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ORIGINAL COMMUNICATION

Cerebral venous thrombosis

3-year clinical outcome in 55 consecutive patients

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Abstract An early diagnosis and heparin therapy have contributed to a decreased mortality in cerebral venous thrombosis (CVT). However, predictors of outcome are difficult to identify, because most studies suffered heterogeneity in diagnostic findings and treatments, retrospective design, and recruitment bias. The aim of this study was to evaluate the clinical outcome in 55 consecutive patients with CVT admitted over a 4-year period. The study population consisted of 42 women and 13 men, with a median age of 39 years (range 16–68). The diagnosis was performed with MRI in 53 patients, and angiography in 2. The outcome was assessed with the modified Rankin scale (mRs). After a median follow-up of 36 months (range: 12–60), 45 patients were independent (mRS 0–2), and 10 were dependent or dead (mRS 3–6). Of 48 survivors, 7 had seizures, 6 motor deficits, 5 visual field defects, 29 headache (migraine in 14, tension headache in 13, other in 2). The logistic regression analysis found focal deficits and cancer at time of diagnosis, as independent predictors of dependence or death at year 3, and isolated intra-cranial hypertension as an independent predictor of survival and independence. Mortality rates are low in the absence of cancer and focal deficits, and more than 80 % of survivors are independent after 3 years. However, 3/4 of survivors have residual symptoms. Therefore, despite a low mortality rate, CVT remains a serious disorder.

Key words cerebral venous thrombosis · ischemic stroke · hemorrhagic stroke · stroke outcome · isolated intra-cranial hypertension

Introduction

Thirty-five to 50 years ago, cerebral venous thrombosis (CVT) were considered as rare and associated with a poor outcome, with mortality rates ranging from 30 % to 50 % [3, 19–21]. This concept was revisited during the last decade for two reasons: (i) computed tomography (CT) [30], then magnetic resonance imaging (MRI) and magnetic resonance angiography (MRA) [23], have re-

placed cerebral angiography, and, nowadays, allow in emergency an accurate and non-invasive diagnosis of CVT, and (ii) heparin has been shown to reduce mortality in patients with CVT [14], although another study did not confirm this therapeutic effect [12].

Nowadays, the mortality rate at the acute stage has been reduced to approximately 5 % to 10 % [1, 2, 6, 8, 12, 15, 25, 26]. The identification of factors associated with a poor prognosis may influence the therapeutic strategy: thrombolysis which has been suggested as a possible treatment in highly selected cases [16, 17], is not appropriate in a patient who has a reasonable chance of having a good outcome under heparin. Previous studies have suggested that factors associated at admission with a poor short-term outcome are coma [2, 6, 12, 21], intracerebral hemorrhage [1, 2, 13, 16, 30], delta sign on CT [30], central nervous system infection [15], focal deficits [6, 8], symptoms of encephalopathy [15], defined as the association of multifocal deficits, seizures, abulia, delirium, stupor or coma, with or without intra-cranial hypertension [15], hemorrhagic cerebrospinal fluid [2], bilateral pyramidal tract signs [2], and generalized seizures [2]. Factors associated at admission with a good short-term outcome are normal consciousness [12], absence of intra-cerebral hemorrhage or infarct [6, 12, 15], younger age [15, 26], absence of encephalopathy [16], isolated intra-cranial hypertension [7, 26], and early heparin therapy [15].

However, the conclusions of most studies evaluating the outcome after CVT are weakened by the following issues: (i) a long period of recruitment leading to heterogeneity in the diagnostic methods and findings, especially in the radiological procedure and treatment [1, 6, 8, 15]; (ii) a retrospective design, leading to incomplete case ascertainment and underestimation of minor sequelae [1, 2, 8, 6, 15, 21, 26]; (iii) a recruitment bias with a high proportion of benign cases such as isolated intracranial hypertension [1, 8, 25]; (iv) the absence of multivariate analysis [2, 26]; or (v) evaluation in the setting of a drug trial [12], *i. e.* in a group of patients who are not always representative of all patients, and are usually treated in highly specialized units. Therefore, the identification of predictors of outcome remains difficult.

The aim of this follow-up study was to evaluate the clinical outcome in 55 consecutive patients with CVT admitted over a 4-year period [1995–1998] and to determine predictors of death and dependence. The radiological follow-up is not reported here.

Patients and methods

We included all consecutive patients admitted over a 4-year period [1995–1998] in the stroke, neurosurgery and intensive care departments of the Lille University Hospital, and of the Hospital of Lens, who met criteria for CVT: (i) a clinical history consistent with that of a CVT, as defined by Bousser and Russel [9], and (ii) evidence of a cerebral sinus or venous occlusion on MRI and MRA, or on conventional angiography, according to the current diagnostic criteria [9]. Patients with isolated cavernous sinus thrombosis, and patients below 15 years of age were not included. Of 57 patients who had inclusion criteria, we excluded 2 who lived in other countries; however, we know they were symptom-free at discharge and, from birth certificates, they were still alive at the end of the follow-up period. Patients were recruited in emergency for a stroke, a rapidly progressive focal deficit, headache, seizures, or any association of these symptoms.

The study population consisted of 55 patients (42 women and 13 men), with a median age of 39 years (range: 16–68). Fifty-three were admitted to a stroke department (50 in Lille, 3 in Lens), and 2 in an intensive care department (Lille). No patient was admitted to the neurosurgery department. The diagnosis of CVT was performed with MRA/MRI in all patients, except in 2 with contraindications, 1 with metallic ocular foreign body, and 1 with severe obesity (175 kg/1.68 m), who underwent a conventional angiography.

The 1st symptom was headache in 54 patients (98.2 %) and a generalized seizure in 1 (1.8 %). At admission, the patients presented with 4 major clinical syndromes: (i) isolated intra-cranial hypertension (defined as any combination of headache, vomiting, papilledema, without any other symptoms or signs, except a VIth nerve palsy) in 17 patients (30.9 %); (ii) isolated and transient acute headache mimicking pure subarachnoid hemorrhage in 6 (10.9 %); (iii) progressive focal neurological deficit in 19 patients (34.5 %), with fever (body temperature > 37°5) in 11 and without in 8; (iv) acute focal neurological deficit in 17 patients (30.9 %), stable at time of diagnosis in 7, and reversible in 10 (within 24 hours in 3). Seizures (focal or generalized) occurred in 28 patients (50.9 %). Two patients had a history of deep venous thrombosis, and 2 of pulmonary embolism. Baseline characteristics are summarized in Table 1.

The management of patients (investigations and treatments) was left to the treating physician. However, all patients but one (who died within 2 hours after admission) were treated by intravenous unfractionated heparin as soon as the diagnosis was confirmed, irrespective of the presence of hemorrhagic changes, followed by oral anticoagulation. The median delay between symptoms onset and heparin therapy was 5 days (range: 1–33). Research of coagulation disorder was performed in 31 cases. Patients included in this study were not included in the previous study of our group [26], nor in the on-going international registry on cerebral venous and sinus thromboses.

The outcome was evaluated between October 1999 and January 2000 for all patients. All survivors and their general practitioners were first contacted by a letter in September 1999. They gave informed consent to be followed-up either at the outpatients clinic or by telephone. Thirty-five patients were reexamined by one of us (G.B) and we specifically looked for the presence of any of the following residual symptoms or signs: headache (migraine, tension headache or other types according to the International Headache Society [18], epileptic seizures, sensory-motor and visual disturbances. In patients interviewed by telephone, the presence of sensorimotor or visual changes was assessed with the following questions: do you feel you still have a motor/a sensory deficit in one limb? Do you feel you still have visual disturbances you never experienced before? In case of positive answer more details were asked. In case of negative answer we considered that the patient was free of these symptoms. We also collected information about CVT recurrence, deep vein thrombosis, pulmonary embolism, any other health problem leading to hospitalization, subsequent pregnancies and current medications. In 13 patients these data were obtained by a telephone interview with the patients and their general practitioner. The outcome was also assessed with the modified Rankin Scale [28] (mRS), patients with mRS scores ≤ 2 were classified as independent survivors, and patients with mRS scores > 2 were classified as dependent or dead. It has been shown [11] that the Oxford Handicap Scale is relevant, simple enough to be used reliably over the telephone and is therefore useful in large studies, without any clear difference between self- or carer-completed questionnaires [21]. Actually, the modified version of the Rankin scale we used is similar to the Oxford Handicap scale, with the addition of a score 6 meaning

Table 1 Baseline characteristics of the patients.

	number n = 55	percent (%)
Demographic data		
Median age (range) (years)	39 (16–68)	
Median delay (range) between onset and heparin (days)	5 (1–33)	
Women	42	76
Clinical findings, n (%)		
Isolated headache	17	30.9
Focal deficits	26	47.3
Focal or generalized seizures	28	50.9
Impaired consciousness (GDS ≤ 13)	10	18.2
Decreased visual acuity	3	5.5
Site of sinus occlusion, n (%)		
Superior sagittal sinus	37	
Lateral sinus	38	
Straight sinus	6	
Brain imaging n (%)		
Cerebral infarct	9	16.4
Cerebral hemorrhage or hemorrhagic infarct	19	34.5
No focal lesion	27	49.1
Causes n (%)		
Unknown	**12**	**21.8**
Local infection	**3**	**5.5**
Cancer	**5**	**9.1**
Puerperium	**3**	**5.5**
Lumbar puncture plus IV steroids	**4**	**7.3**
Any systemic disorder	**2**	**3.6**
systemic lupus erythematosus	1	
Crohn's disease	1	
Any coagulation disorder	**10**	**18.2**
Protein C deficiency	2	
Protein S deficiency	4	
Factor V gene mutation	1	
Prothrombin gene mutation	3	
Oral contraceptive	**24**	**52.2**

death. Major complications of anticoagulant therapy were defined as any cerebral or systemic hemorrhage leading to hospitalization or prolonged hospital stay, or requiring blood transfusion, or leading to neurological worsening or death.

The first step of the statistical analysis consisted of a bivariate analysis with the Mann-Whitney U test, and odds ratios (OR) with 95 % confidence intervals (CI), to compare independent survivors (mRS 0–2) and patients dependent or dead (mRS 3–6) for sex, age, delay of diagnosis, presence at time of diagnosis of isolated intra-cranial hypertension, seizures, focal deficits, decreased visual acuity, or altered consciousness, associated cancer or malignant hemopathy, site of sinus occlusion and presence of cerebral infarct or hemorrhage. The same comparison was also performed between patients with mRS 0–1 and mRS 2–6.

The second step of the statistical analysis consisted of a logistic regression analysis with a stepwise selection procedure, performed with the SAS [27] and SIPINA [32] packages, with mRS score (classified as 0–2 and 3–6) as dependent variables. We included in the model as independent variables all variables with a p value < 0.25 in the bivariate analysis (Mann Whitney U test, Chi square test with Yates correction or Fisher's exact test when appropriate), after having checked the absence of colinearity within variables. p values < 0.05 were regarded as statistically significant.

Results

The median follow-up was 36 months (range: 12 to 60). None of the 55 patient was lost to follow-up.

Oral anticoagulation followed heparin and was stopped after 6 months in 31 patients (56.4 %), of whom 7 were given aspirin by their general practitioner. Seventeen patients (30.9 %) were still under oral anticoagulant therapy at the end of the follow-up period, because of an underlying disease carrying a high thrombotic risk in 6 patients, and without any medical reason in 11 patients, in whom the general practitioner decided to continue despite the neurologist's advice to stop. No major complication of anticoagulant therapy was seen, at the acute stage and during the follow-up.

At the end of the follow-up period, 45 patients (81.8 %) were independent and 10 (18.2 %) were dependent or dead. Detailed characteristics of patients in both groups are presented in Tables 2 and 3. Of 7 patients who died, 4 died within 1 month, from brain herniation in 2, and end-stage cancer in 2, and 3 patients died later, from cancer in 2 and suicide in 1.

Of 48 survivors, 6 (12.5 %) had residual motor deficits, 3 (5.5 %) had visual field defects and 2 (3.6 %) had a decreased visual acuity due to optic nerve atrophy in patients with isolated intra-cranial hypertension. Recurrent epileptic seizures were observed in 7 patients (focal in 2, generalized in 3, both in 2) out of 28 who had had seizures at the acute stage. Twenty-nine patients (52.7 %) had residual headache, fulfilling criteria for migraine in 14, tension headache in 13, and not classified in 2. Twenty-seven patients used analgesic drugs at least once a week. Fifteen (31.3 %) of the 48 survivors were completely asymptomatic. No death occurred in patients admitted without focal deficits.

Three patients (5.5 %), including 2 with protein S deficit, developed a deep venous thrombosis during the follow-up period, but no recurrence of CVT was observed. Of 3 subsequent pregnancies, 2 remained uneventful and 1 led to spontaneous miscarriage within 12 weeks of pregnancy.

The logistic regression analysis found presence of a neurological deficit at time of diagnosis ($p = 0.03$) and presence of a cancer or malignant hemopathy ($p = 0.038$) as independent predictors of dependence or death (mRS ≥ 3) after 3 years, and isolated intracranial hypertension at time of diagnosis as an independent predictor of survival and independence after 3 years (mRS < 2) ($p < 0.01$). This model correctly predicted the outcome in 87 % of the patients: 44 of 45 patients with good outcome and 4 of 10 patients with poor outcome.

Table 2 Baseline characteristics of the 55 patients with cerebral venous thrombosis: comparison of patients with complete objective recovery with or without subjective complaints (modified Rankin Scale [mRS] 0–1) and the remainders (mRS 2–6). NA: not applicable. GCS: Glasgow Coma Scale [29]. OR means odds ratio. CI means confidence interval. An OR > 1 means that patients who have the criterion are more likely to have a poor outcome (mRS 2–6).

	mRS 0–1 (n = 42)	mRS 2–6 (n = 13)	p*	OR (95 % CI.OR)
Demographic data				
Median age (range) (years)	39 (16–68)	41 (28–64)	0.13	
Median delay (range) between onset and heparin (days)	5.5 (1–33)	6 (2–20)	0.62	
Women	35 (83)	7 (54)		1.5 (0.6–4.3)
Clinical findings, n (%)[a]				
Isolated headache	17 (40)	0 (0)		NA
Focal deficits	15 (36)	11 (85)		0.4 (0.2–1.1)
Focal or generalized seizures	19 (45)	9 (70)		0.7 (0.2–1.8)
Impaired consciousness (GDS ≤ 13)	5 (12)	5 (38)		0.3 (0.1–1.2)
Decreased visual acuity	2 (5)[c]	1 (8)[d]		0.6 (0.1–7.4)
Site of sinus occlusion, n (%)				
Superior sagittal sinus	29 (70)	8 (61)		1.1 (0.4–3.0)
Lateral sinus	30 (71)	8 (61)		1.2 (0.4–3.1)
Straight sinus	3 (7)	3 (23)		0.3 (0.1–1.7)
Brain imaging n (%)				
Cerebral infarct	5 (12)	4 (31)		0.4 (0.1–1.7)
Cerebral hemorrhage or hemorrhagic infarct	13 (31)	6 (46)		0.7 (0.2–2.1)
No focal lesion	24 (57)	3 (23)		2.5 (0.6–9.6)
Causes n (%)[b]				
Unknown	7 (21)	5 (23)		0.4 (0.1–1.6)
Local infection	2 (5)	1 (8)		0.6 (0.1–7.4)
Cancer	1 (2)	4 (31)		0.1 (0.0–0.8)
Puerperium	3 (7)	0 (0)		NA
Lumbar puncture plus IV steroids	4 (9)	0 (0)		NA
Any systemic disorder	2 (5)	0 (0)		NA
Any coagulation disorder	9 (21)	1 (0)		2.8 (0.3–24.1)
Oral contraceptive therapy	19 (45)	5 (38)		1.2 (0.4–3.8)

* Mann-Whitney U test. [a] More than 1 clinical finding in several patients. [b] Does not reach 100 % because of unknown causes. [c] in 2 patients with intra-cranial hypertension. [d] not detailed visual symptoms occurring a few hours before coma and death

Table 3 Baseline characteristics of 55 patients with cerebral venous thrombosis: comparison of independent survivors (modified Rankin Scale [mRS] 0–2) and patients dependent or dead (mRS 3–6). NA: not applicable. GCS: Glasgow Coma Scale [29]. OR means odds ratio. CI means confidence interval. An OR > 1 means that patients who have the criterion are more likely to have a poor outcome (mRS 3–6).

	mRS 0–2 (n = 45)	mRS 3–6 (n = 10)	p*	OR (95 % CI.OR)
Demographic data				
Median age (range) (years)	39 (16–68)	43 (39–64)	0.03	
Median delay (range) between onset and heparin (days)	5 (1–33)	7 (3–20)	0.52	
Women	38	4		0.2 (0.0–0.7)
Clinical findings, n (%)[a]				
Isolated headache	17 (38)	0 (0)		NA
Focal deficits	17 (38)	9 (90)		14.8 (1.7–127.5)
Focal or generalized seizures	20 (44)	8 (80)		5.0 (0.95–26.2)
Impaired consciousness (GDS ≤ 13)	6 (13)	4 (40)		4.3 (0.0–20.0)
Decreased visual acuity	2 (4)[c]	1 (10)[d]		2.4 (0.2–29.3)
Site of sinus occlusion, n (%)				
Superior sagittal sinus	30 (66)	7 (70)		1.2 (0.3–5.2)
Lateral sinus	32 (71)	6 (60)		0.6 (0.3–5.5)
Straight sinus	4 (9)	2 (20)		2.6 (0.4–16.1)
Brain imaging n (%)				
Cerebral infarct	6 (13)	3 (30)		4.4 (0.8–24.0)
Cerebral hemorrhage or hemorrhagic infarct	15 (33)	4 (40)		1.3 (0.3–5.5)
No focal lesion	24 (54)	3 (30)		0.4 (0.1–1.6)
Causes n (%)[b]				
Unknown	8 (18)	4 (40)		3.1 (0.7–13.5)
Local infection	2 (5)	1 (10)		2.4 (0.2–29.3)
Cancer	1 (2)	4 (40)		29.3 (2.8–308.0)
Puerperium	3 (2)	0 (0)		NA
Lumbar puncture plus IV steroids	4 (9)	0 (0)		NA
Any systemic disorder	2 (4)	0 (0)		NA
Any coagulation disorder	10 (22)	0 (0)		NA
Oral contraceptive therapy	19 (45)	5 (50)		0.8 (0.3–2.8)

* Mann-Whitney U test. [a] More than 1 clinical finding in several patients. [b] Does not reach 100 % because of unknown causes. [c] in 2 patients with intra-cranial hypertension. [d] not detailed visual symptoms occurring a few hours before coma and death

Discussion

Our study revealed that independent predictors of good outcome are absence of focal deficit, absence of cancer, and presence of isolated intra-cranial hypertension. However, although the mortality rate due to CVT is low, and most patients are independent after 36 months, CVT remains a serious disorder, because of an underlying disorder and frequent sequelæ.

Our study, as most recent studies conducted in western countries [1, 4, 6–8, 10, 12–15, 22–26, 31], was characterized by a prominence of young adults, a high frequency of headache at onset, and rarity of septic CVT. Although our study was conducted in 2 centers, the non-university center accounted for only 3 of the 55 patients, has a stroke unit, and is larger than several French University centers. Therefore, we cannot exclude a recruitment bias due to the weight of the tertiary university center, and the size of the non-university center. Our results, as those from previous studies [1, 4, 6–8, 10, 12–15, 22–26, 31], are valid only for patients recruited and treated in large centers with special interest in CVT. The outcome may be less favorable in hospitals without stroke unit, especially in the absence of neurological expertise or accessibility to early MRI. We should therefore bear in mind that, at the community level, the outcome of patients with CVT is probably worse than that found in studies conducted in specialized centers. Our study population has several specificities when compared with other studies: a short period of recruitment allowing homogeneous diagnostic procedures for the diagnosis of CVT and search for a cause, and a shorter delay between the onset of symptoms and heparin therapy.

Our study was not designed to evaluate the influence of heparin in CVT, but may provide indications on its tolerance: no major complication has been seen, at the acute stage and during the follow-up, even in patients with intra-cerebral hemorrhage or hemorrhagic infarcts at admission. Therefore, our study supports the opinion that heparin followed by oral anticoagulation is safe in patients with CVT, even in the presence of a cerebral hemorrhage. We were also surprised that several general practitioners were reluctant to stop oral anticoagulation after 6 months, or substituted aspirin after cessation of oral anticoagulation, although there is no proof of its potential effect on prevention of recurrences.

A few studies only have evaluated the long-term outcome in patients with CVT. A study conducted in 77 patients showed a good overall outcome with a complete recovery in 86 % of patients [25], but this study was retrospective and probably underestimated sequelæ. A recent prospective study in 59 patients [12] showed a fairly good overall outcome, 83 % of patients being independent after 12 weeks; however, this study was conducted in the setting of a drug trial, and was probably biased by the exclusion of the cases with isolated intracranial hypertension who needed a lumbar puncture. Such a bias is less likely to have occurred in our study, which was observational and also included patients from the intensive care department. However, our study also found a favorable outcome in terms of survival and independence, 81.8 % of patients being independent after a median follow-up of 36 months. The least biased study was probably the Portuguese study, in its prospective part, which was conducted in 20 centers and included University and non-university centers, and also oncology centers [15].

The decreased mortality rates over the last 30 years may be the consequence of: (i) the development of brain MRI allowing an early diagnosis of benign cases of CVT, which may have remained undiagnosed before the era of MRI, and may have spontaneously recovered [10, 12, 14], and (ii) early anticoagulation even in the hemorrhagic cases [23]. In our study, despite inclusion of patients from the intensive care department, mortality at the acute stage was only of 7.3 %, which is similar or lower than in most previous studies: 7 % in the prospective part of the Portuguese study [15], 10.2 % in the British-Dutch study [12], 17.7 % in the retrospective multicentric Dutch study [6]. Moreover, in our study, most deaths were not directly related to the CVT, but to its cause, although we also recruited the most severe cases from the intensive care departments. However, we cannot exclude that other patients admitted in our institution in a very severe state, with prominent systemic disorders, may have had an undiagnosed CVT, and died early after admission. This may be true especially in patients at the end-stage of cancers or malignant hemopathies.

Long-term sequelae identified among the 48 survivors are those usually described: epilepsy, motor deficits, and visual field defect [7, 8, 25]. They were more frequent than in other studies [25]. Epileptic seizures were more frequent in our study, with a rate of 50.9 % at the acute stage and 14.6 % in the 3-year survivors. Of 4 patients with seizures at the acute stage, 1 developed epilepsy in the following 3 years. However, this high rate of seizures was not associated with a worse functional outcome. Ophthalmological sequelæ were frequent, with 3.6 % of visual acuity loss and 5.1 % of visual fields defects, and occurred mainly in patients with isolated intra-cranial hypertension. Persistence of a motor deficit was also more frequent in our study than in previous ones [25] and was associated with a poor outcome. Residual headache was present in 52.7 % of survivors and led to important use of analgesic drugs. Therefore, survival and independence are not appropriate outcome measures in CVT trials, because they do not take into account subtle cognitive and behavioral changes, slight neuropsychological disorders, and epilepsy, which can impair the quality of life. The method used in our study did not allow the analysis of neuropsychological sequelae, as previously did de Bruijn et al. [12].

The 2 independent predictors of poor outcome were the presence of a neurological deficit and cancer. Impaired consciousness and presence of cerebral hemorrhage were not predictors of poor outcome, probably because of the small number of patients with a poor 3-year outcome, leading to a lack of statistical power. In the bivariate analysis there was, however, a tendency towards a worse outcome in patients with impaired consciousness (OR: 4.3) and cerebral hemorrhage (OR: 1.3) but they did not reach the level of significance. The independent predictor of good outcome identified in our study was an initial clinical presentation of isolated intra-cranial hypertension. This finding was identical in other studies [7, 26].

The delay between the first symptoms and the diagnosis has also been suggested as a factor of poor outcome [23]. However, it is difficult to confirm this finding because this effect could be masked by the fact that the most benign cases usually have a delayed diagnosis [26]. This may explain the lack of relationship between the delay of diagnosis and outcome in our study.

The prognosis of deep CVT, i. e. located in the straight sinus or in the vein of Galen, is also considered as poor [27, 32]. Such thromboses are known to induce bithalamic lesions and cognitive dysfunction, coma, and death [5, 22, 27, 32]. However, there are several recent reports of patients who recovered without sequelae [5, 22]. Thus, the outcome of deep CVT appears difficult to identify clearly, the criteria of bad outcome in these forms could be the presence of coma and extreme ages of life [22]. In our study, there was also a tendency towards a poorer 3-year outcome in patients with deep CVT (OR: 2.6) but it did not reach the level of significance, probably because of the small number of cases. In the Dutch-British study, there was also a tendency towards a worse outcome in straight sinus thrombosis [12].

No recurrence of CVT was found, although 11 % of patients had recurrence in Preter et al. study [25], in which one third of recurrences were in-patients with Behçet's disease [25].

One of the major limitations of our study, and of all previous studies on CVT, was the small number of patients, leading to a poor statistical power, and a limited number of factors that could be included in the multivariate analysis. The international cerebral venous thrombosis study, which is currently running, will allow more power in the statistical analysis.

In conclusion, the 3-year mortality rate is low in patients with CVT, in the absence of cancer or focal deficit, and more than 80 % of survivors are independent. However, 3/4 of survivors have residual symptoms. Therefore, despite a low mortality rate, CVT remains a serious disorder, most survivors being symptomatic.

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