# Headaches after a recent pregnancy need an MRI to rule out cerebral thrombosis

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**ABSTRACT** Cerebral venous thrombosis (CVT) is a rare but severe complication of pregnancy and postpartum. Misleading symptomatology characterizes it and the diagnosis must be evoked in the presence of persistent headache after delivery. MRI is the gold standard imaging exam for the diagnosis. Treatment is based on anticoagulation therapy. Functional prognosis is improved if diagnosis is promptly made and treatment started. We report the case of a 25-year-old woman with a delayed diagnosis of postpartum cerebral venous thrombosis. Severe neurological sequelae marked evolution. We emphasize the interest of imaging in the early diagnosis of this pathology.

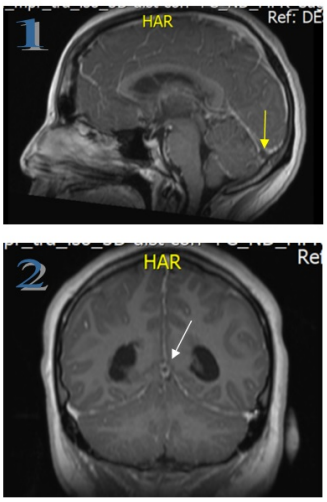
**KEYWORDS** post-partum cerebral venous thrombosis, magnetic resonance imaging

## Introduction

Cerebral venous thrombosis (CVT) is a rare but severe and life-threatening condition. CVT may occur at any age with a peak incidence in young women and it is associated with a hypercoagulation state (combined oral contraceptive treatment, pregnancy and postpartum). We report the case of a young patient with cerebral venous thrombosis following childbirth.

## Case report

This is a 25-year-old woman admitted to the emergency department for abruptly drowsiness and fever since early in the morning. Her past medical history was negative for chronic diseases, and she had a normal delivery two months before. Of note, she had been previously evaluated for headaches in two different emergency departments. In work out, the non-contrast enhanced cerebral tomodensitometry was normal, as well as the blood tests and lumbar puncture. She was treated as a migraine attack and sent back home with oral pain killers.




Figures 1 and 2: Contrast enhanced T1 weighted MRI sequences sagittal and coronal views show the thrombus in the straight sinus (white arrow) to confluence of sinuses (yellow arrow).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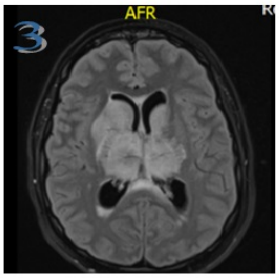


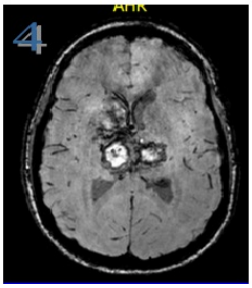


Figures 3,4:

Figure 3 is a T2 FLAIR in axial view. It shows a heterogeneous hypersignal of the thalamus and the head of the caudate nucleus, signifying local edema and swelling.

Figure 4 is a SWI sequence sensitive to bleeding. It shows a tissue deposition of hemoglobin around the thalamus, which refers to a hemorrhagic stroke.

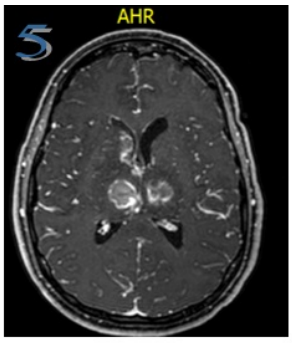


Figure 5 is a T1 contrast enhanced in axial view. It confirms the bilateral infarction of the thalamus and caudate head and demonstrates the disruption of the blood-brain barrier with local pathological enhancement.

At the admission in our emergency department, Glasgow Coma Scale (GCS) score was 12, and she was febrile and hemodynamically stable.

The neurological examination showed symmetrical and reactive pupils, no neck stiffness and osteotendinous hyperreactivity without any motor deficit.

The biological panel showed hyperleukocytosis at 14900 /mm3 and elevated CRP (200 mg/l). Liver and renal function tests were normal. The non-contrast enhanced cerebral tomodensitometry performed was normal. The cerebrospinal fluid was clear with a very low cellularity (8 WBC / mm3 and 70 RBC / mm3), proteins and glycorrhachia were normal.

In this clinical setting, empiric treatment with ceftriaxone (2 gr every 12 hours) and aciclovir (10 mg: kg/d) were initiated. The patient was transferred to the Intensive Care Unit. The electroencephalogram showed no evidence of abnormal activity. PCR for herpes simplex virus (HSV), varicella-zoster virus (VZV), cytomegalovirus (CMV), adenovirus, enterovirus, Epstein Barr virus (EBV) and measles were negative. The serology for rubella, borreliosis, listeriosis, CMV, herpes, VZV and EBV were also negative.

Few hours after admission, the patient presented a deterioration of consciousness (GCS Score 8/15) and anisocoria, needing sedation and intubation.Mannitol and hypertonic sodium chloride (20%) infusion were started. The cerebral magnetic resonance imaging performed demonstrated the presence of deep vein thrombosis associated with extensive ischemic venous lesions in the grey nuclei and several hemorrhagic lesions in the right caudate nucleus. Diffuse edema compressed the third ventricle, lateral ventricles were enlarged. (Figure 1 and 2). This aspect was compatible with cerebral thrombophlebitis of the right sinus associated with intracranial hypertension signs.

After a multidisciplinary discussion, an external ventricular drain (EVD) placement was placed associated with enoxaparine 100 mg/12h. Aciclovir and ceftriaxone were discontinued.

After seven days of treatment in the ICU, the cerebral MRI showed a reduction of intracranial hypertension, the edema of the grey nuclei, and regression of the thrombus and the haemorrhage in the right cerebellum. (Figures 3 and 4) Progressive clinical improvement was noted, EVD was removed, and the patient was extubated. Enoxaparin was switched to acenocoumarol, and the patient was discharged from ICU on day 45. She was unfortunately tetraparesis.

She was reevaluated at the neurology outpatient clinic fourth months after hospital discharge, and she was still on wheelchair and pursuit a neurological revalidation program.

## Discussion

Immediate postpartum cerebral thrombophlebitis is a rare and severe condition that can be life-threatening. Pregnancy and postpartum increase the risk of the thrombotic event as a result of a hypercoagulation state. Among the other causes of CVT, it is mandatory to rule out drugs (oral contraceptive) and hereditary or acquired abnormalities as they require specific management [1].

The average incidence of postpartum cerebral thrombophlebitis is 12/100 000 births. This accounts for 5 to 20% of all CVT. The clinical symptomatology of postpartum CVT is polymorphic. The most common clinical signs reported in the literature are headache (88%), seizures (32%), focal or bilateral neurological disorders (37% to 39%), alteration of consciousness

(15% to 20%), papillary edema (28% to 32%) and psychiatric disorders [1, 2]

In front of this non-specific symptomatology, differential diagnosis with other conditions such as a transient ischemic attack, subarachnoid haemorrhage, tumoral process or meningoencephalitis is mandatory. However, diagnosis of cerebral thrombophlebitis is considered to be radiological.

Contrast-enhanced cerebral computed tomography is the first-line imaging investigation. This could be normal in up to 25% of cases, especially in cases of isolated intracranial hypertension. The best direct sign, visible on a CT after contrast injection is the empty delta sign described in about 25% of cases. The empty delta sign is a finding that is seen on a contrast-enhanced CT (CECT) and was first described in thrombosis of the superior sagittal sinus. The likely explanation is enhancement of the rich dural venous collateral circulation surrounding the thrombosed sinus, producing the central region of low attenuation. Indirect signs of CVT, are venous infarctions, cerebral edema or contrast enhancement of the false brain or tent cerebellum. Cerebral MRI is the gold standard for the diagnosis of CVT because it allows visualizing the thrombus and the associated parenchymal lesions and it is also the best examination for follow-up. [3]

The treatment of CVT has two axes: symptomatic treatment to control intracranial hypertension based on corticosteroids, mannitol, hypertonic sodium chloride, sedation, and neurosurgical treatment by external ventricular drains or decompressive craniotomy [3,4].

Anticoagulation-allows the recanalisation of sinuses or veins secondary limiting the thrombus extension and preventing recurrence. This therapy, which has been controversial for a long time, is widely accepted in CVT even in case of intracerebral hemorrhagic infarct. There is no consensus about the duration of treatment [3,4]. Cerebral venous thromboses, if treated early and efficiently, have a good functional prognosis

The mortality rate varies from 5 to 10%. Neuropsychological sequelae are observed in 10 to 15% of cases, including epilepsy in 5 to 32% of cases. Poor prognosis factors such as the thrombosis location (deep veins, cerebellum and isolated cortical thrombosis), nature of the underlying disease, presence of focal signs, coma, age of onset, or presence of a haemorrhage have been described. [5]

In this case, the patient was treated late, and she presented signs of intracranial hypertension, brainstem ischemia and haemorrhage explaining the severe neurological sequelae.

This case demonstrates the importance of early diagnosis and treatment of CVT to minimize neurological sequelae.

## Conclusion

Postpartum cerebral thrombophlebitis is rare, and its clinical presentation is variable. Early diagnosis improves prognosis MRI is the gold standard for diagnosis and allows early and adequate therapeutic management.

## Conflict of Interest

There are no conflicts of interest to declare by any of the authors of this study.


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