

Good Management of an Ischemic Stroke on Postpartum Complicated Gestational Hypertension and Uterine Rupture

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Abstract

Clinical Case

We present a case of postpartum ischemic stroke due to hypertension of pregnancy complicated by uterine rupture. Thanks to the clinical elements, in particular postpartum seizures, disturbances of consciousness and an immediate CT scan confirmed the diagnosis. The patient was resuscitated and transferred to neurology with hemiparesis as a sequel. We hope that the treatment of these patients can be improved thanks to our experience in this case management.

Keywords: Ischemic stroke; Post-partum; Pregnancy arterial hypertension; Uterine rupture.

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INTRODUCTION

Pregnancy is classically considered to be a risk factor for ischemic and hemorrhagic stroke. There are, during pregnancy physiological changes likely to increase the risk of a vascular event: state of maximum hypercoagulability near term [1], increase in cardiac output and in total blood volume, and changes in the structure of the arterial media.

The occurrence of stroke during pregnancy or postpartum (puerperal-stroke gravid) however, is a rare event, but potentially dramatic by the risk of disability and death among mothers and possible consequences to the fetus or newborn.

Many uncertainties remain about the impact of these stroke risk factors and their causes, maternal and fetal prognosis and obstetrical and neurological management [2-5]. Current data suggest that stroke risk is not increased significantly during the pregnancy itself.

However, the postpartum period is associated with an increased risk of stroke, whether infarction, hemorrhage or cerebral venous thrombosis (CVT). These patients require multidisciplinary care, which makes the central role of the resuscitator undeniable,

particularly in cases of disturbance of consciousness. We report the case of a complicated pregnancy induced hypertension and ischemic stroke diagnosed in a parturient woman.

CLINICAL CASE

This is a 20-year-old housewife, primigest, with no specific medical or surgical pathological history, received at the maternity hospital for abdomino-pelvic pain with the notion of pregnancy-induced hypertension. On admission the blood pressure was 180/120 mmhg, with negative albuminuria, the temperature was 36.2° C.

The examination had shown a foeto-pelvic disproportion and the indication for a cesarean was made with extraction of a newborn male Apgar at 10/10. At H10 post-cesarean section, the patient presented disturbances of consciousness and psychomotor agitation with an arterial pressure of 80 / 60mmhg, negative albuminuria, temperature at 36° and a pulse at 127 beats / min. On examination: pale mucous membranes, distended and painful abdomen, flaccid uterus and on vaginal examination the cervix was dehiscence with abundant bright red blood. The indication for a laparotomy was made and had objected to a posterior uterine rupture requiring a hemostasis

suture. General anesthesia was given. The parturient received 1g of tranexamic acid and intraoperative vascular filling. The post-operative consequences were simple. On postoperative D01, the patient presented with an alteration of consciousness and convulsive seizures with a Glasgow score of 09/15 (E2V3M4) which motivated her referral to intensive care for better management.

On admission, AP = 137 / 92 mm Hg; Albuminuria negative, T ° = 35.8°C; HR = 86 beats / min; FR = 10; SpO2 = 100%; GC = 1.13g / dl /. The examination revealed a non-febrile coma with a Glasgow score of 07/15 (E2V3M4), left pyramidal syndrome and clinical anemia.

The paraclinical examinations carried out at her admission found:

Biology	Hemogram: Hb= 6,2g/dl; RBC= 4.100.000; Ht =16,1%; WBC= 17.060; Platelets =125G/l Serum electrolyts: Na= 138mmol/l; K=3,1mmol/l; Cl=107mmol/l Renal function: Urea = 0,51g/l; Creatinin =19,2mg/l PT/INR= 67% / 1,25 PTT = 32,4 sec Blood type = AA +
Imagery	Brain scan : acute ischemic stroke in medium cerebral artery superficial territory

Hb: hemoglobin
WBC: white blood cells
RBC: Red blood cells
Na: sodium K potassium
Cl: chlorine
PT: Prothrombin time
INR: international normalized ratio
PTT: partial thromboplastin time

The diagnosis of ischemic stroke associated with uterine rupture due to hypertension of pregnancy was retained. The following treatment was carried out:

- Hospitalization and conditioning (urinary probe, scope, peripheral venous route, oxygen)
- Ceftriaxone: 2g / day as a single dose from D1 to
- Injectable metronidazole: 500mg in three doses / day
- Injectable paracetamol
- Injectable oxytocin
- Enoxaparin 0.4mL / D

- Omeprazole 40mg / D
- Aspirin 100mg: 1sachet / D from D2
- Blood transfusion

On Day 2 of admission, the evolution is marked by an improvement in the state of consciousness with a Glasgow score = 13/15 (E4V4M5), good uterine involution, on the other hand we noted a persistence of the left pyramidal syndrome. The course of action was to start physiotherapy, complete the assessment and continue treatment.

Biology	Lipid profile: Total cholesterol =1,83 g/l; Triglycerides=1,11g/l ; HDL=0,52g/l ; LDL=1,08g/l Emmel test negative
Imagery	ECG= Regular sinus tachycardia HB 125 per minute, normal axis

On Day 5 of admission, we noted a good improvement in her clinical condition with normal consciousness, Glasgow score of 15/15 and regression of left hemiplegia in left hemiparesis with upper limb rated at 2/5 and lower limb 4 / 5.

On Day 9 of admission, seeing the good improvement in her clinical picture, the patient was transferred to the neurology department.

DISCUSSION

The occurrence of a cerebrovascular accident (stroke) is a rare event during pregnancy or postpartum (10/100 000 deliveries) [2-5]. All types of stroke can occur, whether they are ischemic (24%), hemorrhagic (74%) or mixed (2%). Pregnancy is probably a more risky situation for stroke although this is debated for

arteriovenous malformations (AVMs), aneurysms and cavernomas [6]. The symptoms and clinical signs of GP-stroke are unremarkable. The positive diagnosis is based on brain imaging. The etiological investigation must be as rigorous as it can be outside of pregnancy, in order to allow appropriate therapeutic management [7]. Apart from the classic vascular risk factors, some authors have described more or less specific risk factors for pregnancy: maternal age over 35, black race, pre-existing or gestational high blood pressure (hypertension), preeclampsia, post-pregnancy complications (haemorrhages and infections), transfusions, fluid and electrolyte disturbances, delivery by cesarean section, onset of migraine during pregnancy, coagulopathy [8-11]. Other risk factors probably remain to be identified. The relative frequency of the causes of GP-stroke remains poorly understood.

It is likely, moreover, it varies by country. Sudden onset focal deficits are frequently observed, associated with the classic signs of headache, seizures and visual disturbances. Only MRI with diffusion sequences can reliably differentiate cerebral infarctions that can complicate eclampsia, reversible posterior encephalopathy lesions (Posterior Reversible Encephalopathy Syndrome [PRES]), which are much more frequently observed. The majority of CVD-GPs occur postpartum, after normal childbirth, mainly in Western countries. CVDs of pregnancy are rarer and can occur at any term. The direct role of GP state in the occurrence of CVD is well established [12, 13], but the frequency of associated contributing factors, such as anticoagulant protein deficiency, especially when CVD occurs during pregnancy, is less well known. After pregnancy, a haemostasis assessment looking for thrombophilia is recommended. Many uncertainties remain as to the necessary therapeutic, neurological and obstetrical management. Apart from pathologies with a high thromboembolic risk (major embologenic heart disease, coagulopathies), the antithrombotic treatment most often proposed in cases of cerebral infarction is aspirin. However, there are few recommendations available [14]. The use of aspirin during the first trimester remains controversial [15]. If indicated for anticoagulant therapy, low molecular weight heparins (enoxaparin, dalteparin, tinzaparin) are the treatment of choice because they do not cross the blood placental barrier. They are easier to use than unfractionated heparin and carry a lower risk of thrombocytopenia. Anti-vitamin K (AVK) are contraindicated between the sixth and the twelfth week because of the teratogenic risk. Fetal brain abnormalities have been described with AVK regardless of the term of pregnancy. AVKs are therefore reserved for exceptional indications (mechanical valves for example). Low molecular weight heparins and warfarin are compatible with breastfeeding. Data on the use of rt-PA in acute cerebral infarction during pregnancy and the immediate postpartum period are still limited. Only a dozen cases have been reported; in particular, the fetal risks are poorly understood [16]. The indication should be made on a case-by-case basis. There is no consensus on how to deliver after a stroke during pregnancy. They must be decided above all on the basis of obstetric criteria, without resorting to a systematic delivery by cesarean section. Maternal mortality linked to stroke represents 5 to 10% of total maternal mortality (4.1% in a recent American study) [9]. Mortality by stroke subtypes is poorly understood; it is higher in cases of cerebral hemorrhage than in the case of a heart attack [8]. Deaths from stroke occurred during active pregnancy in 25% of cases, after spontaneous miscarriage in 5% of cases and postpartum in 70% of cases [6]. The only data concerning the risk of recurrence of a cerebral infarction during subsequent pregnancies come from a French multicenter study [7]. This study did not show a significant increase in the risk of heart attack during pregnancy itself, the postpartum period being associated

with a significant risk of recurrence (RR: 9.7; 95 CI: 1, 2-78.9).

CONCLUSION

The occurrence of a stroke is a rare event during pregnancy or postpartum. All types of stroke can occur, whether ischemic, hemorrhagic or mixed [6]. Pregnancy is probably a more risky situation for stroke. The management of postpartum ischemic strokes must be early, therefore instituted as soon as the focal neurological signs appear suddenly, as any diagnostic delay worsens the prognosis.

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