PARENCHYMAL ABNORMALITIES IN CEREBRAL VENOUS THROMBOSIS: FINDINGS OF MAGNETIC RESONANCE IMAGING AND MAGNETIC RESONANCE ANGIOGRAPHY*

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Abstract OBJECTIVE: To determine the frequency and localization of parenchymal abnormalities in cerebral venous thrombosis on magnetic resonance imaging and magnetic resonance angiography as well as their correlation with the territory and affected venous drainage. MATERIALS AND METHODS: Retrospective analysis (1996 to 2004) of 21 patients (3 male and 18 female) age range between 3 and 82 years (mean 40 years, median 36 years) with clinical and radiological diagnosis of cerebral venous thrombosis on magnetic resonance imaging and magnetic resonance angiography in 2D PC, 3D PC and contrast-enhanced 3D TOF sequences. The statistical analysis was performed with the qui-square test. Four patients had follow-up exams and three patients underwent digital subtraction angiography. RESULTS: Main predisposing factors were: infection, use of oral contraceptives, hormone replacement therapy and collagenosis. Predominant symptoms included: focal deficit, headache, alteration of consciousness level and seizures. Most frequent parenchymal manifestations were: cortical/subcortical edema or infarct, venous congestion and collateral circulation, meningeal enhancement and thalamic and basal ganglia edema or infarct. Occlusion occurred mainly in superior sagittal, left transverse, left sigmoid and straight sinuses. Cavernous sinus and cortical veins thrombosis are uncommon events. CONCLUSION: Cerebral venous thrombosis is an uncommon cause of stroke, with favorable prognosis because of its reversibility. Diagnosis is highly dependent on the radiologist capacity to recognize the presentations of this disease, principally in cases where the diagnosis is suggested by parenchymal abnormalities rather than necessarily by visualization of the thrombus itself. An accurate and rapid diagnosis allows an immediate treatment, reducing the morbidity and mortality rates.
Keywords: Magnetic resonance; Cerebral venous thrombosis; Cerebral infarct; Intracranial hemorrhage.

Resumo *Alterações parenquimatosas na trombose venosa cerebral: aspectos da ressonância magnética e da angiorressonância.*
OBJETIVO: Determinar a freqüência e localização das alterações parenquimatosas da trombose venosa cerebral nos exames de ressonância magnética e de angiorressonância, bem como a correlação com o território e a drenagem venosa comprometida. MATERIAIS E MÉTODOS: Foram analisados exames de 21 pacientes realizados entre 1996 e 2004, com diagnóstico clínico e radiológico de trombose venosa cerebral em exames de ressonância magnética e de angiorressonância nas seqüências 2D PC, 3D PC e 3D TOF com contraste paramagnético. Análise estatística foi realizada com o teste do qui quadrado. Quatro pacientes tinham exames de controle e três realizaram angiografia por subtração digital. RESULTADOS: Dos 21 pacientes, 18 eram mulheres, todos com idade entre três e 82 anos (média de 40 anos e mediana de 36 anos). Os principais fatores etiológicos foram infecção, uso de contraceptivos orais, reposição hormonal e colagenoses. Predominaram os sintomas de déficit focal, cefaléia, alteração do nível de consciência e convulsões. Por freqüência, as manifestações parenquimatosas foram: edema/infarto de distribuição cortical e/ou subcortical, congestão venosa e circulação colateral, realce meníngeo e infarto ou edema dos tálamos e núcleos da base. Os principais seios comprometidos foram o sagital superior, o transverso esquerdo, o sigmóide esquerdo e o seio reto, sendo incomum o acometimento dos seios cavernosos e de veias corticais. CONCLUSÃO: A trombose venosa cerebral é causa incomum de acidente vascular encefálico, com prognóstico favorável pelo caráter reversível das lesões. Seu diagnóstico depende fundamentalmente da capacidade do radiologista reconhecer suas formas de apresentação, principalmente nos casos em que ele é sugerido pelas alterações parenquimatosas e não necessariamente pela visualização do trombo. A precisão e a rapidez no diagnóstico permitem o pronto tratamento, reduzindo a morbi-mortalidade da doença.
Unitermos: Ressonância magnética; Trombose venosa cerebral; Infarto cerebral; Hemorragia intracraniana.

INTRODUCTION

Cerebral venous thrombosis, contrarily to vascular accidents of arterial origin[1], is an uncommon, potentially fatal entity, corresponding to less than 1–2% of cases of

encephalic vascular accidents[2]. Recent studies have reported a mortality rate of 5–15% [3,4]. It is a subdiagnosed condition because of the low rate of clinical suspect, considering non-specificity of signs and symptoms[5–8], which usually may impede an accurate and early diagnosis as well as na early treatment aiming at avoiding sequels for the patient. Therefore, it is essential that the radiologist has the capacity to recognize the parenchymal manifestations of this disease to proceed with the diagnostic investigation.

Currently, magnetic resonance angiography (MRA) is the method of choice for the diagnosis of cerebral venous thrombosis[4,7] although it detects only direct signs of thrombosis and is not capable of demonstrating the related parenchymal abnormalities. Due the existence of cerebral venous system anatomical variations like agenesia and hypoplasia, the correlation between MRA and MR images becomes is essential for the diagnosis confirmation.

The objective of this study was the determination of frequency and localization of parenchymal abnormalities on MRI and MRA in cases of cerebral venous thrombosis, as well as its correlation with the territory and the affected venous drainage.

MATERIALS AND METHODS

In the period between 1996 and 2004, we have retrospectively analyzed MRI and MRA studies of 21 patients in three different institutions (Hospital da Beneficência Portuguesa do Rio de Janeiro, Hospital Quinta D'Or and Hospital São Vicente de Paulo), with clinical and radiological diagnosis of cerebral venous thrombus. Our sample consisted of three men and 18 women, with ages ranging between three and 82 years (mean age 40 years, median 36 years).

In the three institutions, 0.5 T and 1.0T equipment with similar protocols were utilized, including, at least: gadolinium contrast- and non-contrast-enhanced axial SE (spin echo) T1-weighted images; axial, coronal and sagittal FSE (fast spin echo) T2-weighted images; and axial Flair (fast fluid-attenuated inversion recovery) images with slices thickness ranging between 5 mm and 7 mm. In one patient, an axial

GRE (gradient recalled eco) T2-weighted sequence* was performed.

2D PC MRA were performed in 18 patients, contrast-enhanced 3D TOF sequences in five, and 3D PC sequences in two patients. The PC sequences velocity was 20 cm/s. For images reconstruction, the maximum intensity projection (MIP) technique was employed.

Four patients had follow-up MRI and MRA studies. Three patients underwent digital subtraction angiography (DSA).

MRI and MRA studies were independently analyzed by two specialists, and the cases where there was disagreement were reviewed by one of them and included in the casuistic, provided no doubt remained regarding the interpretation of cerebral venous thrombosis signs on the images. The final diagnoses were confirmed by means of MRA studies.

Each patient MRI radiological finding was classified on the basis of the identification of direct signs of the cerebral venous system occlusion as well as indirect signs related to parenchymal and circulatory repercussions in the affected territory, and correlated with epidemiological and predisposing factors, time of disease evolution and clinical manifestations in patients by the time of diagnosis.

The qui-square test was employed for statistical analysis of secondary radiological findings, with a significance degree of $p < 0.05$.

RESULTS

The mean time of symptoms evolution was determined for 16 of 21 patients. Three of them presented symptoms with less than 48 hours, nine patients, between two and 30 days and four, more than 30 days.

Distribution by sex and age range is shown in Figures 1 and 2.

Factors related to the development of cerebral venous thrombosis (Figure 3) were identified in ten of 21 patients with data available in dossiers. Amongst the cases of infection, there were two of otitis, one of sinusitis and one of tonsillitis. Systemic infectious processes included gastroenteritis, pneumonia and urinary tract infection.

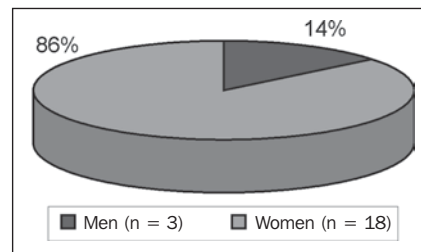Seventeen of 21 patients presented the clinical feature found by the time of diag-

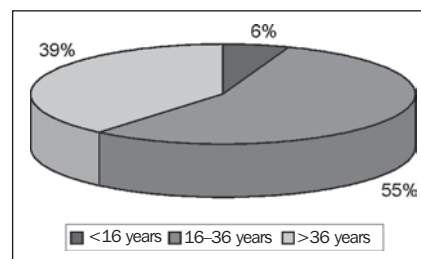**Figure 1.** Distribution of patients by sex.

Figure 2. Distribution of age ranges – Female patients (n = 18).

nosis, as per Figure 4, focal deficit, headaches and consciousness level alterations being the most frequent symptoms. For the remaining patients, this information was not available.

Cerebral venous thrombosis sites are demonstrated in Figure 5. The secondary radiological findings are described in Table 1. The main parenchymal manifestation was cortical and/or subcortical infarct or edema (Figures 6 and 7) in 16 patients (78%), eight (39%) of them presenting a hemorrhagic component (Figure 8). In four of 21 patients, hemorrhagic manifestations also were observed as intraparenchymal hematoma with perilesional edema (19%).

In five of 21 patients (24%) there was a thalamic-nuclear infarct or edema (Figure 9), four of them resulting from the straight sinus thrombosis and one from right transverse and sigmoid sinuses or ipsilateral jugular vein thrombosis.

In cases of straight sinus occlusion, the thrombus was evidenced at MRI of three patients, and in others, the thrombus was detected by MRA.

DISCUSSION

Cerebral venous thrombosis is a relatively rare condition when compared with vascular accidents of arterial origin[1]. Although it may affect individuals in any age
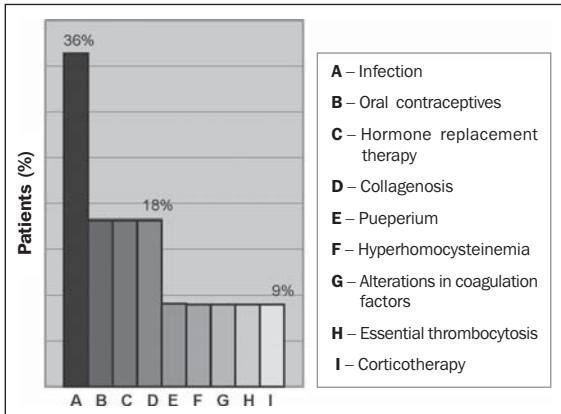
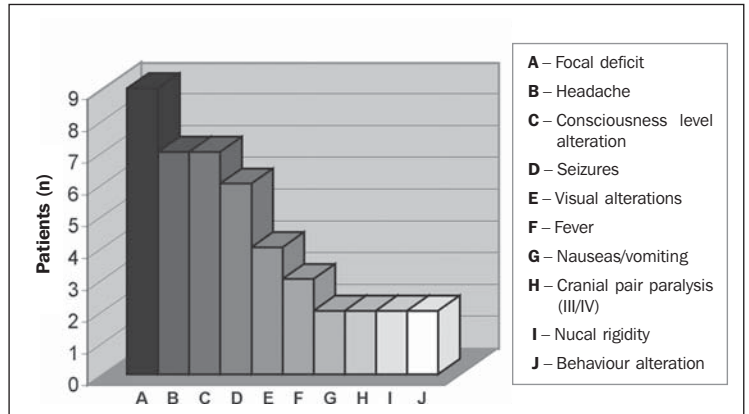**Figure 3.** Frequency of predisposing factors (n = 10).

A – Infection
B – Oral contraceptives
C – Hormone replacement therapy
D – Collagenosis
E – Pueperium
F – Hyperhomocysteinemia
G – Alterations in coagulation factors
H – Essential thrombocytosis
I – Corticotherapy

Figure 4. Frequency of clinical manifestation (n = 17).

A – Focal deficit
B – Headache
C – Consciousness level alteration
D – Seizures
E – Visual alterations
F – Fever
G – Nauseas/vomiting
H – Cranial pair paralysis (III/IV)
I – Nucal rigidity
J – Behaviour alteration
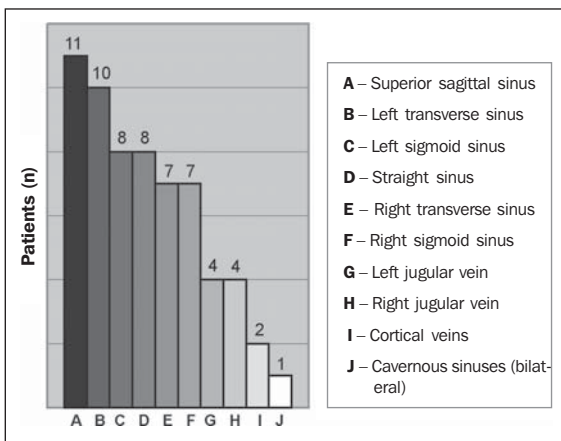
Figure 5. Venous thrombosis localization.

A – Superior sagittal sinus
B – Left transverse sinus
C – Left sigmoid sinus
D – Straight sinus
E – Right transverse sinus
F – Right sigmoid sinus
G – Left jugular vein
H – Right jugular vein
I – Cortical veins
J – Cavernous sinuses (bilateral)

Table 1 MRI and MRA: secondary findings statistical analysis.

Findings	Results		
	Positive	Negative	p
Cortical and/or subcortical edema or infarct	16	5	0.01
Congestion/collateral circulation	13	8	0.001
Meningeal enhancement	5	16	0.82
Thalamic/nuclear edema or infarct	5	16	0.82
Intraparenchymal hematoma	4	17	0.34
White matter lesion	3	18	0.16
Hydrocephaly	1	20	0.08
Intraventricular hemorrhage	1	20	0.08
Subarachnoid hemorrhage	1	20	0.08
Total	49	140	
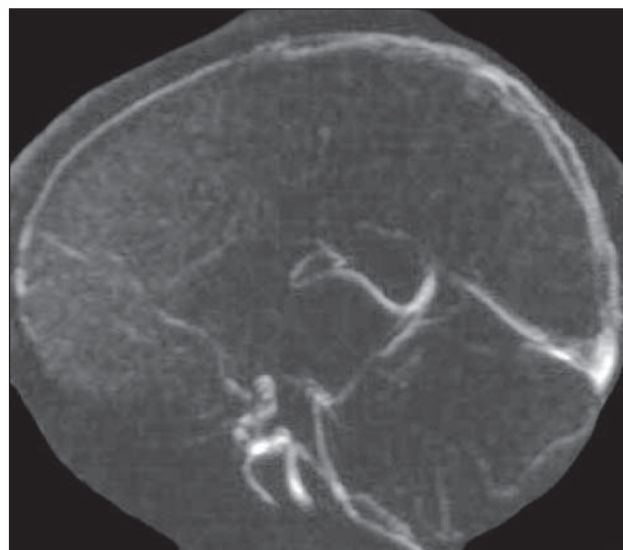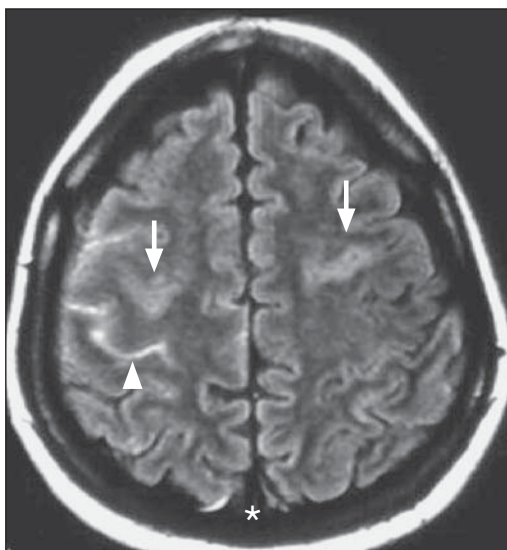
Figure 6. Flair MRI, axial view (**A**) showing bilateral, asymmetrical cortical alterations on frontal lobes, compatible with edema or infarcts (arrow) and presence of subarachnoid hemorrhage (arrowhead). Sagittal sinus presenting signal void inside suggesting flow patency (asterisk). 2D TOF MRA (**B**), showing signal intensity irregularities and alterations in the superior sagittal sinus, compatible with thrombosis and partial recanalization.
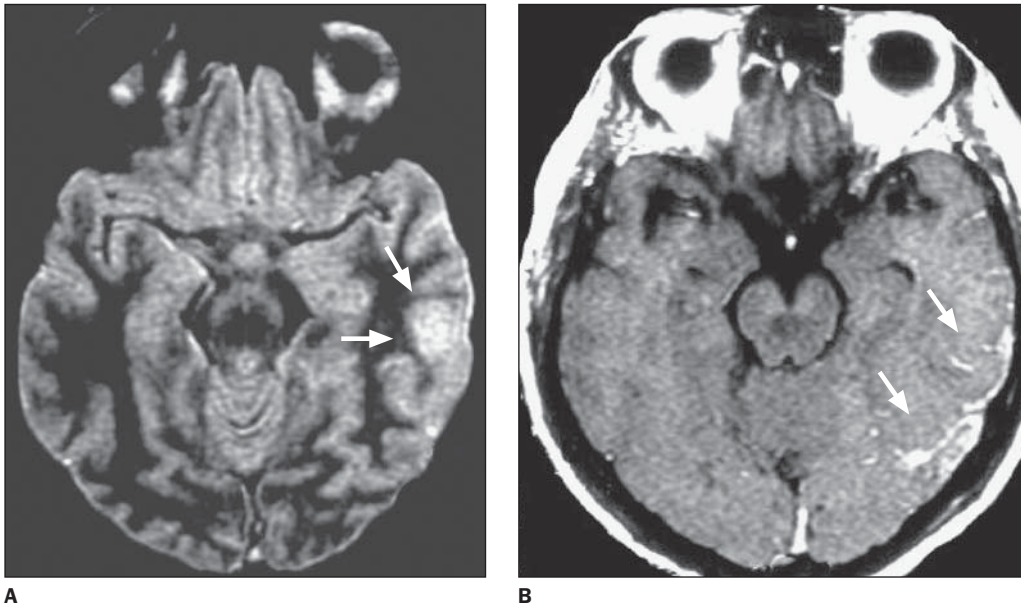
Figure 7. T2-weighted MRI (**A**), axial view showing a small area of cortical hypersignal on the left temporal lobe, compatible with edema or infarct (arrows). Contrast-enhanced T1-weighted MRI (**B**), axial view showing dural enhancement and signs of adjacent venous congestion adjacent to the lesion (arrows).
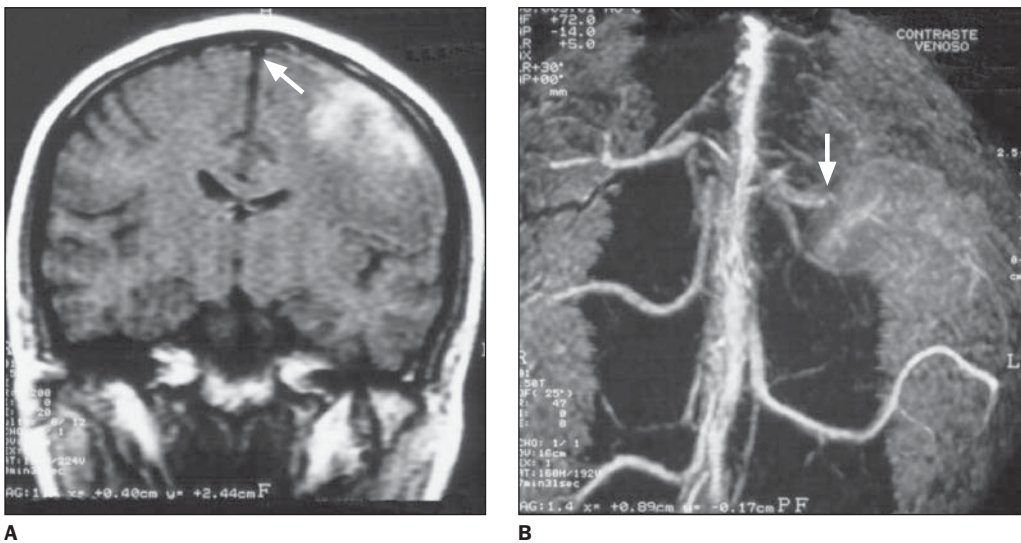
Figure 8. Non-contrast-enhanced T1-weighted MRI, coronal view (**A**) showing cortical-subcortical hemorrhagic infarct on the left frontal lobe. The superior sagittal sinus presents normal signal (arrow). Gadolinium-enhanced 2D TOF MRA (**B**) shows the cortical vein signal interruption at left (arrow), compatible with thrombosis. Patent superior sagittal sinus.

range, in the present study, only one child was affected. There was a prevalence in women between 16 and 36 years of age, differently from the male group (n = 3) were ages ranged between 46 and 59 years. The female prevalence corroborates the results reported in the literature.

Terazzi *et al*.[8] describe the duration time of the cerebral venous thrombosis, according the classification of Bousser *et al*., in acute (< 48 hours), subacute (between 48 hours and 30 days) and chronic (more than 30 days). According this classification, three patients presented an acute clinical picture, nine, subacute and four, chronic. The other could not be classified due the absence of clinical data in the patients dossiers.

The predisposing factors and clinical manifestation identified in the present sample were similar to those described in the literature[3,4,7,9,10].

At a lower rate, hematological alterations like essential thrombocytosis, hyperhomocysteinemia and other non-specified alterations of coagulation factors.

Cases of venous thrombosis in the superficial system predominated, and just in eight (38.0%) of 21 cases the deep venous system was affected. In agreement with the literature, the present study found rare cases of cavernous sinus and cortical veins thrombosis, principally isolated cases[7,10].

Venous thrombosis and parenchymal hemorrhage, either in the form of hemorrhagic infarct or parenchymal hematoma, occurred in 58% of cases. This finding has been described by several authors and is a result of lesion in vein walls, with rupture of the hematoencephalic barrier due intravascular hypertension in the occluded territory causing from petechial hemorrhages to intraparenchymal hematomas[3–7,10,11]. Additionally, in one of these patients, a T2-weighted GRE* sequence demonstrated a large area of hemorrhage within a voluminous infarct and that, on a SE T1-weighted sequence was visualized just as discreet
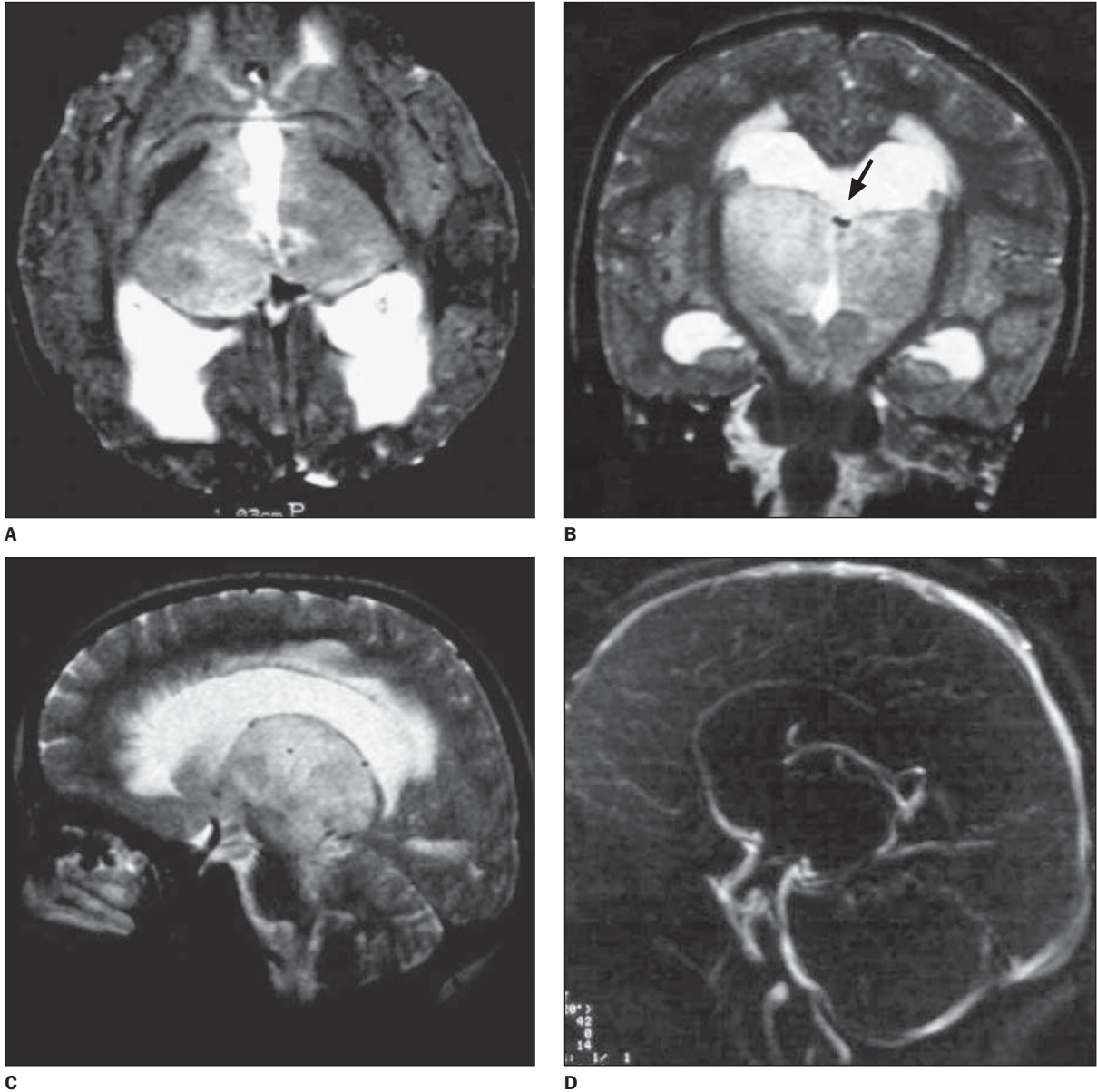
Figure 9. T2 weighted MRI, axial (**A**), coronal (**B**) and sagittal (**C**) views. Bilateral edema in thalami and basal nuclei, causing supratentorial hydrocephaly with signs of transependymal transudation. The internal cerebral veins present normal flow signal (arrow). Straight sinus with absent signal inside, possibly corresponding to hypointense thrombus. 2D TOF MRA (**D**). Absence of flow signal on the straight sinus, indicating thrombosis. Internal cerebral veins with preserved flow.

foci of hypersignal. This finding was in agreement with the literature that reports a higher sensitivity of GRE sequences in relation to conventional SE and Fair sequences for detection of parenchymal of parenchymal hemorrhages - even the small ones -, and in the identification of subarachnoid hemorrhage[7,10].

In the present study, one has not observed parenchymal abnormalities producing a mass effect (characterized by fading of cortical and compression effect over the ventricular system), without parenchymal signal alteration at the T2-weighted sequence, according studies by Tsai *et al*.[6] and Yuh *et al*.[9].

Evidences of infarct or edema affecting the thalamus and basal nuclei were observed in five patients, this finding being related principally to straight sinus thrombosis in four patients. In only one patient it was a result of right transverse and sigmoid sinuses and ipsilateral jugular vein thrombosis.

The second most frequent abnormality was the circulatory one, present in 62% of cases, and depicted by signs of venous congestion and collateral circulation diagnosed at MRI as intravascular enhancement after paramagnetic contrast agent injection[2].

Another frequent manifestation, also related to circulatory alterations resulting from cerebral venous thrombosis was the presence of dural enhancement adjacent to parenchymal lesions (infarct or hematoma). This is explained by the fact that the dura

mater has veins inside which, with the venous thrombosis and slowing of the blood flow, enhance due the venous congestion in the affected territory[2].

A regression of parenchymal alterations was observed in the four patients with follow-up studies and there was just one case of death of a patient presenting left transverse and straight sinuses thrombosis, progressing to a condition of intracranial hypertension and progressive decay of the neurological picture.

Thrombosis of the deep venous system is an uncommon condition with high morbidity and mortality rates, and the absence of typical clinical findings results in diagnostic difficulty, mistakes and delayed treatment start[12,13].

In the present study, thrombosis of the deep venous system involving isolatedly the straight sinus, occurred in only one patient. In all the other seven cases, thrombosis occurred concomitantly in structures of the superficial venous system. This pattern of involvement of multiple structures of both superficial and deep venous systems also has been reported by Lafitte *et al*.[5], Ming *et al*.[12] and Crawford *et al*.[13], and has been attributed to a trend towards retrograde progression of the thrombus present in the superficial venous system.

In most of our cases, the main signs of the presence of thrombosis of the deep venous system were due to the localization of the secondary parenchymal manifestations which were found in all of the patients with cerebral venous thrombosis, similar to data reported by Lafitte *et al*.[5]. In half of cases (four patients) of the present study, there was a thalamic-nuclear infarct or edema, and in the other patients one has observed hemorrhagic infarct of varied distribution in three cases, and diffuse alteration of the white matter in one patient.

The deep venous system present a more constant anatomy and parenchymal alterations occur in a more predictable pattern and may involve the thalami, basal nuclei and the superior portion of the cerebellum and mesencephalon[5,10]. In cases where parenchymal alterations consist basically of bilateral or, more rarely, unilateral signal alterations on the thalami, the deep venous thrombosis and infarct of the basilar artery and thalamus-perforating arteries

(especially the paramedian thalamic arteries of Percheron) are included in the differential diagnosis, especially if the symptoms are recent and with fast progression[14,15].

When alterations are cortical and/or subcortical, the diagnosis of infarct of arterial etiology should be the first hypothesis to be considered, since arterial thromboembolism is much more frequent than cerebral venous thrombosis. However, arterial infarcts are characterized by well defined anatomical territory, which does not occur in venous infarcts involving the superficial system. For this reason, in cases of cortical and/or subcortical infarcts in atypical sites one should suspect cerebral venous thrombosis[4].

The superior sagittal sinus thrombosis should be taken into consideration in the presence of parenchymal alterations near the midline, involving frontal, parietal and occipital lobes. When bilateral, usually they are asymmetrical[3]. An alteration of signal in the posterior region of the temporal lobe should raise suspect of an infarct by transverse sinus thrombosis on the same side of the lesion[3]. The differential diagnosis of lesions in these sites includes arterial infarct, contusion and encephalitis[3].

In the present study, white matter signal alterations were not frequent and were identified in only three of the 21 patients evaluated (14,3%). In one of these cases, a diffuse alteration of the periventricular white matter signal attributed to a vasogenic edema in a 36-year old patient with a history of nephropathy and systemic arterial hypertension presenting signs of venous congestion and extensive collateral circulation observed at MRI, the dural sinuses presenting absent signal. The MRA was inconclusive and the final diagnosis was achieved only by means of cerebral angiography that showed a diffuse thrombosis of dural sinuses and deep venous system with an intense superficial and deep collateral circulation. Despite the scarcity of data in the literature on this subject, the significance of this finding is in the reversible character of these lesions which was observed in follow-up studies of this patient after diagnosis and an appropriate treatment.

Only one patient presented hydrocephaly attributed to accentuate edema in

thalami and basal nuclei, causing compression over the third ventricle and supratentorial hydrocephaly similar to those described in studies of Crombé *et al*.[16] and Crawford *et al*.[13]. Although the cerebral venous thrombosis physiopathology indicates the possibility of ventricular system dilatation during the progression of the disease, this was not what we found in the present study[9].

Pitfalls were observed on MR images of three patients. In two of them there was a signal void inside the thrombosed dural sinus mimetizing flow patency. These patients presented a period of symptoms development longer than two months and the radiological investigation occurred belatedly.

In another patient, despite the presence of cortical infarcts in the frontal lobes, there was no superior sagittal sinus signal alteration, with signal void on all the weighting images. The final diagnosis of cerebral venous thrombosis was achieved by means of MRA and cerebral angiography compatible with thrombosis with partial recanalization of this sinus.

Both Lafitte *et al*.[5] and Zaiqiang *et al*.[17] emphasize the importance of a radiological investigation to be performed within up to two weeks from the first clinical manifestations, a period where the thrombus may be iso- or hyperintense on T1-weighted sequences. During the chronic phase there is a progressive loss of signal intensity on T1- and T2-weighted sequences and the thrombus presents a poorly defined aspect and may be partial or completely recanalized, resulting in flow-void. This may generate hyposignal on MR images, and is an acknowledged cause of false-negative results which reduces the method sensitiveness.

MRI provides superior anatomical detail when compared with CT, but is limited as regards differentiation between the hypersignal generated by the slowed blood flow and the presence of thrombosis, and, besides, the signal intensities change along time, with possible false-negative results both in hyperacute and chronic cases[18].

In the present study, despite several secondary radiological findings related to cerebral venous thrombosis, only the presence of cortical/subcortical infarct or

edema, or the existence of venous congestion and collateral circulation demonstrated statistical significance (Table 1). It is possible that these findings indicate a higher clinical and radiological suspicion of cerebral venous thrombosis. Especially in these cases, the assessment by means of MRA is indicated to define the certainty diagnosis.

An investigation for cerebral venous thrombosis should start with an evaluation of the parenchyma through conventional MRI with a protocol including T1- and T2-weighted sequences in at least two mutually orthogonal views to avoid possible artifacts. The MRA is a non-invasive method that allows visualization of both deep and superficial systems, confirming the diagnosis in suspect or inconclusive cases diagnosed by MRI.

CONCLUSION

Cerebral venous thrombosis is an uncommon cause of encephalic vascular accidents, with a favorable prognosis because of the lesions reversibility. However, its diagnosis is highly dependent on the radiologist capacity to recognize the disease presentations, principally in cases where the diagnosis is suggested by parenchymal alterations rather than necessarily by visualization of the thrombus itself. An accurate and rapid diagnosis allows an immediate treatment, reducing the morbidity and mortality rates.

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