



Original Research Article

Clinicoradiological profile and functional outcome of acute cerebral venous thrombosis

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Abstract

Objectives: To determine clinicoradiological profile and functional outcome among patients of acute cerebral venous thrombosis.

Background: Acute cerebral venous thrombosis (CVT) may result in a variety of clinical presentations, with headache being the most common. The relationship between clinical and neuroradiological characteristics in acute CVT patients is still not univocally characterized.

Materials and Methods: We enrolled all patients aged ≥ 18 years old admitted to the emergency department (ED) of Chengalpattu government medical college, and discharged between January 1, 2020, and September 30, 2021, with a diagnosis of CVT (study period = 21 months). exclusion criteria in our study is presence of stroke/ICSH/seizure disorder, metabolic encephalopathy.

Results: All patient (100%) are associated with concomitant neurological symptoms/signs (74%) and superior sagittal sinus thrombosis involved in 33 (66%) patients. Headache is most common presenting symptoms (84%).

Conclusions: Our study confirms and enriches available data on the clinicoradiological profile of patients with acute CVT venous ischemia are independently associated with poor outcomes. We confirmed and enriched available data on acute CVT clinicoradiological profiles in a consecutive series of patients admitted to our ED. New-onset, sub-acute, moderate to severe, and persistent headache represents a major clinical red flag that should prompt urgent investigation for CVT.

Keywords: Cerebral venous thrombosis, Headache, Neuroimaging, Outcome, Prognosis.

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1. Introduction

Cerebral venous thrombosis (CVT) is a rare cerebrovascular disease with an estimated incidence of 5/1,000,000 people per year.¹ Although accounting for less than 1% of all strokes,^{1,2} CVT-related complications (e.g. venous ischemia (VI), intracranial hypertension (IH), parenchymal hemorrhage (PH), subarachnoid hemorrhage (SAH)) account for important morbidity and mortality rates (about 15%).³ CVT has a three-fold higher incidence in women younger than 50 years old, reflecting sex-specific risk factors such as pregnancy, contraceptive use, and estrogen fluctuations.^{1,2,4,5} Other less common risk factors include genetic or paraneoplastic thrombophilia, infectious or autoimmune disorders, and traumatic skull injury.^{1,2,4,5} CVT can present with a multitude of neurological symptoms depending on many factors such as age and the anatomical location of the thrombus.^{6,7} Headache is notoriously the most common

symptom of CVT (up to 90% of all cases)⁷ and can result from either mechanical stretching of trigeminal nerve fibers in the walls of the occluded sinus or from cortical and dural inflammation.^{1,2} However, CVT-related headache location and characteristics are extremely heterogeneous, ranging from thunderclap pain to less specific features (e.g. throbbing pain with nausea, vomit, phono/photophobia-mimicking migraine, or other primary headache disorders).^{6,8} Few studies attempting to identify a recurrent headache pattern have failed to provide univocal data^{6,7} so that the clinical suspicion of acute CVT still relies on the concomitant presence of risk factors and neuroimaging findings.^{1,2,4,5,6,7,8} Furthermore, the relationship between headache and neuroimaging findings has been not univocally characterized, and the only reported association is between occipital pain and sigmoid sinus thrombosis.^{9,10}

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With this background, our study aimed to investigate the clinicoradiological profile and to identify potential associations with the functional outcome in a retrospective cohort of CVT patients.

2. Materials and Methods

2.1. Study design and participants

We enrolled all patients aged ≥ 18 years old admitted to the emergency department (ED) of Chengalpattu government medical college, and discharged between January 1, 2020, and September 30, 2021, with a diagnosis of CVT (study period = 21 months).exclusion criteria in ovr study is presence of stroke/ICSOL/seizure disorder, metabolic encephalopathy. Additionally, we interrogated our hospital database in order to check the inclusion of all CVT cases with the following International Classification of Diseases, Ninth Revision Clinical Modification (ICD-9 CM), overall, patients underwent several neurological evaluations during admission. Clinical, neuroimaging, treatment, and outcome data were extrapolated from medical records, neurological, and the mortality registry.

Definitions, classifications, and diagnostic procedures CVT was classified according to the time from the onset as acute (<48 hours), including thunderclap headache, subacute (48 hours - 1 week), and chronic (>1 week) on the basis of onset of headache. Unenhanced brain CT scan was considered consistent with CVT in the presence of a hyper-attenuating clot in the cerebral vein system, signs of VI (infarct not conforming to a conventional artery territory or spanning ≥ 1 territory with sparing of the cortex, multiple or bilateral lesions with/without hemorrhage).¹¹ CVT diagnosis was confirmed according to the American Health Association/American Stroke Association (AHA/ASA) international guidelines for CVT diagnosis and management.⁵

2.2. Statistical analysis

Patients were stratified into different groups based on clinical variables. Demographics and neuroimaging patterns were compared between patients dichotomized according to the presence or absence of the specific clinical feature in each of these groups. Categorical variables were summarized as frequencies and percentages while continuous variables were presented as median (interquartile range - IQR). Categorical variables were compared using the chi-square test Results were presented as odds ratio (OR) with a 95% confidence interval (CI). A p-value of ≤ 0.05 was considered statistically significant. Statistical analysis was performed with SPSS software.

3. Results

Fifty five patients aged >18 years old with a diagnosis of CVT were included in the study. Five patients were excluded because of secondary referral to our hospital for CVT complications. Therefore, we included 50 patients in the study. (hyperhomocysteinemia, antiphospholipid antibodies, antithrombin III deficiency, protein S/C deficiency, factor V Leiden mutation, prothrombin mutation, activated protein C resistance). Among gender-specific risk factors, the assumption of oral contraceptives was the most prevalent condition (60%), followed by pregnancy (10%). The presence of hereditary thrombophilia was the most frequent non-gender-specific risk factor (5%). For five patients (25%), we could not identify any possible prothrombotic factor. In our study headache found in 42 (84 %).Headache is most common presenting symptoms in our study. Second most common presenting symptom is seizure, found in 25 (50%). (Table 1)

Table 1: Distribution of CVT patients according to clinical sign /symptoms

Symptoms & Sings	No. of patients (n-50)	Percentage (%)
Headache	42	84%
Vommiting	17	34%
Fever	6	12%
Seizure	25	50%
Altered sensorium	10	20%
Focal deficit	15	30%
Papilloedema	11	22%
CN involvement 6 th , 7 th	10	20%
	4	8%
	6	12%

Table 2: Distributions of CVT patients according to MRV finding

Sinus onvoled	No. of patients (N-50)	Percentage (%)
Superior sagittal sinus(SSS)	33(Isolated in 5PTS)	66%
Transverse singus(TS)	30	60%
Sigmoid sinus (SS)	28(Isolated in 2PTS)	56%
Straight sinus	2	4%
Cortical veins	19(Isolated in 3PTS)	38%
Deep veins	3(Isolated in 1PTS)	6%

Table 3: Correlation between clinical features and MRV findings

Site of thrombus on MRV	Mode of onset			Clinical presentaions						P value
	AC	SA	CR	HA	CON	FD	AL	CN	PAP	
SSS	2	0	4	14	3	4	3	2	2	
TS	0	2	2	2	3	1	0	0	1	
CV	1	0	2	2	1	0	1	1	0	
TS+SIS+IJV	1	0	0	1	1	1	2	1	0	>0.05
SSS+TS+SIS	2	1	5	6	5	1	1	4	4	
SSS+TS	5	0	3	4	1	0	2	1	2	
SSS+CV	2	0	1	2	2	1	2	0	0	
TS+SIS	1	2	4	7	5	0	0	0	0	
SSS+TS+ICV+CV	1	0	1	2	1	0	1	0	1	
SSS+TS+SIS+ICV	2	1	2	1	1	1	1	0	1	
SSS+TS+ISI+STS+IJV	1	0	2	1	2	1	1	1	0	

Chi square test showed no significant difference between clinical parameters and site of venous occulision (p value more than.05). (Table 3)

Table 4: Correlation between clinical presentation and structural imaging

Parameters		Patients without parenchymal lesions	Patients with parenchymal lesions	P value
Mode of onset	Acute	4	15	
	Sub-acute	9	16	
	Chronic	3	3	
Clinical presentation	Headache	16	28	0.073
	Seizure	2	21	0.001
	Sensorium	3	7	0.880
	Focal deficit	3	11	0.318
	Hemiparesis	3	12	0.446
	CN palsy	4	6	0.063
	Papilloedem	6	5	0.70
Sinus involved	SSS	17	16	0.650
	TV	11	19	0.671
	SIS	6	22	0.136
	Cortical vein	8	11	0.243

4. Discussions

We analyzed a prospective cross sectional study of consecutive CVT patients admitted to the ED of our hospital over a period of 21 months. Our study outlines some relevant clinoradiological profile and functional outcomes, especially with regard to acute CVT-related headache

characteristics, which, to date, are still not univocally defined.

Epidemiologically, the higher prevalence of women 36 (72%) observed in our population, as well as the distribution of risk factors (mostly gender-specific, e.g., oral contraceptives and pregnancy >70%), are in line with

observations from larger cohort studies (e.g. International Study on Cerebral Vein and Dural Sinus Thrombosis – ISCVT,³ Cerebral Venous Sinuses Thrombosis Study – VENOST).¹² Remarkably, unenhanced head CT scan resulted positive for direct (e.g. cord sign, dense triangle sign) or indirect (e.g. venous ischemia, subcortical hemorrhagic infarction, brain swelling) signs consistent with acute CVT in a great proportion of patients (83%), suggesting great value in the emergency diagnosis of CVT.^{15,16,17}

As expected, headache was the most prevalent presenting symptom of acute CVT 42 (84%), although with heterogeneous intensity and location (either holocranial, “band-like,” or hemicranial). Patients presenting with headache tended to be younger in comparison to patients reporting different onsets. This finding may be due to cerebral atrophy in the elderly, attenuating effects of IH, as well as to diminished pain reactivity.⁶ Notably, the classical thunderclap onset was never reported. Isolated headache as a CVT inaugural manifestation reasonably delayed the first-aid access of about a week compared with focal neurological defects at onset. Despite the heterogeneous intensity and location, the recurrence of some features seems to indicate a more frequent pattern for CVT-related headaches should arouse suspicion, particularly in the presence of an underlying prothrombotic condition, as stated by the current international classification for headache disorders - ICHD criteria,¹⁸ to avoid possible delay of treatment initiation.

With reference to neuroimaging findings, the distribution of involved sinuses slightly differed from results of the ISCVT cohort,³ which reported a more frequent involvement of the SSS 33 (>66%). Second most common involved sinus is transverse sinus in 30 (60%). This clinico-radiological association was observed independently from the concomitant presence of ICH or symptoms of IH, supporting the hypothesis that cortical irritation and inflammation due to VI might consistently contribute to the development of headache in CVT.^{19,20,21} SSS thrombosis was more frequently observed in patients with focal neurological defects ($p=0.043$), likely for the concomitant involvement (50%) of cortical and/or deep cerebral veins, not allowing the development of adequate collateral outflows,^{22,23} thus leading to transient dysfunction of eloquent parenchymal areas. The more frequent presence of vomit in patients with straight sinus thrombosis ($p=0.05$) is a novel finding and may be interpreted as an early sign of IH due to the initial development of impaired cerebrospinal fluid circulation when structures neighboring the III ventricle outlet (e.g. thalami) are involved.^{1,2,6} Concerning the prognosis, acute CVT was associated with a good overall functional outcome. In our study 3 patients expired. Our study has several limitations. First, the small number of enrolled subjects (reflecting disease rarity) might have hampered the inferability of our findings and masked otherwise relevant associations. Second, the retrospective design represents a known source of possible information bias. However, clinical

data were mainly obtained from neurological visits carried out by expert neurologists during the hospital stay, thus downplaying possible information biases.

5. Conclusions

Our study suggests that acute CVT, although accounting for a small part of all cerebrovascular accidents, could be more frequent than previously reported. We confirmed and enriched available data on acute CVT clinico-radiological profiles in a consecutive series of patients admitted to our ED. New-onset, subacute, moderate to severe, and persistent headache represents a major clinical red flag that should prompt urgent investigation for CVT, especially in the presence of concomitant neurological defects and/or well-known risk factors. Unenhanced head CT scan showed great sensitivity as the first-line ED diagnostic investigation, followed by CT/MR venography. Prompt and adequate recognition and management led to an excellent outcome in most of the patients (>85%), whereas older age and presence of VI were associated with poor functional outcomes. Given the rarity of CVT, a disease registry should be implemented to achieve a better and univocal characterization of acute CVT clinico-radiological profile in order to favor early diagnosis and the identification of robust prognostic predictors, which still represent an important challenge.

6. Treatment and Outcome

All patients underwent immediate anticoagulation with low-molecular-weight heparin (LMWH). Thereafter, anticoagulation therapy was continued with warfarin (90 %) or LMWH (10%) according to current guidelines for CVT treatment.⁵ Specifically, 37 out of 50 patients (74%) were treated with anticoagulants for <12 months while 10 patients (20%) underwent anticoagulation for 12-24 months. 3 patient expired in our study. No case of CVT recurrence was recorded during the entire follow-up.

7. Ethics

Consent was obtained or waived by all participants in this study. All authors have confirmed that this study did not involve animal subjects or tissue.

8. Source of Funding

None.

9. Conflict of Interest

None.

References

1. 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.

2. Idiculla PS, Gurala D, Palanisamy M, Vijayakumar R, Dhandapani S, Nagarajan E. Cerebral venous thrombosis: a comprehensive review. *Eur Neurol*. 2020;83(4):369–79. doi: 10.1159/000509802.
3. Ferro JM, Canhão P, Stam J, Boussier MG, Barinagarrementeria F. Prognosis of cerebral vein and dural sinus thrombosis. Results of the International Study on Cerebral Vein and Dural Sinus Thrombosis (ISCVT). *Stroke*. 2004;35(3):664–70. doi: 10.1161/01.STR.0000117571.76197.26.
4. Boussier MG, Ferro JM. Cerebral venous thrombosis: An update. *Lancet Neurol*. 2007;6(2):162–70. doi: 10.1016/S1474-4422(07)70029-7.
5. Saposnik G, Barinagarrementeria F, Brown RD Jr, Bushnell CD, Cucchiara B, Cushman M, et al. Diagnosis and management of cerebral venous thrombosis: a statement for healthcare professionals from the American Heart Association/American Stroke Association. *Stroke*. 2011;42(4):1158–92. doi: 10.1161/STR.0b013e31820a8364.
6. Mehta A, Danesh J, Kuruvilla D. Cerebral venous thrombosis headache. *Curr Pain Headache Rep*. 2019;23:47. doi: 10.1007/s11916-019-0786-9.
7. Song SY, Lan D, Wu XQ, Meng R. The clinical characteristic, diagnosis, treatment, and prognosis of cerebral cortical vein thrombosis: a systematic review of 325 cases. *J Thromb Thrombolysis*. 2021;51(3):734–40. doi: 10.1007/s11239-020-02229-x.
8. Ferro JM, Boussier MG, 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(10):1203–13. doi: 10.1111/ene.13381.
9. Wasay M, Kojan S, Dai AI, Bobustuc G, Sheikh Z. Headache in cerebral venous thrombosis: incidence, pattern and location in 200 consecutive patients. *J Headache Pain*. 2010;11:137–9. doi: 10.1007/s10194-010-0186-3.
10. Sparaco M, Feleppa M, Bigal ME. Cerebral venous thrombosis and headache - a case-series. *Headache*. 2015;55(6):806–14. doi: 10.1111/head.12599.
11. Ghoneim A, Straiton J, Pollard C, Macdonald K, Jampana R. Imaging of cerebral venous thrombosis. *Clin Radiol*. 2020;75(4):254–64. doi: 10.1016/j.crad.2019.12.009.
12. Duman T, Uluduz D, Mide 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(8):1848–7. doi: 10.1016/j.jstrokecerebrovasdis.2017.04.020.
13. Janghorbani M, Zare M, Saadatnia M, Mousavi SA, Mojarad M, Asgari E. Cerebral vein and dural sinus thrombosis in adults in Isfahan, Iran: frequency and seasonal variation. *Acta Neurol Scand*. 2008;117(2):117–21. doi: 10.1111/j.1600-0404.2007.00915.x.
14. Coutinho JM, Zuurbier SM, Aramideh M, Stam J. The incidence of cerebral venous thrombosis. A cross-sectional study. *Stroke*. 2012;43(12):3375–7. doi: 10.1161/STROKEAHA.112.671453.
15. Linn J, Pfefferkorn T, Ivanicova K, Müller-Schunk S, Hartz S, Wiesmann M. et al. Noncontrast CT in deep cerebral venous thrombosis and sinus thrombosis: comparison of its diagnostic value for both entities. *AJNR Am J Neuroradiol*. 2009;30:728–735. doi: 10.3174/ajnr.A1451.
16. Buyck PJ, De Keyser F, Vanneste D, Wilms G, Thijs V, Demaerel P. CT density measurement and H:H ratio are useful in diagnosing acute cerebral venous sinus thrombosis. *AJNR Am J Neuroradiol*. 2013;34(8):1568–72. doi: 10.3174/ajnr.A3469.
17. Tayyebi S, Akhavan R, Shams M, Salehi M, Farrokhi D, Yousefi F et al. Diagnostic value of non-contrast brain computed tomography in the evaluation of acute cerebral venous thrombosis. *Sci Rep*. 2020;10(1):883. doi: 10.1038/s41598-020-57867-1.
18. Headache Classification Committee of the International Headache Society (IHS). The International Classification of Headache Disorders, 3rd edition. Cephalgia. 2018;38:1–211. doi: 10.1177/0333102417738202.
19. Botta R, Donirpathi S, Yadav R, Kulkarni GB, Kumar MV, Nagaraja D. Headache patterns in cerebral venous sinus thrombosis. *J Neurosci Rural Pract*. 2017;8(Suppl 1):S72–7. doi: 10.4103/jnpr.jnpr_339_16..
20. Nascimento FA, Sória MG, Rizelio V, Kowacs PA. Cerebral venous thrombosis with migraine-like headache and the trigeminovascular system. *Case Rep Neurol Med*. 2016;2016:2059749. doi: 10.1155/2016/2059749.
21. Agostoni E. Headache in cerebral venous thrombosis. *Neurol Sci*. 2004;25(Suppl. 3):S206–10. doi: 10.1007/s10072-004-0287-3.
22. Stolz E, Gerriets T, Bödeker RH, Hügens-Penzel M, Kaps M. Intracranial venous hemodynamics is a factor related to a favorable outcome in cerebral venous thrombosis. *Stroke*. 2002;33:1645–1650. doi: 10.1161/01.str.0000016507.94646.e6.
23. Tong LS, Guo ZN, Ou YB, Yan-Nan Y, Xiao-Cheng Z, Jiping T, et al. Cerebral venous collaterals: A new fort for fighting ischemic stroke?. *Prog Neurobiol*. 2018;163:172–93. doi: 10.1016/j.pneurobio.2017.11.001.

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