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ORIGINAL ARTICLE

Evaluation of risk factors and clinical and radiological characteristics in cortical vein thrombosis accompanying cerebral venous sinus thrombosis

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ABSTRACT

Objectives: The study aimed to determine the clinical relevance of cortical vein thrombosis (CVT) accompanying cerebral venous sinus thrombosis (CVST) and to investigate the risk factors.

Patients and methods: This retrospective study included 176 patients (52 males, 124 females; mean age: 41.8±14.5 years; range, 16 to 72 years) with CVST between January 2015 and January 2021. The radiological, demographic, and clinical features and risk factors of patients with and without CVT were com-pared.

Results: When the clinical demographic and radiological features associated with CVT were evaluated; there was a significant relationship between age, clinical onset symptom, connective tissue disease, post-partum period, presence of hypercoagulopathy, superior sagittal sinus (SSS) thrombosis, transverse sinus thrombosis, sigmoid sinus thrombosis, non-hemorrhagic venous infarct, small juxtacortical hemorrhage and large parenchymal hematomas, time to diagnosis (days), and poor clinical outcome. When significant risk factors for CVT were analyzed by binary logistic regression, the post-partum period and SSS thrombosis were independent risk factors.

Conclusion: In this study, the rate of CVT accompanying CVST was 30.7%, which is higher than the studies in the literature. Cortical vein thrombosis accompanying CVST is associated with parenchymal lesions and poor clinical outcome; SSS thrombosis and the postpartum period are independent risk factors. Multicenter prospective studies are recommended for more precise information.

Keywords: Cranial, neurological deficits, puerperal disorders, venous sinus thrombosis.

Cerebral venous sinus thrombosis (CVST) is a cerebrovascular disease that can cause morbidity and mortality by leading to damage in the brain parenchyma.[1] The incidence of CVST is 5 to 16 per million.[2,3] The clinical presentation of CVST is variable.[4] In CVST, the occlusion of the cerebral sinuses disrupts the drainage of the cerebrospinal fluid (CSF), which can lead to signs of intracranial hypertension. In cortical vein thrombosis (CVT), the disruption of cortical drainage increases capillary hydrostatic pressure, resulting in dilated vessels, petechial hematoma, ischemic neuronal damage, intraparenchymal hematoma, and vasogenic edema. When CVT accompanies CVST, the clinical presentation may include focal neurological deficits.[5-7]

The frequency of CVT accompanying CVST was determined to be 17.1%; however, due to the small size of the cortical venous structures, diagnosing CVT can be challenging.[8] With the advancement of modern imaging technologies in recent years, CVT has become more widely recognized. A limited number of studies in the literature examine whether CVT accompanies CVST, and the clinical significance

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of accompanying CVT is not clearly known. Hence, this study aimed to determine the clinical significance of CVT accompanying CVST and to investigate the risk factors.

PATIENTS AND METHODS

This retrospective study included 176 patients (52 males, 124 females; mean age: 41.8±14.5 years; range, 16 to 72 years) with CVST followed at the neurology department of the Uludağ University, Faculty of Medicine, between January 2015 and January 2021. Patient consent was not required due to the retrospective design. The inclusion criteria for the study were defined as being diagnosed with CVST by contrast-enhanced cranial magnetic resonance imaging (MRI) and magnetic resonance venography, completing all investigations regarding the etiology of CVST, completing a monthly follow-up of three months after diagnosis at the stroke clinic of the Uludağ University, Faculty of Medicine, and having documented clinical outcomes. The exclusion criteria were defined as the absence of noncontrast cranial computed tomography (CT), contrast-enhanced cranial MRI, and magnetic resonance venography and the inability to determine clinical outcomes at three months due to incomplete monthly follow-up. Clinical and radiological data of the patients included in the study were obtained from the hospital's automation system. The initial complaints, symptom durations, neurological examination findings, and medical histories of the patients were evaluated by a neurology specialist during their emergency department visit and recorded in the discharge summaries. The initial symptoms were evaluated in three categories: intracranial hypertension, focal neurological deficit, and epileptic seizure. All patients were investigated for Behçet's disease, connective tissue diseases, and causes of hypercoagulopathy regarding the etiology of CVST, and treatment was arranged according to the European Stroke Organization's cerebral venous thrombosis guidelines.[9] The clinical outcome of the patients, who were regularly followed and treated in the neurology outpatient clinic after discharge, was evaluated at three months using the modified Rankin Scale (mRS). The mRS scores were dichotomized as follows: scores of 0 to 2 were considered a good clinical outcome, and scores of 3 to 6 were considered a poor clinical outcome.[10]

The cranial CT, MRI, and magnetic resonance venographies of the patients were evaluated by a neuroradiologist who was blinded to the clinical findings. Whether there was parenchymal damage secondary to venous occlusion was determined on noncontrast cranial CT or T2-weighted MRI images. Parenchymal damages were classified into two categories: hemorrhagic and nonhemorrhagic venous infarcts. Hemorrhagic venous infarcts were further classified into four groups: small juxtacortical hemorrhages, subarachnoid hemorrhages, large parenchymal hematomas, and subdural hematomas.[11,12]

Patients with CVST were compared based on the presence or absence of accompanying CVT regarding demographic, clinical, and radiological characteristics, as well as predisposing and precipitating factors.

Statistical analysis

Statistical analysis was performed using the IBM SPSS version 25.0 software (IBM Corp., Armonk, NY, USA). To determine whether the data conformed to a normal distribution, the Shapiro-Wilk test, histograms, and Q-Q plots were utilized. Continuous variables were expressed as mean and standard deviation, while categorical variables were presented as frequency and percentage. The Mann-Whitney U test was used to compare age and symptom duration between groups. Categorical variables were compared using Fisher exact test, Pearson's chi-square test, and the continuity-corrected chi-square test. Independent risk factors for CVT in patients with CVST were determined using binary logistic regression analysis. A p-value <0.05 was considered statistically significant.

RESULTS

The mean age of female patients was 40.7±13.2 years, and the mean age of male patients was 44.6±16.9 years. There was no statistically significant difference in age between female and male patients (p>0.05).

The symptoms at clinical onset were complaints related to intracranial hypertension in 121 (68.7%) patients, focal neurological deficits in 27 (15.4%) patients, and epileptic seizures in 28 (15.9%) patients.

The distribution of patients according to the thrombosed sinus structures was as follows: superior sagittal sinus (SSS) in 70 (39.8%) patients, transverse sinus in 128 (72.7%) patients, sigmoid sinus in 103 (58.5%) patients, inferior sagittal sinus in 11 (6.2%) patients, jugular vein in 54 (30.7%) patients, and straight sinus in nine (5.1%) patients. Fifty-four (30.7%) patients had CVT accompanying CVST. There were no patients with isolated CVT.

The distribution of parenchymal lesions was as follows: nonhemorrhagic venous infarcts in 28 (15.9%) patients, small juxtacortical hemorrhages in 22 (12.5%) patients, subarachnoid hemorrhages in five (2.8%) patients, and large parenchymal hematomas in 12 (6.8%) patients.

In the evaluation of predisposing and precipitating factors for CVST, 25 (14.2%) patients had connective tissue diseases, 16 (9.1%) patients had Behçet's disease, 14 (7.9%) patients were using oral contraceptives, and 53 (30.1%) patients had hypercoagulopathy [19 (10.79%) patients with factor V Leiden mutation, 38 (21.59%) patients with methylenetetrahydrofolate reductase gene mutation, 37 (21.02%) patients with plasminogen activator inhibitor gene mutation, and four (2.27%) patients with factor II gene mutation]. Additionally, 21 (11.9%) patients were in the puerperium, five (2.8%) patients were pregnant, malignancy was present in 23 (13.1%) patients, infection was present in 10 (5.7%) patients, and 22 (12.5%) patients had a history of lumbar puncture. At three months, 36 (20.5%) had poor clinical outcomes, whereas 140 (79.5%) had good clinical outcomes.

There was a statistically significant relationship between accompanying CVT and age (p=0.024), initial clinical symptoms (p<0.001), presence of connective tissue disease (p=0.042) and hypercoagulopathy (p=0.008), puerperium (p<0.001), superior sagittal (p=0.001), transverse (p=0.049),

TABLE 1
Demographic and clinicoradiological characteristics of patients with CVST with and without accompanying CVT

	CVST patients with CVT (n=43)			CVST patients without CVT (n=81)			
	n	%	Mean±SD	n	%	Mean±SD	*p*
Age (year)*			38.0±13.8			43.5±14.5	**0.024**
Sex							
Female	43	79.62		81	66.39		>0.05
Clinical onset†							
Isolated intracranial hypertension	32	59.25		108	8.52		**<0.001**
Focal neurological deficit	35	64.81		16	13.11		**<0.001**
Epileptic seizure	34	62.96		15	12.29		**<0.001**
Risk factors†							
Behçet's disease	2	3.70		14	11.47		>0.05
Vasculitis	3	5.55		22	18.03		**0.042**
Hypercoagulopathy	23	42.59		30	24.59		**0.008**
Oral contraceptive use	7	12.96		7	5.73		>0.05
Pregnancy	2	3.70		3	2.45		>0.05
Postpartum period	16	29.62		6	4.91		**<0.001**
Infection	2	3.70		8	6.55		>0.05
Head trauma, lumbar puncture, mechanic factors	7	12.96		15	12.29		>0.05
Malignancy	5	9.25		18	14.75		>0.05
Thrombosed structure†							
Superior sagittal sinus	34	62.96		36	29.50		**<0.001**
Transverse sinus	33	61.11		95	77.86		**0.049**
Sigmoid sinus	23	42.59		80	65.57		0.020
Straight sinus	5	9.25		4	3.27		>0.05
Inferior sagittal sinus	5	9.25		6	4.91		>0.05
Jugular vein	12	22.22		42	34.42		>0.05
Parenchymal lesions							
Nonhemorrhagic venous infarct†	17	31.48		11	9.01		**<0.001**
Hemorrhagic infarct†	33	61.11		5	4.09		**<0.001**
Small juxtacortical hemorrhage†	21	38.88		1	0.81		**<0.001**
Subarachnoid hemorrhage†	3	5.55		2	1.63		>0.05
Subdural hematoma†	0	0.00		1	0.81		>0.05
Large parenchymal hematoma†	10	18.51		2	1.63		**<0.001**
Time to diagnosis (day)*			3.90±5.37			38.17±61.54	**<0.001**
Clinical outcome (poor clinical outcome)†	24	44.44		12	9.83		**<0.001**

CVST: Cerebral venous sinus thrombosis; CVT: Cortical vein thrombosis; SD: Standard deviation. Significant differences were shown in bold. * Mann-Whitney U test; † Pearson' chi-square test/continuity-corrected chi-square test/Fisher exact test.

TABLE 2
Evaluation of significant risk factors for CVT in patients with CVST using binary logistic regression analysis

			95% CI	
	p	Odds ratio	Low	High
Age	0.929	0.999	1.027	0.929
Superior sagittal sinus thrombosis	**0.008**	2.798	1.301	6.021
Transverse sinus thrombosis	0.989	0.993	0.374	2.641
Sigmoid sinus thrombosis	0.118	0.505	0.214	1.190
History of rheumatologic disease	0.269	0.472	0.125	1.786
Being in the postpartum period	**0.021**	3.715	1.216	11.353
Presence of hypercoagulopathy	0.073	2.133	0.932	4.881

CVST: Cerebral venous sinus thrombosis; CVT: Cortical vein thrombosis; CI: Confidence interval; Significant differences were shown in bold. Model significance: p<0.001.

and sigmoid (p=0.020) sinus thrombosis, presence of nonhemorrhagic venous infarct (p=0.001), small juxtacortical hemorrhage (p<0.001), and large parenchymal hematoma (p<0.001), symptom duration (p<0.001), and poor clinical outcome (p<0.001). There was no statistically significant relationship between CVT and sex, Behçet's disease, oral contraceptive use, pregnancy, infection, head trauma, malignancy, presence of straight sinus, inferior sagittal sinus, and jugular vein thrombosis, and presence of subarachnoid hemorrhage and subdural hematoma (p>0.05, Table 1).

Binary logistic regression analysis revealed that puerperium (p=0.021) and SSS thrombosis (p=0.008) were independent risk factors for CVT (Table 2).

DISCUSSION

To date, the presence of CVT accompanying CVST has been the subject of a limited number of studies. In this study, 30.7% of patients were found to have CVT accompanying CVST, which is higher than a previously reported rate.[6] A possible reason for this discrepancy is that contrast-enhanced magnetic resonance venography is required for the diagnosis of CVT, whereas most studies have used noncontrast magnetic resonance venography for diagnosis in contrast to our study.

In the ISCVT (International Study on Cerebral Vein and Dural Sinus Thrombosis), the most frequently thrombosed venous sinus was reported to be the SSS.[6] However, in the VENOST study, the largest cohort study from Türkiye involving 1,144 patients, the most common location of thrombosis was the transverse sinus.[13] Consistent with the VENOST study data, our study also identified the transverse sinus as the most frequently thrombosed sinus.

Our findings indicate that the presence of CVT accompanying CVST is associated with parenchymal lesions. In the presence of CVT, the clinical presentation is more likely to involve focal neurological deficits and epileptic seizures rather than intracranial hypertension, and the likelihood of poor clinical outcomes increases. The formation of parenchymal lesions in CVT can be explained by the drainage of the cortex and subcortical white matter through the superficial medullary veins, which have poor collaterals, into the cortical veins.[14] The occlusion of superficial medullary veins due to CVT can lead to nonhemorrhagic or hemorrhagic venous infarcts.[7,8,15,16] In the absence of accompanying CVT, the good collaterals between the venous sinuses and cortical vessels may protect against the development of parenchymal damage.[4,7,8,15-17] Therefore, efforts should focus on the early diagnosis and treatment of CVST to prevent the spread of thrombi from the cerebral venous sinuses to the cortical veins, reduce the risk of nonhemorrhagic and hemorrhagic venous infarcts, and improve prognosis.[18]

In the current study, SSS thrombosis and puerperium were identified as independent risk factors for CVT accompanying CVST. Pregnancy is a chronic hypervolemic condition, and the hypervolemic state rapidly regresses during the postpartum period. This regression can lead to reduced venous return, decreased blood volume in the veins, and development of stasis.[19] According to Virchow's triad, the reduction in blood flow results in endothelial damage and hypercoagulability.[20]

In our study, 22 patients developed CVST following lumbar puncture. Twenty-one patients were in the postpartum period. Spinal anesthesia was administered to 12 (57.1%) of the postpartum patients during childbirth. Dural puncture performed during spinal anesthesia can lead to intracranial hypotension.[21] Intracranial hypotension can cause the dilation of venous sinuses and a slowing of the flow within the sinuses.[22-24] This mechanism may explain why cortical veins very small in diameter become thrombosed during the puerperium rather than the cerebral sinuses.

This study identified puerperium as an independent risk factor for CVT accompanying CVST, a finding that has not been previously reported. It is known that estrogen increases endothelial nitric oxide synthase via its receptors in cerebral vessels.[25] The rapid decrease of elevated levels of estrogen and progesterone during pregnancy in the puerperium leads to a decrease in endothelial nitric oxide synthase activity, resulting in an increased risk of thrombosis. This process may affect cortical veins more significantly, thereby increasing the risk of CVT.

Our findings indicate that SSS thrombosis is an independent risk factor for CVT. The SSS is a major sinus that drains both cerebral hemispheres and functions as a common midline venous structure receiving blood from the drainage vessels of the cortical hemispheres. There are numerous variations in the venous vessels draining into the SSS, and the number of these vessels range from 13 to 19 for each hemisphere. This number is generally equal on both sides for any given individual. The SSS drains into the transverse and sigmoid sinuses. The most important vessel draining into it, is the vein of Trolard, which connects the superficial middle cerebral vein to SSS. Another cortical branch is the Rolandic vein, which drains the primary motor and sensory cortices.[26-29] The potential reasons why SSS thrombosis is a risk factor for the development of CVT may be its role as the major sinus draining the hemispheres and its drainage of numerous cortical veins.

There was also a difference in the time from symptom onset to diagnosis between patients with and without CVT. A possible reason for this finding is that the presence of CVT leads to the formation of parenchymal lesions, making the clinical presentation more apparent.

The ISCVT study, a pioneer in prospective studies, identified CVT as a poor prognostic factor.[6] In another recent study, CVT accompanying CVST was associated with increased mortality.[30] Our findings support the association of CVT with adverse clinical outcomes.

This study was limited by its retrospective single-center design and the small sample size. Prospective and multicenter studies are needed to reach more definitive conclusions.

In conclusion, it was determined that CVT accompanying CVST is associated with parenchymal lesions and poorer clinical outcomes. Superior sagittal sinus thrombosis and puerperium were identified as independent risk factors for CVT.

Ethics Committee Approval: The study protocol was approved by the Bursa Uludağ University Faculty of Medicine Clinical Research Ethics Committee (date: 01.08.2023, no: 2023-15/22). The study was conducted in accordance with the principles of the Declaration of Helsinki.

Patient Consent for Publication: A written informed consent was obtained from each patient.

Data Sharing Statement: The data that support the findings of this study are available from the corresponding author upon reasonable request.

Author Contributions: Idea/concept, design, references and fundings, materials: Y.D.; Control/supervision, critical review: M.B., B.H.; Analysis and/or interpretation, literature review: R.Ö.; Writing the article: R.Ö., Y.D.

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