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Intracranial venous collaterals in cerebral venous thrombosis: clinical and imaging impact.

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Introduction

The underlying mechanism of symptomatic recovery in patients with cerebral venous thrombosis (CVT) remains poorly understood. It is conceivable that the role of intracranial venous collaterals is to prevent venous stasis, thrombus propagation, cerebral edema, venous infarct, and intracerebral hemorrhage. Parenchymal lesions are an unfavorable prognostic sign and will influence recovery.[However, only a few studies have examined collateral formation in patients with CVT. Therefore, the aim of this study was to analyze the influence of the baseline intracranial collateral pattern on the presence and severity of parenchymal lesions (infarcts of hemorrhages) and on the clinical outcome, controlling for known prognostic factors.

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Materials and Methods

Magnetic resonance images from consecutive patients with acute CVT were retrospectively analyzed. The category system described by Qureshi was used to assess the pattern of venous collaterals. Clinical and imaging features and outcomes were analyzed using bivariate and multivariate models to assess the association of collateral patterns with the type of parenchymal lesion and clinical outcome (modified Rankin Scale: mRs) at 30 and 90 days.

MRI and MRV images were evaluated jointly by a board-certified neuroradiologist (CM, with 6 years of neuroimaging review experience) and 2 stroke neurologists (AA, with 20 years of neuroimaging review experience, and MAB, with 5 years of neuroimaging review experience) on a high-resolution monitor.

We performed bivariate (two-tailed chi-square statistics with Yates correction) and multivariate (logistic regression; p in 0.05, p out 0.1) analyses adjusted for potential modifiers such as gender, age, GCS, malignancy and CNS infection. The fitness of the model was evaluated using the Hosmer-Lemeshow goodness-of-fit test, which was considered reliable if $p>0.2$

Results

One hundred patients were included (77 women; median age 32 years; and median of 18 months of follow-up). Venous collaterals were present in 88% of the patients; type I collaterals in 3 patients; type II collaterals in 27 patients; and type III collaterals in 58 patients. Twelve patients did not exhibit any collaterals. Cohen's Kappa coefficient between evaluators was 0.86. In the bivariate analysis, type III collaterals were associated with isolated intracranial hypertension and complete recovery, whereas type I collaterals were associated with encephalopathy. However, in the multivariate regression analysis, the collateral pattern was not associated with clinical presentation, type of brain lesion or outcome.

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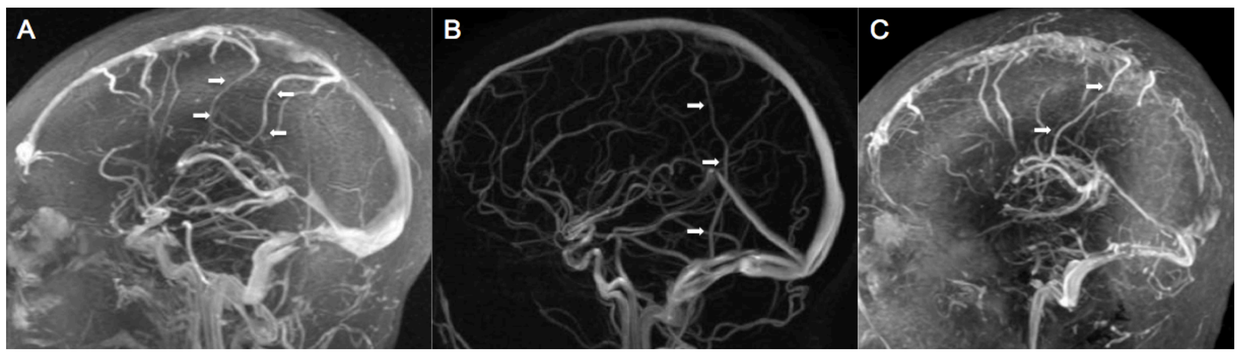
Figure 1. Types of collaterals according to Qureshi's classification



(A) Grade I collaterals: Sagittal MRV with thrombosis of the superior sagittal sinus (SSS) with a loop that bypasses the occluded segment and connects to a proximal segment within the same sinus (white arrows). (B) Grade II collaterals on a sagittal view of an MRV that connect the SSS with the transverse sinus (white arrows). (C) Thrombosis of the SSS on a sagittal view of an MRV with a grade III collateral that bypasses the occluded segment of the dural sinus and connects to a different circulation (white arrows).

Table 1. Clinical characteristics of the participants

	Female (%) n=77	Male (%) n=23	Total (%) n=100	*P*
Age (years, mean ± SD)	32.6±12.7	38.1±15.4	33.8±13.5	0.29
Hypertension	9 (11.7)	2 (8.7)	11 (11)	0.34
Smoking	11 (14.3)	9 (39.1)	20 (20)	0.009
Time at diagnosis (days, mean ± SD)	16.8±44.7	59.5±98.1	26.5±62.8	<0.001
Clinical conditions				
Isolated intracranial hypertension	25 (32.9)	8 (34.8)	33 (33)	0.86
Seizures at onset	36 (46.8)	10 (43.5)	46 (46)	0.78
Focal syndrome	55 (71.4)	14 (60.9)	69 (69)	0.33
Encephalopathy	7 (9.1)	3 (13.1)	10 (10)	0.61
Follow-up (months, mean ± SD)	26.6±26.7	29.8±27.1	27.3±26.7	0.93
Type of collateral				
None	10 (13)	2 (8.7)	12 (12)	0.58
Type I	2 (2.6)	1 (4.3)	3 (3)	0.66
Type II	21 (27.3)	6 (26.1)	27 (27)	0.91
Type III	44 (57.1)	14 (60.9)	58 (58)	0.75
Outcome				
Death	3 (3.9)	0	3 (3)	-
Rankin 0-2 (30-day)	65 (84.4)	16 (69.6)	81 (81)	0.11
Rankin 0-2 (90-day)	69 (89.6)	19 (82.6)	88 (88)	0.36
Remote seizures	13 (16.9)	2 (8.7)	15 (15)	0.49

Table 2. CVT collateral patterns and imaging features.

	Collateral type							
	None (%) n= 12	*p*	Type I (%) n=3	*p*	Type II (%) n=27	*p*	Type III (%) n=58	*p*
Brain parenchymal lesion								
Non-hemorrhagic lesions	5 (41.7)	0.19	1 (33.3)	0.70	6 (22.2)	0.80	12 (20.7)	0.36
Hemorrhagic lesions	3 (25.0)	0.14	2 (66.7)	0.44	15 (55.6)	0.19	25 (43.1)	0.65
Lesion size >6 cm	1 (8.3)	0.39	0	0.53	3 (11.1)	0.98	7 (12.1)	0.68
None	4 (33.3)	0.16	0	0.23	3 (11.1)	0.20	20 (34.5)	0.13
Drainage system affected								
Superior sagittal sinus	7 (58.3)	0.10	2 (66.7)	0.80	23 (85.2)	0.12	41 (70.7)	0.42
Unilateral transverse sinus	8 (66.7)	0.40	0	0.79	12 (44.4)	0.36	30 (51.7)	0.84
Bilateral transverse sinus	1 (8.3)	0.76	1 (33.3)	0.24	1 (3.7)	0.14	9 (15.5)	0.24
Straight sinus	0	0.17	0	0.63	0	0.10	7 (12.1)	0.07
Jugular vein	2 (16.7)	0.29	0	0.58	0	0.06	7 (12.1)	0.32
Galen's vein	1 (8.3)	0.69	1 (33.3)	0.07	3 (11.1)	0.29	2 (3.4)	0.22
Mixed system (deep/superficial)	1 (8.3)	0.19	1 (33.3)	0.32	4 (14.8)	0.81	8 (13.8)	0.62

Conclusion

Intracranial venous collaterals are frequently found in patients with CVT during the acute phase. However, they do not have an independent effect on the type of brain damage, clinical manifestations or prognosis.