Clinical and therapeutic aspects of cerebral thrombophlebitis of postpartum

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Abstract

Cerebral thrombophlebitis are rare pathologies (1/5000 births). Pregnancy and postpartum are favorable circumstances: their clinical symptomology is entertained and varied dominated by headaches, convulsions and neurological deficits but no specific signs. The physical examination is often poor and ready for confusion with many other affections.

The diagnosis of certainty can only be neuroradiological. MRI is currently the reference method; it allows the visualization of venous thrombus and the monitoring of its evolution. The treatment of these TPC is mainly medical based on anticoagulants.

In four observations of TPC, postpartum and a literature review, we will focus on the importance of early diagnosis and appropriate therapeutic management.

Keywords: cerebral thrombophlebitis • postpartum • cerebral MRI • convulsion • heparinothherapy

Introduction

Cerebral thrombophlebitis (TPC) are rare but formidable pathologies. They include thrombosis of the venous sinuses of the dura mater and superficial and deep cerebral veins. Its frequency is estimated at 1/3000 to 1/10000 births and 0.5% to 1% of all strokes [1].

Apart from the classic risk factors such as the hypercoagulability of pregnancy and postpartum as well as the presence of thrombophilia, the impact of other factors favoring in particular age, parity and the conditions of delivery remain unclear.

In four post-partum TPC observations and a literature review, we will focus on the importance of early diagnosis and appropriate therapeutic management.

Case Presentation (Patient and Observation)

■ Observation 1:

A 22-year-old patient with no significant pathological history, G1P1, was admitted on the 18th day of postpartum for extreme psychomotor agitation. The beginning of the symptomatology was on the 7th day of the post-partum associating headaches and insomnia followed by the installation of a delusional syndrome and psychomotor agitation. She was initially hospitalized in the psychiatric department for suspicion of puerperal psychosis and put on neuroleptics but without improvement. The initial clinical examination showed no motor deficit or meningeal stiffness. The brain CT scan was normal, along with the lumbar puncture and fundus. In front of the worsening neurological picture requiring assisted ventilation. Cerebral MRI showed a TPC-compatible appearance at the expense of the left transverse sinuses without associated parenchymal reshaping **(FIGURE 1)**.

The patient was on curative dose anticoagulant therapy with anti-convulsive therapy. The evolution was favorable allowing awakening and desintubated.

The patient was on curative dose anticoagulant therapy with anti-convulsive therapy. The evolution was favorable allowing awakening, ventilator weaning and the exit of resuscitation on the 6th day. A relay with antivitamin K was started after 14 days of heparin therapy. Seen at three months of treatment, the patient kept no functional sequelae.

■ Observation 2

Mrs. Z.G, 43 years old, multipara, with no significant pathological history, was admitted to resuscitation 32 days after a caesarean section

for agitation and confusion associated with tonic movements, loss of consciousness and tongue bite without fever. Cerebral CT was normal, as was the lumbar puncture. Despite the anticonvulsant treatment, the evolution was marked by the persistence of the tonic attacks that required its mechanical ventilation with deep sedation. A brain MRI was urgently requested, revealing cerebral thrombophlebitis **(FIGURE 2).**

A curative heparin therapy was started, allowing the patient to wean and extubate after three days of mechanical ventilation. Heparin therapy was relayed after 10 days by the anti-vitamin K. She left the hospital after 15 days without sequelae.

■ Observation 3

G3P2, a 29-year-old patient with no particular pathological history, had consulted for headache, vomiting and disorder of consciousness. Examination revealed a Glasgow score of 11/15, temporo-spatial disorientation, deep tendon reflex was sharp, high blood pressure 18/9, and renal-type edema.

In front of this table of severe pre-eclampsia, the patient received a load dose of 4 g of magnesium sulphate in IVL maintained by 1g/h at the electric syringe pump associated with 1 mg/h of Loxen.

Pelvic ultrasound showed a complicated retro placental hematoma. The biological assessment did not show any particular abnormalities. The patient had a cesarean section for maternal rescue. She was put on magnesium sulphate at the dose of 1g/h and loxen at the push electric syringe.

The evolution was marked by the appearance of a tonic-clonic seizure that occurred two hours after the operation and given up by 10 mg of Valium. The immediate post-operative assessment revealed DIC with Hb 8 g/dl, Ht at 21%, thrombocytopenia at 47,000, TP at 45%, and fibrinemia<0.8 g/l. She had received a transfusion of two globular units, 6 CP, 6 PFC and 4 g fibrinogen. The control balance showed: Hb at 7.5 g/dl, Ht at 23%, platelet at 63,000, TP at 95% and Fib at 3.05 g/l.

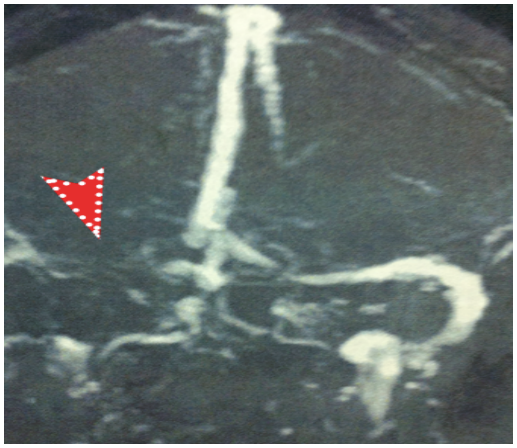

FIGURE 1. Angio IRM showing thrombosis dependent on left transverse sinus.

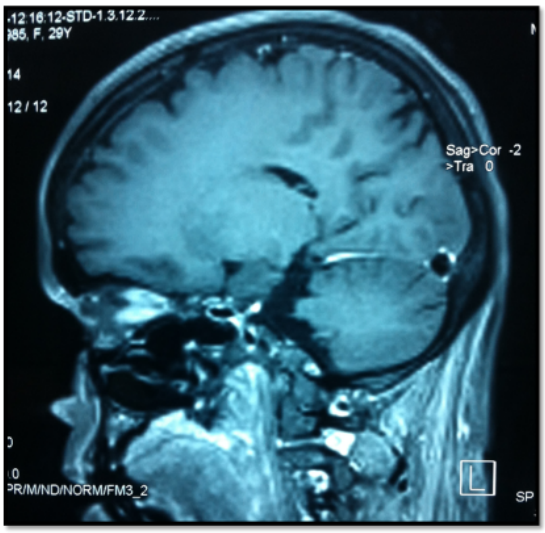

FIGURE 2. Frontal Angio IRM section shwoing thrombosis dependent on left transverse sinus.

Clinical and biological improvement was noted. At day two post-op, the patient had an altered state of consciousness, a Glasgow 13/15 with dysarthria. An emergency brain scan revealed a spontaneous hypo density of two external capsules evoking cerebral thrombophlebitis that was confirmed by a brain MRI.

The patient was treated with curative-dose heparin therapy. The progression was marked by an improvement in the neurological condition. An overlap with AVK was started on the 5th day. The patient had left the service without sequelae with an INR at 2.26 and a TP at 38%.

■ Observation 4

A 26-year-old patient with no significant pathological history, G1 P1 was admitted to resuscitation at 17 days of postpartum for agitation, behavioural disorder and fever, followed by altered consciousness. Initial clinical examination showed anisocoria, Glasgow score at 10/15, right hemiplegia and left facial paralysis. The evolution was marked by the rapid worsening of the state of consciousness requiring intubation and mechanical ventilation.

The brain CT scan revealed a focal spot of left temporal venous softening that is the site of intra-lesional hemorrhage. A brain MRI was urgently requested revealing a cerebral thrombophlebitis dependent on the upper longitudinal sinus with a hemorrhagic softening of a left posterior parietal venous infarction. She had benefited from deep sedation with an optimization of her hemodynamic state and put under a curative anti-coagulation. She had also benefited from a decompressive left cranial flap that was kept under the adipose tissue of the abdomen.

The evolution was marked by a neurological and respiratory improvement at the expense of a resuscitation stay requiring heavy and prolonged care. Recovery after 64 days of conscious resuscitation, Glasgow score 15/15 on AVK. 6 months later, she had received a repositioning of her cranial limb.

Discussion

Pregnant women are at increased risk of developing cerebral venous thrombosis due to their hypercoagulability, increased clotting factors (VII, VIII, IX, XI, XII) fibrinogen and platelets and decrease in fibrinolytic activity. Postpartum is also a favorable circumstance for the occurrence of this complication. Indeed, in more than half of the cases, these TPC occur in the first week [2]. This risk also increases with maternal age, cesarean delivery, and the presence of IHT, infection and pregnancy vomiting [2, 3].

The clinical symptomatology of these PTCs is very polymorphic. The main thing is to make an early diagnosis. Their installation can be early (less than 48 hours of delivery) in 28% of cases, sub-acute (between 48 hours and 30 days) in 47% of cases or late (more than 30 days) in 25% of cases [2-11].

Cerebral thrombophlebitis should be suspected when the parturient develops symptoms associating to varying degrees intra-cranial hypertension (headache, vomiting, papillary edema, disorders of consciousness) and/or focal neurological deficit and/or seizures [3]. Headaches are the most common symptom, found in 75% of cases, they have no specific characteristics or evolutionary profile. Papillary edema is present in about 50% of cases, neurological deficit in 34% to 70% of cases, seizures in 20% to 57% of cases, disorders of consciousness in 34%-70% of cases or psychiatric disorders that are sometimes in the foreground and mark the rest of the symptomatology [5]. Our four patients had a sub-acute facility. Their symptomatology combines signs of cranial hypertension and agitation in 03 cases, neurological deficit in one case and seizures in 02 cases.

This clinical symptomatology poses a problem of differential diagnosis with eclampsia, hemorrhage sus arachnoid, a transient ischemic accident or even puerperal psychosis as is the case with our first observation[8,4].

The positive diagnosis can only be neuro-radiological [1]. Brain CT without and with contrast injection is still performed at first intention. It remains normal in 4% to 25% of patients with TPC [3]. The typical appearance is the presence of delta sign, found in about 25% of cases. It appears as a hypodense area surrounded by a contrast setting. Another direct sign is fresh thrombus which appears as a spontaneous hyperdensity at the thrombosis vein [9]. The indirect signs visible on the cerebral scanner are essentially venous infarction but also the existence of cerebral edema. In our work, cerebral CT was normal in the first two observations, showed venous infarction in the 3rd and the presence of venous thrombosis in the 4th case.

Brain MRI is currently the reference method for the diagnosis of TPC. The usual sequences are the T1 and T2 weighted echo and spin sequences, the FLAIR sequences for the parenchyma study. The T2* sequence is sensitive to the presence of blood and more recently the diffusion and perfusion weighted sequences. MRI allows the visualization of venous thrombus and the monitoring of its

evolution. The MRI was performed in three of our patients, had allowed the diagnosis of TPC while the brain CT was normal.

Magnetic resonance angiography is a complement to brain MRI. It allows visualization of venous circulation and thrombosis [10].

The search for thrombophilia is systematic to detect a possible deficit in anti-thrombin III, responsible for 20% to 40% of thrombosis during pregnancy or a deficiency in protein C or S responsible respectively of 7% to 22% of thrombosis in the post-partum [5-7].

There are two components to the processing of PCTs:

The treatment of the thrombotic process is based on anticoagulation whose objective is to prevent the extension of thrombosis in order to allow the development of collateral circulation and the prevention of venous infarction. The theoretical risk is that of massive bleeding within an infractus, willingly spontaneously hemorrhagic. Two randomized therapeutic trials evaluated the benefit/risk ratio of placebo anticoagulant therapy in patients with proven CPD, showed a statistically significant benefit in favour of heparin and without major risk of hemorrhagia [3].

Treatment with fibrinolites is difficult to recommend in the absence of randomized studies that compare the risk/benefit ratio [1].

Symptomatic treatment to combat intra-cranial hypertension based on drug treatments such as corticosteroids, mannitol, deep sedation and sometimes surgical treatment including decompressive craniopathy which can be discussed in case of major edema. The anti-epileptic treatment is systematic in case of seizures.

The classic factors of poor prognosis are the reaching of deep veins, the existence of a coma and advanced age [2].

The evolution is towards healing in the majority of cases, permanent sequelae are observed in 10% to 30% of cases [1]. For our patients, the progression was good due to the rapid introduction of heparin therapy and adequate management of intra-cranial hypertension.

Conclusion

TPC postpartum is a rare but serious condition. The clinical diagnosis remains difficult, you must know how to think about it even in front of signs little evocative and request neuro radiological examinations to confirm it especially the MRI. The benefit of heparin therapy is now well sought. The prognosis remains good if the diagnosis is made on time and if treatment is started early.

Competing Interests

No conflicts of interest

Authors' Contributions

Mhelheli Riadh, Hafsi Montacer: Data acquisition and data interpretation, article writing.

Ben Moumen Olfa, Gomri Emna, Hachicha Sarra: Article Writing

Ragmoun Houssem: Supervision, correction and critical review of the article

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