Neurology Asia 2025; 30(3) : 699 – 706

Study on the changes in blood biochemical indicators of patients with Cerebral venous sinus thrombosis and their correlation with imaging examination results

Li Zhao, Ping Yang, Juan Yang, Fang Li, Xiaolin Hou

Department of Neurology, General Hospital of Ningxia Medical University, Yinchuan, China

Abstract

Objective: This study investigates the implications of combining blood biochemical indicators with imaging for Cerebral venous sinus thrombosis (CVST) diagnosis, aiming to enhance diagnostic precision and speed. *Methods:* We retrospectivity analyzed data from 120 CVST patients between August 1, 2016, and July 31, 2023, examining clinical, laboratory, and cranial MRI and MRV imaging data. Statistical methods assessed the diagnostic significance of various blood indicators and their correlation with CVST. *Results:* Headache was the most common symptom, followed by changes in vision, epileptic seizures, and neurological deficits. D-dimer levels, fibrinogen, plateletcrit, mean corpuscular volume (MCV) and platelet distribution width were higher in CVST patients compared to controls. Subgroup analysis revealed that gender and age might influence certain blood test indicators. Notably, a significant difference in D-dimer levels between patient groups with and without complications was observed, suggesting its potential as a biomarker for complication risk. Logistic regression model analysis further confirmed the significant positive correlation between elevated D-dimer levels and CVST risk, also noting that increases in plateletcrit and platelet distribution width were positively correlated with CVST risk. Moreover, through correlation analysis using point biserial correlation coefficient and Spearman's correlation coefficient, significant correlations were found among blood test indicators, and between these indicators and CVST status. MRI and MRV, as key imaging tools for diagnosing CVST, directly display thrombus formation, further enhancing diagnostic accuracy. *Conclusion:* Blood biochemical markers and imaging (MRI and MRV) are vital in diagnosing CVST, aiding in faster, accurate clinical decision-making and better patient outcomes.

Keywords: CVST, blood biochemical indicators, D-dimer, imaging studies

INTRODUCTION

Cerebral venous sinus thrombosis (CVST) is a rare but serious cerebrovascular disorder that typically results from thrombus formation within the cerebral venous sinuses, causing obstruction of blood circulation and thereby affecting the blood supply and drainage of the brain.[1,2] The formation of a thrombus can block the venous sinuses, leading to ischemia or infarction of brain tissue, and triggering a range of neurological symptoms and complications. CVST predominantly occurs in young and middle-aged adults, especially women.[3-5] Epidemiological data suggest that while the incidence of CVST is relatively low, there has been a gradual increase in recent years. Studies indicate an annual incidence rate of CVST of about 2.4 to 7.1 cases per million people, with an increasing trend, especially among certain populations such as pregnant women and oral contraceptive users.[6,7] The deleterious effect of CVST lies in its potential to cause serious complications such as cerebral edema, intracranial hypertension, brain infarction, and even death. Timely diagnosis and treatment of CVST are crucial in preventing severe complications.[8] Accurate diagnosis enables doctors to implement timely and effective treatment measures to prevent further deterioration of the condition. Therefore, enhancing understanding of CVST and exploring effective diagnostic and treatment methods are important.

In recent years, with the advancement of medical technology, abnormal changes in blood-related indicators may indicate a risk of

Address correspondence to: Li Zhao, No. 804 Shengli South Street, Xingqing District, 750001, Yinchuan, China. Tel:860951-6743237. Email: zhaoli1232024@163.com

Date of Submission: 10 May 2025; Date of Acceptance: 20 May 2025

https://doi.org/10.54029/2025hiu

thrombosis formation. Research has found that blood indicators in CVST patients often show significant differences from those in the healthy population, aiding physicians in identifying the risk of CVST.[9,10] Moreover, imaging studies such as Magnetic Resonance Imaging (MRI) and Magnetic Resonance Venography (MRV) are widely used to detect the presence and extent of CVST. MRI can provide high-resolution images to help physicians observe brain structure and vasculature, while MRV directly displays the presence and extent of CVST, aiding in diagnosis and treatment planning.[11,12]

This study aims to explore the application of laboratory test indicators in combination with imaging in the diagnosis of CVST. By analyzing the changes in these indicators among CVST patients, we can improve the accuracy and timeliness of CVST diagnosis, providing a more accurate basis for clinical diagnosis for physicians, and aiding in the timely implementation of effective treatment measures to reduce patient mortality and disability rates.

METHODS

Patient clinical information

This study retrospectively collected data from 120 patients with CVST treated from August 1, 2016, to July 31, 2023. Inclusion criteria were: complete clinical data and compliance with the "2019 Guidelines for the Diagnosis and Treatment of CVST in China". Exclusion criteria were: clinical symptoms, signs, and imaging studies not consistent with the diagnosis of the disease, or patients with severe heart, brain, or lung diseases. Patient general data (such as age, gender, medical history), clinical manifestations (including but not limited to headache, vision changes, neurological deficits), and other relevant data were reviewed through the hospital's medical record system.

To assess the clinical characteristics and specificity of test indicators more accurately in CVST patients, a control group of individuals undergoing health examinations at our hospital's health examination center during the same period was also selected. The control group was matched as closely as possible with the study group in terms of age and gender distribution to enable effective comparative analysis.

Laboratory tests

Patients' laboratory test indicators were collected, including complete blood count: fibrinogen (FIB), platelet count, plateletcrit (PCT), mean platelet volume (MPV), platelet volume distribution width (PDW) and D-dimer levels.

Imaging data

Patients' cranial MRV and MRI data were collected. The imaging data were obtained from the hospital's electronic medical record system, with inclusion criteria mainly including CVST patients who underwent cranial MRV or MRI examinations, and whose imaging reports clearly indicated the location, size, affected venous sinuses, and any potential complications. The collected imaging data were integrated with patients' clinical presentations, laboratory test results, and treatment response data for analysis to identify correlations between imaging features and clinical outcomes, such as the relationship between the size and location of the thrombus and the degree of neurological deficits.

Data analysis

Collected data were inputted into a Microsoft Excel database. Descriptive statistical methods were used to summarize the basic information and clinical characteristics of the patients. SPSS 26.0 statistical software was used for statistical analysis, comparing differences in clinical test indicators between CVST patients and the healthy control group using t-tests or chi-square tests. In addition, univariate analysis of clinical presentations and laboratory indicators in CVST patients was performed to screen for factors significantly associated with the disease. Lastly, multivariate logistic regression analysis was conducted to further explore independent risk factors influencing the occurrence of CVST.

RESULTS

Patient clinical information

This study conducted a retrospective analysis of 120 patients with CVST treated from from August 1, 2016, to July 31, 2023 (Table 1). The results indicated that the age of onset for CVST patients primarily ranged between 21 and 40 years, with an average age of 38.64 years, highlighting the disease's prevalence in the young and middle-aged population. In terms of onset characteristics, acute onset was the predominant type, while chronic onset was less common. This finding suggests that in the young and middle-aged population, patients presenting with acute headaches accompanied by dizziness, nausea, vomiting,

Table 1: Clinical information of patients

	Total n=240		CVST patients n=120		Controls n=120		F	p
D-dimer	1.53±2.96	(1.15-1.91)	2.81±3.78	(2.13-3.49)	0.25±0.12	(0.22-0.27)	54.912	0.000
FIB	3.93±13.45	(2.22-5.65)	3.39±1.22	(3.17-3.61)	4.48±19.01	(1.04-7.92)	.395	0.000
platelet count	248.01±67.86	(239.38-256.64)	261.42±80.23	(246.92-275.92)	234.6±49.48	(225.65-243.54)	9.717	0.002
PCT	0.25±0.05	(0.24-0.25)	0.25±0.07	(0.24-0.26)	0.24±0.04	(0.23-0.24)	4.914	0.166
MCV	10.07±1.12	(9.93-10.21)	9.94±0.91	(9.77-10.10)	10.20±1.29	(9.96-10.43)	3.267	0.007
PDW	11.39±1.94	(11.14-11.64)	11.11±1.92	(10.75-11.45)	11.67±1.93	(11.32-12.02)	5.193	0.024

Abbreviations: CVST, Cerebral Venous Sinus Thrombosis

and other symptoms should be highly suspected of having CVST. The most common symptoms included headache (80% of patients), changes in vision (45%), seizures (30%), and neurological deficits (25%), indicating that headache is the most common clinical manifestation of CVST, with vision changes and seizures also serving as important indicative symptoms.

D-dimer levels were significantly higher in CVST patients (mean±SD: 2.81±3.78) compared to controls (mean±SD: 0.25±0.12) (F=54.912, p<.0001). Fibrinogen (FIB) levels did not show significant differences between CVST patients (mean±SD: 3.93±13.45) and controls (mean±SD: 4.48±19.01) (F=0.395, p<.0001). Platelet count was significantly lower in CVST patients (mean±SD: 248.01±67.86) compared to controls (mean±SD: 234.6±49.48) (F=9.717, p=.002). Plateletcrit (PCT) levels did not show significant differences between CVST patients (mean±SD: 0.25±0.05) and controls (mean±SD: 0.24±0.04) (F=4.914, p=.166). Mean corpuscular volume (MCV) was significantly higher in CVST patients (mean±SD: 10.07±1.12) compared to controls (mean±SD: 10.20±1.29) (F=3.267, p=.007). Platelet distribution width (PDW) was significantly higher in CVST patients (mean±SD: 11.39±1.94) compared to controls (mean±SD: 11.67±1.93) (F=5.193, p=.024).

Subgroup analysis

In the subgroup analysis divided by gender, we utilized the Mann-Whitney U test to compare differences in blood test indicators between male and female patients. The comparison between genders revealed statistically significant differences in PCT(p=0.017) and PCT, p=0.004, indicating that gender may influence these blood test indicators.

Regarding age, FIB showed slight statistical significance between patients under 40 and those 40 or older (p=0.043), suggesting age may have an impact on fibrinogen levels. However, other blood test indicators like D-dimer, platelet count, PCT, MPV, and PDW did not show significant differences across age groups.

Comparing patient groups with and without complications, we observed a highly significant difference in "D-dimer" levels (p<0.0001), indicating a strong correlation between D-dimer and the presence of complications. PCT and MPV also showed statistically significant differences, p=0.042 and p=0.044, respectively. Although PDW did not reach statistical significance

Table 2: Mann-Whitney U test compares the differences in blood test indexes among different groups

	P-values by sex	P-values by age	P-values by complications
D-dimer	0.631	0.932	<0.0001
FIB	0.055	0.043	0.725
platelet count	0.017	0.555	0.042
PCT	0.004	0.485	0.103
MCV	0.731	0.337	0.044
PDW	0.987	0.328	0.078

Abbreviations:
D-dimer, D-dimer;
FIB, Fibrinogen;
PCT, Plateletcrit;
MCV, Mean Corpuscular Volume;
PDW, Platelet Distribution Width.

(p=0.078), it showed a trend towards difference (Table 2).

Risk factor analysis

According to the logistic regression model analysis (Table 3, Figure1), we evaluated the role of different clinical variables and blood test indicators as potential risk factors for CVST. The model revealed key findings: a coefficient of 0.068 for gender, indicating that males (coded as 1) have a slightly increased risk of CVST compared to females (coded as 0), though this effect is relatively minor. The coefficient for age was -0.151, suggesting a slight decrease (PDW) 0.393, further emphasizing the importance of platelet-related indicators in assessing the risk of CVST. Specifically, an increase in PDW is significantly associated with an increased risk of CVST, while larger MPV values seem to correlate with a lower risk.

Table 3: Values of model coefficients

Variable	Coefficient
Gender	0.068
Age	-0.151
D-dimer	5.088
FIB	-0.164
Platelet count	0.311
PCT	0.082
MCV	-0.342
PDW	0.393

Abbreviations:
D-dimer, D-dimer;
FIB, Fibrinogen;
PCT, Plateletcrit;
MCV, Mean Corpuscular Volume;
PDW, Platelet Distribution Width.

Correlation analysis

To further explore the correlations among blood test indicators, between these indicators and other clinical variables such as age, and between health status and CVST, we conducted a correlation analysis (Table 4 and 5). The analysis revealed correlations between multiple blood test indicators, age, gender, and CVST status (healthy vs. CVST), as well as between these variables themselves. Using the point biserial correlation coefficient (for correlations between binary and continuous variables) and Spearman's correlation coefficient (for correlations among continuous variables), we identified significant correlations between CVST status and various factors. The correlation between D-dimer levels and CVST status was the most significant (correlation coefficient = 0.435, p-value < 0.0001), indicating

Table 4: Point two column correlations between CVST state and continuous variables

Variable	Correlation	p-value
Gender	0	1
Age	-0.062	0.341
D-dimer	0.433	<0.0001
FIB	-0.041	0.53
Platelet count	0.198	0.0021
PCT	0.142	0.028
MCV	-0.116	0.072
PDW	-0.146	0.0236

Abbreviations:
CVST, Cerebral Venous Sinus Thrombosis;
D-dimer, D-dimer;
FIB, Fibrinogen;
PCT, Plateletcrit;
MCV, Mean Corpuscular Volume;
PDW, Platelet Distribution Width.

Table 5: Spearman correlation analysis

Variable 1	Variable 2	Spearman	P
D-dimer	FIB	0.384	<0.0001
Platelet count	MCV	-0.433	<0.0001

a significant association between elevated levels of D-Dimer and increased risk of CVST. PCT also showed positive correlations with CVST status, with moderate (correlation coefficient = 0.195, p = 0.0024) and lower (correlation coefficient = 0.140, p = 0.030) levels of correlation, respectively. Age and gender showed no significant correlation with CVST status, suggesting these factors have a minor impact on differentiating health status from CVST in this dataset.

In terms of inter-variable correlations, there was a very strong positive correlation between PCT (correlation coefficient = 0.930, p-value < 0.0001), indicating a close relationship between these two blood indicators, reflecting the overall activity and functional status of platelets. MPV and PDW also showed an extremely strong positive correlation (correlation coefficient = 0.928, p-value < 0.0001), suggesting a physiological link between platelet size and its distribution width. The positive correlation between D-dimer and FIB (correlation coefficient = 0.384, p-value < 0.0001) reflects two different aspects of the coagulation system state, potentially indicating that activation of the coagulation and fibrinolytic systems is related to the development of CVST.

Imaging examination

In our study, we have employed multimodal magnetic resonance imaging (MRI) to meticulously investigate a case with a suspected cerebrovascular event. We have selected three significant images that directly display the abnormal signal in the right parietal lobe and localized thrombus formation in the superior sagittal sinus.

Figure 2A features a Diffusion Weighted Imaging (DWI) which reveals a hyperintense signal in the right parietal lobe, indicative of restricted diffusion in that region. Such signal enhancement typically suggests an acute ischemic stroke, where the area of increased signal corresponds to the affected brain tissue. Figure 2B is a Fluid Attenuated Inversion Recovery (FLAIR) sequence that demonstrates a corresponding hyperintense signal in the same region of the right parietal lobe. The FLAIR sequence, by suppressing cerebrospinal fluid signals, enhances the contrast of abnormal tissues, corroborating the presence of acute ischemia, as observed on the DWI scan. Figure 2C utilizes Magnetic Resonance Venography (MRV), which is dedicated to visualizing the venous system of the brain. The absence of signal in the expected

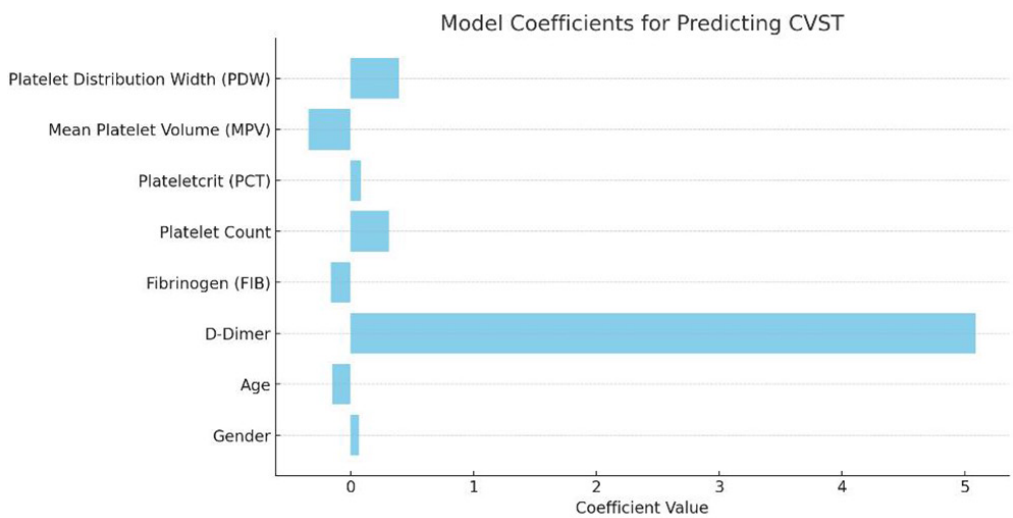

Figure 1. Model coefficients for predicting CVST

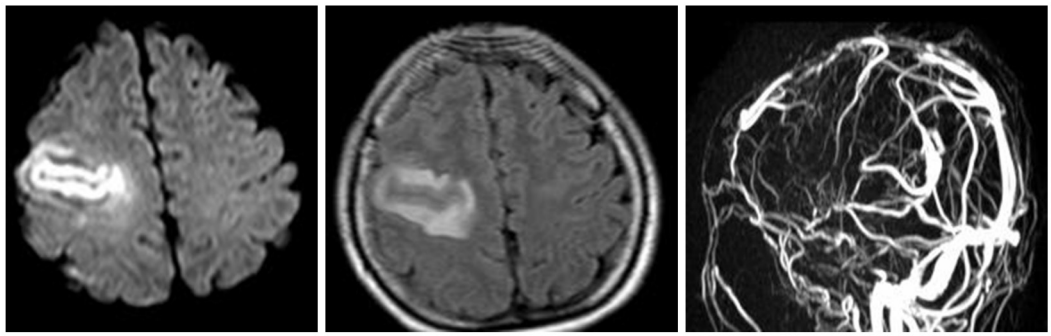

Figure 2. Abnormal signals in the right parietal lobe (A, B), and localized thrombus formation in the superior sagittal sinus (C).

area of the superior sagittal sinus, which should normally be filled with flowing blood, signifies the presence of a thrombus, indicating localized venous sinus thrombosis.

The results illustrate that the patient experienced an acute ischemic stroke in the right parietal lobe region, accompanied by thrombus formation in the superior sagittal sinus. This has significant implications for clinical intervention and treatment planning. Our findings underscore the importance of integrating various imaging techniques for the diagnosis of cerebrovascular pathologies.

DISCUSSION

CVST is a unique type of venous stroke and a significant cause of stroke in young and middle-aged adults. The majority of patients are between 16-50 years of age, with less than 10% over 65 years old.[6-8] CVST is more common in women of childbearing age, linked to gender-specific risk factors such as the use of oral contraceptives, pregnancy and the puerperium, and hormone replacement therapy, which contribute to a hypercoagulable state. Studies have shown that women taking oral contraceptives have an approximately 6-fold increased risk of developing CVST, and this risk may further increase in obese women using oral contraceptives.[13,14] The etiology and risk factors of CVST are diverse and are generally considered closely related to factors causing venous stasis, endothelial damage, and a hypercoagulable state. Existing research indicates that oxidative stress, inflammatory responses, blood-brain barrier disruption, and cerebral edema are involved in the onset and progression of CVST, leading to symptoms and neurological damage. In particular, the inflammatory response plays a key role by causing blood-brain barrier disruption, cerebral edema, and venous cerebral infarction, ultimately leading to poor neurological outcomes in CVST patients.[15-18]

This study demonstrates that CVST primarily occurs in young and middle-aged individuals, consistent with previous research findings. For instance, research by Coutinho et al. also highlighted that CVST predominantly affects young adults, especially women, further confirming the high incidence of CVST in the young and middle-aged population and its diverse clinical presentations. Additionally, acute onset was the most common type, and headache was the most frequent symptom, aligning with findings by Ferro et al. CVST can cause a wide range of clinical symptoms, from headache in outpatient settings to coma in emergency situations. The clinical symptoms of CVST can be classified into four different syndromes: isolated intracranial hypertension, focal syndrome, encephalopathy, and cavernous sinus syndrome. Patients with isolated intracranial hypertension commonly present with headache, often accompanied by nausea, vomiting, papilledema, visual impairment, and tinnitus. The headache worsens when lying down and may be associated with transient visual disturbances, usually occurring during coughing or sneezing. Focal syndrome, caused by thrombosis in the superficial venous system and brain parenchymal lesions, often involves hemorrhagic or ischemic venous infarction, leading to central limb motor disorders, sensory deficits, aphasia, and often seizures. Patients with deep cerebral venous system thrombosis, especially those with extensive venous infarction or bilateral basal ganglia and thalamic edema, exhibit severe clinical symptoms, including psychiatric disturbances, consciousness disorders, and even coma. Cavernous sinus syndrome is less common

in CVST patients, primarily manifesting as orbital pain, ocular pain, and ophthalmoplegia.

The diagnosis of CVST relies primarily on clinical symptoms, lumbar puncture, and imaging studies.[19-21] Lumbar puncture can help differentiate CVST from meningitis. Increased pressure is a common feature in CVST patients, with more severe clinical manifestations when the pressure exceeds 300mmH2O. MRI sequences are the preferred imaging studies for screening CVST in suspected patients, excluding arterial strokes, tumors, and abscesses. Acute MRI and MR venography are the diagnostic methods of choice for CVST. When MRI and MR venography are not feasible, CT venography can be used as a non-invasive alternative. Digital subtraction angiography, although the gold standard for vascular imaging, is limited in clinical application due to its invasiveness and is only performed when CVST diagnosis is unclear or during endovascular treatment. The International Study on Cerebral Vein and Dural Sinus Thrombosis (ISCVST) is one of the most influential studies on CVST prognosis.

The changes in PCT and D-dimer in this study were significantly associated with the occurrence of CVST. Especially D-dimer, which showed significant differences between patient groups with and without complications, indicating its potential as an important predictor of CVST complications. This finding is consistent with previous studies, such as the report by Lalive *et al*. on the significant role of D-dimer in diagnosing CVST. An increase in PCT also showed a positive correlation with CVST risk, reflecting the activation state of platelets during thrombus formation.

D-dimer, a unique marker of fibrinogen degradation, has a molecular weight of about 62ku and a half-life of approximately 8 hours in the body, primarily cleared by the kidneys and metabolized by the reticuloendothelial system.[22] Since D-dimer is produced only during the formation and degradation of cross-linked fibrin, it serves as a direct marker of the activation of the coagulation and fibrinolytic systems and an indirect marker of thrombosis. D-dimer is the most reliable indicator for assessing a hypercoagulable state, clinically used in the evaluation of suspected disseminated intravascular coagulation, venous thromboembolism (VTE), including pulmonary embolism and deep vein thrombosis. Elevated levels of D-dimer are common in patients with acute venous thrombosis. Studies have found that patients with atrial fibrillation have higher levels of D-dimer compared to those without atrial fibrillation, and the levels of D-dimer decrease following anticoagulation treatment or successful cardiac rhythm normalization. Patients with atrial fibrillation and other stroke risk factors, such as hypertension, diabetes, or heart failure, have higher levels of D-dimer than those without risk factors, indicating that D-dimer can predict the risk of stroke in patients with atrial fibrillation. The significant differences in D-dimer levels between patient groups with and without complications in this study further highlight its potential value in assessing the risk of CVST complications.

Moreover, MRI and MRV, as key imaging tools in this study, can directly display the formation of thrombi, crucial for the diagnosis of CVST.[23,24] This has been widely recognized in previous studies, and our findings further validate this point, emphasizing the value of combining platelet indicators, D-dimer testing, and imaging studies to enhance the accuracy of CVST diagnosis.

In conclusion, this study not only summarizes the clinical characteristics and significance of laboratory test indicators in CVST patients but also explores the application value of blood indicators and D-dimer combined with imaging in the diagnosis of CVST. These findings are consistent with previous research results and further deepen our understanding of the importance of diagnosis in CVST, providing valuable references for future clinical practice.

DISCLOSURE

Ethics: This study was approved by the Ethics Committee of General Hospital of Ningxia Medical University (2020-521) and written informed consent was obtained from the patient.

Data availability: The data used to support the findings of this study are included within the article.

Financial support: None

Conflict of interests: None

REFERENCES

1. Ferro JM, Aguiar de Sousa D. Cerebral venous thrombosis: an update. *Curr Neurol Neurosci Rep* 2019;19(10):74. doi:10.1007/s11910-019-0988-x
2. Cohen O, Pegoraro S, Ageno W. Cerebral venous thrombosis. *Minerva Med* 2021;112(6):755-66. doi:10.23736/S0026-4806.21.07353-5
3. Silvis SM, de Sousa DA, Ferro JM, Coutinho JM. Cerebral venous thrombosis. *Nat Rev Neurol* 2017;13(9):555-65. doi:10.1038/nrneurol.2017.104

4. Mehta A, Danesh J, Kuruvilla D. Cerebral venous thrombosis headache. *Curr Pain Headache Rep* 2019;23(7):47. doi:10.1007/s11916-019-0786-9
5. Aguiar de Sousa D. Cerebral venous thrombosis: What's new?. *Hamostaseologie* 2021;41(1):25-30. doi:10.1055/a-1332-3042
6. Ferro JM, Bousser MG, Canhão P, *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(10):1203-13. doi:10.1111/ene.13381
7. Martinelli I, Passamonti SM, Rossi E, De Stefano V. Cerebral sinus-venous thrombosis. *Intern Emerg Med* 2012;7 Suppl 3:S221-S225. doi:10.1007/s11739-012-0806-9
8. Dmytriw AA, Song JSA, Yu E, Poon CS. Cerebral venous thrombosis: state of the art diagnosis and management. *Neuroradiology* 2018;60(7):669-85. doi:10.1007/s00234-018-2032-2
9. Shatzel JJ, O'Donnell M, Olson SR, *et al*. Venous thrombosis in unusual sites: A practical review for the hematologist. *Eur J Haematol* 2019;102(1):53-62. doi:10.1111/ejh.13177
10. Kristoffersen ES, Harper CE, Vetvik KG, Faiz KW. Cerebral venous thrombosis - epidemiology, diagnosis and treatment. *Tidsskr Nor Laegeforen* 2018;138(12). doi:10.4045/tidsskr.17.1047
11. Ferro JM, Canhão P, Aguiar de Sousa D. Cerebral venous thrombosis. *Presse Med* 2016;45(12 Pt 2):e429-e450. doi:10.1016/j.lpm.2016.10.007
12. Hartel M, Kluczewska E, Gancarczyk-Urlik E, Pierzchała K, Bień K, Zastawnik A. Cerebral venous sinus thrombosis. *Phlebology* 2015;30(1):3-10. doi:10.1177/0268355514526712
13. Ranjan R, Ken-Dror G, Sharma P. Pathophysiology, diagnosis and management of cerebral venous thrombosis: A comprehensive review. *Medicine* (Baltimore). 2023;102(48):e36366. doi:10.1097/MD.0000000000036366
14. Goyal M, Fladt J, Coutinho JM, McDonough R, Ospel J. Endovascular treatment for cerebral venous thrombosis: current status, challenges, and opportunities. *J Neurointerv Surg* 2022;14(8):788-93. doi:10.1136/neurintsurg-2021-018101
15. Hisada Y, Mackman N. Cancer-associated pathways and biomarkers of venous thrombosis. *Blood* 2017;130(13):1499-506. doi:10.1182/blood-2017-03-743211
16. Borhani-Haghighi A, Hooshmandi E. Cerebral venous thrombosis: a practical review. *Postgrad Med J* 2024;100(1180):68-83. doi:10.1093/postmj/qgad103
17. Kuiper L, Sánchez van Kammen M, Coert BA, *et al*. Association between dural AVFs and cerebral venous thrombosis. *AJNR Am J Neuroradiol* 2022;43(12):1722-9. doi:10.3174/ajnr.A7652
18. Zhang D, Wang J, Zhang Q, He F, Hu X. Cerebral venous thrombosis in spontaneous intracranial hypotension: A report on 4 cases and a review of the literature. *Headache* 2018;58(8):1244-55. doi:10.1111/head.13413
19. Yang X, Yu P, Zhang H, *et al*. Deep learning algorithm enables cerebral venous thrombosis detection with routine brain magnetic resonance imaging. *Stroke* 2023;54(5):1357-66. doi:10.1161/STROKEAHA.122.041520
20. Franchini M, Testa S, Pezzo M, *et al*. Cerebral venous thrombosis and thrombocytopenia post-COVID-19 vaccination. *Thromb Res* 2021;202:182-3. doi:10.1016/j.thromres.2021.04.001
21. Bossoni AS, Peres MFP, Leite CDC, Fortini I, Conforto AB. Headache at the chronic stage of cerebral venous thrombosis. *Cephalalgia* 2022;42(14):1476-86. doi:10.1177/03331024221113825
22. Nakamura M, Sakon M, Sasako M, *et al*. Association of D-dimer level with thrombotic events, bleeding, and mortality in Japanese patients with solid tumors: a Cancer-VTE Registry subanalysis. *Int J Clin Oncol* 2024;29(4):407-16. doi:10.1007/s10147-024-02475-6
23. Alharthi A, Alghamdi GA, Alghamdi BS, Alghamdi GS, Alzahrani NK. Tuberculous otitis media with cerebral venous thrombosis: A rare and challenging diagnostic case. *Cureus* 2024;16(2):e54391. doi:10.7759/cureus.54391
24. Sajjad Z. MRI and MRV in cerebral venous thrombosis. *J Pak Med Assoc* 2006;56(11):523-6.