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Psychiatric Signs Revealing Cerebral Thrombophlebitis: A Case Report

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Abstract **Case Report**

Cerebral thrombophlebitis (CT) corresponds to an obstruction of a cerebral vein by a clot. It is a less frequent pathology than cerebrovascular accidents (CVA) but is certainly not rare. Its diagnosis is more radiological (brain MRI being the reference examination) than clinical, given the heterogeneity of the symptoms. In most frequent cases, CVT is revealed by headaches, papilledema, epileptic seizures, or isolated intracranial hypertension. In other cases, the clinical signs are less obvious and can be presented by psychiatric signs, as in our patient's case. The treatment was primarily based on anticoagulant therapy and the evolution was marked by an improvement of all the symptoms.

Keywords: Cerebral thrombophlebitis, CT scan, psychiatric signs.

INTRODUCTION

Cerebral thrombophlebitis (or venous thrombosis) was considered for a long time, cerebral thrombophlebitis (or venous thrombosis) was considered a rare condition with an unfavorable prognosis; the confirmation of the diagnosis being made by the autopsy, only the severe manifestations were known.

The exact incidence of cerebral venous thrombosis is unknown; it is estimated at 5 per 1 million. The CT represents less than 1% of strokes and is of particular interest to young women. They are characterized by their clinical and etiological polymorphism [1].

The incidence of CVTs is undoubtedly greater and not rare, as evidenced by the increasing number of cases published in the literature in recent years [2].

The diagnosis of CVT remains difficult, due to the extreme heterogeneity of the clinical pictures and the need for neuro-imaging examinations to confirm the diagnosis.

The progress and accessibility of non-invasive imaging currently allow an early diagnosis of CVT, which makes it possible to begin the etiological investigation as quickly as possible, from which the prognosis and specific treatment derive.

At first, the patient experiences moderately severe headache attacks. Gradually, the headaches become permanent and more intense. The patient then begins to show other signs pointing to intracranial hypertension syndrome. These disorders include vomiting, abnormal vision, and seizures. Some patients also have signs of hemispheric localization caused by cerebral ischemia among other neurological and/or psychiatric symptoms.

In recent years, therapeutic advances have been recorded, thanks to studies on the effectiveness of anticoagulants or thrombolytics [3].

Today the revolution in imaging procedures has essentially enabled the reliable diagnosis of CVT and has greatly contributed to the understanding of the clinical picture.

We report an observation of CVT in a young male adult, and we would like to focus on the clinical polymorphism, the etiological aspect, the place of imaging precisely of CT scans, and finally the prognosis of his condition.

CASE REPORT

In our case, the patient, a young man of 21 years old, came to our facility with his parents due to a sudden change of mood and a break from his previous clinical state. He reports mood sadness, anhedonia,

severe headaches, two suicide attempts fourteen days before his admission to the emergency department, verbalization of delusions with impaired judgment and insight, and insomnia.

The patient has no particular pathological history. The initial biological assessments were: blood count, complete blood ionogram, fasting blood sugar, liver function tests, thyroid function tests, renal function tests, lipid function tests, a search for drugs in the urine initially, and then a cerebral CT afterward.

The diagnoses evoked at first were psychiatric: brief psychotic episodes, depression with psychotic characteristics, induced psychiatric disorder, and then an organic cause remains to be eliminated.

Three days after his hospitalization, the clinical examination revealed a fever of 39°C, high blood pressure at 180 mm Hg systolic and 100 mm Hg diastolic, partial neurological deficit involving all four limbs, segmental muscle rigidity concerning his upper members, intense sedation and slight disturbances in his initial biological work-up, fasting blood sugar is the most disturbed: 1.78g/L.

The neuroleptic malignant syndrome was first suspected as the patient was put on antipsychotic and antidepressant medication (olanzapine, paroxetine, and diazepam).

The treatment was stopped and the patient was put on diazepam with plenty of fluids and close and strict monitoring of his constants and psychomotor behavior. A thorough biological work-up was carried out, in particular the determination of creatine phosphokinase (CPK), which came back high: 2468U/L.

This led to his transfer to the medical emergency room where his hemodynamic and respiratory status was stabilized and an emergency CT scan was subsequently performed, which revealed a CVT.

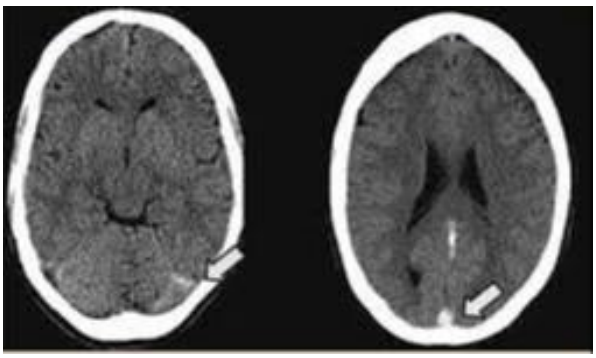

Figure 1: Brain scan without injection of contrast product

DISCUSSION

The clinical diagnosis of CVT remains difficult because the clinical spectrum of CVT and its evolution over time are varied. On the etiological level, we observe in the foreground the young subject in addition to local causes such as meningitis, and hemostasis disorders.

The pathophysiology remains difficult to understand. Two mechanisms explain the symptomatology and the mode of installation: Venous obstruction, with increased capillary pressure, and dysfunction of the parenchyma. It can be responsible for bleeding. Depending on the venous disposition and collateral drainage pathways, vasogenic (potentially reversible) or cytotoxic edema may appear, responsible for parenchymal lesions. Or the increase in intracranial pressure (ICP) by edema and the deficit of resorption of cerebrospinal fluid (CSF) by the arachnoid granules [8].

Infectious cerebral venous thrombosis is distinguished from non-infectious forms and the list of risk factors for non-infectious cerebral venous thrombosis is long. It should be noted that in 15% of cases the etiology is not determined [2]. Rosenstein *et al.*, in 2002, in a retrospective study of 27 cases of cerebral venous thrombosis, reported that an anomaly of hemostasis was found in 41% of patients [8].

The clinical diagnosis however is difficult and the symptomatology is polymorphic and often misleading [5]. It can manifest as headache (41 to 100%), intracranial hypertension syndrome (30 to 70%), neurological deficit (34 to 70%), seizures (20 to 57%) consciousness (34 to 70%), or psychiatric disorders which are sometimes in the foreground and mask the rest of the symptomatology [6].

This is exactly the case of our patient, the psychiatric signs masked the thrombosis and the cause has not been able to be determined.

The treatment of CVT is based on heparin therapy combined with symptomatic treatment based on anti- oedematous and anticonvulsants. However, thanks to early diagnosis and treatment, survival without sequelae has been reported in several cases [7], as is the case for our patient.

The benefit of heparin therapy has been well demonstrated and the prognosis is good if the diagnosis is made early and if the treatment is administered in time. MRI and venous angio-MRI are the reference neuroradiological examinations, but computed tomography without and with the injection of contrast product can be an interesting alternative for diagnosis [4].

CONCLUSION

It is important to recognize and diagnose CVT because the prognosis is good after a treatment and the clearance of symptoms is complete.

Thanks to this case, we were able to shed light on the place of the thorough neurological examination in psychiatric pathology and of the somatic examination in general. The sequelae of the anomaly can be serious because the cerebral venous sinus is one of the main elements allowing blood to drain from the brain. If not diagnosed and treated on time, the prognosis can be fatal, resulting in chronic impairment and even death.

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