

Non-Traumatic Cerebral Venous Thrombosis, an Entity with Complex Radioclinical Aspects in Adults. About 33 Cases

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DOI: [10.36347/sasjm.2021.v07i12.008](https://doi.org/10.36347/sasjm.2021.v07i12.008)

| Received: 07.11.2021 | Accepted: 11.12.2021 | Published: 19.12.2021

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Abstract

Original Research Article

Cerebral venous thrombosis (CVT) is a rare condition that can be life-threatening. It is characterized by clinical and radiological polymorphism. We carried out a retrospective study over a period of 34 months, from January 01, 2019 to October 31, 2021 ; from the files of patients referred for magnetic resonance imaging (MRI) or CT scan exploration in cerebral neurological symptoms context, with as judgment criterion the demonstration of a thrombus in a venous sinus or cerebral vein. We collected thirty-three (33) patients, with an average age of 39.06 years old and as extreme ages 18 years old and 76 years old. We noted a female predominance with a sex ratio of 1.2 in favor of female sex. The most of patients were explored with MRI, in 60% of cases. Our patient history was dominated by epilepsy in 13% of cases, followed by deep vein thrombosis of the lower extremities, toxic habits, cerebral venous thrombosis and combined contraception represented in 8% of cases each. The symptomatology presented by the patients was intracranial hypertension in 23% followed by headache in 20% of cases. The etiologies implicated were infectious in 28% of cases. The most affected sinus was the superior sagittal sinus in 41% of cases. Cerebral parenchymal complications were mainly consisted of ischemia with hemorrhagic infarction in 24% of cases.

Keywords: Thrombosis, sinuses, veins, brain.

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INTRODUCTION

Venous thrombosis mainly affects the lower extremities, but can rarely involve other areas of the veins, such as the veins and sinuses of the brain. Among the rare sites of venous thrombosis, cerebral circulation is one of the most fatal. In the vast majority of patients, thrombosis develops concomitantly in the sinuses and veins.

The goal of our study is

- To show the radiological characteristics in magnetic resonance imaging (MRI) and in computed tomography of symptomatic cerebral venous thrombosis.
- Enlighten clinicians on the details of the localization and associated lesions.

MATERIALS AND METHODS

We carried out a retrospective study over a period of 34 months, from January 01, 2019 to October

31, 2021 ; from the files of patients referred for magnetic resonance imaging (MRI) or CT scan exploration in cerebral neurological symptoms context, with as judgment criterion the demonstration of a thrombus in a venous sinus or cerebral vein.

Data processing were performed using Excel software.

RESULTS

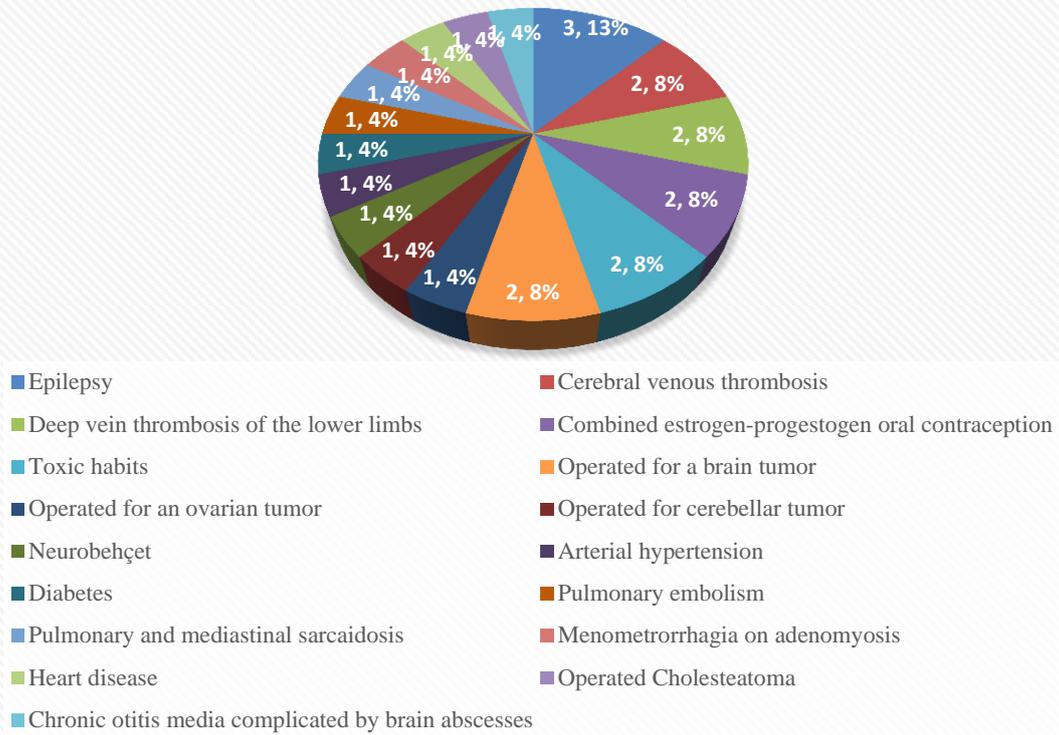
Thirty-three (33) patients were collected. The average age was 39.06 years old, with the extreme ages 18 years old and 76 years old.

Gender: 18 patients were female and 15 were male.

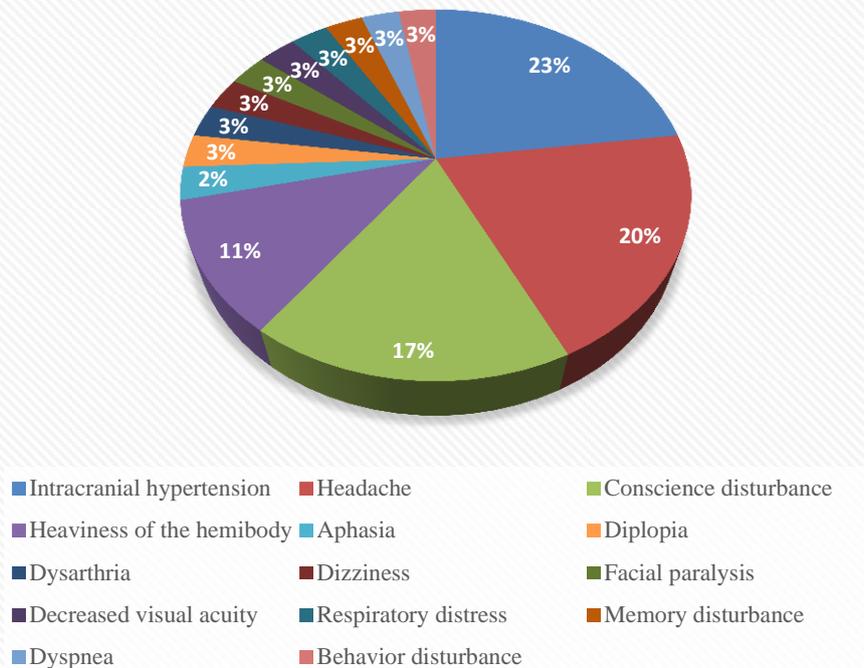
The exploration modality were: cerebral MRI n = 20, cerebral CT scan n = 7,

ARM n = 4 and CT angiography n = 2.

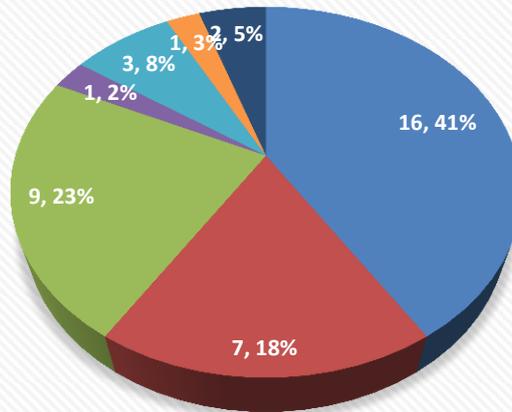
Distribution of patients by history



Distribution patients by symptomatology

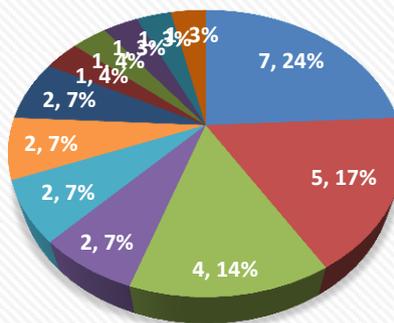


Distribution of patients by affected sinus



■ Superior sagittal sinus ■ Right lateral sinus ■ Left lateral sinus ■ Right sigmoid sinus
 ■ Left sigmoid sinus ■ Internal cerebral vein ■ Cortical vein

Distribution of patients by associated anomalies



■ Ischemia with hemorrhagic infarction ■ Ischemia without hemorrhagic infarction
 ■ Hematomas ■ Empyemas
 ■ Brain abscesses ■ Subarachnoid haemorrhage
 ■ Filling of mastoid cells ■ Pansinustitis
 ■ Meningoencephalitis with pyoventriculitis ■ Recurrent cholestatoma
 ■ Chronic subdural hematomas ■ Brain tumor process

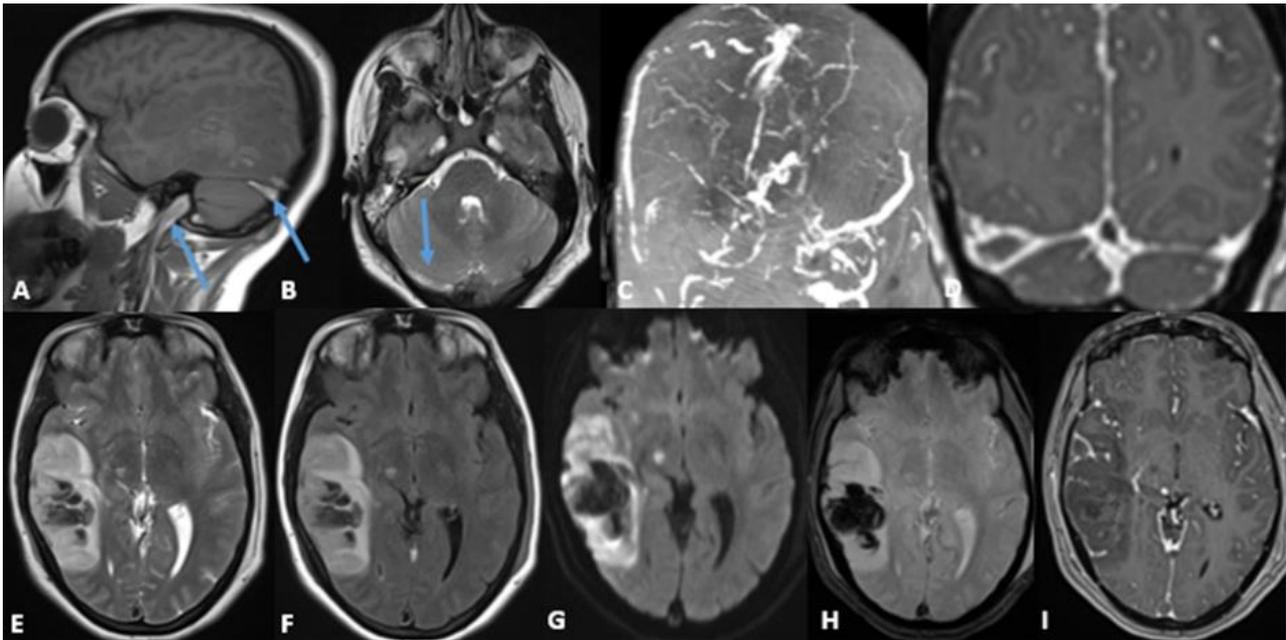


Fig-1: Angio-MRI of the brain. T1 Sagittal (A), T2 axial (B and E), angiographic venous (C), coronal and axial reconstructions after gadolinium injection (D and I), axial FLAIR (F), axial diffusion (G) and T2 * axial (H): T2 hypersignal aspect of the right lateral sinus, extended to the right jugular vein and to the posterior part of the superior sagittal sinus (blue arrows), with absence of flow within them on the venous angiographic sequence, without enhancement after injection of gadolinium. It is associated with a cortico-subcortical right temporal area in T1 hypointense areas in T1 hypersignal, heterogeneous T2 hypersignal, FLAIR and diffusion, with highlighting of signal voids on the T2 * sequence, not enhanced by the contrast medium. We also note a T2 nodular hypersignal, FLAIR and diffusion of the right thalamus, related to a thrombosis of the right lateral sinus, extended to the ipsilateral jugular vein and the posterior part of the superior sagittal sinus complicated by ischemic vascular accident with hemorrhagic infarction in a 28 years old female patient with estrogen-progestogen contraception since 3 years.

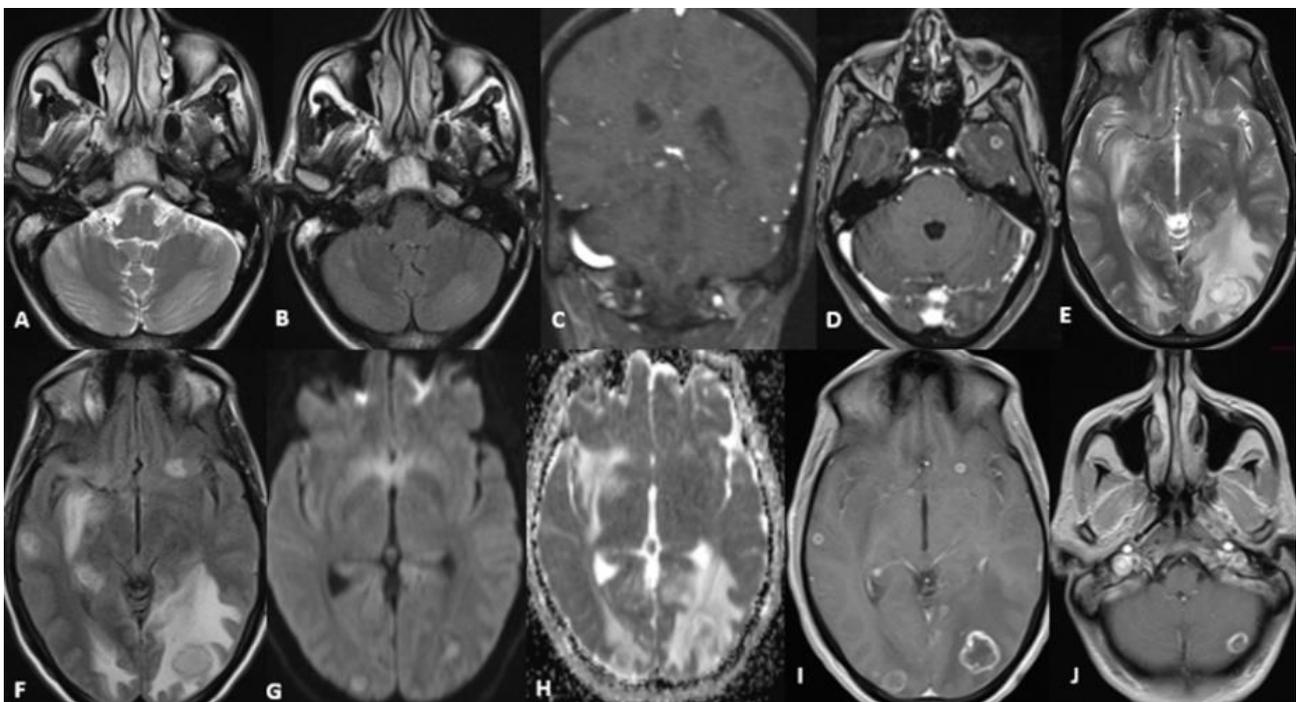


Fig-2: Angio-MRI of the brain. T2 Axial sequences (A and E) axial FLAIR (B and F), coronal angiographic venous (C), T1 axial after injection of gadolinium (D, I and J), axial diffusion sequence with ADC mapping (G and H): Aspect in T2 and FLAIR hypersignal of the left sigmoid sinus, with absence of flow in the venous angiographic sequence, without enhancement after injection of gadolinium. It is associated with supra and sub-tentorial lesions in heterogeneous T2 hypersignal, FLAIR and diffusion, with high ADC, enhanced at the periphery after injection of gadolinium, in relation to a thrombosis of the left sigmoid sinus on upper and sub-tentorial cerebral abscesses in a 46-year-old patient with toxic habits.

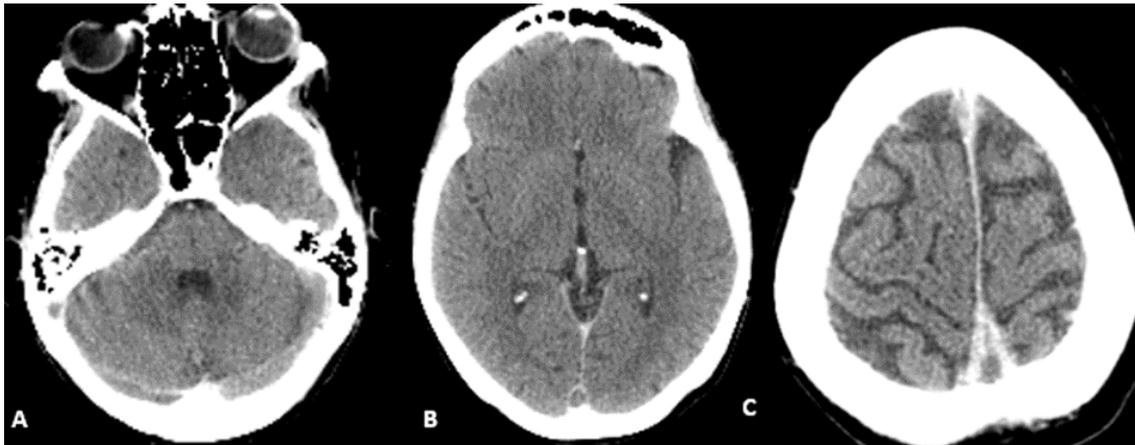


Fig-3: Brain scanner after injection of contrast product, axial slices. Aspect of an empty triangle of the upper sagittal sinus related to thrombosis in a 30-year-old patient operated on for a mature teratoma.

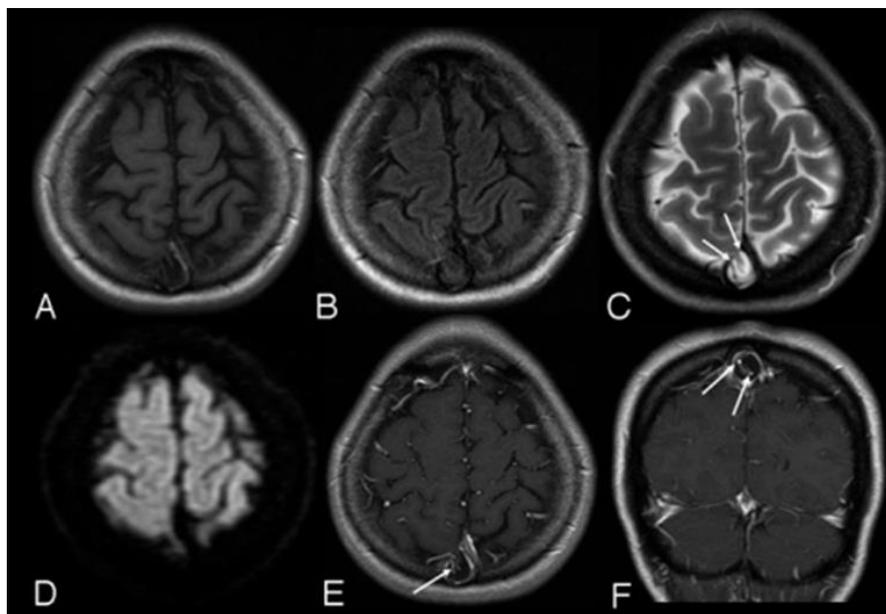


Fig-4: Brain MRI. T1 Axial sequences (A), FLAIR (B), T2 (C), (D); T1 axial and coronal after injection of gadolinium (E and F): a large Pacchioni granulation (arrows, C) along the right margin of superior the sagittal sinus

DISCUSSION

Thrombosis of the dural venous sinus or of the cerebral veins (CVT) is a particular anatomical location of venous thrombosis and a particular type of stroke, less common and quite distinct from arterial stroke [1].

According to recent studies, cerebral venous thrombosis has an annual incidence of 13.9 to 20.2 per million. Older studies have reported a lower frequency of the disease; the increased incidence of CVT over the years is likely due to improved diagnostic techniques and clinical knowledge of the disease [2].

It affects women more than men, especially women between the ages of 20 and 50. The gender difference in CVT is likely due to fluctuations in estrogen, oral contraceptive use, and pregnancy / puerperium [3].

Infectious etiologies have become rarer and probably represent less than 10% in adults; the classic causes are represented by locoregional infections (sinusitis, otomastoiditis, meningitis, empyemas, brain abscesses, septicemia). Among the non-infectious causes, it is necessary to distinguish the local etiologies (tumor invasion, trauma, jugular thrombosis on catheter), hormonal disturbances (immediate postpartum, oral contraceptives), certain drug treatments (androgens, danazol, L-asparaginase, corticosteroids), autoimmune and inflammatory diseases (Crohn's disease, lupus, Behçet, Gougerot-Sjögren, Wegener, sarcoidosis), visceral neoplasias and hemopathies (leukemia, lymphomas, polycythemia, thrombocythemia, etc.), thrombophilia and hemostatic disorders (congenital thrombophilias, antithrombin, protein C or protein S deficiencies, antiphospholipid antibody syndrome, cryoglobulinemia) and dehydration in infants. Head trauma with vault or base fracture that radiates to the venous sinus is associated with venous

thrombosis in 40% of cases. In 25% to 35% of cases, the etiology of CVT is not known [4].

The most frequent locations of thrombosis are the upper sagittal sinus (62% of patients) and the transverse sinus (40 to 45%), but in two thirds of cases more than one sinus is involved. The symptoms are varied and related to the venous structure involved. When the thrombosis affects the cortical veins, localized edema and parenchymal infarction usually develop. Large-scale infarction or intracranial hemorrhage results in stupor or coma in 15% of patients with CVT. Overall, intracranial hemorrhage complicates 14–39% of cerebral venous thrombosis [5].

The clinical presentation of CVT is highly variable and often nonspecific, attributing to late diagnosis. Additionally, symptoms may vary due to varying degrees of venous hypertension, recanalization, and concomitant thrombosis, causing fluctuations in clinical presentation. Common signs and symptoms are: headache, which is the most common symptom (75-95% of patients), papillary edema, seizures, focal neurological deficits, and altered mental status. Other symptoms described at the onset of CVT include occipital and neck pain which is usually associated with sigmoid sinus involvement. The clinical presentation may vary depending on the extent of the thrombosis, the structures involved and the concomitant clinical circumstances [6].

The diagnosis of CVT is based on visualization of the thrombus and venous occlusion by neuroimaging techniques. The non-injected CT scan may reveal indirect signs, such as the "dense triangle sign" associated with the occlusion of the superior sagittal sinus in its posterior part, or the "cord sign" in cases of cortical venous thrombosis. In addition, it can rule out a tumor, ischemic stroke, cerebral hemorrhage or abscess. After injection, we can find the "sign of the empty triangle or delta" corresponding to an absence of filling of the torcular. Conventional angiography has lost its diagnostic role and is only used in cases of suspected thrombosis of a cortical vein with normal MRI, even if its interpretation may prove difficult due to the great intra- and inter-individual variability [7-9].

Brain MRI is the reference method for diagnosing CVT and must be requested systematically, the usual sequences are the spin echo sequences weighted in T1 and T2, the FLAIR sequence for the study of the parenchyma, the T2* sequence sensitive to the presence of blood and more recently the weighted diffusion and perfusion sequences. It allows the visualization of the venous thrombus and the monitoring of its evolution, it is also more sensitive than the scanner to detect cerebral edema and venous infarction. To confirm venous sinus thrombosis, the thrombus must be visible in several slices in different planes. It is indeed essential to multiply the sequences

to eliminate any flow artefacts. The venous thrombus evolves in three stages. The first three to five days, the thrombus appears iso-intense in T1, hypo-intense in T2 and the flow can simulate a normal sinus. Then, it becomes hyper intense in T1 and T2. After two to three weeks, the appearance varies according to the degree of repermeabilization of the sinus: MRI may be normal or show a heterogeneous signal (iso-intense in T1, iso or hyper intense in T2). MRI is also more sensitive than a CT scan in detecting brain edema and venous infarction. The latter are readily hemorrhagic and, compared to arterial infarctions. Contrast enhancement is rare and edema is prolonged. The hypersignals visible on the T2-weighted sequences may correspond to infarctions but are most often the indicator of venous engorgement, which explains the frequent normalization of subsequent MRIs. MRI can also detect a possible local cause such as a tumor or infection. Finally, it is the technique of choice for differentiating between thrombosis and hypoplasia of a lateral sinus. The latter results, on a sagittal slice, by a clear asymmetry in size of the transverse portion of the lateral sinus without signal anomaly [10].

Reading errors can lead to misinterpretation, on the one hand because the dural sinuses are frequently located at the edges of an image rather than in the central part and, on the other hand, because the images of the sagittal sinus higher are at the end of the series of images [11].

The differential diagnosis is mainly made with the Pacchioni granulations that are localized within the superior sagittal sinus and the transverse sinuses. They are responsible in CT angiography and MRA for opacification defects; these should not be confused with a thrombus. They present densities and a signal close to those of the cerebrospinal fluid and do not determine any obstruction of the vascular lumen. Multiplanar reformations obtained from MRA or 3D T1 after injection of gadolinium allow a detailed study of the arachnoid Pacchioni granulations and the septa developed within the sinus (Fig. 4) [12].

CONCLUSION

Cerebral venous thrombosis (CVT) is a rare condition that can be life-threatening. Because of clinical polymorphism and the variability of the data of the imaging in sections, the radiologist is required to confirm the diagnosis by showing signs of occlusion of the venous structure by a clot, to assess the suffering of the cerebral parenchyma secondary to this thrombosis by looking for signs of cerebral venous ischemia and finally, attempting to show an origin or a pathology associated with this thrombosis.

Magnetic resonance imaging and CT scan, by the use of venous angiographic sequences and multiplanar reconstructions make it possible to presume this pathology by highlighting indirect signs,

supplemented by the injection of contrast product, highlighting the thrombus, specifying its exact location.

Competing interests

The authors declare no conflict of interest.

Contributions from authors

All the authors contributed to the conduct of this work. They also state that they have read and approved the final version of the manuscript.

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