ORIGINAL ARTICLE

Cerebral Blood and CSF Flow Patterns in Patients Diagnosed for Cerebral Venous Thrombosis – An Observational Study

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ABSTRACT

Background and Purpose: Recent studies of the organization of the cerebral venous system in healthy subjects using phase contrast magnetic resonance imaging (PC-MRI) show its structural complexity and inter-individual variations. Our objective was to study the venous blood and CSF flows in cerebral venous thrombosis (CVT). **Materials and Methods:** PC-MRI sequences were added to brain MRI conventional protocol in 19 patients suspected of CVT, among whom 6 patients had CVT diagnosis confirmed by MR venography. Results were compared with 18 healthy age-matched volunteers (HV). **Results:** In patients without CVT (NoCVT) confirmed by venography, we found heterogeneous individual venous flows, and variable side dominance in paired veins and sinuses, comparable to those in healthy volunteers. In CVT patients, PC-MRI detected no venous flow in the veins and/or sinuses with thrombosis. Arterial flows were preserved. CSF aqueductal and cervical stroke volumes were increased in a patient with secondary cerebral infarction, and decreased in 4 patients with extended thrombosis in the superior sagittal and transverse sinuses. These results suggest the main role of the venous system in the regulation of the dynamic intracranial equilibrium. **Conclusions:** CVT produces highly individualized pattern of disturbance in venous blood drainage. Complementary to MRI venography, PC-MRI provides non-invasive data about venous blockage consequences on CSF flow disturbances.

Key words: Cerebral venous thrombosis, CSF, hydrodynamics, PC-MRI, veins

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INTRODUCTION

In adults, regulation of intracranial pressure (ICP) results from the dynamic interaction between the arterial, venous, cerebrospinal fluid (CSF), and brain parenchyma compartments.[1-3]

The role of the arterial system has been assessed through human studies, and confirmed using mathematical models.[4,5] The arterial intracerebral systolic inflow is commonly considered now as the "driving force,"[2] which initiates a mechanical coupling between intracerebral mobile compartments, resulting in aqueductal, subarachnoid CSF, and venous venting during the systole. The aqueductal and cervical subarachnoid CSF stroke volumes represent the mobile compensatory reserve of respective compartments, and their modulation depends on the "driving current," in order to preserve ICP throughout the cardiac cycle (CC).

The role played by the venous system in the volume regulation remains controversial. For a long time, veins have been considered as passive conduits, modulating their cross-sectional area according to the blood flow and transmural pressure (cerebral venous pressure minus ICP). Anatomic configuration in veins is different from in arteries, because venous walls do not contain smooth muscle layers and therefore they cannot change their lumen actively.

Although the pressure waveforms are highly attenuated from the arteries when passing in the capillary bed and reaching the venous system, pulsation of ICP is reflected by pulsation of intracranial venous pressure. Specific modifications of venous flow pattern have been suggested to be associated with abnormalities of CSF dynamic in patients with hydrocephalus,[6] and the authors hypothesized that the venous system may originate numerous neurological disorders, such as Idiopathic Intracranial Hypertension or Normal Pressure Hydrocephalus.[6,7]

Although veins are supposed to play a crucial role in regulating intracranial dynamics, only a few studies have been devoted to the cerebral venous system physiology and pathology.[8,9] Veins functioning remained difficult to study non-invasively *in vivo* until the advent of phase contrast magnetic resonance imaging (PC-MRI).

In a recent study,[10] it has been shown that PC-MRI enables reliable, reproducible, and rapid measurement of venous flows (in both dural sinuses and cervical veins), and of their interactions with arterial and CSF flows. The heterogeneity of venous drainage and the variability of side dominance in paired veins and sinuses in healthy young adults were concordant with previous anatomic, angiographic, and PC-MRI studies.[11,12] The role of accessory venous pathways, mainly epidural, in drainage of venous blood from the cranium, was also emphasized, independently of commonly described postural[13,14] or respiratory factors.[15]

Cerebral venous thrombosis (CVT) can be considered as a model of dynamic disorder in the intracerebral venous system. Reports on the evaluation of venous dynamics in CVT are anecdotal,[16] and quantitative evaluations of intracerebral venous flows have never been published.

Our purpose was to study the variations and the dynamic coupling between vascular and CSF compartments in patients with CVT.

MATERIALS AND METHODS

1 – Participants

We studied successive patients admitted via the emergency department, presenting with neurological symptoms suggesting CVT, and who consequently underwent a brain MRI. Phase contrast sequences were added to the conventional imaging protocol used by the neuroradiologists with the intention to visualize the region of "no flow" in thrombosis section of venous bed. According to the results of the MRI protocol, this population was then divided into 2 groups: patients with confirmed thrombosis (CVT) or those without (NoCVT).

The NoCVT group consisted of 13 patients (9 women and 4 men) with a mean age of 34 ± 13 years, in whom diagnosis different from CVT were finally confirmed (migraine, multiple sclerosis, meningitis, etc.)

The CVT group consisted of 6 female patients (mean age: 32 ± 11 years) who were admitted for unusual headaches or/and seizures, and in whom cranial MRI showed CVT of dural sinuses or of a cortical vein.

Flow results were compared in CVT, NoCVT, and an age-matched control population, who underwent the same PC-MRI examination protocol. The control population, hereby called HV, consisted of 18 healthy young volunteers (9 women and 9 men), who underwent cine PC-MRI. The mean age was 31 ± 7 years. The exclusion criteria were any neurological, psychiatric or severe general disease, alcoholism, or abnormalities detected with clinical MRI exams.

All of the participants (patients and controls) gave their informed consent to participate in the study.

2 – Data acquisition

All MRI exams were performed using a 3-Tesla machine (Signa; General Electric Medical System, Milwaukee, WI). Subjects were supine.

Conventional morphologic sequences were acquired in each patient with CVT suspicion according to the protocol

routinely used in our clinic. This protocol includes the following sequences: Sagittal T1 Flair, Axial FSE (Fast Spin Echo) T2, T2*, Flair, diffusion-weighted, SE (Spin Echo) T1 before and after contrast-enhancing agent injection, 3D TOF (Time-of-Flight), coronal 2D TOF, and cerebral MR venography.In all subjects, flow images were acquired with a 2D fast cine PC-MRI pulse sequence with retrospective peripheral gating, so that the 32 frames analyzed covered the entire CC. The MRI parameters were as follows: echo-time TE = 11-17 ms; repetition time TR = 29-43 ms; 4 views per segment, flip angle: 25° for vascular flows, 20° for CSF flows; field of view (FOV) = 14×14 mm²; matrix 256 × 128; slice thickness 5 mm. Velocity (encoding) sensitization was set at 80 cm/s for the vessels, 15 cm/s for the aqueduct and 5 cm/s for the cervical subarachnoid space. Sagittal scout view sequences were used to localize the anatomical levels for flow quantification. The acquisition planes were selected perpendicular to the presumed direction of the flow, and are represented on Figure 1. Sections through the C2-C3 subarachnoid spaces level (a1), and Sylvius aqueduct (b) were used for CSF flow measurement. By varying the velocity encoding, the same cervical section level (a2) was used to measure vascular flows in left and right internal carotids (ICA), vertebral arteries (VA), and internal jugular veins (IJV). By using 3D TOF sequence as a localizer, and with adjusted velocity encoding, the intracerebral vascular plane (c) was used to measure arterial flows in the basilar artery (BA) and in the intrapetrous ICA, and venous flows in the superior sagittal sinus (SSS) and the straight sinus (SRS). Finally, a coronal section (d) enabled measurement of the venous flows in the SSS, and in the left and right lateral transverse sinuses (TS).

Black pixels represent the flows entering in the section plane, white pixels represent flows directed out of the section plane, and gray pixels correspond to immobile tissues.

The acquisition time for each flow series was about 40 seconds, with a slight fluctuation that depended on the participant's heart rate, resulting in a total additional examination time of 5 minutes.

3 - Data analysis

Data were analyzed using validated image processing software[3] with an optimized CSF and blood flows segmentation algorithm, which automatically extracts the region of interest (ROI) at each level, and calculates its flow curves over the 32 segments of the CC.

Then, the venous, arterial, and CSF flow curves were generated within one CC.

For the arterial flows, we calculated the mean cerebral arterial total blood flow (CBF) in milliliters per minute, at cervical and cerebral levels. The cervical CBF was evaluated as the sum of arterial flows in left and right ICA and VA, and the cerebral CBF as the sum of arterial flows in left and right intrapetrous ICA and in the BA. Similarly, the cervical cerebral venous flow was calculated by summing the venous flows in left and right Internal jugular vein (IJV) and epidural veins (EV). The cerebral venous blood flow was calculated as the sum of mean venous flows in the SSS and SRS.

Similarly, CSF flow curves at aqueductal and cervical levels were reconstructed, and CSF stroke volumes, which represent the CSF volumes displaced in both directions through the considered ROI at each level.[17,18]

4 - Statistical analysis

Comparison of vascular flows and CSF stroke volumes between the 3 populations (HV, CVT, and NoCVT) was
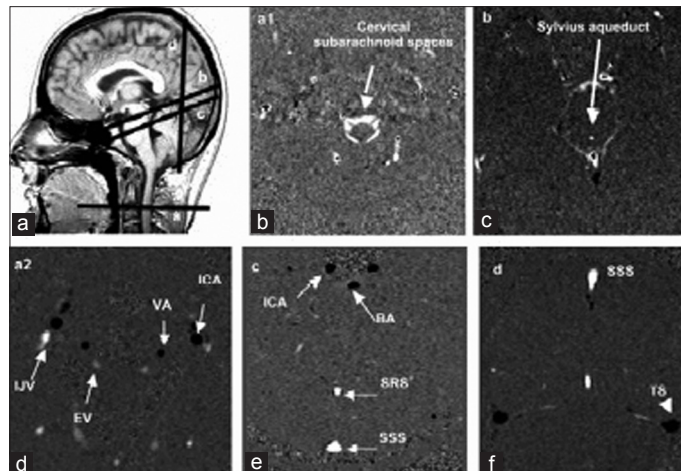

Figure 1: Data acquisition. Sagittal scout view sequences were used to localize the anatomical levels for flow quantification. The acquisition planes were selected perpendicular to the presumed direction of the flow. Sections through these different levels, for each flow series are represented.

performed using multivariate analysis. The level for statistical significance (*P* value) was set at 0.05.

RESULTS

1 - General

Table 1 compares mean flow values between the 3 studied groups (volunteers – HV, NoCVT patients confirmed with MRI venogram, and CVT patients).

The mean arterial blood flows in each arterial vessel are shown, expressed in mL/min. Comparison showed no side difference for even arteries.

Mean CBF comparison between the 3 groups showed no statistical difference, at both cervical and cerebral levels.

Similarly there were no significant differences between cerebral and cervical venous CBF. Although mean value in CVT patients was lower than in HV and NoCVT groups, standard deviation was greater, which makes testing insignificant. CVT group seemed to have much greater heterogeneity in patterns of venous CBF than remaining groups.

2 - Symptomatic patients without confirmed thrombosis (NoCVT)

All of the patients were admitted to the emergency room for unusual headaches, and cerebral MRI venography

Table 1: Parameters comparison in the studied populations

	HV	NoCVT	CVT	P
Arterial				
Cervical level				
R ICA	269 ± 49	277 ± 103	201 ± 61	0.1
L ICA	265 ± 59	239 ± 85	246 ± 122	0.5
R VA	77 ± 32	72 ± 30	81 ± 65	0.9
L VA	104 ± 41	99 ± 64	105 ± 49	0.9
Cervical arterial CBF	714 ± 124	686 ± 233	615 ± 235	0.63
Cerebral level				
R ICA	238 ± 71	250 ± 7	240 ± 73	0.9
L ICA	251 ± 89	254 ± 93	257 ± 103	0.9
BA	161 ± 62	157 ± 110	166 ± 49	0.9
Cerebral arterial CBF	649 ± 178	661 ± 233	663 ± 270	0.98
Venous				
IJV	519 ± 205	398 ± 242	535 ± 357	0.35
EV	52 ± 54	107 ± 92	72 ± 28	0.1
Cervical venous CBF	506 ± 137	472 ± 164	316 ± 356	0.16
Cerebral venous CBF	457 ± 80	443 ± 183	316 ± 245	0.19
CSF stroke volume				
Cervical	460 ± 149	443 ± 104	320 ± 242	0.47
Aqueductal	41 ± 22	43 ± 39	30 ± 35	0.65

HV = healthy volunteers, NoCVT = symptomatic patients without cerebral venous thrombosis, CVT = patients with confirmed CVT, R = right, L = left, ICA = internal carotid artery, VA = vertebral artery, BA = basilar artery, CBF = cerebral blood flow, IJV = internal jugular vein, EV = epidural vein, CSF = cerebrospinal fluid. Arterial and venous flows are expressed in mL/min. CSF stroke volumes are expressed in μl per cardiac cycle *P* level for significance <0.05

excluded the diagnosis of CVT. Nevertheless, they were hospitalized for etiological exploration (lumbar puncture, biological exams, etc.).

Table 2 shows the individual flow values in each vein and sinus in NoCVT patients. The averaged venous flows are summarized in the bottom rows, with large standard deviations, indicating aforementioned heterogeneity of venous flows.

In agreement with previous results published in healthy young controls,[19] we found:

i)   Significant right-sided predominance of venous flows in the IJV and in the TS

ii)   However, a high heterogeneity of venous drainage was detected [Table 2], in both quantitative evaluations (represented with value dispersion and high standard deviations), and in lateralization. In addition, 5 subjects had a strictly unilateral right IJV collecting the cerebral venous blood flow, with contralateral IJV flow equal to 0, and in 4 of them, the homolateral TS flow was significantly reduced (<20%) or null (patients P2, P4, P5, and P10).

iii)   Significant preponderance of the jugular venous drainage (left + right IJV mean flow: 398 ± 242 mL/min) over the epidural drainage (107 ± 92 mL/min) in a significant way (*P* = 0.001). Cervical drainage was exclusively jugular in 4 patients, with absent flows in both epidural veins.

CSF flow curves analysis provided the CSF stroke volumes at the aqueductal (43 ± 39 μL/min) and cervical (443 ± 104 μL/min) levels [Table 1], which were not statistically different from those obtained in HV (*P*: 0.65 and 0.47, respectively).

PC-MRI analysis found unusual results in 1 patient (P13), who had a dramatic reduction of CBF (total arterial flow: 387 mL/min; total venous flow: 278 mL/min), with a significant increase of aqueductal CSF stroke volume (146 μL/CC) and a blockage of cervical CSF circulation. This 32-year-old young female had serious neurological dysfunction (coma, severe headaches) related to bacterial meningitis.

3 - Patients with confirmed thrombosis (CVT group)

Six female patients were diagnosed with CVT, with different single or multiple thrombosis on MR venography.

Arterial flows were rather normal in these patients, with a mean cerebral mean flow of 663 ± 270 mL/min, and a mean cervical cerebral flow at 615 ± 235 ml/min, not statistically

Table 2: Venous blood flows in patients with symptoms but with no confirmed Cerebral Venous Thrombosis (NoCVT)									
Patient Number	R IJV	L IJV	R EV	L EV	SSS	SSS (c)	SRS	R TS	L TS	Diagnosis
P1	206	161	0	0	198	175	58	199	182	CTTH
P2	587	0	14	26	409	425	128	386	64	Migraine
P3	33	66	25	66	301	306	88	281	152	Idiopathic HT
P4	189	0	170	78	511	463	98	607	186	CTTH
P5	475	177	8	10	409	383	93	386	0	Alcohol
P6	392	0	125	137	399	561	111	550	218	Migraine
P7	409	116	0	0	343	244	NC	529	111	Hemorrhage
P8	427	32	62	78	376	380	104	621	145	CTTH
P9	461	0	0	0	192	170	107	215	114	Hypophyse A
P10	197	0	23	28	226	205	102	265	0	MS
P11	417	189	55	0	721	679	199	635	443	Chemotherapy
P12	69	233	39	17	253	243	53	161	208	Dental algia
P13	55	283	0	0	142	133	136	35	253	Meningitis
CBFv ± SD	301 ± 184	157 ± 83	58 ± 55	55 ± 43	345 ± 156	336 ± 165	106 ± 38	375 ± 199	189 ± 100	
	R > L: 9 (69%)		R > L: 3 (23%)					R > L: 9 (69%)		
	L > R: 3 (23%)		L > R: 2 (15%)					L > R: 1 (7%)		
	E: 1		E: 8					E: 3		

R = right, L = left, IJV = internal jugular vein, EV = epidural vein, SSS = superior sagittal sinus, SSS (c) = SSS measured in a coronal acquisition plane, SRS = straight sinus, TS = transverse sinus, CBFv ± SD = venous mean cerebral blood flow ± Standard Deviation, expressed in mL/min, R>L = right side dominance, L>R = left side dominance, E = equal drainage to both sides, CTTH = chronic type tension headaches, HT = intracranial hypertension, Hypophyse A = adenoma, MS = multiple sclerosis, NC = non-available data for technical reasons. Gray shaded squares correspond to veins with no detected flow

different from those obtained in the other populations (*P*: 0.98 and 0.63, respectively).

CSF stroke volumes were more heterogeneous, and are represented in Figure 2 for cervical and aqueductal CSF stroke volumes. We found CSF flow abnormalities in 5 of these patients.

DISCUSSION

Veins

Our observational study confirms the heterogeneity in venous flows [Tables 1 and 2] in NoCVT group, similar to the results previously published.[10] This heterogeneity shows the difficulty in distinguishing between the physical side dominance and partial venous thrombosis, as shown in Table 3. In patients T1 and T2, flow is not null in the IJV but highly asymmetric as compared to contralateral vein. The blockage of these IJV was confirmed on venography. On the contrary, in patients T4, T5, and T6, the venous flow was bilaterally absent in the IJV although venography did not show thrombosis at this level. In patients T5 and T6, the absence of flow can be functional, related to the stenosis in the upstream flow in the ipsilateral TS.

Our results showed higher levels of lateralized venous flows in both IJV and TS, in the NoCVT population. This side dominance in paired venous pathways has been described in previous studies with PC-MRI technique[9,10] or with angiography[19] with different percentages probably related to the used technique.

PC-MRI enables quantification of venous flows in SSS, SRS,

TS, as well as in cervical veins, with a good detection rate, as previously shown in HV.[10,15,20]

Anatomical, and physiological publications have stressed the preponderant role played by the jugular system in cephalic venous drainage in physiological states. Although the vertebral and cervical epidural venous drainage have been considered as accessory until recently, it is now admitted that epidural veins can enlarge in some pathological states as intracranial hypotension,[21] and that they can play a crucial role in the venous drainage of the cephalic extremity in the upright position.[13]

Arteries

The small individual variability of arterial bed helps reproducibility of these studies in large populations.[8] Recent studies have used MRI techniques, with comparable and reproducible results.[22,23] Buijs et al.,[24] studied CBF values in a large population, and found a constant contribution of the right, left ICA, and BA (41%, 40% and 19% of total CBF, respectively), which was stable through ageing, although associated with a proportional decrease in CBF. In our study, total CBF measured at the cervical and intracerebral levels in HV and NoCVT were comparable to those obtained in healthy adults in a previous study,[12] and contribution of each intracranial (right ICA: 36% / left ICA: 40%, BA: 24%) vessel was comparable to published results.

Observations in patients with confirmed CVT

CVT offers the opportunity to investigate intracerebral dynamic modifications initiated with venous dysfunction. The limit of this study is the small size of CVT patients,

Table 3: Cerebral flows in patients with Cerebral Venous Thrombosis (T1 to T6)						
	T1	T2	T3	T4	T5	T6
Arterial cerebral flow	815	972	727	501	533	427
Arterial cervical flow	713	952	747	551	419	309
Venous cerebral flow	107	181	476	331	91	197
SSS	0	553	304	218	0	0
SRS	107	181	145	102	91	197
R TS	TP	TP	566	277	101	0
L TS	TP	TP	172	161	0	409
Venous cervical flow	384	947	419	34	18	91
R IJV	17	884	293	0*	0*	0*
L IJV	294	63	55	0*	0*	0*
R EV	0*	0*	34	17	18	44
L EV	73	0*	37	17	0*	47
Thrombosis Location	RTS + RIJV + SSS	LTS + LIJV (+H)	L frontal cortical vein (+H)	R parietal cortical vein (+H)	LTS + SSS + SSI	RTS + SSS + R parietal cortical vein
Cervical CSF stroke volume (µl/CC)	412	714	401	49	123	221
Aqueductal CSF stroke volume (µl/CC)	39	93	0	34	5	6

R = right, L = left, IJV = internal jugular vein, EV = epidural vein, SSS = superior sagittal sinus, SRS = straight sinus, TS = transverse sinus. All flows are expressed in mL/min, except CSF stroke volumes (in µL/CC). Cerebral flow is calculated by summing flows in SSS and SRS, and cervical flow by summing bilateral epidural and jugular flows. 0 means absence of visualized flow: in a gray square, it corresponds to blocked veins or sinus on MR venography followed by a star * in a white square, it corresponds to a non blocked vessel on MR venography, and probably due to physiologic heterogeneity of venous drainage TP: technical problem at acquisition (+H) corresponds to the presence of an associated hemorrhagic infarct

especially with different sites of sinus and / or vein occlusion, which makes it difficult to generalize.

Furthermore, all CVT patients were female, but cerebral blood and CSF published results with PC-MRI in healthy young or elderly subjects have failed to show sex-related differences.[10,25] Nevertheless, the study has rather an observational nature. Our aim was not to prove PC-MRIs' superiority to MR venogram or standard MR protocols in CVT diagnosis, but to suggest that these techniques could be used complementarily to understand intracranial dynamics.

Firstly, the absence of flow in SSS (T1, T5, and T6) was correlated to MR venography results. On the contrary, technical problems in TS flows acquisition were encountered in T1 and T2, which makes it difficult to reach a conclusion.

Interestingly, although very weak, a venous flow was measured in IJV that were considered as blocked on MR venography for 2 patients (T1 and T2). In these 2 patients, the cervical level of IJV flow analysis was caudal to the thrombosis level, which suggests the development of a collateral drainage pathway.

Jugular drainage was absent in 3 patients (T4, T5, and T6), without visualized jugular blockage on MR venography. This is probably related to the blockage of cerebral venous structures above. More surprisingly, epidural drainage was not increased in these patients, which suggests the implication of other accessory venous drainage pathways (as pterygoid plexus and ophthalmic veins). Furthermore, in CVT patients, arterial to venous blood flows ratios were elevated [Table 1], which suggests that when venous blockage occurs in principal venous pathways, drainage in the accessory veins becomes preponderant.

Mean CSF flows were comparable to those in healthy adults, but individual analysis showed normal results in only 1 patient (T1) and major alterations in 5 patients.

Aqueductal and Cervical CSF stroke volumes were increased in 1 patient (T2), with obstruction of left venous drainage pathways (TS and IJV) and hemorrhagic infarct. In this patient, although venous outflow was exclusively drained by the right IJV, cervical venous flow was significantly elevated. These results suggest a hyper-dynamic functioning in the 3 outflow systems (subarachnoid, ventricular, and jugular), which according to the Monro-Kellie doctrine, must be related to a raise of ICP, provoked by the hemorrhage.

In patient T3, cervical CSF stroke volume was within normal values, while the aqueductal flow was null. The re-examination of conventional MRI images visualized blockage at the aqueductal level [Figure 3], probably related to secondary intraventricular hemorrhage.

In the other 3 patients, we observed a decrease in CSF stroke volume either in the cervical (T4) or in both cervical and aqueductal (T5 and T6) compartments. We did not find any clinical, chronological, or radiological specificity in these patients, and correlations to occlusion site were not possible. These results were surprising, as it is logically expected to observe hyper-dynamic flows, related to elevated ICP. In these patients, attenuation of CSF oscillations was probably related to hemorrhage extension to the cerebral SAS [Figure 3], which would
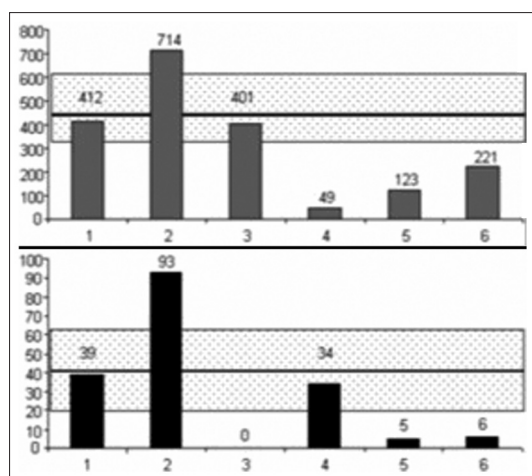
Figure 2: Cervical (upper) and aqueductal (lower) CSF represented in patients with CVT. CSF stroke volumes are represented in µL per cardiac cycle in each 1 of the 6 patients. The dashed rectangle represents the average ± 1 standard deviation, observed in the control HV's population.
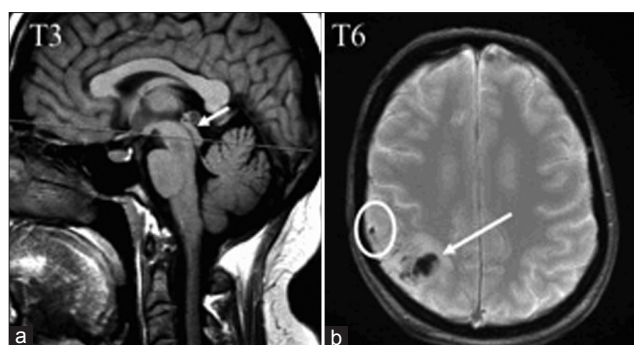

Figure 3: In patient T3, a sagittal T1 view shows the aqueductal stenosis (arrow), probably secondary to the diffusion of hemorrhage in the ventricular space. In patient T6, an axial T2* image shows the parietal cortical vein thrombosis (in a circle), and cortical venous infarct (arrow).

increase resistance to CSF flows in the SAS, and thus reduce the compliance of the subarachnoid CSF compartment.

CONCLUSION

CVT produces highly individualized patterns of disturbances in the venous blood drainage.

The results obtained in 5 out of 6 patients with CVT stress the importance of dynamic interaction between brain compartmental components, especially the role played by the venous system in the volume regulation, and thus in CSF flow dynamics.

Due to the small size of CVT cohort, these results need to be confirmed in a larger population.

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