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Diagnostic Utility of Clinical Scoring System and D-Dimer Assay in Suspected Cerebral Venous Thrombosis: A Prospective Observational Study

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ABSTRACT

Objective: This study aimed to evaluate the diagnostic accuracy of a clinical scoring system, both alone and in combination with D-dimer assay, for suspected cerebral venous thrombosis (CVT) in the emergency department.

Methods: This prospective observational study was conducted in the Emergency Department of a tertiary care teaching hospital in India. Patients who met the inclusion criteria were assessed using a CVT scoring system. They underwent non-contrast computed tomogography of the brain to rule out other types of strokes and to detect signs of CVT. D-dimer testing was also performed. The diagnosis of CVT was subsequently confirmed by magnetic resonance venography (MRV).

Results: Among the75 study subjects, the frequency of CVT, as confirmed by MRV, was 53.33%. Elevated D-dimer levels were observed in 32 out of 75 patients, of whom 26 (65%) patients had CVT. A low-probability clinical score demonstrated a sensitivity of 73.33% and a pecificity of 71.1% for ruling out CVT. When a low-probability score was combined with D-dimer values below 500 μg/L, the sensitivity for ruling out CVT improved to 92.5%.

Conclusion: The present study indicated that moderate- and high-probability clinical scores were associated with a higher likelihood of CVT. In this cohort, a low-probability clinical score combined with a negative D-dimer assay had a high negative predictive value for CVT. While a moderate or high-probability score warranted urgent imaging, it could not confirm the diagnosis on its own. This approach is particularly useful in resource-limited settings for triaging and referring patients for the early initiation of treatment.

Keywords: Cerebral venous thrombosis; D-dimer, Cerebral venous thrombosis score; magnetic resonance venography.

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Introduction

Cerebral Venous thrombosis (CVT) is a rare neurological emergency, accounting for 0.5 % of all strokes [1]. Patients often present to emergency department (ED) with a wide variety of symptoms and are subsequently diagnosed with the condition. Due to its diverse etiologies and clinical presentations, CVT is frequently missed in the early phase of the disease, which can result in significant morbidity and even death [2].

It is essential, therefore, to consider CVT as a possible differential diagnosis in a cohort of patients with risk factors. A clinical score that guides the emergency physician in identifying patients who need urgent imaging would be highly appropriate, as it could facilitates early diagnosis and risk stratification [3].

CVT encompasses occlusions of the dural venous sinus, superficial (cortical) vein, and deep veins. The disease is more commonly seen in individuals younger than 50 years and predominantly affects the female population. The risk factors for CVT include the use of oral contraceptives and prothrombotic conditions such as deficiency of protein C, protein S, or antithrombin III; Factor V Leiden mutation, the presence of antiphospholipid and anticardiolipin antibodies; hyperhomocysteinemia; puerperium, pregnancy; metabolic causes such as dehydration and thyrotoxicosis and infections such as mastoiditis and meningitis [4].

The clinical features arise from a combination of impaired venous drainage, intracerebral haemorrhage, and focal brain injury from venous ischemia. Headache is the most common symptom. Patients may also present with diplopia (due to abducens nerve palsy), focal sensory and motor deficits, aphasia and seizures [5, 6]. Early institution of anticoagulant therapy is lifesaving in CVT cases [7].

Non-contrast computed tomography (NCCT) of the head is the initial screening test for all patients upon arrival to the ED. The highly specific direct signs of CVT on NCCT include the cord sign (a hyperdense thrombosed cortical vein) and the dense triangle sign (hyperdensity in the superior sagittal sinus due to fresh thrombus) [8, 9]. However, NCCT brain has been reported to be normal in many patients, particularly those presenting without focal neurological deficits [1]. The definitive investigation for diagnosing CVT is either CT venography or magnetic resonance venography (MRV) [10, 11].

D-dimer is an acute phase reactant that has been shown to be associated with inflammatory states. Elevated D-dimer levels indicate a pathologically high level of fibrin degradation products, which is related to thrombosis formation [12]. While D-dimer concentrations are raised in most patients with CVT, a normal D-dimer level does not rule out the condition, particularly in patients with isolated headaches. Consequently, D dimer is considered a marker that may serve as a useful screening tool to determine the necessity and urgency of obtaining magnetic resonance imaging (MRI) in patients presenting with symptoms suggestive of CVT [13]. High levels of D-dimer correlate with extensive thrombosis and acute onset of symptoms [14, 15].

The confirmation of a CVT is made using MRV. Recognizing the need for early diagnosis and the constraints of resource-limited settings, we applied a clinical score to triage potential cases of CVT and substantiated it with serum D-dimer levels.

Materials and Methods

This prospective observational study was conducted in the ED of a tertiary care teaching hospital, Nizams Institute of Medical Sciences, in India from December 2020 to November 2021, after obtaining approval from the institutional ethics committee. The sample size was calculated based on the following formula:

$$\text{Sample size (N)} = \frac{Z^2 * SD * (1\text{-}SD)}{ME^2}$$

Z=1.645 at a confidence level of 90%
SD=Suggestive standard difference of 0.05
ME=Margin of error of +/- 1% or 0.01

The calculated sample size was 67.6 subject, which was rounded to 75 to account for a 10% attrition rate. This study was designed as an exploratory diagnostic accuracy study. The sample size was considered sufficient to estimate sensitivity with reasonable precision. Assuming an expected sensitivity of approximately 90% for the combined clinical score and D-dimer (as reported by Heldner *et al*.,), a minimum of 35-40 CVT-positive cases would allow estimation of sensitivity with a 95% confidence interval width of approximately±10%. Given the observed CVT prevalence of 53.3%, a total sample size of 75 patients was considered adequate for preliminary validation.

Data were collected from consenting patients who met the inclusion criteria and presented to the ED of Nizams Institute of Medical Sciences during the study period.

Patients above 18 years of age of both sexes presenting with clinical features of isolated headache or headache associated with focal neurological deficits, seizures and altered sensorium of less than 30 days' duration were included in the study after obtaining written consent. Pregnant women, patients with head injury, patients on anticoagulants, and those with a history of deep vein thrombosis (DVT), pulmonary embolism, ischemic stroke, or myocardial infarction within 3 months before admission were excluded from the study. Additionally, patients with thrombophilic disorders, defined as a previously established diagnosis of hereditary or acquired thrombophilia documented in medical records, were excluded.

In patients fulfilling the inclusion criteria, the CVT scoring system based on the study by Heldner *et al.,* [3] was applied. They were informed of the need for an additional D-dimer test with values above 500 µg/L considered elevated (done under Department of Biochemistry of the Institute). After neurological examination, patients underwent non-contrast CT of the brain to rule out other types of strokes and to detect direct and indirect signs of CVT. Clinical scoring was performed based on 6 variables, as shown in Table 1.

The clinical score was applied prospectively at the time of presentation in the ED, before any neuroimaging was performed. The MRV findings were interpreted independently by neuroradiologists who were blinded to the clinical score, and the results were compared with the clinical scoring and D-dimer levels. The association between anemia and regular alcohol intake with CVT was also studied [16]. The WHO definition of anemia was used, with hemoglobin levels below 13 g/dL in men and below 12 g/dL in women considered anemic. The patient flow diagram is shown in Figure 1.

Table 1. Variables used in clinical scoring

Clinical variable	Score
Seizures at presentation	4
Known thrombophilia [a]	4
Use of Oral contraceptives	2
Duration of symptoms > 6 days	2
Worst headache ever	1
Focal neurological deficit	1
Score based probability	
Probability	Score
Low CVT probability	0-2 points
Moderate CVT probability	3-5 points
High CVT probability	> 6 points

[a]Thrombophilic disorders were defined as a previously established diagnosis of hereditary or acquired thrombophilia, documented in medical records.

Data were entered into an Excel sheet and analyzed using SPSS software Version 28. Categorical variables were presented as numbers and percentages (%), and continuous variables were presented as mean±SD and median (IQR) depending on the distribution of the data after assessing normality by the Kolmogorov-Smirnov test. The sensitivity and specificity of the clinical scores in the predicting CVT, and the effect of adding D-dimer to increase the predictability of diagnosing CVT, were analyzed. Receiver operating characteristic (ROC) analysis was performed for D-dimer and total clinical score in relation to CVT (Figure 2). The correlation between D-dimer and clinical score was analyzed using the Spearman correlation coefficient, and a p-value < 0.05 was considered statistically significant.

Results

A total of 75 patients were included in the study. The frequency of CVT, as confirmed by MRV, was 53.3%.

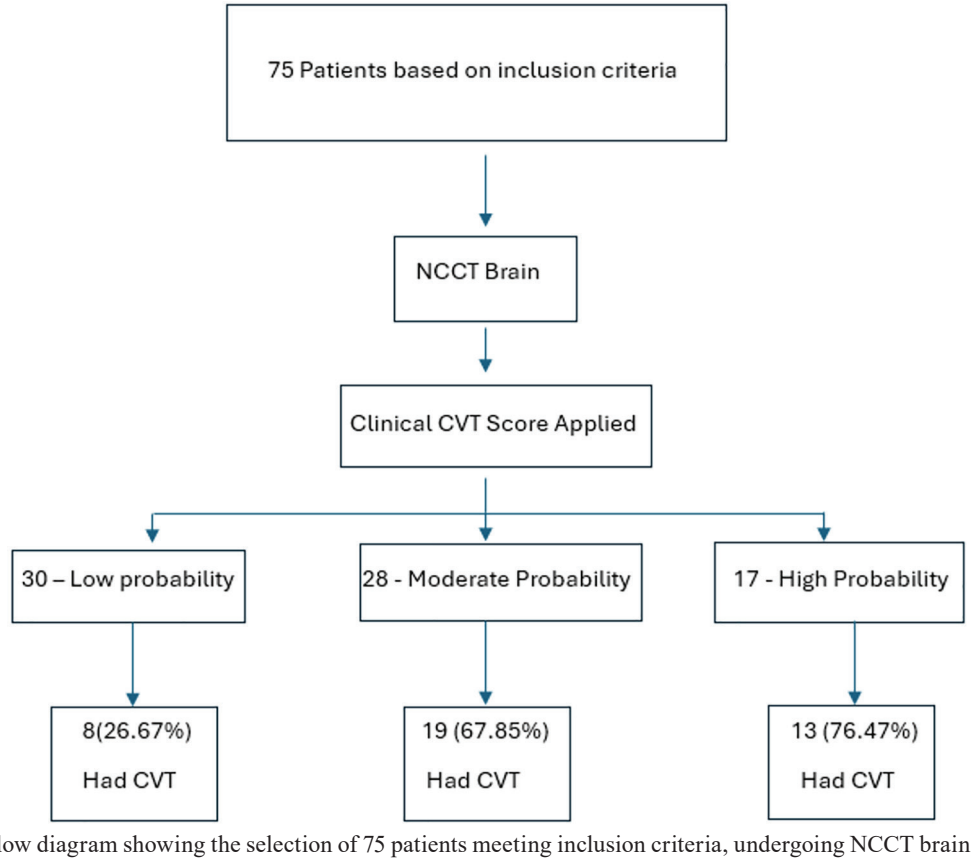

Fig. 1. Patient flow diagram showing the selection of 75 patients meeting inclusion criteria, undergoing NCCT brain and application of the clinical CVT score and their distribution into low (n=30), moderate (n=28) and high (n=17) CVT probability groups with corresponding proportions ultimately diagnosed with CVT (26.6%, 67.8% and 76.4%, respectively). NCCT: Non-contrast computed tomography; CVT: Cerebral venous thrombosis

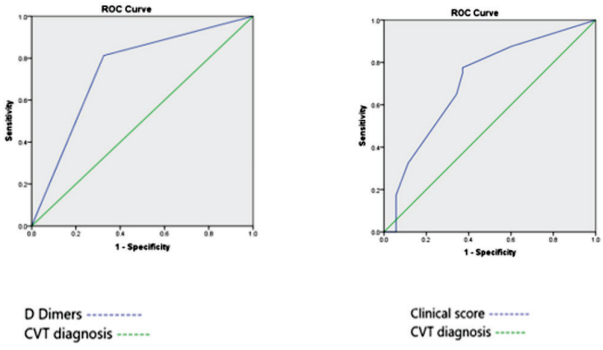

Fig. 2. ROC curves for D-dimers and clinical scores in CVT

Among the other diagnoses, 18.6% of patients were diagnosed with acute intracerebral haemorrhage, while 12% had subarachnoid hemorrhage. Of note, 8% of patients were diagnosed with acute ischemic stroke. There was one case each of chronic infarction, hydrocephalus, posterior reversible encephalopathy syndrome (PRES), subdural empyema, and tuberculoma. In one case, a definitive diagnosis could not be made.

The mean age of patients with CVT was 37.8±11 years, and the mean age of patients with causes other than CVT was 41.57±14 years (p=0.21). Notably, 60% of the patients diagnosed with CVT were less than 40 years of age. In our study, 41 patients were men (54.66%), and 34 were women (45.33%). A total of 61% of male patients had CVT, whereas only 44.1% of females had CVT. This might be attributable to the exclusion of pregnant women from the study.

Regarding seizure presentation, the number of CVT patients was significantly higher than that of non-CVT patients (28 [70%] CVT patients vs. 12 [34%] non-CVT patients, p=0.003]. However, no statistically significant difference was observed between the CVT and non-CVT groups concerning thrombophilic disorders (1 [2%] CVT patients vs. 3 [8.5%] non-CVT patients, p=0.33]. Moreover, we found a substantial difference in the history of oral contraceptive (OCP) use between CVT and non-CVT patients (5 [12.5%] CVT patients vs. 0 [0%] non-CVT patients). There were no cases of OCP use in the non-CVT group.

Table 2. Demographic, clinical, and laboratory characteristics of study participants

Parameter	CVT (n=40)	non-CVT (n=35)	*p*-value
Age			
Less than 40 years	24	16	0.21
More than 40 years	16	19	
Total	40	35	
Mean Age [a]	37.8±*11*	41.57±13.9	
Sex			
Male	25	16	0.16
Female	15	19	
Total	40	35	
Seizures at presentation			
Yes	28	12	0.003
No	12	23	
Total	40	35	
History of thrombophilia			
Yes	1	3	0.33
No	39	32	
Total	40	35	
History of contraceptive usage			
Yes	5	0	0.05
No	35	35	
Total	40	35	
History of worst ever headache			
Yes	28	30	0.16
No	12	5	
Total	40	35	
Focal neurological deficits			
Yes	13	12	0.9
No	27	23	
Total	40	35	
D-dimer levels (µg/mL)			
More than 500	26	6	<0.001
Less than 500	14	29	
Total	40	35	

a: Mean age is represented as the mean±standard deviation.

Table 3. Association between clinical score and D-dimer values with CVT

Variable	Area	SE	95% CI		*p*-value
			Lower	Upper	
Clinical score	0.709	0.06	0.58	0.83	<0.001*
D-Dimer	0.743	0.05	0.62	0.85	<0.001*

Table 4. Diagnostic performance of clinical probability levels and D-dimer in CVT

Clinical Probability	Sensitivity(%)	Specificity(%)	PPV (%)	NPV (%)	P-value
Low probability (0–2)	73.3	71.1	62.9	80	<0.001
Low probability + D-dimer <500 µg/L	92.5	54.3	65.5	86.4	<0.001
Moderate probability (3–5)	47.5	74.3	67.9	55.3	0.06
High probability (≥6)	32.5	88.6	76.5	53.4	0.05

PPV: Positive predictive value; NPV: Negative predictive value

Therefore, a statistical significance between CVT and consumption of OCPs was not found in our study (p=0.05). The OCP was observed exclusively in the CVT group (5 vs. 0). Although this showed a strong association with CVT, statistical significance was borderline (P=0.05), likely due to THE sparse data.

Headache was a common symptom, and 48.3% of patients who presented with the worst headache of their life were diagnosed with CVT. Clinically, headache appreaed to be equally common in both groups (CVT and non-CVT). Therefore, the statistical significance was not achieved in our study population (p=0.16). Only 25 patients (33.3%) had a neurological deficit at the time of ED presentation, and among them, 13 were diagnosed with CVT, a finding that was statistically insignificant (p=0.9). A detailed demographic, clinical, and laboratory profile of the study participants is presented in Table 2.

A total of 32 patients showed a significant increase in D-dimer values, among whom 65 % had CVT, while 19% had elevated values in the non-CVT group. This was a significant observation (p<0.001). Furthermore, a D-dimer levels >500 µg/L identified CVT with 65% sensitivity and 82.8% specificity, with a positive predictive value of 81.2% and a negative predictive value of 67.4%. D-dimer alone should not be used to rule out CVT, but the combination of low clinical probability and a negative D-dimer might be useful.

The ROC curve (Figure 2) and analysis in Table 3 showed that the clinical score had an area under the curve (AUC) of 0.70, with a standard error of 0.06 and a 95% confidence interval of 0.58 to 0.82, indicating a moderate ability to differentiate CVT from non-CVT cases. This association was statistically significant (p<0.001). Similarly, D-dimer demonstrated a slightly higher diagnostic performance, with an AUC of 0.74, a standard error of 0.05, and a 95% confidence interval of 0.62 to 0.85, which was also statistically significant (p<0.001). These findings supported the use of clinical scoring for initial risk stratification, with D-dimer serving as a valuable adjunct to improve diagnostic confidence and guide early neuroimaging in patients with suspected CVT.

Based on the clinical scoring system used in our study, a score of 0-2 points was defined as indicating a low probability of CVT. Thirty of the 75 patients (40%) fell into this category, of whom 22 did not have CVT. As shown in Table 4, the low-probability category demonstrated a high sensitivity (73.3%) and negative predictive value (80%), indicating its usefulness in ruling out CVT. When low clinical probability was combined with a D-dimer level <500 µg/L, diagnostic performance improved substantially, with sensitivity increasing to 92.5% and NPV to 86.36%, highlighting the added value of D-dimer in safely excluding CVT.

A clinical score of 3-5 points was considered to indicate a moderate probability of CVT and was observed in 28 patients (37.3%). Among these, 19 patients (67.8%) were diagnosed with CVT; however, this association did not reach statistical significance (p=0.06). Correspondingly, the moderate-probability group showed intermediate sensitivity and specificity in Table 4, reflecting its limited ability to rule in or rule out CVT independently. Notably, a majority of CVT patients in this group (13 of 19) had D-dimer levels greater than 500 µg/L, suggesting that biomarker elevation might help refine risk stratification within this intermediate category.

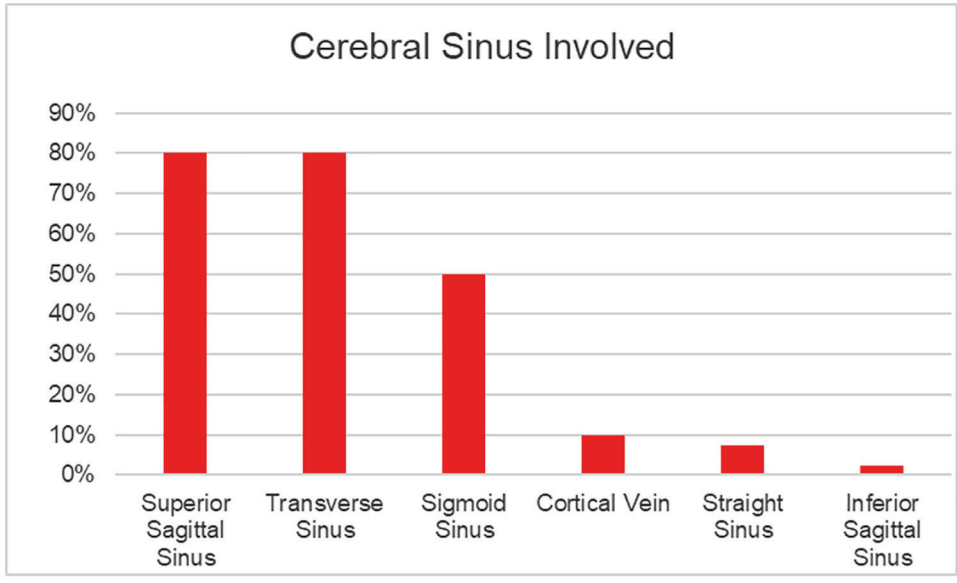

Fig. 3. Cerebral venous sinuses involved

Scores of 6-14 points defined the high-probability group, which included 17 patients (22.6%). Of these, 13 patients (76.4%) were confirmed to have CVT, yielding a statistically significant association (p=0.05). As presented in Table 4, the high-probability group demonstrated high specificity (88.6%) and positive predictive value (76.5%), supporting its role in ruling in CVT. A significant positive correlation was also observed between the total clinical score and D-dimer levels in patients diagnosed with CVT, further supporting the biological plausibility of the scoring system.

Although the high-probability group showed a statistically significant association with CVT, the moderate-probability group did not. This finding should be interpreted with caution, as the limited sample size and small subgroup distributions might have reduced the statistical power to detect a true association. Overall, the diagnostic performance metrics summarized in Table 4 demonstrate the complementary role of clinical probability scoring and D-dimer testing: a low probability combined with a low D-dimer effectively excludes CVT, while a high probability strongly predicts its presence. Figure 3 illustrates the cerebral venous sinus involved.

Discussion

Recent studies have shown that the incidence of CVT may be as high as 1.32-1.57 per 100,000 person-years [17, 18]. Increased awareness and the availability of diagnostic methods have led to a rise in thereported incidence of CVT. In our study, out of 75 suspected CVT patients, 40 were confirmed to have CVT. The condition is predominantly seen in young and middle-aged adults. CVT is three times more common in women compared to men, and earlier studies reported an increased incidence during pregnancy and postpartum period, with a higher incidence in the third trimester and the first 6 weeks after delivery [5, 19]. Predisposing conditions include prothrombotic states, infections, trauma, vasculitis, malignancies, and dehydration [20].

Our sample size (n=75) was adequate for the initial validation of the scoring system; however, it led to smaller subgroup distributions, which might limit the generalizability of the present study. A major limitation was the exclusion of pregnant and postpartum women, despite these groups being well-established high-risk populations for CVT. Their exclusion restricted the generalizability of our findings and might have contributed to the higher proportion of CVT among men in our sample. Among the 34 female patients in our study, five had a history of OCP intake. The OCP use was observed exclusively in the CVT group. Although this finding showed a strong association, statistical significance was borderline, likely due to sparse data. Earlier studies stated that OCPs were one of the most common predisposing factors of dural sinus thrombosis [21]. Amoozegar *et al.,* reported that the incidence of CVT in females taking OCP was seven-fold higher than in the rest of the female population [22].

CVT has varied clinical presentations. Headache is the most common and least specific of all presentations, making the diagnosis challenging. In our study, 70% of the patients diagnosed with CVT had a headache, often describing it as the worst headache of their life. The pathophysiological mechanisms of headache in CVT are raised intracranial pressure (ICP), stretching of nerve fibers in the walls of the occluded sinuses, and local inflammation caused by mediators released from the clot. Seizures are more frequently observed in CVT than in arterial stroke, with a reported frequency of 35-50% [23, 24]. Our study showed that 70% of the patients with CVT presented with seizures. Focal neurological deficits were the next most common presentation in our study, which was in concordance with earlier studies [1, 5].

Our study utilized a clinical score previously developed in the study by Heldner *et al.,* [3]. Heldner *et al.,* demonstrated that the performance of the score significantly improves when combined with D-dimer levels, and our results closely mirror this pattern. In our study, the predictive value of the CVT score in the low probability group was further increased from 73.3% to 92.5% by adding serum D-dimer levels. Thirty out of 75 patients (40%) had a low pre-test probability score for CVT. Post-MRV, 73.3% of patients in the low-probability group were diagnosed as not having CVT, which implies that the scoring system can be used as a pretest score to rule out CVT if the score values is less than and equal to 2. Eight out of the thirty patients in the low-probability group were diagnosed with CVT, and five of these patients had D-dimer levels greater than 500 μg/L.

In our study, 68% of the patients in the moderate-probability group had CVT. D-dimer assays are known to be highly sensitive, but their specificity is moderate to low. They often yield false positive results in conditions such as malignancy, recent surgery, trauma, other acute illnesses, renal failure, pregnancy or postpartum state. Although D-dimer alone is highly sensitive, its specificity is variable. Several studies suggested that integrating clinical pre-test probability with a biochemical marker such as D-dimer strengthens diagnostic accuracy [3, 15]. Six out of 35 patients not diagnosed with CVT had elevated D-dimers. Plasma D-dimer values, when added to the low-probability group, were found to increase the probability of ruling out CVT. However, we could not statistically confirm the role of D-dimer in the patient population with moderate to high probability scores. Based on our statistical analysis, high plasma levels of D-dimer were not specific for diagnosing CVT. Among patients in the moderate to high-probability group, a substantial proportion had elevated D-dimer levels; however, this did not reliably discriminate between CVT and non-CVT diagnoses. Overall, six of the 35 patients without CVT had elevated D-dimer levels, reflecting the known limitation of false-positive results.

Alcoholism predisposes individuals to cerebrovascular accidents. Regarding CVT, some studies suggest dehydration and resultant hyperviscosity induced of blood induced by heavy alcohol consumption as a possible mechanism for thrombosis [25]. However, we did not find a statistical correlation between the two variables. Anemia is often considered a risk factor for CVT [26]. Iron-deficiency anemia can contribute to a hypercoagulable state and predispose individuals to CVT, especially in children [27]. Iron-deficient red cells have a structural deformity that can result in turbulent flow, activating the coagulation cascade and promoting thrombi formation. However, we did not find any statistically significant correlation between anemia and CVT in our study population.

The study had several limitations. First, it was conducted in a single emergency department, and therefore, the patient characteristics might differ across different centers. Moreover, we had a small sample size, and the spectrum of patient population was narrow. The subjective judgment of the patient can influence the CVT clinical score. Hence, the overall reliability of identifying CVT based on the scoring can sometimes be fallacious. The representation of pregnant and postpartum women was low in our study. Therefore, targeted studies should focus on underrepresented groups, such as pregnant and postpartum females, to address demographic-specific pathophysiological differences and outcomes. Another limitation was the inability to statistically confirm the independent role of D-dimer in patients with moderate and high clinical probability scores, which is likely attributable to the limited sample size and small subgroup distributions, resulting in reduced statistical power. We were also unable to associate thrombophilia with the diagnosis of CVT, as a majority of our patients had never been diagnosed with thrombophilia before the present episode. The score is not meant to replace the diagnostic imaging.

In conclusion, it is possible to apply a simple score and estimate D-dimer levels during emergency hours in patients with a history suggestive of CVT. A low probability based on the score will require additional D-dimer testing to rule out CVT. Based on the clinical score, moderate- and high-probability scores were associated with a higher likelihood of CVT. However, they did not demonstrate sufficient specificity or positive predictive value to definitively rule in the diagnosis. They help clinicians prioritize patients for urgent imaging but cannot be used as a substitute for MRV. Clinical scoring, in conjunction with D-dimer levels, might help support triage decisions in a hospital with access to MRV. The score cannot replace the gold standard investigation, MRV; however, it can assist in early triaging and identifying patients with probable CVT.

Declaration

Ethics approval and consent to participate: Written prior approval obtained from the Institutional Ethics Committee of Nizam's Institute of Medical Sciences, Hyderabad, India (reference number 1106/2021). We confirmed that all methods were performed in accordance with the ethical standards outlined in the Declaration of Helsinki and its later amendments or comparable ethical standards. Written informed consent was obtained from each patient.

Consent for publication: Consent from all authors has been obtained, and the consent form is attached.

Conflict of Interest: The authors declared no conflicts of interest with respect to the research, authorship, and publication of this article.

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Data availability: The datasets used and/or analyzed during the present study are available from the corresponding author upon reasonable request.

Author's Contribution: CAAR: Study conception and design and data acquisition; AS: Data analysis, drafting, and revision; IN: Fata interpretation and supervision; SAJ: Editing and Proof-editing.

References

1. Bousser MG, Ferro JM. Cerebral venous thrombosis: an update. *Lancet Neurol*. 2007;**6**(2):162-70.
2. Ferro JM, Canhão P, Stam J, Bousser MG, Barinagarrementeria F, Massaro A, et al. Delay in the diagnosis of cerebral vein and dural sinus thrombosis: influence on outcome. *Stroke*. 2009;**40**(9):3133-8.
3. Heldner MR, Zuurbier SM, Li B, Von Martial R, Meijers JC, Zimmermann R, et al. Prediction of cerebral venous thrombosis with a new clinical score and D-dimer levels. *Neurology*. 2020;**95**(7):e898-909.
4. Stam J. Thrombosis of the cerebral veins and sinuses. N Engl J Med. 2005;352(17):1791-8.
5. 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**(3):664-70.
6. Mangshetty B, Reddy KN. Clinical and neuroimaging correlation in patients with cerebral sinus venous thrombosis. *Al Ameen J Med Sci*. 2015; **8**(1):64-71
7. 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.
8. Tanislav C, Siekmann R, Sieweke N, Allendörfer J, Pabst W, Kaps M, et al. Cerebral vein thrombosis: clinical manifestation and diagnosis. *BMC Neurol*. 2011; 11:69
9. van Dam LF, van Walderveen MAA, Kroft LJM, Kruyt ND, Wermer MJH, van Osch MJP, et al. Current imaging modalities for diagnosing cerebral vein thrombosis: a critical review. *Thromb Res*. 2020; *189*:132-9.
10. Idbaih A, Boukobza M, Crassard I, Porcher R, Bousser MG, Chabriat H. MRI of clot in cerebral venous thrombosis: high diagnostic value of susceptibility weighted images. *Stroke*. 2006;37(4):991-5.
11. 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): S19-3.
12. Weitz JI, Fredenburgh JC, Eikelboom JW. A test in context: D-dimer. *J Am Coll Cardiol*. 2017;**70**(19):2411-20.
13. Meng R, Wang X, Hussain M, Dornbos D 3rd, Meng L, Liu Y, et al. Evaluation of plasma D-dimer plus fibrinogen in predicting acute CVST. *Int J Stroke*. 2013;**9**(2):166-73.
14. Kosinski CM, Mull M, Schwarz M, Koch B, Biniek R, Schläfer J, et al. Do normal D-dimer levels reliably exclude cerebral sinus thrombosis? *Stroke*. 2004;**35**(12):2820-5.
15. Hiltunen S, Putaala J, Haapaniemi E, Salonen O, Tatlisumak T. D-dimer and clinicoradiologic features in cerebral venous thrombosis. *J Neurol Sci*. 2013;**327**(1-2):12-4.
16. Singh H, Paulomi T, Ray S, Gupta V. A rare presentation of acute alcohol intoxication, subarachnoid haemorrhage, and cortical venous thrombosis. *J Neurol Res*. 2012; **2**(2):62-4.
17. Silvis SM, de Sousa DA, Ferro JM, Coutinho JM. Cerebral venous thrombosis. Nat Rev Neurol. 2017;13(9):555-65.
18. Devasagayam S, Wyatt B, Leyden J, Kleinig T. Cerebral venous sinus thrombosis incidence is higher than previously thought. *Stroke*. 2016;**47**(9):2180-2.
19. Silvis SM, Lindgren E, Hiltunen S, Devasagayam S, Scheres LJ, Jood K, et al. Postpartum period is a risk factor for cerebral venous thrombosis. *Stroke*. 2019;**50**(2):501-3.
20. Silvis SM, Middeldorp S, Zuurbier SM, Cannegieter SC, Coutinho JM. Risk factors for cerebral venous thrombosis. *Semin Thromb Hemost*. 2016;**42**(6):622-31.
21. Saadatnia M, Tajmirriahi M. Hormonal contraceptives as a risk factor for cerebral venous and sinus thrombosis. *Acta Neurol Scand*. 2007;**115**(5):295-300.
22. Amoozegar F, Ronksley PE, Sauve R, Menon BK. Hormonal contraceptives and cerebral venous thrombosis risk: a systematic review and meta-analysis. *Front Neurol*. 2015;**6**:7.
23. Masuhr F, Busch M, Amberger N, Ortwein H, Weih M, Neumann K, et al. Risk and predictors of early epileptic seizures in acute cerebral venous and sinus thrombosis. *Eur J Neurol*. 2006;**13**(8):852-6.
24. Ferro JM, Correia M, Rosas MJ, Pinto AN, Neves G. Seizures in cerebral vein and dural sinus thrombosis. *Cerebrovasc Dis*. 2003;**15**(1-2):78-83.
25. Prasad P. Alcohol : A provocating factor in male cerebral venous thrombosis – A Prospective Analysis In Western Rajasthan. *International Journal Of Scientific Research*. 2022; 59-61.
26. Saadatnia M, Fatehi F, Basiri K, Mousavi SA, Mehr GK. Cerebral venous sinus thrombosis risk factors. *Int J Stroke*. 2009;**4**(2):111-23.
27. Maguire JL, deVeber G, Parkin PC. Association between iron-deficiency anemia and stroke in young children. *Pediatrics*. 2007;**120**(5):1053-7.