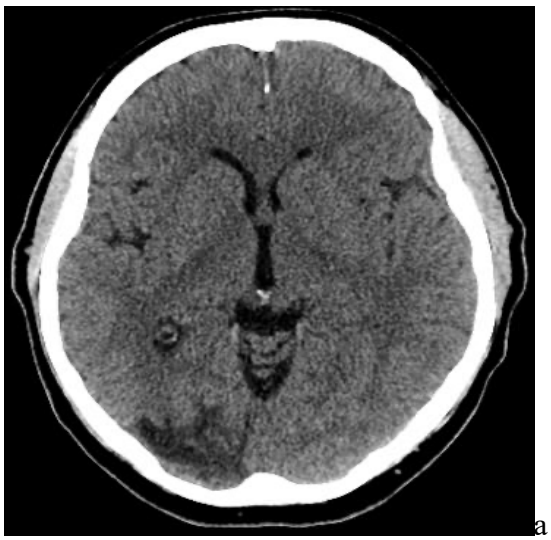
Figure (15) MRI of the Brain transverse plane (a,b). Coronal plane (c).

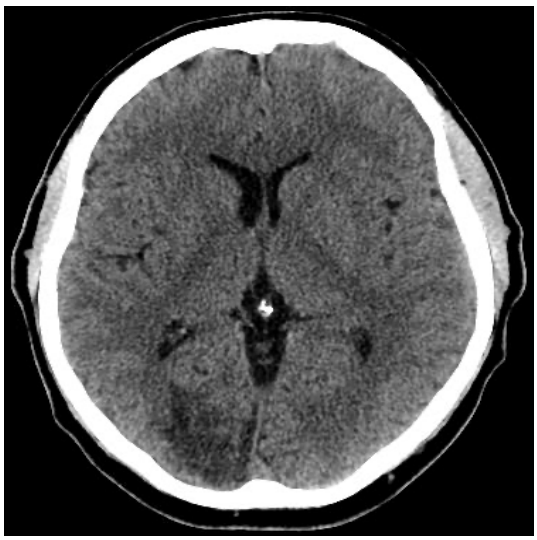
Due to the pathological findings mentioned above, possibly residual findings after the previous subdural hematoma. Thus, AVD fistula cannot be ruled out and regular follow up scans are necessary. During this period, the patient was treated with Warfarin (5/7.5 mg) after consulting the haematology department. Rehabilitation medicine was also consulted regarding

the inpatient rehabilitation plan and was assessed using Barthel Index (80 points at discharge). The patient was discharged on the 11th of august 2014.

One month later, on the 22 of September, it was suspected that the patient may have thrombophilia. After obtaining all test required, the haematologist recommended further testing as well as stopping the Warfarin treatment. On the 17th of November, the patient was hospitalised. Upon admission the patient was conscious, communicative, oriented. Moderate peripheral paresis of the facial nerve on the right. CT scan (a, b, c) and angiogram (d) shown below are consistent with the following:

- Thrombosis of the superior sagittal, right and left transverse sinuses, thrombosis of the left transverse sinus may be suspected.
- Arterial aneurysms and dural arteriovenous fistula (dAVF) are not visible.
- In the temporal/occipital lobe of the right cerebral hemisphere, a wide area of encephalomalacia after previous ICA and ischemia.
- Cerebral angiogram confirmed obstruction of the right sigmoid sinus and jugular vein at the level of the jugular foramen with visible flow distally.


a


b

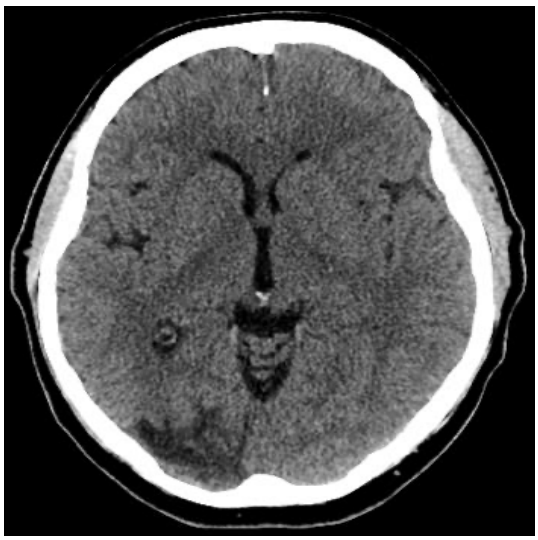
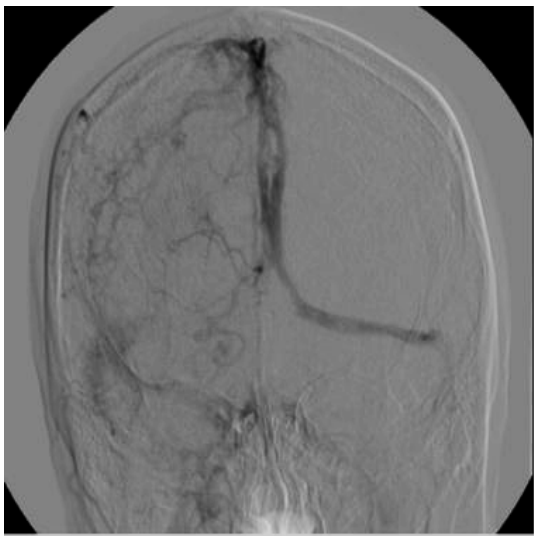

c

d

Figure (16) CT scan (a,b,c) angiogram (d)

On the 29th of august 2018, controll visit without any complaints, no thrombotic events and normal control tests except for factor VIII that showed increased activity dynamics by 321% which is generally associated with a higher risk of thrombosis. When planning surgical interventions (especially orthopedic), in case of prolonged immobilization a longer and more aggressive treatment with low molecular weight heparin (LMWH). An increase in FVIII activity is associated with an increased risk of thrombosis. It is appropriate to monitor the change in D-dimers in dynamics. If it increases, consider renewing anticoagulants. In case of rethrombosis, long-term, lifelong anticoagulant administration is recommended.
Last imaging of the brain was performed in may 2017 showing no acute pathological focal density changes or signs of intracranial hemorrhage are visible in the brain and Right encephalomalacic changes after a previous venous cerebral infarction. As visible in figure 17 (a,b,c).

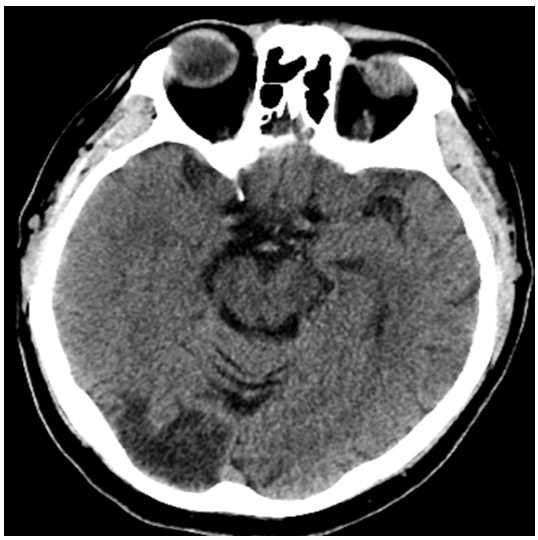
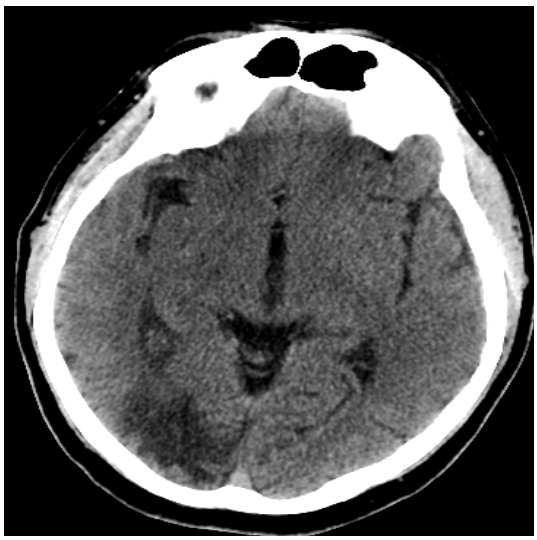
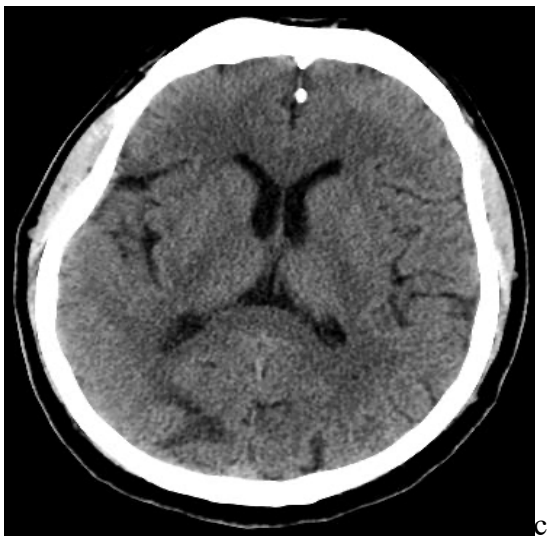

a

b

c

Figure (17) CT scan of the brain.

17. Conclusion

Cerebral venous thrombosis (CVT) is the formation of a thrombus in the cerebral venous system resulting in impaired venous drainage. Arterial obstruction or partial occlusion being the most common type of stroke, CVT is considered a less common type of stroke. Epidemiologically, CVT is more common in women than in men thus new studies suggest a shift in that matter. Symptoms may vary and are unspecific. Considering headache is the most common symptom, other neurological deficits may be present with a wide variety depending on the site of the thrombus/ thrombi. The initial assessment of the patient depends on the severity of the symptoms, clinical setting as well as the time frame of symptom debut. Computed tomography venography (CTV) is often the first choice but other imaging modalities like magnetic resonance venography (MRV) are of similar or higher diagnostic value. Invasive modalities like cerebral venous angiogram might be considered if an intervention is planned. Early detection and treatment including rehabilitation, risk factor identification and control influence the outcome positively together with other specialties consultation, regular follow ups aiming to assess the patients function, further optimising the risk factors and treatment as needed.

18. Reference:

1. Weimar C, Beyer-Westendorf J, Bohmann FO, Hahn G, Halimeh S, Holzhauer S, Kalka C, Knoflach M, Koennecke HC, Masuhr F, Mono ML, Nowak-Göttl U, Scherret E, Schlamann M, Linnemann B. New recommendations on cerebral venous and dural sinus thrombosis from the German consensus-based (S2k) guideline. Neurol Res Pract. 2024 Apr 19;6(1):23. doi: 10.1186/s42466-024-00320-9. PMID: 38637841; PMCID: PMC11027218.
2. Bayot ML, Reddy V, Zabel MK. Neuroanatomy, Dural Venous Sinuses. [Updated 2023 Aug 8]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK482257/
3. **Novel insights into cerebral venous thrombosis book page 8**
4. Diagnostic imaging: brain. 4th ed. Miral D. Jhaveri, MD, MBA; 2021.
5. Sobotta Anatomy Textbook. Elsevier Health Sciences; 2018. (6) Diagnostic Imaging: Brain. 4th ed. 2020.
6. Neuroradiology- Images vs Symptomes. Martina Špero, Hrvoje Vavro; 2006.
7. Otite FO, Patel S, Sharma R, Khandwala P, Desai D, Latorre JG, Akano EO, Anikpezie N, Izzy S, Malik AM, Yavagal D, Khandelwal P, Chaturvedi S. Trends in incidence and epidemiologic characteristics of cerebral venous thrombosis in the United States. Neurology. 2020 Oct 20;95(16):e2200-e2213. doi: 10.1212/WNL.0000000000010598. Epub 2020 Aug 26. PMID: 32847952; PMCID: PMC7713788.
8. Thrombotic thrombocytopenia after CHADOx1 NCOV-19 vaccination. The New England Journal of Medicine. 2021 Apr 9; (10) Scully M, Singh D, Lown R, Poles A, Solomon T, Levi M, Goldblatt D, Kotoucek P, Thomas W, Lester W. Pathologic Antibodies to Platelet Factor 4 after ChAdOx1 nCoV-19 Vaccination. N Engl J Med. 2021 Jun 10;384(23):2202-2211. doi: 10.1056/NEJMoa2105385. Epub 2021 Apr 16. PMID: 33861525; PMCID: PMC8112532.
9. Schultz NH, Sørvoll IH, Michelsen AE, Munthe LA, Lund-Johansen F, Ahlen MT, Wiedmann M, Aamodt AH, Skattør TH, Tjønnfjord GE, Holme PA. Thrombosis and Thrombocytopenia after ChAdOx1 nCoV-19 Vaccination. N Engl J Med. 2021 Jun 3;384(22):2124-2130. doi: 10.1056/NEJMoa2104882. Epub 2021 Apr 9. PMID: 33835768; PMCID: PMC8112568.
10. Scully M, Singh D, Lown R, Poles A, Solomon T, Levi M, Goldblatt D, Kotoucek P, Thomas W, Lester W. Pathologic Antibodies to Platelet Factor 4 after ChAdOx1 nCoV-19 Vaccination. N Engl J Med. 2021 Jun 10;384(23):2202-2211. doi: 10.1056/NEJMoa2105385. Epub 2021 Apr 16. PMID: 33861525; PMCID: PMC8112532.
11. Ken-Dror G, Cotlarciuc I, Martinelli I, Grandone E, Hiltunen S, Lindgren E, Margaglione M, Duchez VLC, Triquenot AB, Zedde M, et al. Genome-wide association study identifies first locus associated with susceptibility to cerebral venous thrombosis. *Ann Neurol*. 2021;90:777–788. doi: 10.1002/ana.26205
12. Ranjan R, Ken-Dror G, Martinelli I, Grandone E, Hiltunen S, Lindgren E, Margaglione M, Le Cam Duchez V, Bagan Triquenot A, Zedde M, Mancuso M, Ruigrok YM, Worrall B, Majersik JJ, Putaala J, Haapaniemi E, Zuurbier SM, Brouwer MC, Passamonti SM, Abbattista M, Bucciarelli P, Lemmens R, Pappalardo E, Costa P, Colombi M, Aguiar de Sousa D, Rodrigues S, Canhao P, Tkach A, Santacroce R,

Favuzzi G, Arauz A, Colaizzo D, Spengos K, Hodge A, Ditta R, Han TS, Pezzini A, Coutinho JM, Thijs V, Jood K, Tatlisumak T, Ferro JM, Sharma P. Age of onset of cerebral venous thrombosis: the BEAST study. Eur Stroke J. 2023 Mar;8(1):344-350. doi: 10.1177/23969873221148267. Epub 2023 Jan 6. PMID: 37021156; PMCID: PMC10069208.

13. Kalita, J., Misra, U.K. & Singh, R.K. Do the Risk Factors Determine the Severity and Outcome of Cerebral Venous Sinus Thrombosis?. *Transl. Stroke Res.* **9**, 575–581 (2018). https://doi.org/10.1007/s12975-017-0607-6
14. Weimar C, Beyer-Westendorf J, Bohmann FO, Hahn G, Halimeh S, Holzhauer S, Kalka C, Knoflach M, Koennecke HC, Masuhr F, Mono ML, Nowak-Göttl U, Scherret E, Schlamann M, Linnemann B. New recommendations on cerebral venous and dural sinus thrombosis from the German consensus-based (S2k) guideline. Neurol Res Pract. 2024 Apr 19;6(1):23. doi: 10.1186/s42466-024-00320-9. PMID: 38637841; PMCID: PMC11027218.
15. Stam, J. (2005) 'Thrombosis of the cerebral veins and sinuses', *New England Journal of Medicine*, 352(17), pp. 1791–1798. doi:10.1056/nejmra042354.
16. Tadi P, Behgam B, Baruffi S. Cerebral Venous Thrombosis. [Updated 2023 Jun 12]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK459315/
17. Miraclin TA, Prasad JD, Ninan GA, Gowri M, Bal D, Shaikh AIA, Benjamin RN, Prabhakar AT, Sivadasan A, Mathew V, et al. Cerebral venous sinus thrombosis: changing trends in the incidence, age and gender (findings from the CMC Vellore CVT registry). *Stroke Vasc Neurol*. 2023:svn-2023-002351. doi: 10.1136/svn-2023-002351
18. Ferro JM, Canhão 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;35:664–670. doi: 10.1161/01.STR.0000117571.76197.26
19. Otite FO, Patel S, Sharma R, Khandwala P, Desai D, Latorre JG, Akano EO, Anikpezie N, Izzy S, Malik AM, et al. Trends in incidence and epidemiologic characteristics of cerebral venous thrombosis in the United States. *Neurology*. 2020;95:e2200–e2213. doi: 10.1212/WNL.0000000000010598
20. Duman T, Uluduz D, Midi I, Bektas H, Kablan Y, Goksel BK, et al. A multicenter study of 1144 patients with cerebral venous thrombosis: the VENOST Study. J Stroke Cerebrovasc Dis. 2017;26:1848–1857. doi: 10.1016/j.jstrokecerebrovasdis.2017.04.020.
21. Ferro JM, Canhão P, Stam J, Bousser M-G, 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;35:664–670. doi: 10.1161/01.STR.0000117571.76197.26.
22. Ferro JM, Canhão P, Bousser M-G, Stam J, Barinagarrementeria F, ISCVT Investigators Cerebral vein and dural sinus thrombosis in elderly patients. Stroke. 2005;36:1927–1932.
23. Lindgren E, Silvis SM, Hiltunen S, Heldner MR, Serrano F, de Scisco M, et al. Acute symptomatic seizures in cerebral venous thrombosis. Neurology. 2020;95:e1706–e1715. doi: 10.1212/WNL.0000000000010577.

24. Gokey R, Das JM. Venous Sinus Thrombosis. [Updated 2023 Jul 31]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK560598/
25. Kevci R, Lewén A, Ronne-Engström E, Velle F, Enblad P, Svedung Wettervik T. Lumbar puncture-verified subarachnoid hemorrhage: bleeding sources, need of radiological examination, and functional recovery. Acta Neurochir (Wien). 2023 Jul;165(7):1847-1854. doi: 10.1007/s00701-023-05640-4. Epub 2023 May 25. PMID: 37227503; PMCID: PMC10319674.

26. Alet M, Ciardi C, Alemán A, Bandeo L, Bonardo P, Cea C, Cirio J, Cossio J, Cuculic M, Esnaola MM, et al. Cerebral venous thrombosis in Argentina: clinical presentation, predisposing factors, outcomes and literature review. *J Stroke Cerebrovasc Dis*. 2020;29:105145. doi: 10.1016/j.jstrokecerebrovasdis.2020.105145
27. GBD 2019 Stroke Collaborators (2021) Global, regional, and national burden of stroke and its risk factors, 1990–2019: a systematic analysis for the Global Burden of Disease Study 2019. *Lancet Neurol* 20: 795–820.
28. Stam J. Thrombosis of the cerebral veins and sinuses. *N Engl J Med*. 2005;*352*:1791–1798.
29. Murphy SJ, Werring DJ. Stroke: causes and clinical features. *Medicine (Abingdon).* (2020) 48:561–6. doi: 10.1016/j.mpmed.2020.06.002
30. Duman T, Uluduz D, Midi I, Bektas H, Kablan Y, Goksel BK, et al. A multicenter study of 1144 patients with cerebral venous thrombosis: the VENOST study. *J Stroke Cerebrovasc Dis.* (2017) 26:1848–57. doi: 10.1016/j.jstrokecerebrovasdis.2017.04.020
31. Ferro JM, Bousser MG, Canhão P, Coutinho JM, Crassard I, Dentali F, di Minno M, Maino A, Martinelli I, Masuhr F, Aguiar de Sousa D, Stam J; European Stroke Organization. European Stroke Organization guideline for the diagnosis and treatment of cerebral venous thrombosis - endorsed by the European Academy of Neurology. Eur J Neurol. 2017 Oct;24(10):1203-1213. doi: 10.1111/ene.13381. Epub 2017 Aug 20. PMID: 28833980.
32. Weimar C, Beyer-Westendorf J, Bohmann FO, Hahn G, Halimeh S, Holzhauer S, Kalka C, Knoflach M, Koennecke HC, Masuhr F, Mono ML, Nowak-Göttl U, Scherret E, Schlamann M, Linnemann B. New recommendations on cerebral venous and dural sinus thrombosis from the German consensus-based (S2k) guideline. Neurol Res Pract. 2024 Apr 19;6(1):23. doi: 10.1186/s42466-024-00320-9. PMID: 38637841; PMCID: PMC11027218.
33. van Dam LF, van Walderveen MAA, Kroft LJM, Kruyt ND, Wermer MJH, van Osch MJP, Huisman MV, Klok FA. Current imaging modalities for diagnosing cerebral vein thrombosis: a critical review. *Thromb Res*. 2020;189:132–139. doi: 10.1016/j.thromres.2020.03.011
34. Manolidis S, Kutz JW. Diagnosis and management of lateral sinus thrombosis. *Otol Neurotol*. 2005;*26*:1045–1051.
35. Girot M, Ferro JM, Canhão P, Stam J, Bousser MG, Barinagarrementeria F, Leys D; ISCVT Investigators. Predictors of outcome in patients with cerebral venous thrombosis and intracerebral hemorrhage. *Stroke*. 2007;*38*:337–342.

36. Ford K, Sarwar M. Computed tomography of dural sinus thrombosis. *AJNR Am J Neuroradiol*. 1981;*2*:539–543.
    a. (38) Bousser MG, Ferro JM. Cerebral venous thrombosis: an update. *Lancet Neurol*. 2007;*6*:162–170.
37. Dentali F, Crowther M, Ageno W. Thrombophilic abnormalities, oral contraceptives, and risk of cerebral vein thrombosis: a meta-analysis. *Blood*. 2006;*107*:2766–2773. (36) Ford K, Sarwar M. Computed tomography of dural sinus thrombosis. *AJNR Am J Neuroradiol*. 1981;*2*:539–543.
38. Bousser MG, Ferro JM. Cerebral venous thrombosis: an update. *Lancet Neurol*. 2007;*6*:162–170.
39. Leach JL, Fortuna RB, Jones BV, Gaskill-Shipley MF. Imaging of cerebral venous thrombosis: current techniques, spectrum of findings, and diagnostic pitfalls. *Radiographics*. 2006;*26*(suppl 1):S19–S41.
40. Leys D, Cordonnier C. Cerebral venous thrombosis: update on clinical manifestations, diagnosis and management. *Ann Indian Acad Neurol*. 2008;*11*:S79–S87.
41. Wasay M, Bakshi R, Bobustuc G, Kojan S, Sheikh Z, Dai A, Cheema Z. Cerebral venous thrombosis: analysis of a multicenter cohort from the United States. *J Stroke Cerebrovasc Dis*. 2008;*17*:49–54.
42. Tsai FY, Kostanian V, Rivera M, Lee KW, Chen CC, Nguyen TH. Cerebral venous congestion as indication for thrombolytic treatment. *Cardiovasc Intervent Radiol*. 2007;*30*:675–687.
43. Tsai FY, Nguyen B, Lin WC, Hsueh CJ, Yen A, Meng K, Kostanian V. Endovascular procedures for cerebrovenous disorders. *Acta Neurochir Suppl*. 2008;*101*:83–86.
44. Stroke Unit Trialists' Collaboration. Organised inpatient (stroke unit) care for stroke. *Cochrane Database Syst Rev*. 2007 Oct 17;(4):CD000197.
45. Saposnik G, Kapral MK, Coutts SB, Fang J, Demchuk AM, Hill MD; Investigators of the Registry of the Canadian Stroke Network (RCSN) for the Stroke Outcome Research Canada (SORCan) Working Group. Do all age groups benefit from organized inpatient stroke care? *Stroke*. 2009;*40*:3321–3327.
46. Linnemann, B., Blank, W., Doenst, T., Erbel, C., Isfort, P., Janssens, U., Kalka, C., Klamroth, R., Kotzerke, J., Ley, S., Meyer, J., Mühlberg, K., Müller, O. J., Noppeney, T., Opitz, C., Riess, H., Solomayer, E.-F., Volk, T., Beyer-Westendorf, J. (2023). Diagnostik und Therapie der tiefen Venenthrombose und Lungenembolie – AWMF-S2k-Leitlinie. Retrieved 11.04.2023, from https:/register.awmf.org/de/leitlinien/detail/065–002.
47. von Kummer R, Broderick JP, Campbell BCV, Demchuk A, Goyal M, Hill MD, et al. The Heidelberg Bleeding Classification: classification of bleeding events after ischemic stroke and reperfusion therapy. Stroke. 2015;46:2981–2986. doi: 10.1161/STROKEAHA.115.010049.
48. Weimar C, Beyer-Westendorf J, Bohmann FO, Hahn G, Halimeh S, Holzhauer S, Kalka C, Knoflach M, Koennecke HC, Masuhr F, Mono ML, Nowak-Göttl U, Scherret E, Schlamann M, Linnemann B. New recommendations on cerebral venous and dural sinus thrombosis from the German consensus-based (S2k) guideline. Neurol Res Pract. 2024 Apr 19;6(1):23. doi: 10.1186/s42466-024-00320-9. PMID: 38637841; PMCID: PMC11027218.

49. Erkens PM, Prins MH. Fixed dose subcutaneous low molecular weight heparins versus adjusted dose unfractionated heparin for venous thromboembolism. Cochrane Database Systematic Reviews. 2017;2(2):CD001100. doi: 10.1002/14651858.CD001100.pub4.
50. Coutinho JM, Zuurbier SM, Bousser MG, Ji X, Canhao P, Roos YB, Crassard I, Nunes AP, Uyttenboogaart M, Chen J, Emmer BJ, Roosendaal SD, Houdart E, Reekers JA, van den Berg R, de Haan RJ, Majoie CB, Ferro JM, Stam J, investigators, T.-A. Effect of Endovascular Treatment With Medical Management vs Standard Care on Severe Cerebral Venous Thrombosis: The TO-ACT Randomized Clinical Trial. JAMA Neurology. 2020;77(8):966–973. doi: 10.1001/jamaneurol.2020.1022.
51. Nepal G, Kharel S, Bhagat R, Ka Shing Y, Ariel Coghlan M, Poudyal P, Ojha R, Sunder Shrestha G. Safety and efficacy of Direct Oral Anticoagulants in cerebral venous thrombosis: A meta-analysis. Acta neurologica Scandinavica. 2022;145(1):10–23. doi: 10.1111/ane.13506.
52. Ferro JM, Bousser MG, Canhao P, Coutinho JM, Crassard I, Dentali F, di Minno M, Maino A, Martinelli I, Masuhr F, et al. European Stroke Organization guideline for the diagnosis and treatment of cerebral venous thrombosis. *Eur Stroke J*. 2017;2:195–221. doi: 10.1177/2396987317719364
53. Mahale R, Mehta A, Varma RG, Hegde AS, Acharya PT, Srinivasa R. Decompressive surgery in malignant cerebral venous sinus thrombosis: what predicts its outcome? *J Thromb Thrombolysis*. 2017;43:530–539. doi: 10.1007/s11239-017-1489-x
54. Wilson JT, Hareendran A, Grant M, Baird T, Schulz UG, Muir KW, et al. Improving the assessment of outcomes in stroke: use of a structured
55. Li M, Wan S, Wang N, Chen J, Duan J, Chen J, Zhang X, Meng R, Ji X. Development and Validation of a Clinical-Based Severity Scale for Patients with Cerebral Venous Thrombosis. Int J Gen Med. 2023 Oct 25;16:4783-4794. doi: 10.2147/IJGM.S437457. PMID: 37904904; PMCID: PMC10613448.
56. Zubkov AY, McBane RD, Brown RD, Rabinstein AA (2009) Brain lesions in cerebral venous sinus thrombosis. Stroke 40:1509–1511
57. Aguiar de Sousa D, Lucas Neto L, Arauz A, Sousa AL, Gabriel D, Correia M et al (2020) Early recanalization in patients with cerebral venous thrombosis treated with anticoagulation. Stroke 51:1174–1181
58. Jiang YY, Chen LJ, Wu XJ, Zhou GQ, Mo DC, Li XL et al (2022) Efficacy and safety assessment of rivaroxaban for the treatment of cerebral venous sinus thrombosis in a Chinese population. Clin Appl Thromb Hemost 28:10760296221144038
59. Yang Q, Duan J, Fan Z, Qu X, Xie Y, Nguyen C et al (2016) Early Detection and quantification of cerebral venous thrombosis by magnetic resonance black-blood thrombus imaging. Stroke 47:404–409
60. Wang, Z., Dandu, C., Guo, Y. *et al.* A novel score to estimate thrombus burden and predict intracranial hypertension in cerebral venous sinus thrombosis. *J Headache Pain* **24**, 29 (2023). https://doi.org/10.1186/s10194-023-01562-9
61. Anand A, Crowley SC, Srivatsan A, Srinivasan VM, Chintalapani G, Kan

P et al (2022) A retrospective anatomical study of the cerebral dural

venous sinus outflow pathways utilizing three-dimensional rotational venography. Brain Circ 8:38–44

62. Zhou LW, Yu AYX, Ngo L, Hill MD, Field TS. Incidence of Cerebral Venous Thrombosis: A Population-Based Study, Systematic Review, and Meta-Analysis. Stroke. 2023 Jan;54(1):169-177. doi: 10.1161/STROKEAHA.122.039390. Epub 2022 Nov 7. PMID: 36337058.
63. *The venous drainage of the Central Nervous System* (no date) *TeachMeAnatomy*. Available at: https://teachmeanatomy.info/neuroanatomy/vessels/venous-drainage/?form=MG0AV3 (Accessed: 20 March 2025).
64. Alimohammadi A, Kim DJ, Field TS. Updates in Cerebral Venous Thrombosis. Curr Cardiol Rep. 2022 Jan;24(1):43-50. doi: 10.1007/s11886-021-01622-z. Epub 2022 Jan 13. PMID: 35028817; PMCID: PMC8757924.
65. Saposnik G, Kapral MK, Coutts SB, Fang J, Demchuk AM, Hill MD; Investigators of the Registry of the Canadian Stroke Network (RCSN) for the Stroke Outcome Research Canada (SORCan) Working Group. Do all age groups benefit from organized inpatient stroke care? *Stroke*. 2009;*40*:3321–3327.
66. Ferro JM, Bousser M-G, Canhão P, Coutinho JM, Crassard I, Dentali F, et al. European Stroke Organization guideline for the diagnosis and treatment of cerebral venous thrombosis - endorsed by the European Academy of Neurology. Eur J Neurol. 2017;24:1203–1213. doi: 10.1111/ene.13381.
67. Neuroradiology: A Case-Based guid. 1st ed. Swati Goyal; 2021.
68. Coutinho J, de Bruijn SF, Deveber G, Stam J. Anticoagulation for cerebral venous sinus thrombosis. Cochrane Database Syst Rev. 2011 Aug 10;2011(8):CD002005. doi: 10.1002/14651858.CD002005.pub2. PMID: 21833941; PMCID: PMC7065450.
69. Saposnik G, Bushnell C, Coutinho JM, Field TS, Furie KL, Najibah Galadanci, et al. Diagnosis and Management of Cerebral Venous Thrombosis: A Scientific Statement From the American Heart Association. Stroke. 2024 Jan 29;55(3).
70. Sciencedirect.com. [cited 2025 Mar 26]. Available from: https://www.sciencedirect.com/topics/neuroscience/superficial-cerebral-veins

18.2 Additional references on table nr 1.

5. Bousser M-G, Ross Russell RW. Cerebral venous thrombosis. London: W.B. Saun-
Ders, 1997

6. deVeber G, Andrew M. Cerebral sinovenous thrombosis in children. N Engl J Med
2001;345:417-23.

7. Deschiens MA, Conard J, Horellou MH, et al. Coagulation studies, factor V Leiden, and anticardiolipin antibodies in 40 cases of cerebral venous thrombosis. Stroke 1996; 27:1724-30.
8.Enevoldson TP, Russell RW. Cerebral venous thrombosis: new causes for an old syndrome? Q J Med 1990;77:1255-75.
9. Martinelli I, Sacchi E, Landi G, Taioli E, Duca F, Mannucci PM. High risk of cerebral-vein thrombosis in carriers of a prothrombin-gene mutation and in users of oral contraceptives. N Engl J Med 1998; 338:1793-7.
11. Weih M, Junge-Hulsing J, Mehraein S, Ziemer S, Einhaupl KM. Hereditare Thrombophilien bei ischamischem Schlaganfall und Sinusvenenthrombosen: Diagnostik, Therapie und Meta-Analyse. Nervenarzt 2000;
71:936-45.
12. Reuner KH, Ruf A, Grau A, et al. Prothrombin gene G20210˚A transition is a risk factor for cerebral venous thrombosis. Stroke 1998;29:1765-9.
13. Hillier CE, Collins PW, Bowen DJ, Bowley S, Wiles CM. Inherited prothrombotic risk factors and cerebral venous thrombosis. QJM 1998;91:677-80.
14. Cantu C, Alonso E, Jara A, et al. Hyperhomocysteinemia, low folate and vitamin B12 concentrations, and methylene tetrahydrofolate reductase mutation in cerebral venous thrombosis. Stroke 2004;35:1790-4.
15. Carhuapoma JR, Mitsias P, Levine SR. Cerebral venous thrombosis and anticardiolipin antibodies. Stroke 1997;28:2363-9.
16. Cantu C, Barinagarrementeria F. Cerebral venous thrombosis associated with pregnancy and puerperium: review of 67 cases. Stroke 1993;24:1880-4.
17. Lanska DJ, Kryscio RJ. Risk factors for peripartum and postpartum stroke and intracranial venous thrombosis. Stroke 2000; 31:1274-82.
18. Vidailhet M, Piette JC, Wechsler B, Bousser MG, Brunet P. Cerebral venous thrombosis in systemic lupus erythematosus. Stroke 1990;21:1226-31.

20. farah S, Al-Shubaili A, Montaser A, et al. Behcet's syndrome: a report of 41 patients with emphasis on neurological manifestations. J Neurol Neurosurg Psychiatry 1998;64:382-4.
21.
Wermes C, Fleischhack G, Junker R, et al. Cerebral venous sinus thrombosis in children with acute lymphoblastic leukemia carrying the MTHFR TT677 genotype and further prothrombotic risk factors. Klin Padiatr 1999;211:211-4.
22.
Hillmen P, Lewis SM, Bessler M, Luzzatto L, Dacie JV. Natural history of paroxysmal nocturnal hemoglobinuria. N Engl J Med 1995;333:1253-8.
23. de Bruijn SF, Stam J, Koopman MM, Vandenbroucke JP. Case-control study of risk
of cerebral sinus thrombosis in oral contraceptive users and in [correction of who are] carriers of hereditary prothrombotic conditions. BMJ 1998;316:589-92. [Erratum, BMJ 1998;316:822.]
24. Stiefel D, Eich G, Sacher P. Posttraumatic dural sinus thrombosis in children. Eur J Pediatr Surg 2000;10:41-4.
25. Wilder-Smith E, Kothbauer-MargreiterI, Lammle B, Sturzenegger M, Ozdoba C, Hauser SP. Dural puncture and activated protein C resistance: risk factors for cerebral venous sinus thrombosis. J Neurol Neurosurg Psychiatry 1997;63:351-6.