

# Suspicion of peripartum cardiomyopathy in a preeclamptic near term primigravid complicated with pulmonary edema and placental abruption: Case report

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## ABSTRACT

Although peripartum cardiomyopathy (PPCM) is known since 1800s its etiology is still unclear and its differential diagnosis and management is a challenge for the clinician. This task becomes even more complicated when preeclampsia comes into play because the current guidelines state that hypertensive crises and preeclampsia ought to rule out PPCM. Nevertheless, new scientific evidence points out a link between preeclampsia and PPCM. We present the case of a 31-year-old female patient admitted at 35 weeks of gestation who required an emergent cesarean section delivery due to acute pulmonary edema and preeclampsia. Following hospitalization, signs of possible peripartum cardiomyopathy were found – although such possibility was later ruled out because of ESC 2018 guidelines. This case should make us reflect on the possibility of underdiagnosis, and thus undertreatment of PPCM due to not-so-up-to-date guidelines.

## KEYWORDS

Emergency cesarean section, acute pulmonary edema, placental abruption, peripartum cardiomyopathy, preeclampsia

## Introduction

Cardiomyopathies of many types, including peripartum, hypertensive, dilated, tako-tsubo disease, toxic cardiomyopathies, and storage illnesses, can occur during or after pregnancy. The diagnostic criteria for peripartum cardiomyopathy listed in the European Society of Cardiology (ESC) 2018 Guidelines <sup>[1]</sup> rely on an echocardiographic determined left ventricular ejection fraction of less than 45% in patients with heart failure that develops in the last month of pregnancy or within 5 months of delivery after the exclusion of other causes for heart failure.

Arrhythmia, cardiac arrest, and abrupt decompensated heart failure are only a few of the symptoms that can occur. Patients with a left ventricular ejection fraction lower than 30%, left ventricular enlargement, and right ventricular involvement have a worse prognosis. The mortality rate of a peripartum cardiomyopathy (PPCM) is between 2% and 24% which depends on the follow-up period. Patients whose ejection fraction is not greater than 55% are discouraged from having another child because of the risk of recurrence exists <sup>[1]</sup>.

For the patient profiled in this case report, a diagnosis of PPCM was initially postulated. This case report aims to highlight the challenges in composite diagnosis of acute heart failure at the end of pregnancy, especially due to the overlap of two entities such as preeclampsia and PPCM. Further studies investigating the co-relationship between PPCM and preeclampsia are needed in order to optimize the diagnosis, and therefore the subsequent treatment of patients.

## Article history

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## Case report

A 31-year-old Caucasian woman at 35 weeks of pregnancy – primigravida, with a diagnosis of preeclampsia at 34 gestational weeks, obesity, and chronic obstructive pulmonary disease with an active smoking history during pregnancy – was hospitalized due to acute hypertensive pulmonary edema and bilateral pleural effusion, with a blood pressure of 180/100 mmHg. She had been under treatment with labetalol (100 mg BID) and methyldopa (500 mg TID) since 30 weeks of pregnancy when she has been diagnosed with gestational hypertension. The patient reported a first-degree relative (mother) with a history of hypertension but no family history for cardiomyopathies or sudden cardiac death.

In the emergency room she was administered a diuretic and a bronchodilator and oxygen therapy with CPAP was administered. She also received magnesium sulfate (MgSO<sub>4</sub>) in prevention of eclampsia. An emergent cesarean section was subsequently performed under general anesthesia and orotracheal intubation due to maternal worsening clinical conditions. A healthy child was delivered with an Apgar scoring of 6, 7, 8 at 1, 5, 10 minutes

respectively. The placenta showed signs of abruption. The mother was taken postoperatively to the intensive care unit (ICU), where she received thromboembolic prophylaxis, non-invasive ventilation (NIV), antihypertensive medication, a diuretic, a uterotonic agent and postoperative analgesic therapy.

Upon her admission in the ICU, N-terminal pro-B-type natriuretic peptide (NT-pro-BNP) was dosed resulting in 5,306 ng/L, clear sign of myocardia dysfunction. An ECG recorded “sinus rhythm, 81 bpm, poor R wave progression in precordial leads” which ruled out left bundle branch block and other cardiomyopathies. Myocardial infarction was also ruled out by a troponin assay.

After 7 days since the delivery an echocardiography revealed a mildly dilated left ventricle with eccentric hypertrophy of its wall and a global systolic function mildly reduced by diffuse hypokinesia. There was an ejection fraction of 47%, diastolic filling from impaired relaxation, mild mitral regurgitation, severe left atrial dilation and no direct signs of pulmonary hypertension. Cardiologic consultation postulated PPCM as a possible diagnosis. The patient’s previous antihypertensive therapy was halted and heart failure medication was prescribed as follows: captopril (25 mg/day) bisoprolol (2.5 mg/day), potassium canrenoate (25mg/day) and furosemide (25 mg/day for seven days, then 25 mg every other day).

Cleared for discharge with regards to gynaecological and neonatological matters eight days after delivery, hypertension and

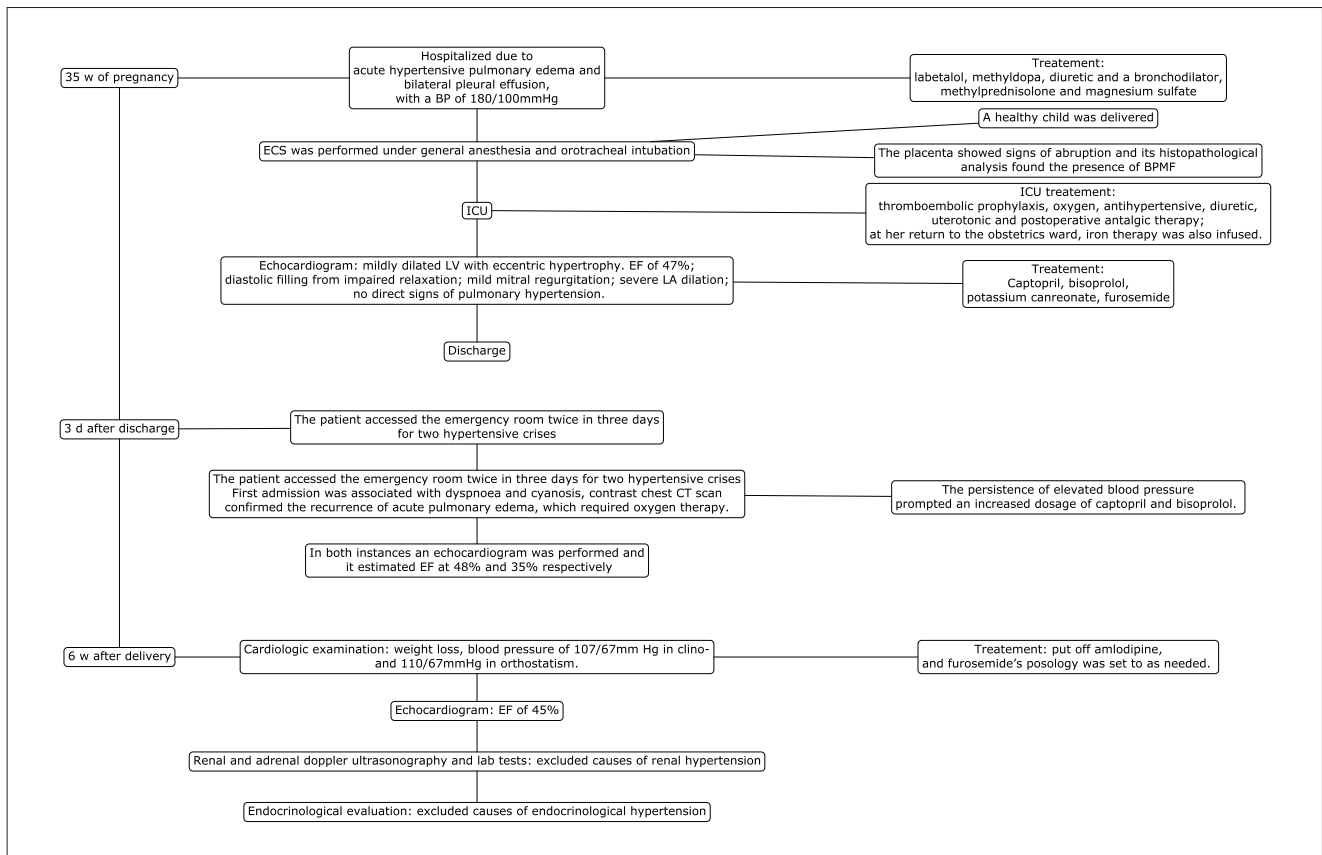
mild peripheral oedema persisted. Three days after discharge, the patient accessed the emergency room twice in three days for two hypertensive crises. The first episode was once more associated with dyspnoea and cyanosis, and a chest CT scan confirmed the recurrence of acute pulmonary oedema, which required oxygen therapy. In both instances an echocardiogram was performed and it estimated her ejection fraction at 48% and 35% respectively, in addition to confirming the morphological features already observed.

A week later an echocardiogram was repeated, estimating an ejection fraction of 45%. After further cardiologic consultation, olmesartan (20 mg/day) and amlodipine (5 mg/day) were added to the patient’s treatment plan. At the cardiologic examination scheduled six weeks after delivery, the cardiopulmonary physical examination findings appeared within range. She was therefore put off amlodipine, and furosemide’s posology was set to as needed. After an additional echocardiogram, the patient’s clinical picture was deemed ostensibly ascribable to a dilated cardiopathy secondary to hypertension with mild left ventricular dysfunction (left ventricular ejection fraction [LVEF]: 45%).

Causes of secondary hypertension were inquired by means of renal and adrenal Doppler ultrasonography and urinary metanephrine levels — and subsequently excluded. Aldosteronism and hypokalaemia were also ruled out upon endocrinological evaluation, as well as any other sources of secondary hypertension. A flow chart representing the subsequent stages of the patient’s care pathway is shown in Figure 1.

**Figure 1** Histogram with distribution of appendicular skeleton mass for both groups.

Abbreviations: BP, blood pressure; ECS, emergent cesarean section; ICU, intensive care unit; w, weeks; d, days; EF, ejection fraction.



## Discussion

The management of PPCM, as well as its diagnostic criteria, are controversial. It is well known that it is a rare condition, which may be under-diagnosed<sup>[1]</sup> and on which few data are available. The patient profiled in this document received a tentative diagnosis of PPCM – although guidelines criteria state that hypertensive crises and preeclampsia ought to rule it out – because her condition worsened in a short period of time, and such event was followed by evidence of reduced ventricular ejection fraction. Moreover, the patient reported no personal or family history of prior heart disease and other causes of heart failure. It is also difficult to ascertain whether her echocardiographic findings – hypertrophic cardiomyopathy, systolic dysfunction, and severe left atrial dilation – are more ascribable to an ostensible hypertension pre-existent to pregnancy rather than related to the preeclamptic syndrome.

The ESC 2018 Guidelines for the management of cardiovascular diseases during pregnancy establish that a LVEF lower than 45%, with or without peripartum left ventricular cavity dilation, is indicative of PPCM<sup>[1]</sup>. Indeed, our patient had an estimated LVEF of 47% followed by a finding of 35%, to then regain her function at 47% a month later. We postulate these changes were related to the patient's composite condition of hypertensive cardiomyopathy and PPCM, because of the recurrence of acute pulmonary edema after delivery and despite treating the first. In other words, it is unclear whether her hypertensive issues are consequential to, or causative of her reduced ejection fraction.

Another significant factor associated with PPCM is obesity – which our patient was affected by before and during her pregnancy – as Putra *et al.*<sup>[2]</sup> have demonstrated in their meta-analysis.<sup>2</sup> Furthermore, a meta-analysis from Bello and co-authors<sup>[3]</sup> demonstrates how preeclampsia and hypertensive issues – which both affected our patient – are included as important risk factors for the development of PPCM. Indeed, women with PPCM have a significantly higher incidence of preeclampsia compared to the general population. Their findings support the hypothesis that preeclampsia and PPCM have a shared pathogenesis and they stress the importance of being cognizant of their overlap<sup>[3]</sup>.

Patterns of left ventricular remodeling and recovery of its function have been described to be distinctly different in PPCM patients with preeclampsia when compared to PPCM patients without it. Moreover, the conjunction of PPCM with preeclampsia is associated with higher morbidity and mortality in the short term but it is a more favorable condition in the long-term<sup>[3]</sup>.

Regarding our patient's treatment, bromocriptine was not added because of the uncertain diagnosis. Bromocriptine is recommended in PPCM, because it leads to considerably greater survival and left ventricular ejection fraction improvement [4]. The treatment used in our patient was based on the ESC guidelines for heart failure. Although it is true that guidelines should be followed to provide an adequate therapy for patients, the lack of women recruited into heart failure clinical trials (25% of cohorts) and the fact that treatment guidelines are mostly based on data gathered from male subjects underlie the present sex disparity in the needs of patients affected by heart failure. Optimal pharmacological dosages for women, sex-specific device treatment parameters, and sex-specific processes all have

significant information gaps<sup>[5]</sup>. A minimum of one year has also been thought to be a fair length of time for medical therapy, even if the ideal time frame for PPCM patients with regained LVEF is still unknown<sup>[6]</sup>. Secondly, the risk of recurrent hospitalization or mortality in individuals with previous diagnosis of PPCM associated with preeclampsia remains significant after full normalization of LVEF<sup>[7]</sup>.

## Conclusions

ESC guidelines define PPCM as heart failure secondary to left ventricular systolic dysfunction with a LVEF <45%, which occurs towards the end of pregnancy or in the months following delivery, with no other identifiable causes of heart failure. This last clause makes it a diagnosis of exclusion, which is indicative of two issues: firstly, how little is known about PPCM and how identifying connotative features which would distinguish PPCM from other cardiomyopathies would be an instrumental line of inquiry. Secondly, the fact that PPCM is overall underdiagnosed because diagnostic criteria do not conceive the concomitance of PPCM with other causes of heart failure, or because there is overlap with conditions such as e.g. preeclampsia, obesity, etc, in a potentially syndromic scenario, even though new evidence has been published since guidelines were last updated in 2018. Ultimately, a re-evaluation of guidelines is warranted.

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### Declaration of interest

The authors declare having no conflicts of interest regarding the publication of this case report.

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### Patient consent

Consent for the publication of this case was obtained from the patient.