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MECHANICAL THROMBECTOMY IN CEREBRAL VENOUS SINUSOIDS THROMBOSIS – CASE REPORT

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Abstract: *Cerebral venous sinus thrombosis (CVST) is a relatively uncommon cause of stroke and other neurologic complications who may lead to venous infarction, a different entity of other ischemic stroke. The widespread availability of noninvasive brain imaging has led to an increase in its diagnosis. Incidence is much higher in younger patients with a female predominance. The initial treatment for CVST is systemic anticoagulation. Endovascular procedures are considered for patients who exhibit progressive neurological decline despite therapeutic anticoagulation it can be a lifesaving therapy. We present the clinical case of a young patient with CVST with important risk factors unresponsive to medical treatment and treated by venous sinus thrombectomy.*

INTRODUCTION

Cerebral venous thrombosis (CVT) is an uncommon condition that accounts for 0.5 to 1% of all strokes in the adult population (1) caused by an occlusion of the cerebral venous sinuses (CVST - cerebral venous sinus thrombosis) or the other cortical veins (cortical vein thrombosis).(2) CVST typically affects young adults with an average age of 35 years and is more common in women (2.2:1) because of sex-specific risk factors.(3)

According to The International Study on Cerebral Vein and Dural Sinus Thrombosis (4) about 85% of adult patients have at least one risk factor and the most prevalent was use of oral contraceptives, followed by a prothrombotic condition (more often genetic than acquired).

Important risk factors for CVT are estrogen-containing oral contraceptives, prothrombotic conditions (genetic or acquired thrombophilias), pregnancy, postpartum period, infections, associated cancer, head trauma of venous structures and inflammatory diseases. Antiphospholipid antibody syndrome should be considered in cases of CVT with an unknown cause.(4,5)

Many patients have multiple risk factors for CVT, so unless a specifically cause is found, an investigation for all possible risk factors is needed, especially for thrombophilias, which is frequently underdiagnosed, as clinical manifestations are not specific and include a wide range of symptoms.(2,5)

In the VENOST study (6), a multicenter retrospective study made in Turkey, which collected 1144 patients, onset of symptoms were acute in 47%, subacute in 34%, and chronic in 19%. Most frequent clinical symptoms and signs were headache (87%), nausea and vomiting in 28%, seizures in 24%, visual field defects in 27%, other focal neurological deficits in 18%, altered consciousness in 18%, and cranial nerve palsies in 18%. The clinical presentation of CVT varies according to the age of the patient.(7)

Brain imaging is fundamental in detecting brain venous thrombosis and possible associated complications that could modify the prognosis and the therapeutic approach.(1,8)

The ESO guidelines suggest magnetic resonance venography (MRV) or computed tomography venography (CTV) for confirming the diagnosis. Compared to digital subtraction angiography (DSA), a gold standard in diagnosis of cerebral thrombosis, CT venography has a very good diagnostic accuracy (sensitivity of 95% and specificity of 91%). Also, CTV can show absent flow in thrombosed veins or sinuses and partial circumferential enhancement of thrombosed venous sinuses (e.g., the empty delta sign).(2)

Both CTV and MRV are highly accurate in diagnosing CVT when the two imaging modalities are compared directly with each other or with digital subtraction angiography (DSA).(9)

The 2017 European Stroke Organization guidelines (10) suggest initiation of heparin therapy as soon as possible after the diagnosis has been established. In the acute phase, all CVT patients should be anticoagulated parenterally with either unfractionated IV heparin or low molecular weight heparin (LMWH). LMWH is preferable, unless the patient is clinically unstable, or a lumbar puncture or surgery is planned.(7) Patients who do not respond to anticoagulant therapy, who are associated with risk factors for incomplete recovery despite treatment, or those who present a contraindication to heparin treatment may benefit from chemical and/or mechanical endovascular therapy.(11)

The US Guidelines state that endovascular treatment can be considered for patients who are comatose or who deteriorate despite anticoagulation and have no parenchymal lesion with significant mass effect.(2)

CASE REPORT

We present the case of a 43-year old patient with following risk factors: hormone replacement therapy (Desogestrel), overweight (BMI 27), smoking for 10 years (60 packs/year) quit in 2003, cerebral thrombophlebitis in 2000, positive lupus circulating antibodies, history of hormonal contraceptives. She presented with unusual headaches, disorientation, episodes of vomiting and loss of urine. At the

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emergency services, the patient's condition deteriorated and she presented a comatose state (Glasgow 8).

A cerebral CT scan was performed which proved an image of thrombosis on the confluence of the sinuses and the right transverse sinus. Lumbar puncture was made and the curative anticoagulation was administrated.

A cerebral MRI was performed showing a cerebral venous thrombosis extended to the entire deep cerebral venous system with acute cerebral suffering (wide cytotoxic edema), mass effect with exclusion hydrocephalus of the lateral ventricles and punctiform ischemic lesions (figure no. 1, figure no. 2).

Figure no. 1. (A) MIP 3D TOF, MRI enhanced Venography showing cerebral venous thrombosis in the ampulla of Galen and right sinus that extended to the entire deep cerebral venous system with complete occlusion of the internal cerebral veins and basal veins of Rosenthal, (B) DSA, venous phase, lateral view – thrombosis of the internal cerebral veins, basal veins of Rosenthal, Gallen vein and straight sinus

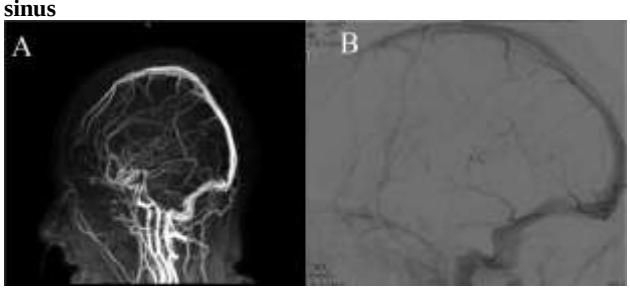
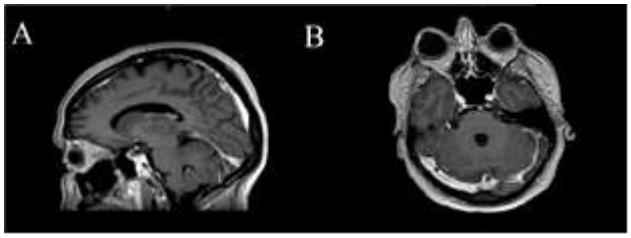
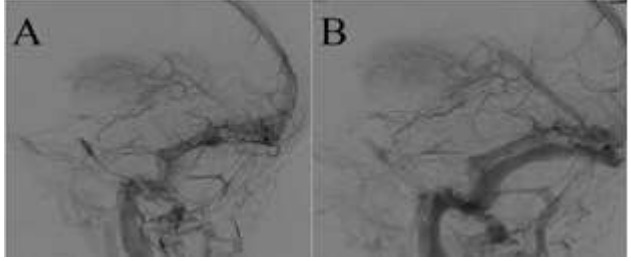


Figure no. 2. (A) Cerebral MRI, sagittal T1 post-contrast shows thrombosis of the straight sinus and Herophili torcular, (B) Cerebral MRI, T1 weighted contrast enhanced, shows venous thrombosis at the level of the left transverse sinus



In this context, mechanical thrombectomy (MT) is required considering the progressive worsening of the patient's condition and lack of response to anticoagulant treatment.

Figure no. 3. DSA of the vertebral arteries, venous phase, lateral view, before (A) and after (B) mechanical thrombectomy – A. Thrombosis of the left transverse sinus, sigmoid sinus. Complete occlusion of the vein of Galen and right sinus. B. Complete recanalization of the veins after mechanical thrombectomy was performed.

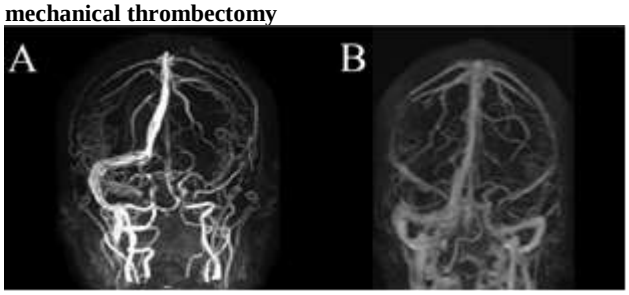


Cerebral venous thrombectomy shows complete recanalization of the right sinus, the left lateral sinus and the jugular vein. Persistence of stenosis at the level of the torcular confluence probably by a mass of an old thrombus. For this reason, balloon angioplasty of the torcular confluence and the origin of the right sinus was used (figure no 3).

The 24-hour post-procedure CT scan does not reveal any hemorrhagic lesions, with persistence of collapse of the 3rd ventricle and a discreet non-obstructive thrombus in the left transverse sinus only.

The 7-day follow-up cerebral MRI shows a significant radiological improvement with partial regression of the vasogenic edema, of the mass effect on the 3rd ventricle and complete regression of the hydrocephalus. The non-occlusive thrombus is found in the left transverse sinus and the internal cerebral veins, with complete repermeabilization of the sigmoid sinus and the left internal jugular vein as well as the right sinus (figure no. 4).

Figure no. 4. TOF 3D MRI venography – A. Occlusion of the left transverse, sigmoid sinuses and left internal jugular vein. B. Recanalization of the described veins after mechanical thrombectomy



DISCUSSIONS

This patient presents important risk factors for CVT such as: feminine gender, hormone replacement therapy, history of hormonal contraceptives and cerebral thrombophlebitis, positive lupus circulating antibodies. According to The International Study on Cerebral Vein and Dural Thrombosis, the most common risk factor is the use of oral contraceptives. Antiphospholipid antibody syndrome is also an important (and treatable) cause of CVT (2) and along with clotting disorders may require indefinite anticoagulation.(12)

The goals of CVT management are to prevent the progression of venous thrombosis, re-establish venous flow, and manage underlying cause(s) of any prothrombotic state to prevent recurrence of CVT.(9)

Anticoagulation continues to remain the gold standard treatment of CVT. Current recommendations from the European Federation of Neurological Societies endorses low molecular-weight heparin (LMWH) in the management of acute CVT.

There are several reasons for anticoagulation therapy in CVT: to prevent thrombus growth, to facilitate recanalization, and to prevent deep venous thrombosis or pulmonary embolism. Controversy has ensued because cerebral infarction with hemorrhagic transformation or intracerebral hemorrhage is commonly present at the time of diagnosis of CVT, and it may also complicate treatment.(1)

The optimal duration of anticoagulant therapy for secondary prevention of CVST should be decided for the single patient, evaluating the risk–benefit ratio. The absolute risk of recurrent thrombosis is low and long-term anticoagulation is reserved for patients with persistent and unmodifiable risk factors (e.g. severe thrombophilia, or solid or hematological neoplasms) and those with recurrent CVST.(3)

In patients receiving anticoagulation, the rate of venous recanalization in the follow-up is around 85%. Analysis of the available cohort studies indicates that this process predominantly occurs in the first few months after CVT, although it can take up to 1 year. Recanalization of CVST can

be considered among the criteria, potentially helping the decision on the optimal duration of anticoagulant therapy. Repeat imaging (CT or MRI) is recommended at 3–6 months from debut or in the case of persistent or recurrent symptoms suggestive of CVST during anticoagulation therapy.(3)

However, anticoagulation may not be the only treatment choice for complicated CVT patients. In patients who show signs of rapid deterioration, mechanical thrombectomy is feasible and has shown benefits with good recanalization rates.(13)

Considering the aggressive deterioration of the patient's clinical condition, from headache to coma in 3 days, the lack of response to medical treatment and the accumulation of risk factors for CVT, emergency endovascular intervention was decided as lifesaving therapy. Following the mechanical thrombectomy, the recanalization was almost complete, with indication for continuation of the anticoagulant therapy, the patient's clinical condition improving.

Figure no. 5. DSA of the left internal carotid artery, venous phase, lateral incidence, before (A) and after (B) mechanical thrombectomy - The right sinus and deep venous system are completely thrombosed (A) and repermeabilized (B) after mechanical thrombectomy

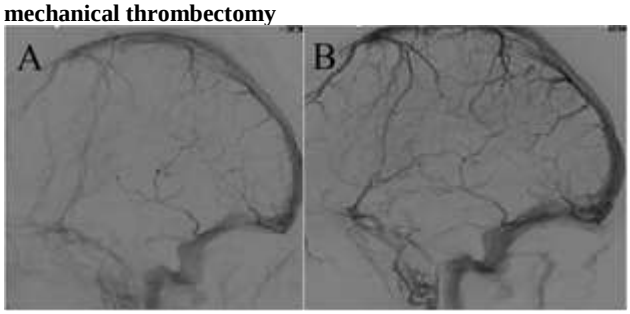
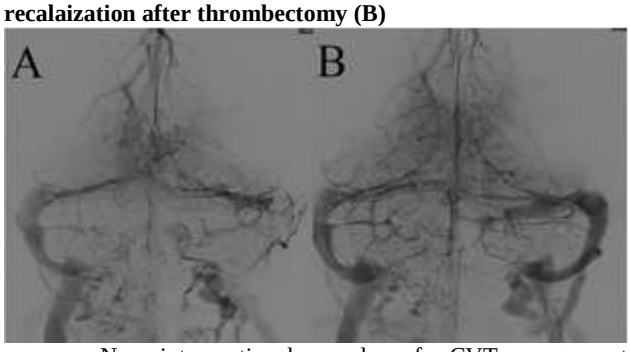


Figure no. 6. DSA of the vertebral arteries, venous phase, posterior incidence, before (A) and after (B) mechanical thrombectomy - Filling defects of the left transverse and sigmoid sinuses and the left internal jugular vein (A) and recalaization after thrombectomy (B)



Neurointerventional procedures for CVT management are considered in patients with major contraindications to anticoagulation, clinical deterioration despite medical treatment, progression of intracerebral hemorrhage or venous infarction despite anticoagulation, severe stupor, comatose presentations, deep CVT, posterior fossa involvement.(9)

ESO recommend that endovascular treatment should only be considered in patients with a high pre-treatment risk of poor outcome and severe cases of CVT that do not improve or deteriorate despite anticoagulant therapy; it is probably most effective for acute rather than chronic thrombosis.(2) Our patient met the criteria for endovascular treatment according to these criteria.

In 17 studies totalling 235 patients with cerebral sinus venous thrombosis with endovascular treatment, approximately 70-80% achieved functional independence up to 42 months after treatment, with procedural complications and aggravation or de novo intracerebral hemorrhage <10% from patients.(14)

CONCLUSIONS

CVT is an uncommon but serious and life threatening cause of stroke, so is a disease with potential for high morbidity and mortality.

Its diagnosis is challenging because of many and varied symptoms or causes, and depends on rapid and appropriate neuroimaging. Venographic imaging with CT (CTV) or MRI (MRV) are efficient to demonstrate obstruction of the venous sinuses or cerebral veins by thrombus.

It is necessary to make a careful search, once CVT is diagnosed, for an underlying cause, for example, oral contraceptive use, thrombophilia or antiphospholipid antibody syndrome.

The current standard of care remains anticoagulation, but in patients who show signs of rapid deterioration, MT is technically feasible and has shown benefit with good recanalization rates.

The particularity of this case lies in the fact that the patient has recurrent cerebral venous thrombosis, the first episode starting in 2000, and subsequently the risk factors related to the patient contributed to the reactivation and worsening of the initial venous thrombosis that was unresponsive to medical treatment, a fact that made endovascular treatment necessary.

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