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Cerebral Venous Thrombosis in the Intensive Care Unit

Yahya Lahrichi[1*], Fatimazahra Haddari[1], Hiba Essolaymani[1], Ibrahim Bechri[1], Ali Derkaoui[1], Abdelkarim Shimi[1], Mohammed Khatouf[1]

[1]Anesthesiology and Intensive Care Department A1, Hassan II University Hospital, Fez, Morocco

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***Corresponding author:** Yahya Lahrichi
Anesthesiology and Intensive Care Department A1, Hassan II University Hospital, Fez, Morocco

Abstract **Original Research Article**

Cerebral venous thrombosis or cerebral thrombophlebitis is rare forms of cerebrovascular accidents that affect the venous sinuses and/or cerebral veins. These thromboses are responsible for an obstacle to cerebral venous return and cause suffering of the cerebral parenchyma, ranging from isolated intracranial hypertension (ICHT) to ischemia or parenchymal hemorrhage. It is a rare but serious pathology with an estimated incidence of 5 to 6 cases per million inhabitants. They are characterized by their great diversity in etiology and clinical presentation with a polymorphic and misleading symptomatology. Thanks to the development of neuroradiological means, the positive diagnosis of cerebral thrombophlebitis has progressed spectacularly. Cerebral CT is the tool of first choice. MRI, when available, remains the examination of choice for the diagnosis of CVT. The initiation of anti-coagulant treatment associated with the treatment of HTIC is the mainstay of treatment. The early treatment of CVT is no longer a pathology with a fatal outcome nowadays, with a clear reduction in morbi-mortality. The objective of our work is to describe the epidemiological, clinical, paraclinical, therapeutic and evolutionary characteristics through a retrospective study over a period of 2 years from January 2021 to December 2022, including all patients admitted to the in the intensive Care Department A1 of the Hassan II University Hospital of Fez for the management of CT.

Keywords: Cerebral venous thrombosis, MRI, anticoagulant treatment.

INTRODUCTION

Cerebral venous thrombosis (CVT) is a relatively rare but serious condition, responsible for a significant morbidity and mortality. It is characterized by polymorphism of clinical presentations, often misleading. Through a retrospective study of two years, we report the epidemiological, clinical, paraclinical and therapeutic aspects of patients admitted for management of cerebral venous thrombosis (CVT) in the intensive Care Department A1 of the Hassan II University Hospital of Fez.

PATIENT AND METHODS

After the agreement of the local ethics committee, we performed a retrospective study in the intensive Care Department A1 of the Hassan II University Hospital of Fez, over a period extending from January 2021 to December 2022. Our study included all patients admitted to the A1 intensive care unit for management of cerebral thrombophlebitis whose records were usable. All patients admitted to the department for a neurological pathology other than cerebral thrombophlebitis, cases of CVT not hospitalized in ICU. The data are collected from the files archived in the department, based on a pre-established exploitation form and the use of Excel software for the analysis of the results.

RESULTS

During the study period, from January 2021 to December 2022, the Hassan II University Hospital of Fez recorded sixteen cases of CVT out of a total of 1498 hospitalizations, that is, a prevalence of 1%. The average age of our patients was 31 years with extremes of 20 and 51 years (Figure 1); there was a female predominance: eleven women and 5 men, i.e., a sex ratio of F/H of 2.2.Different risk factors are identified in our study (Figure 2). Anemia was found in 14 patients, i.e. in 68% of cases. Infection was found in 5 of our patients, i.e. 31.25% of cases. Oral contraception and previous miscarriages were present in four patients (25%). Only one of our patients had a CVT on day 11 of the postpartum period. The clinical pictures of our patients are varied, dominated by consciousness disorders, which are present in 11 of our patients, seizures, noted in eight patients (50%), occurred in a

febrile context in 3 patients. Cerebral computed tomography (CT) with injection of contrast medium was performed in all our patients, completed for seven of them by a cerebral MRI. Imaging revealed thrombosis of the sigmoid sinus in 11 patients (68% of cases), of the superior longitudinal sinus (SLS) in 56% of cases, of the transverse sinus in seven patients (43%) and of the lateral sinus in five patients (31%). Other abnormalities were also noted, including cerebral edema and venous infarction in ten patients (62.5%), hemorrhagic changes in 50% of cases, and meningeal hemorrhage in one patient with thrombosis of the frontal cortical vein. Signs of involvement in nine patients (56%) (Figure 4-7).

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Severe CVT were admitted to the intensive care unit; they benefited from symptomatic measures: artificial ventilation with sedation by midazolam associated with fentanyl in 81.25% of cases to control intracranial hypertension associated with osmotherapy by mannitol in five patients (31% of cases). Antiepileptic treatment with sodium valproate was instituted systematically in all our patients, associated with clobazam in 7 patients and phenobarbital in 4 patients with status epilepticus. Two of our patients underwent decompression surgery (12.5% of cases). Endovascular treatment consisting of thromboaspiration after diagnostic angiography was performed in three patients in our series with a good outcome in 66.6% of cases. All patients received curative anticoagulation during the entire duration of hospitalization in the intensive care unit with LMWH 100 IU/kg/12H with a relay by AOD in 5 patients and a relay by AVK in 31.25%. An adapted antibiotic therapy was instituted in ten patients (63.5%) whose etiology of the CVT was infectious or a co- infection was retained. The short-term evolution of our patients was marked by death in six of our patients (37.5% of the cases), in a picture of intracranial hypertension with encephalic death in 4 patients and refractory septic shock in 2 patients. On the other hand, six patients recovered a normal state of consciousness with a Glasgow score of 15, neurological sequelae such as seizures were noted in 4 of our patients.

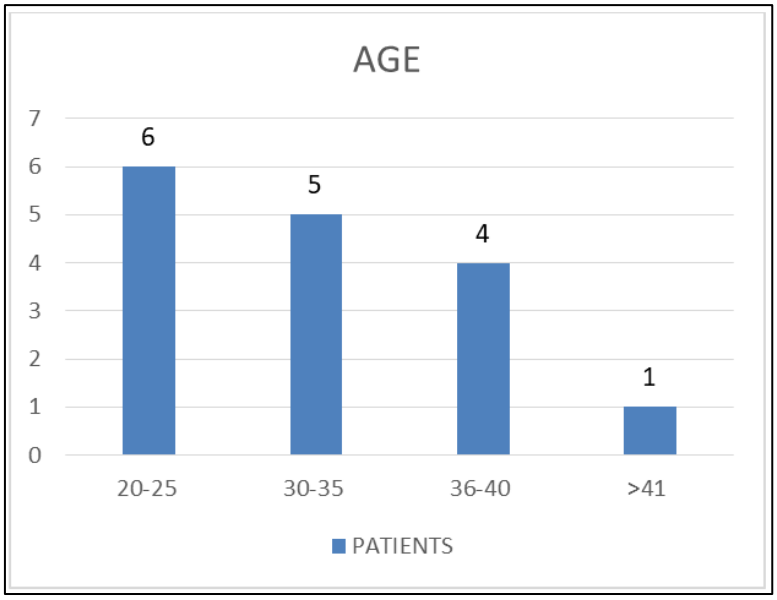

Figure 1: Age distribution of patients

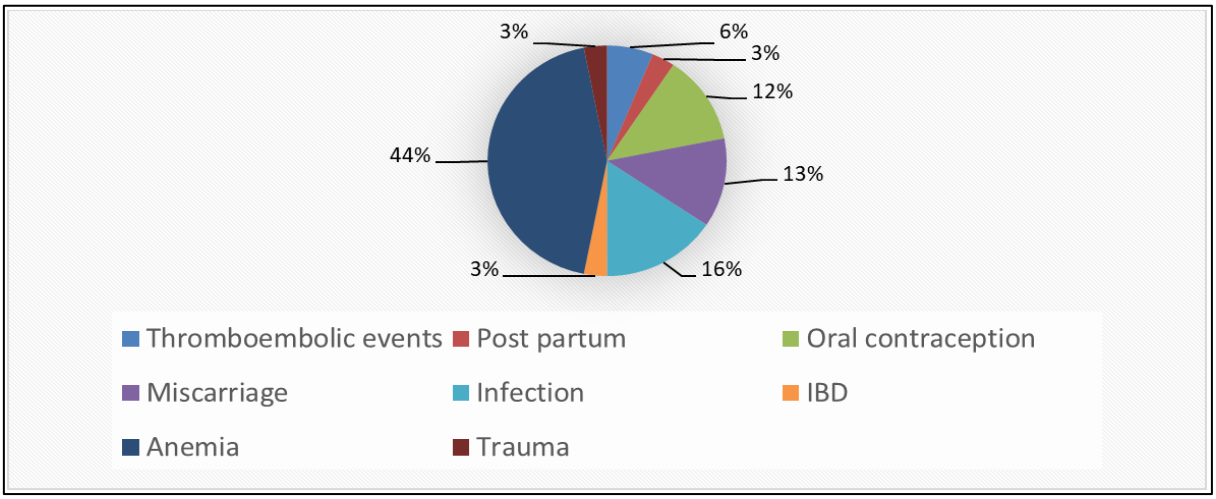

Figure 2: History of our patients

Reason for initial consultation	
Headaches	50 %
Complete HTIC sd	6%
Convulsive seizures	50%
Neurological deficiency	31%
CFP	18%
Meningial syndrome	18%
Fever	56%
Confusion	68%
Visual impairment	6%

Figure 3: Reason for patient consultation

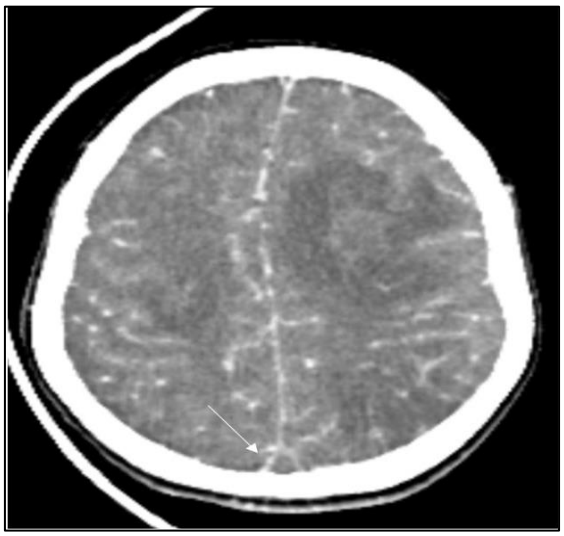

Figure 4: C+ brain CT in axial section: opacification defect of the superior longitudinal sinus, with intense enhancement of its wall, realizing the "Delta" or "empty triangle" sign (arrow)

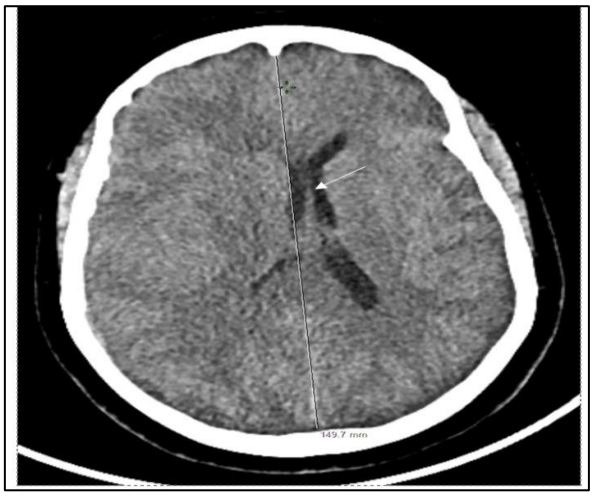

Figure 5: C-axial slice brain CT: obliteration of the cortical sulci at the sustentorial level, related to cerebral edema, with deviation of the midline to the left, related to subfalcoral involvement (arrow)

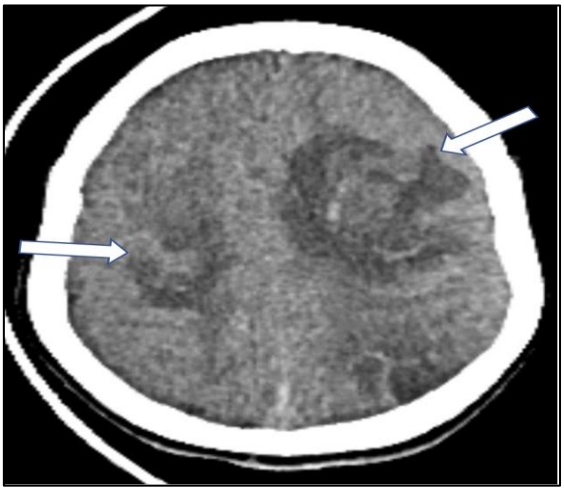

Figure 6: C- brain CT: Hypodense cortico-subcortical folds, mainly subcortical, left fronto-parietal and right frontal, without arterial systematization, containing spontaneously hyperdense areas, related to hemorrhagic remodeling (arrows)

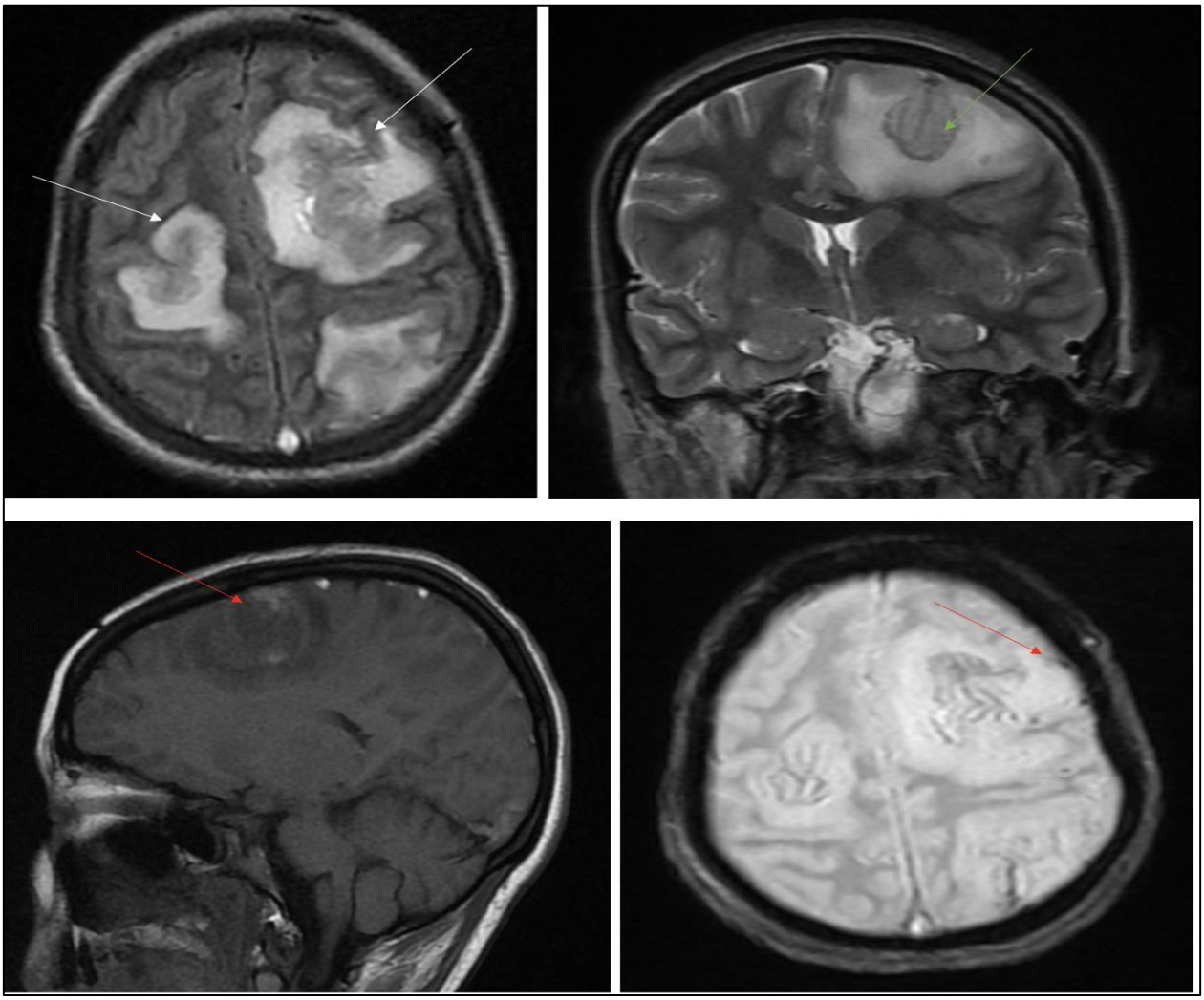

Figure 7: FLAIR, T2, T1 and T2* sequences of a cerebral MRI: Cortico-subcortical folds mainly subcortical at the left fronto-parietal and right frontal level, without arterial systematization, described in FLAIR hypersignal (white arrows) and T2 hypersignal (green arrow), in T1 hyposignal, containing within them areas in T1 hypersignal and T2* hypersignal, in relation to hemorrhagic reorganizations (red arrows)

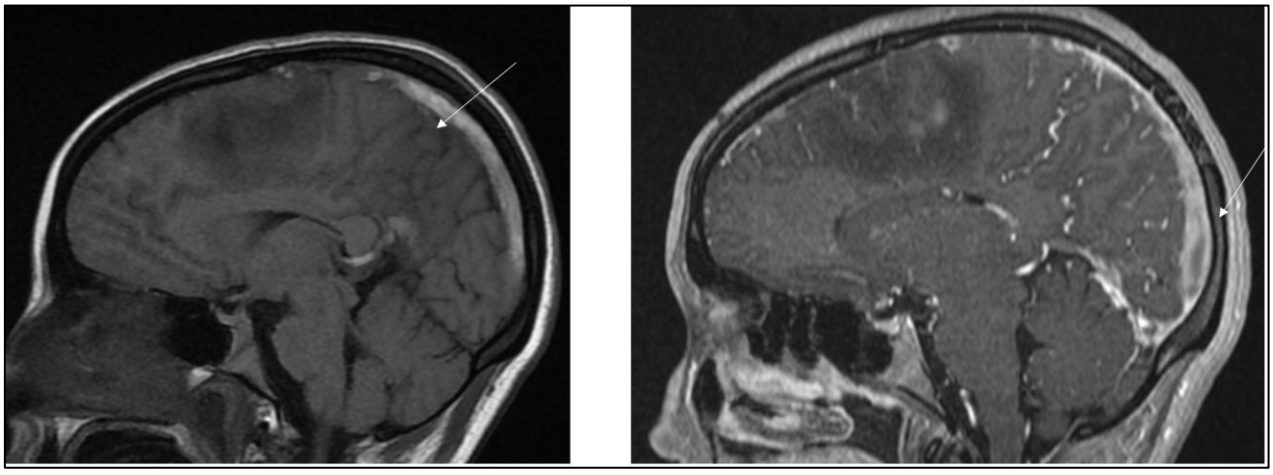

Figure 8: T1 brain MRI: Presence of a T1 hypersignal content in the superior longitudinal sinus, corresponding after injection to an extensive thrombus of the superior longitudinal sinus (arrows)

Table 1: Table comparing the etiologies found in the different series

	UH MARRAKECH FZ HADID[17]	UH FÈS A.DERKAOUI 20 [1]	Germany REUNER [18]	Netherlands E. Libourel [20]	Pays bas E. Libourel [20]	Our series
OC		50%	58%	5.9%	56	25%
PP	21.6%	25%	8%	5.9%	16	6.25%
Anemia	-	-	-	5.9%	-	68%
Dysthyroidia	-	-	-	-	-	
Infection	17.76%	25%	4%	59%	13%	43.75%
Neoplasia		12%				-
Constitutional abnormalities of haemostasis	6.6%	-	28%	11.7%	88%	31.25%
Behçet	17.7%	-	-	-	-	-
Trauma	-	-	-	-	8%	6.25%
inderterminate	33.3%	12%	20%	5.9%	24%	25%

DISCUSSION

CVT is a rare condition and a significant cause of stroke. It is definited by the more or less complete obstruction of a brain vein that drains into the venous sinuses of the dura mater [1]. The annual incidence of CVT is about 5.7 cases per million inhabitants in adults [2]; the incidence of our results is in line with the literature. We found, as in our study, a unanimous female predominance in the literature [3, 4].The average age of patients in our series was 31.3 years, consistent with the series of U.G. Cabrera in the ICU of the National Institute of Neurology and Neurosurgery in Mexico City [5]. Cabrera in the ICU of the National Institute of Neurology and Neurosurgery in Mexico City [5]. The pathophysiological mechanisms of CVT are the same as deep vein thrombosis, it can be triggered by injury to the vascular endothelium, venous dilatation with venous blood flow stasis or hypercoagulability of the blood, it is the "Virchow triad". Cerebral vein occlusion leads to local venous stasis and tissue hypoxia due to a decrease in cerebral blood flow, which in turn leads to ischemia and reactive cytotoxic edema leading to an increase in intracranial pressure [6]. In the literature, multiple predisposing factors for cerebral thrombophlebitis have been identified. These include pregnancy, contraceptive use, dehydration, certain medications, inherited coagulation disorders, systemic disease, head trauma, locoregional or systemic infection, and idiopathic causes [7]. The etiologies of CVTs are numerous and often interrelated. Septic CVTs represent about 12% of CVTs and most often complicate a local infection (sinusitis, otitis, pharyngitis, cellulitis of the face, brain abscess, subdural empyema or meningitis) or, more rarely, a remote infection (pneumopathy) [1]. Non-septic CVTs are secondary to local or general causes. CVTs related to a local cause (head trauma, arteriovenous malformation, jugular catheterization or lumbar puncture) represent only about 4% in some series [9]. General causes are dominated by three conditions: gravidopuerperal status, coagulation abnormalities, and estrogen/progestin [12]. Other causes are classic, but rarer: cancers (myeloproliferative syndromes or solid tumors), vasculitis or systemic diseases (Behçet's disease or systemic lupus erythematosus, for example). 69% of the patients in our series had a subacute onset, followed by an acute onset (25%) and then a chronic onset (6%), which is in line with the data in the literature. CVTs are characterized by a clinical polymorphism that reflects the multiplicity of presentations and problems of differential diagnosis. They have very varied clinical presentations associating to varying degrees signs of intracranial hypertension when the occlusion is limited to the dural venous sinuses and focal signs when the thrombosis involves the cortical veins with the appearance of focal edema or the formation of a venous infarct. Concerning the symptomatology, we find Headache, which is the most frequent symptom found in the literature [10] (in more than 80% of cases), focal deficits, convulsions (generalized or not), papilledema and consciousness disorders, ranging from confusion to deep coma. All conditions with signs of intracranial hypertension can mimic CVT. In addition to meningitis, the differential diagnosis also includes encephalitis (e.g. In addition to meningitis, the differential diagnosis also includes encephalitis (e.g., herpes encephalitis, septic encephalitis), brain abscess, cerebral hemorrhage (subdural hematoma, subarachnoid hemorrhage, primary intracerebral hemorrhage), ischemic infarction, brain tumors, as well as benign intracranial hypertension, migraine, hypertensive encephalopathy, eclampsia, and psychiatric illnesses [11]. The definite diagnosis of CVT is based on neuroimaging of the cerebral veins. Cerebral CT scan is most often requested first. It allows to confirm the diagnosis by showing a spontaneous hyperdensity of the thrombosed sinus without injection of contrast medium and after injection the significant enhancement of the sinus wall contrasting with the non-injection of the thrombosed lumen (delta or triangle sign). This is the most frequent sign, present in 30 to 46% of cases from the fifth day [8]. Nevertheless, the CT scan may remain normal in 20% of cases, which therefore does not eliminate the diagnosis [12]. MRI is certainly the best examination for the diagnosis of CVT with good sensitivities and

specificities. It allows to see the extent and the precise localization of the thrombosis and to analyze the impact on the brain tissue with a sensitivity to the speed of the flow in the vessels and the quasi-specificity of the signal given by the products of degradation of the hemoglobin during the phenomena of thrombosis or hemorrhage [13]. MRI was performed in 43.7% of the cases in our series. Cerebral angiography has lost its diagnostic value and is useful when MRI is insufficient, especially for the diagnosis of cortical stroke. Angiography has never been used in our series for diagnostic purposes only. It was performed in 3 of our patients for therapeutic purposes with thrombo aspiration and endovascular fibrinolysis, i.e. in 18.75% of cases. [13]. Lumbar puncture can be orienting by the observation of hyperpressure and by an analysis of cellularity and biochemistry (hyperproteinorachy generally < 1 g/L, number of red blood cells > 20/mm3, pleiocytosis). The electroencephalogram is abnormal in 75% of cases and shows nonspecific signs (generalized slowing or sometimes epileptic activity) [14, 15]. [Treatment is based on symptomatic, etiological and thrombolytic management. Symptomatic treatment ensures vital functions with the management of consciousness disorders, seizures and ICP [16]. Treatment of ICP depends on its severity, ranging from osmotherapy with mechanical ventilation to surgical evacuation of a compressive hematoma, or decompression craniotomy and CSF bypass under strict monitoring of intracranial pressure [22]. The efficacy of corticosteroids in the treatment of cerebral thrombophlebitis has never been studied in controlled trials [22]. The treatment of seizures is based on the use of effective antiepileptic drugs. On the other hand, some authors use antiepileptic drugs in the acute phase in a systematic way, but this practive is controversial in the literature[23]. In our study, 50% of patients received anticonvulsant treatment in the acute phase following the occurrence of seizures. The treatment was based on the single use of sodium valproate - associated, in one patient, with phenobarbital and clobazam following the onset of status epilepticus. Mannitol-based osmotherapy was used in 37.5% of cases, assisted ventilation with sedation in 82% and corticosteroid therapy in 68.75%. Finally, surgical decompression was reserved for HTIC refractory to medical treatment. It was used in 2 patients, the evolution was excellent with a recovery without sequelae and perfect autonomy in both cases. Antithrombotic treatment was based on anticoagulation initiated in the acute phase with intravenous heparin or subcutaneous low-molecular-weight heparin depending on body weight. This attitude is based on three small randomized studies, several case series and the large ICVST (International Study on Cerebral Vein and Dural Sinus Thrombosis) in which more than 80% of patients received anticoagulants. After the acute phase, patients may be given an oral anticoagulant (VKA or AOD) for a period of three to twelve months. A study was conducted involving 16 patients with CVT [24]: 7 patients were treated with Rivaroxaban, 9 with VKAs. Recanalization rates and clinical outcomes were excellent in both groups. No relevant and statistically significant difference between the VKA group and the FXaI group was found. In our series, AODs were used in 5 patients, i.e. in 31.25% of cases, and no bleeding complications were reported in these patients. Etiological treatment must be initiated rapidly and adapted to the underlying cause. In the case of septic cerebral venous thrombosis, antibiotic therapy is targeting the most frequently responsible germs, which are Staphylococcus aureus, streptococci including S. pneumoniae, anaerobic germs and gram-negative bacilli [25]. Invasive endovascular treatments (local thrombolysis, mechanical recanalization) should be reserved for isolated cases that worsen under optimal drug treatment. In our series, 3 patients underwent thromboaspiration with in situ fibrinolysis following non-response to a well-conducted anticoagulant treatment. The clinical evolution and prognosis of CVT is unpredictable on an individual basis. The clinical course and prognosis of CVT is unpredictable for each individual case, and some cases may progress within a few days to a fatal outcome, or to a complete recovery, or to the persistence of sequelae. Mortality varies between 4 and 38% depending on the series [22]. The main cause of death in ISCVT [20] was neurological, most often transtentorial herniation due to a focal hemorrhagic lesion or to multiple lesions with diffuse edema. In contrast to cerebral arterial infarction, CVTs generally have a good prognosis if diagnosed and treated early. Various studies have reported coma on admission and cerebral hemorrhage as predictive factors for an unfavorable outcome and mortality in patients with seizures has been described as three times higher than in patients without epilepsy. The data from our series find the same factors.

CONCLUSION

Although widely documented in the literature, CVTs are often misunderstood in daily practice because of their sometimes atypical presentation. The etiologies and differential diagnoses are extremely numerous. The contribution of magnetic resonance imaging and the introduction of early heparin therapy have led to a significant reduction in morbidity and mortality. Delayed treatment is a factor of poor prognosis, especially since atypical forms often lead to misdiagnosis.

CONFLICT OF INTEREST

No potential conflict of interest relevant to this article was reported.

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