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Assessment of the Congestion of Emissary Veins and Scalp Edema in Cerebral Venous Thrombosis

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Abstract

Aim of the study: The impact of cerebral venous thrombosis on the emissary veins is not well documented. Our study evaluates the presence of scalp edema and the congestion of the emissary veins on magnetic resonance imaging in patients with cerebral venous thrombosis, idiopathic intracranial hypertension and multiple sclerosis in order to determine whether these signs could represent a marker of cerebral venous thrombosis.

Patients and methods: The magnetic resonance imaging of 102 patients with cerebral venous thrombosis (cases) was analyzed and compared with those of 93 idiopathic intracranial hypertension patients and 63 multiple sclerosis subjects. Imaging analysis was performed by two blinded entities and recorded the presence of the congestion of the emissary veins and of scalp edema, its location and its evolution using the following scale: Stage 1: Thickening of the scalp not exceeding double its thickness; Stage 2: Thickening of the scalp exceeding double but not more than three times its thickness, and or detachment of the scalp from the bone; Stage 3: Thickening of the scalp exceeding three times its thickness.

Results: Scalp edema was present in 60 (58.8%) cerebral venous thrombosis patients vs. 38 (40.9%) idiopathic intracranial hypertension patients and 20 (31.7%) multiple sclerosis subjects. Its location was correlated to the location of the thrombosis in 55 (91.7%) cases. Follow-up MRI was performed in 17 (17.2%) cerebral venous thrombosis cases. Edema regressed in 41.7% of cases. The congestion of emissary veins was independently associated with cerebral venous thrombosis compared to idiopathic intracranial hypertension and multiple sclerosis subjects ($p = 0.042$ and $P < 0.001$) respectively.

Conclusion: The scalp edema and congestion of emissary veins are radiological signs that could be interesting to support the diagnosis of CVT. However, their diagnosis value remains to be proven by further studies to allow stronger evidence.

Keywords

Emissary veins, Scalp edema, Cerebral venous thrombosis, Idiopathic intracranial hypertension, Congestion

Abbreviations

CVT: Cerebral Venous Thrombosis; IIH: Idiopathic Intracranial Hypertension; MS: Multiple Sclerosis; MRI: Magnetic Resonance Imaging; MRV: Magnetic Resonance Venography; PPV: Positive Predictive Value; NPV: Negative Predictive Value

Introduction

Cerebral venous thrombosis (CVT) is an uncommon condition that accounts for 0.5 to 1% of all strokes in the adult population; but remains frequently under diagnosed [1]. Its diagnosis can be challenging because it can have a very similar presentation to idiopathic intracranial hypertension (IIH), with some overlap between the two entities [1,2]. Enlarged occipital emissary veins; a definite sign of generalized increased intracranial pressure was first described by Lindblom in 1936 but has not received adequate attention then [3]; and was only documented in CVT in 1985 by Bousser, et al. [4]. Yet, the congestion of emissary veins could theoretically disrupt the venous drainage of the scalp and cause edema. Therefore, it could constitute a radiological sign

that helps distinguish between CVT and its differential diagnoses, especially IIH. Our aim was to evaluate the presence of scalp edema and the congestion of the emissary veins on magnetic resonance imaging (MRI) in patients with CVT, IIH and multiple sclerosis (MS) subjects in order to determine whether these signs could represent a marker of CVT.

Patients and Methods

Patients

We performed a retrospective, single center case-control study at the neurology department of the University Hospital of Casablanca, Ibn Rochd from 2000 to 2019. The department registry was screened to identify patients with a diagnosis of:

- Cases: patients with radiologically proven CVT on MRI (those with computed tomography were excluded);

- Controls: Two populations were studied: 1) Patients hospitalized for IIH selected on the Modified Dandy Criteria including normal Magnetic resonance venography (MRV) [5] and 2) Another control group consisting of Multiple Sclerosis patients. This later group was chosen as a control because their age of

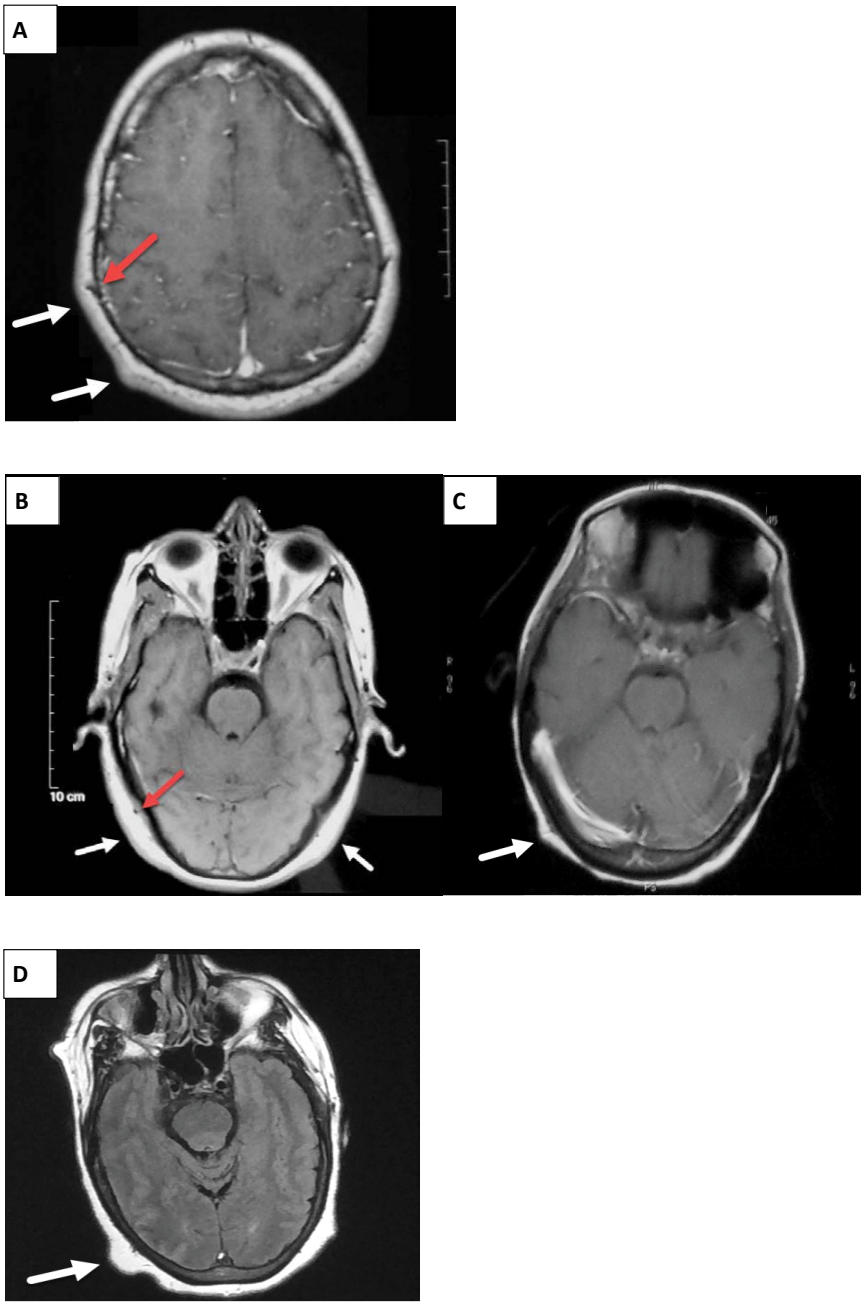

Figure 1: Brain MRI: A) White arrows: Stage 1 scalp oedema: Thickening of the scalp not exceeding double its thickness; Red arrow: Congestion of emissary veins; B) White arrows: Stage 2 scalp edema: Thickening of the scalp exceeding double but not more than three times its thickness; Red arrow: Congestion of emissary veins. C) Detachment of the scalp from the bone, Red arrow: Congestion of emissary veins; D) White arrow: Stage 3 scalp oedema: Thickening of the scalp exceeding three times its thickness.

onset and gender are close to those of IIH and because intracranial hypertension syndrome is not observed in this condition.

Data

To study the association of CVT with the presence of scalp edema, we analyzed the MRI of CVT patients and control patients diagnosed with IIH and MS subjects. Imagery analysis was performed by two blinded entities and recorded the presence of the congestion of the emissary veins and of scalp edema, its location, evolution on follow-up imagery when available (Figure 1) as well as its severity, quantified using the following scale:

- Stage 1: Thickening of the scalp not exceeding double its thickness;
- Stage 2: Thickening of the scalp exceeding double but not more than three times its thickness, and or detachment of the scalp from the bone;
- Stage 3: Thickening of the scalp exceeding three times its thickness.

We included possible confusion variables (age, gender, pregnancy or postpartum period, delay between the onset of symptoms and MRI).

Statistical analysis

We compared the presence of the two signs in cases and controls. Statistical analysis was done using SPSS 23.0 for Windows. Quantitative variables were reported as mean, standard deviation and range. Qualitative variables were reported as number and percentage (%). Bivariate analysis was performed using χ^2 test or Fisher exact test for categorical variables and using Student t test for continuous variables; as well as multivariate (logistic regression). Subgroup analysis of the CVT versus IIH and CVT versus MS subjects were performed. Two-tailed p values < 0.05 were considered significant.

Results

Two hundred and fifty eight cases were included: 102 CVT patients (women: 77.5% with an average age of 35.9 years [6-69]); 93 IIH patients (women: 93.5% with an average age of 28.5 years) and 63 MS subjects (women: 79.4% with an average age of 33.8 years). (Results are described in Table 1 and Table 2).

Scalp edema was present in 60 (58.8%) CVT patients, 38 (40.9%) IIH patients, 20 (31.7%) MS subjects and its location was correlated with that of the venous thrombosis in 55 (91.7%) cases (Figure 1). Follow-up MRI was performed in 17 (17.2%) CVT cases. Edema regressed in

Table 1: Baseline characteristics and occurrence of scalp edema and the congestion of emissary veins in CVT patients compared to MS subjects.

	MS (n = 63)	CVT (n = 102)	P (univariate model)	P (multivariate model)
Gender, n (%)			0.772	-
Female	50 (79.4)	79 (77.5)		
Male	13 (20.6)	23 (22.5)		
Age, mean ± SD	33.8 ± 10.3	35.9 ± 13.2	0.280	-
Scalp edema, n (%)	20 (31.7)	60 (58.8)	**0.001**	0.490
Congestion of emissary veins, n (%)	18 (28.6)	73 (73.7)	**< 0.001**	**< 0.001**

CVT: Cerebral venous thrombosis ; MS: Multiple sclerosis

Table 2: Baseline characteristics and occurrence of scalp edema and the congestion of emissary veins in CVT patients compared to IIH patients.

	IIH (n = 93)	CVT (n = 102)	P (univariate model)	P (multivariate model)
Gender, n (%)			**0.002**	-
Female	87 (93.5)	79 (77.5)		
Male	6 (6.5)	23 (22.5)		
Age, mean ± SD	28.5 ± 10.1	35.9 ± 13.2	**< 0.001**	**0.012**
Scalp edema, n (%)	38 (40.9)	60 (58.8)	**0.012**	**0.302**
Congestion of emissary veins, n (%)	42 (45.2)	73 (73.7)	**< 0.001**	**0.042**

CVT: Cerebral venous thrombosis ; IIH: Idiopathic intracranial hypertension

41.7% of cases. The existence of scalp edema in CVT cases was not correlated to the presence of hemorrhage (p = 0.063), the presence of infarct (p = 0.905), the number of sinuses affected (p = 0.400), or duration of evolution (p = 0.961); (Table 2).

The congestion of the emissary veins was independently associated with CVT compared to IIH and MS subjects (p = 0.042 and P < 0.001) respectively.

Discussion

Several authors have stressed the importance of the emissary veins acting as obligatory outflow pathways for encephalic drainage [6,7]. The superficial drainage of the scalp is provided by scalp veins, which are connected to the diploic veins and thus to the intracranial venous sinuses via the valvless emissary veins. Occlusion of the venous sinuses can give way to collateral circulation (represented by the emissary veins), resulting in an involvement of the scalp veins as was reported by Bousser, et al. [5]. In theory, the congestion of the emissary veins as a result of dural thrombosis may affect the scalp venous drainage and result in edema in the form of thickening of the scalp on neuroimaging. This later could constitute a radiological sign that further helps distinguish CVT and IIH.

In our study, analysis of initial MRI revealed that scalp edema was present in 60 (58.8%) CVT vs. 30 (40.9%) IIH cases and 20 (31.7%) MS subjects. However, this sign was not independently associated with CVT. Otherwise, the congestion of emissary veins was independently associated with CVT compared to IIH and MS subjects. Their sensitivity and specificity were good, but didn't help differentiate CVT and IIH (specificity 54.8%). Hedjoudje, et al. [8] in their study comparing 46 IIH versus 92 healthy controls found that occipital emissary veins are more frequent and larger in patients with pseudotumor cerebri syndrome.

Our study has some limitations regarding its retrospective design with non-standardized MRI protocols; unmatched populations and no precise measurement of scalp thickness. However, to the best of our knowledge, there is no study that evaluated the presence of the congestion of emissary veins and scalp edema in a series of patients with CVT. Commonly, the assessment of scalp edema is not relevant if MRI/MRV is performed for the diagnosis of CVT, but it could be useful in some situations when the MRV is not conclusive (As is often the case with cortical vein thrombosis) and if there is no parenchymal involvement.

Conclusion

The scalp edema and congestion of emissary veins are radiological signs that could be interesting to support the diagnosis of CVT. However, their diagnosis value remains to be proven by further studies to allow stronger evidence.

Declaration of Conflicting Interests

No potential competing interest relevant to this article was reported.

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References

1. Dmytriw AA, Song JSA, Yu E, et al. (2018) Cerebral venous thrombosis: state of the art diagnosis and management. Neuroradiology 60: 669-685.
2. Peralta GM, Cestari DM (2018) An update of idiopathic intracranial hypertension. Curr Opin Ophthalmol 29: 495-502.
3. Chynn KY (1963) Occipital Emissary Vein Enlargement A Sign of Increased Intracranial Pressure. Radiology 81: 242-247.
4. Bousser MG, Chiras J, Bories J, et al. (1985) Cerebral Venous Thrombosis - A Review of 38 Cases. Stroke 16 : 199-213.
5. Friedman DI, Liu GT, Digre KB (2013) Revised diagnostic criteria for the pseudotumor cerebri syndrome in adults and children. Neurology 81: 1159-1165.
6. Louis RG Jr, Loukas M, Wartmann CT, et al. (2009) Clinical anatomy of the mastoid and occipital emissary veins in a large series. Surg Radiol Anat 31: 139-144.
7. San Millan Ruíz D, Gailloud P, Rufenacht DA, et al. (2002) The craniocervical venous system in relation to cerebral venous drainage. AJNR Am J Neuroradiol 23: 1500-1508.
8. Hedjoudje A, Piveteau A, Gonzalez-Campo C, et al. (2019) The Occipital Emissary Vein: A Possible Marker for Pseudotumor Cerebri. AJNR Am J Neuroradiol 40: 973-978.