



Case Report

Stroke and Multi-Organic Infarction Revealing Peri-Partum Cardiomyopathy, an Unusual Presentation: About a Case

Antoine Salambo Mabila^{1,2,3*}, Donald Accrombessi¹, Patrick Lisasi², Job Osango^{2,4}, Elisé Kisioko^{2,4}, Patron Mulumba², Robert Mutombo², Aurélien Darrigol⁵, Arcel WOGA¹, Martial Zossou¹, Anne Landais¹, Annie Lannuzel¹

¹Neurology Department of the CHU of Guadeloupe, Guadeloupe

²Neuropsychopathological Center (CNPP), University of Kinshasa/ DRC, Congo

³Medical School, Notre-Dame du KASAYI University/DRC, Congo

⁴Medical School, University of KIKWIT/DRC, Congo

⁵Cardiology Department of the Guadeloupe University Hospital, Guadeloupe

***Corresponding author:** Antoine Salambo Mabila, Neuropsychopathological Center (CNPP), University of Kinshasa/ DRC, Congo

Citation: Salambo Mabila A, Accrombessi D, Lisasi P, Osango J, Kisioko E, et al. (2023) Stroke and Multi-Organic Infarction Revealing Peri-Partum Cardiomyopathy, an Unusual Presentation: About a Case. Int J Cerebrovasc Dis Stroke 6: 160. DOI: <https://doi.org/10.29011/2688-8734.100160>

Received Date: 28 August, 2023; **Accepted Date:** 30 August, 2023; **Published Date:** 02 September, 2023

Summary

Peripartum cardiomyopathy is a rare cause of pregnancy-related strokes.

We present a case of cerebral infarction with multi-organ ischemic damage complicating peripartum cardiomyopathy. A 25-year-old patient, with no cardiovascular risk factor, was admitted to Neurology at the University Hospital of Guadeloupe, for phasic disorders occurring eleven weeks postpartum of a pregnancy marked by gravidic hypertension. The assessments carried out revealed cerebral, splenic, renal infarctions and mesenteric thrombosis on cardiomyopathy with 25% LVEF and left intraventricular thrombus.

Curative anticoagulation and treatment of heart failure were initiated on the firstday of hospitalization, with a favorable outcome.

A cardiological follow-up was organised, noting a progressive clinical improvement at a distance.

Our case recalls the importance of a systematic medical follow-up in postpartum, and raises the possible gravity of the infarctions related to the CMPP, requiring an optimal care involving Cardiologists and Neurologists.

Keywords: Stroke; PPCM; Multi-organ infarction

Introduction

Pregnancy and the puerperium confer an increased risk of ischemic and hemorrhagic stroke, with incidence rates three times higher than in non-pregnant women [1].

Potential causes of stroke identified in the literature include those that can occur in young non-pregnant people and those that are exclusive to pregnancy. Diagnoses that are not specific to pregnancy include but are not limited to venous sinus thrombosis, cardioembolism, systemic vasculitis. Those that are more specific complications of pregnancy include preeclampsia/eclampsia, amniotic fluid embolism, and peripartum cardiomyopathy [2].

Peripartum cardiomyopathy (PPCM) is a rare cause of heart failure. It can lead to cardioembolism or, less commonly, watershed infarction due to hypotension [3]. It is a disorder of the myocardium which is most common in the age group of 22 to 33 years, where the heart muscle is structurally and functionally abnormal in the absence of coronary artery disease, hypertension, disease valve and congenital heart disease [4].

Risk factors for PMPC include older age, parity, African American race, hypertension, preeclampsia, eclampsia, infection, smoking, diabetes, and cesarean delivery [5].

Ischemic strokes are reported in only 5% of patients with peripartum cardiomyopathy, although the incidence of systemic thromboembolic complications is high at 25-40% [6,7].

We report an unusual case of peripartum cardiomyopathy revealed by multiple cerebral infarctions and multi-organic infarctions, motivating a particular therapeutic attitude. The patient has given informed consent for the publication of this case report, including the images.

Case Presentation

This is a 25-year-old patient, mother of two children aged 8 years and 77 days respectively, admitted to the Neurology Department of the Guadeloupe University Hospital on January 14, 2023.

The patient presented on 08/01/2023 with dizziness, nausea and vomiting; regressive after 24 hours without medical treatment. On 01/12/2023, phasic disorders occur, with complete regression the following day, then resumption of phasic disorders 24 hours later, motivating the transfer to the Emergency Department, regulated on thrombolysis alert. It should be noted that the patient has no known morbid history, apart from pregnancy-induced hypertension during the last pregnancy. She also reports a notion of exertional dyspnoea since childbirth, without medical follow-up.

On arrival at the CHU, the blood pressure was 151/125 mm Hg, with tachycardia at 112 beats per minute and oxygen saturation

at 100% in ambient air.

The neurological examination found an NIHSS (National Institutes of Health Stroke Scale) of 8 with, in particular, severe aphasia, left visual neglect, left facial paralysis and paresis and ataxia of the left upper limb.

A cerebral MRI is performed and finds multiterritorial infarcts of different ages (right superficial sylvian, left superficial sylvian and left PICA) (Figure 1).

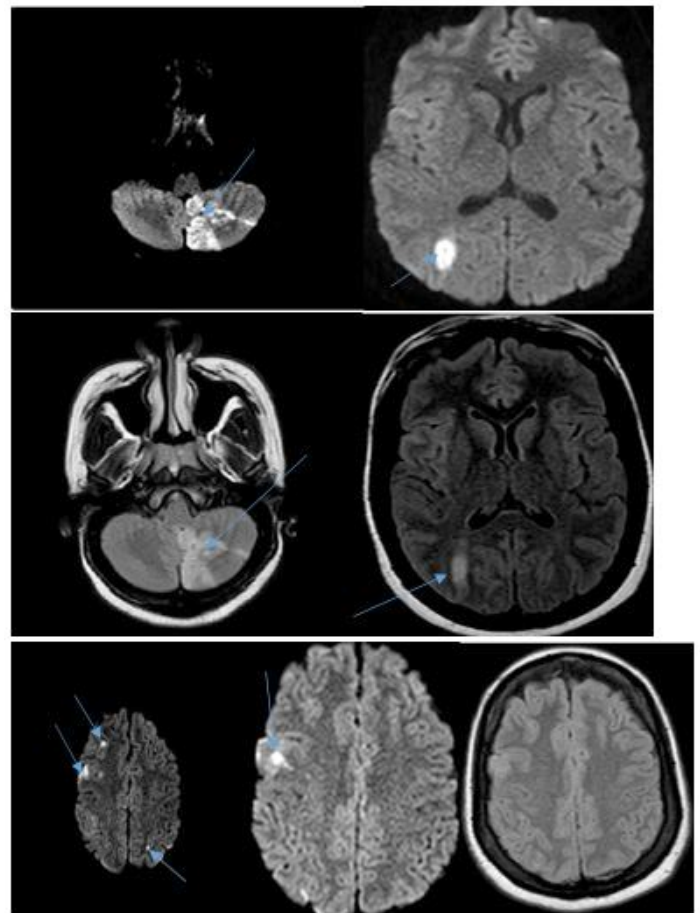


Figure 1: Multiterritorial infarcts, some of which not visible in FLAIR sequence.

A transthoracic echocardiography is performed, finding in particular global hypokinesia, a very altered left ventricular ejection fraction (LVEF) estimated at 25% in Simpson biplane (SB) and a large 5.2 cm² thrombus adhered to the apical wall of the left ventricle, a thrombus also found on the thoraco-abdomino-pelvic (TAP) CT scan (Figure 2).

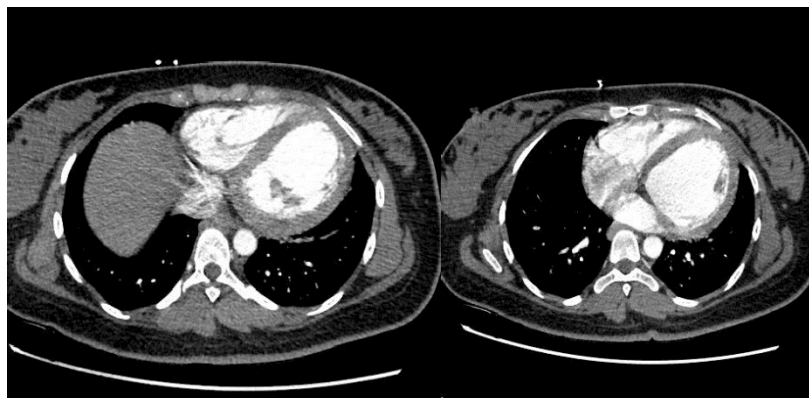


Figure 2: Left intra-ventricular thrombus, on TAP CT scan.

The TAP scan also found a non-occlusive thrombus of the superior mesenteric artery and its left divisions, several foci of splenic infarction and multiple foci of infarction of both kidneys (Figure 3).

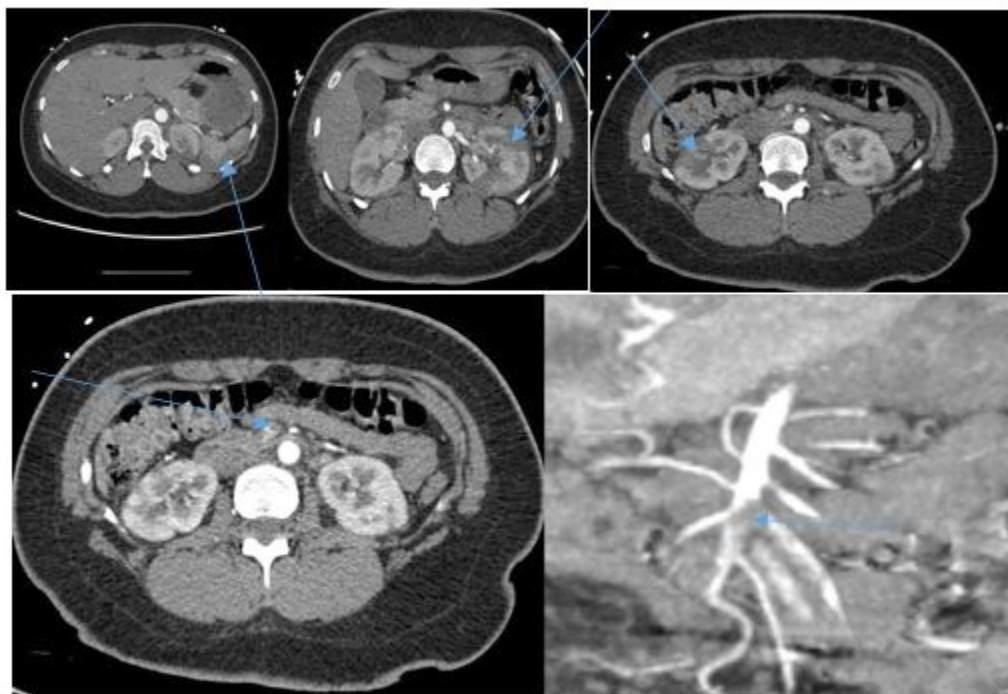


Figure 3: TAP CT scan: splenic infarcts, kidney infarct foci, mesenteric thrombus.

The rest of the etiological assessment of the cerebral infarction was without abnormality.

The patient was put on curative anticoagulant treatment with heparin sodium by electric syringe pump for 48 hours, followed by Enoxaparin 100 IU per kg of weight every 12 hours. Heart failure treatment was initiated with Entresto (sacubitril/valsartan), spironolactone, furosemide, Bisoprolol and dapaglifozine.

A follow-up echocardiography performed on January 20 found a severely altered LVEF at 20%, with no intraventricular thrombus.

The clinical evolution was favorable justifying a return home on January 25 with NIHSS of 1 on a slight aphasia (lack of the word).

A Cardiological follow-up was organized for the rest of the care, noting a good evolution:

- Cardiac MRI of 02/15/2023: hypokinetic dilated heart disease, non-specific septal fibrosis without evidence for myocarditis with LVEF 39%; echogenicity: correct.
- ETT of 07/03/2023 = dilated LV 93 ml/m², not hypertrophied. Global hypokinesia. LVEF estimated at 42% in SB. No left intraventricular thrombus. Absence of significant valve disease.
- The last cardiological consultation (May 2023) indicates an overall satisfactory evolution: no chest pain, no syncope or fainting, no palpitation, appetite and sleep correct, no signs of heart failure ECG: sinus, normal PR, fine QRS, small lower q waves, left ventricular hypertrophy, negative T waves in V1.

Discussion

Maternal stroke, defined as stroke occurring during pregnancy or the postpartum period, is increasingly recognized as a major cause of maternal morbidity and mortality.

A review of maternal strokes at a stroke referral center in New York found that more than 70% of strokes occurred after childbirth [8].

Peripartum cardiomyopathy, left ventricular dysfunction during the last month of pregnancy or within 5 months postpartum in women without preexisting heart disease or alternative etiology, is another possible cause of maternal cardioembolic stroke, with few cases documented in the literature [9].

Our case meets this definition, with multi-territorial cerebral infarctions occurring 11 weeks after delivery with left ventricular failure in a young patient, without known heart disease.

It should be noted that almost all of the articles published on the incidence of pregnancy-related strokes cover the period of pregnancy and are limited to six weeks after delivery [1,10].

We did not find publications of case series on stroke secondary to peripartum cardiomyopathy.

Very few cases of stroke complicating peripartum cardiomyopathy are found in the literature.

Among the published cases, rare are those diagnosed within six weeks of delivery: 10 days postpartum for the case of Hang et al and 4 days for that of WooFUI et al. [7,11].

The majority of the other cases listed were diagnosed, like ours, beyond 6 weeks after delivery: Nasa et al. (two months after delivery); Kumbham et al. (8 weeks postpartum), Pradhan et al. (7 weeks); Jiménez-Ruiz et al. (12 weeks) [4,12-14].

Thus, not only are strokes complicating postpartum cardiomyopathy rare, but they are also underrepresented in pregnancy-related stroke series.

Our case highlights the lack of postpartum medical follow-up in a patient who presented exertional dyspnoea since childbirth. This picture of dyspnoea was witness to a cardiomyopathy of the peripartum developing until being expressed eleven weeks later by signs of mesenteric and cerebral infarction.

Although the majority of peripartum cardiomyopathy occurs in normotensive women, the prevalence of hypertensive disorders of pregnancy in women with peripartum cardiomyopathy is increased [9].

The notion of pregnant hypertension during the last pregnancy, followed by exertional dyspnea after childbirth, could lead to the early diagnosis of peripartum cardiomyopathy and indicate medical follow-up with appropriate management.

We did not find in the literature a case of peripartum cardiomyopathy with multi-organ ischemic involvement.

There are no guidelines available for the management of stroke in these patients. There is also controversy regarding the use of anticoagulation to prevent stroke recurrence in patients with peripartum cardiomyopathy due to the risk of hemorrhagic transformation of cardioembolic stroke [4,15].

Despite this obvious risk of hemorrhagic transformation in our patient, curative anticoagulation was initiated with close monitoring. This management was guided by the high risk of recurrence in the presence of left intraventricular thrombus, and the vital risk linked to multi-organ ischemic damage.

Conclusion

Pregnancy can, through CMPP, constitute a risk factor for serious forms of cerebral infarction in young patients, without cardiovascular risk factors.

This is, to our knowledge, the first published case of peripartum cardiomyopathy complicated by multi-territorial cerebral infarction, with multi-organ ischemic damage.

This case reminds us of the need for rigorous postpartum medical follow-up. It also allows the Neurologist to be made aware of the possibility of the coexistence, in the context of complications of PPCM, of ischemic cerebral and multi-organ damage, threatening the vital prognosis and involving consequent therapeutic adaptations.

Conflict of Interest

The authors declare that the research was conducted in the absence of any commercial or financial relationship that could be construed as a potential conflict of interest.

Author Contributions

All authors contributed to the conduct of this work. All authors also declare that they have read and approved the final version of the manuscript.

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