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Cerebral Venous Thrombosis: Convulsions and Convulsive Status Epilepticus About 2 Cases

Boudiaf Rym 1.2, Touahria Asma 2, Boulefaat.M 2, Ait Mouheb Tahar 1.2

- 1*University of Medical Science, Department of Medicine, Algiers, Algeria
- ^{2*} Mustapha Pacha Universtiy Hospital, Algiers, Algeria

Summary

CVT is a rare but potentially serious pathology, requiring rapid and individualized management. We report 02 cases of cerebral venous thrombosis hospitalized in the medical emergency department of the Mustapha University Hospital Center during the same month.

Both cases, aged between 32 and 37, had been taking combined oral contraceptives of the estrogen-progestin type for several years.

The diagnosis of the disease in these two cases was made by brain scan without injection of contrast agent.

Both patients were put on curative anticoagulation upon admission to the department.

On the first day, both patients developed convulsions requiring the introduction of anticonvulsants; one of them stopped convulsing on the second day, unlike the second patient.

The second patient, aged 37, presented complications after the 4th day, marked by the appearance of a frontal hematoma detected on the 2nd brain scan following the reappearance of headaches.

Both patients left the department after receiving full treatment and improvement in their health.

Introduction

Cerebral venous thrombosis (CVT) is a rare but serious neurological condition caused by occlusion of cerebral veins or venous sinuses. It can present with a variety of clinical manifestations, including headaches, focal neurological deficits, seizures, or signs of intracranial hypertension, thus complicating diagnosis. (Coutinho et al., 2012; Saposnik et al., 2024)

Treatment is based on early anticoagulation to limit the spread of the thrombus and prevent serious complications. (Dmytriw et al., 2018; Saposnik et al., 2024)

Although the prognosis for CVT is often favorable with prompt treatment, some cases can become complicated.

Patient and observation

Case 01:

Mrs. BF, 32 years old, housewife with a history of cervicobrachial neuralgia under investigation, put on codeine and a colchicine-derived muscle relaxant, sinusitis declared cured, with a gynecological history of 03 miscarriages, G6P3, put oncombined oral contraceptive of the estrogen-progestin type for several years, admitted for the management of hemiparesis of the right side of the body with paresthesia of the right upper limb and headaches in a postpartum context of approximately 4 months.

The admission examination revealed profound asthenia, average general condition, hemiparesis of the right side of the body and right paresthesia, dysarthria and headaches, reactive isocoral pupils, preserved swallowing reflex, impaired consciousness scored 12/15 on the Glasgow scale (OY=04, RV=02, RM=06). There were no signs of dehydration or malnutrition, presence of calf roll without edema of the lower limbs, nor signs of deep vein thrombosis, negative Homans sign, the rest of the clinical examination was unremarkable.

The hemodynamic constants were good: BP = 110/07, Fc = 103 Bpm, SpO2 = 100% in room air, $T^{\circ} = 37.7^{\circ}$, Diuresis preserved, Capillary blood glucose = 1.4 g/l.

Toxic dosage returned positive for opiates.

Brain CT scan without injection of contrast agent reveals a spontaneously hyperdense appearance revealing an interesting CVT:

- Anterior two-thirds of the sagittal sinus
- · Some cortical veins.

The chest X-ray was normal.

Blood count with hypochromic microcytic anemia with hemoglobin 10.4 g/dl, CRP at 39.2 mg/l, DDimers at 1.85 ug/ml, the rest unremarkable.

Assessment of antiphospholipid syndrome was done.

Thrombophilia assessment will be carried out 3 months after the onset of thrombosis.

The patient presented three fever peaks between 38.2° and 38.7° and a generalized convulsive status epilepticus on day 2 of hospitalization and since then she has not had any more seizures.

A cytobacteriological study of the urine was carried out to look for the triggering factor which returned without any particularity.

We noted unilateral right-sided blurred vision that lasted a few minutes on day 6 of hospitalization, from which a CT scan with injection was performed, which showed a known cerebral venous thrombosis without complications.

Patient put on heparin therapy with support stockings and painkillers if in pain.

Discharged on day 15 of hospitalization with INR that does not want to return to the target, referred to cardiology who increased the dose to 2 Sintrom tablets®, patient stable to date.

Case 02:

Mrs. OS aged37 years old, with no notable medical or surgical history. Her obstetric history reported two vaginal deliveries (G2P2). The patient had been using combined estrogen-progestin oral contraception for several years.

She was referred to the emergency department for severe headaches and tingling-like paresthesias that began in the left upper limb and gradually spread to the left lower limb. These symptoms were followed by a generalized seizure.

After regaining consciousness, a new convulsive seizure localized to the left side of the body occurred, accompanied by balance disorders.

A brain scan with injection of iodinated contrast agent was performed. The results revealed an interesting cerebral venous thrombosis:

- The superior and inferior longitudinal sinus,
- The lateral and sigmoid sinuses,
- The proximal part of the internal jugular veins,
- Some cortical veins.

The admission examination found an average general condition, pConscious, cooperative patient, scored 15/15 on the Glasgow Coma Scale. Presence of convulsive seizures located in the left side of the body. After treatment with clonazepam injection (1 mg), left hemiplegia was observed.

Hemodynamic constants: Tachycardia at 135 beats per minute, Hypertension at 165/77 mmHg, Oxygen saturation (SpO2) at 99% on room air.

Other observations: Presence of a fever peak at 38°C, Vomiting, No notion of blurred vision, Absence of clinical signs of deep vein thrombosis. The rest of the clinical examinationwas unremarkable.

The biological assessment showed an inflammatory syndrome: Leukocyte count: $16.01 \times 10^3 / \mu L$, Polymorphonuclear neutrophils (PNN): $12.93 \times 10^3 / \mu L$, Eosinophils: $0.00 \times 10^3 / \mu L$, Basophils: $0.03 \times 10^3 / \mu L$, Lymphocytes: $1.9 \times 10^3 / \mu L$, Monocytes: $1.07 \times 10^3 / \mu L$, CRP: 37.9 mg/L

No evidence of anemia, platelet count: Normal

Fibrinogen: 3.94 g/L, D-dimer: 1.35 µg/mL (moderate elevation).

Therapeutic-dose anticoagulant therapy was initiated within the first 24 hours. The patient received enoxaparin (Lovenox®) 0.8 mg twice daily.

Additionally, antiepileptic treatment was initiated with phenobarbital (Gardénal®) at a dose of 5 mg per day.

Sintrom® (acenocoumarol) was introduced on the 3rd day of hospitalization at a dose of 1/2 tablet per day, with regular monitoring of the INR (International Normalized Ratio) for dose adjustment.

On day 3 of hospitalization, anticonvulsant treatment was discontinued. The seizures ceased; however, the left hemiplegia persisted, with no notable improvement in the neurological deficit. This suggests that, although the seizures were controlled, the cerebral venous thrombosis and its neurological consequences continued to affect motor function.

On day 7 of hospitalization, the patient presented with headaches, for which a cerebral CT angiography was performed, which revealed a right frontal parafalcic intraparenchymal hematoma of (9x14mm), anticoagulation was stopped for 24 hours.

On day 8 of hospitalization, the patient improved her motor function of the left upper limb with monoparesis of the left lower limb, reintroduction of anticoagulation

On day 11 of hospitalization, 2 INRs within the target, discontinuation of heparin therapy (Lovenox®), continuation of the taking of the Sintrom®(acenocoumarol) in two different doses (3/4 tablet and 1/2 tablet) alternately.

The patient showed significant improvement with regression of symptoms and partial regression of the thrombus on follow-up CT scan. The patient was discharged on day 15 of hospitalization.

Discussion

According to recent studies, approximately 20 to 30% of patients with CVT may experience seizures. (Kalita et al., 2012; Saposnik et al., 2024), but only 5 to 10% of these cases progress to convulsive status epilepticus.

Although CVT is a rare pathology, it requires increased clinical vigilance, particularly in young women using hormonal contraception.(Zuurbier et al., 2016)

Use of combined oral contraceptives is associated with an increased risk of developing CVT. (Saposnik et al., 2024)

Headaches are the most common symptom of CVT, present in more than 90% of patients. (Idiculla et al., 2020)

Convulsive status epilepticus, in the context of cerebral venous thrombosis, is a clinical warning sign, although the pathophysiological mechanisms involved remain partially elucidated.

Regarding diagnosis, some studies have used brain CT without injection of contrast agent to detect CVT(Linn et al., 2007; Saposnik et al., 2024), however contrast-enhanced CT angiography is generally preferred to confirm the diagnosis, as it allows more precise visualization of venous structures and occlusion.(Leach et al., 2006; Saposnik et al., 2024)

MRI is also recommended for its superior sensitivity in detecting venous thrombi and associated parenchymal lesions. (Uluivi et al., 2020; Saposnik et al., 2024)

The choice of imaging modalities must be adapted according to the available resources and the clinical characteristics of the patient. (Idiculla et al., 2020; Dmytriw et al., 2018)

Treatment of CVT is primarily based on anticoagulation, which aims to prevent spread and promote its resolution. (Silvis et al., 2017; Saposnik et al., 2024)

Treatment for seizures usually includes anticonvulsants to control seizures and prevent recurrences. (Tadi et al., 2025)

Management of CVT remains challenging due to the variability of clinical presentation and the need for rapid intervention to prevent serious complications.(Tadi et al., 2025)

Further research is needed to deepen understanding of the underlying mechanisms and optimize therapeutic strategies.

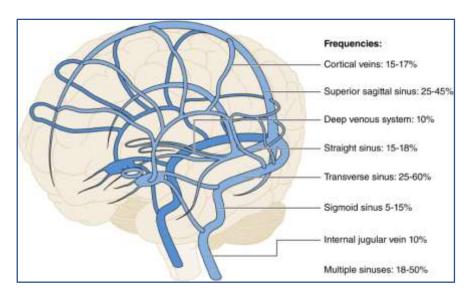


Figure 1.Anatomy of the cerebral venous system and distribution of CVT.

Prevalence of sinus involvement in CVT. Percentages may be greater than 100%, as many patients may have more than one affected sinus.

Please note that internal jugular vein thrombosis represents its concomitant prevalence with CVT (not isolated). CVT indicates cerebral venous thrombosis. SAPOSNIK, G. ET AL. STROKE. 2024; 55(3): e78. DOI: 10.1161/STR.0000000000000456.

Conclusion

Status epilepticus associated with cerebral venous thrombosis, although rare, represents a major clinical challenge. Early diagnosis, adequate seizure management, and effective anticoagulation are essential to improve neurological prognosis and reduce long-term complications.

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