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CASE REPORT
Heart failure

Catastrophic presentation of peripartum cardiomyopathy: a case report of a challenging diagnosis

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Background Peripartum cardiomyopathy (PPCM) usually affects women in the last month of pregnancy or in the first months following delivery and typically presents with signs and symptoms of heart failure (HF), although catastrophic presentations may be the initial manifestation.

Case summary A 36-year-old woman reported intense fatigue that began after delivery. Three months following delivery, she suffered a cardiac arrest at home, in shockable rhythm, with a total estimated time of 70 min before return of spontaneous circulation. Electrocardiogram presented diffuse ST-segment depression and transthoracic echocardiography showed severe left-ventricle (LV) dysfunction with global hypokinesis. In the catheterization laboratory, she underwent a triple rule out (no aortic, coronary, or pulmonary vascular disease); ventriculography was not suggestive of Takotsubo syndrome. She was transferred to the cardiac intensive care unit, under invasive mechanical ventilation and vasopressor support. A presumptive diagnosis of PPCM was made and bromocriptine and heparin were started. In three days, she was weaned from haemodynamic support and extubated, with good neurologic outcome. Cardiac magnetic resonance showed no signs of inflammation or fibrosis. Cardiomyopathy genetic test was negative. PPCM diagnosis was assumed, HF therapy was introduced and a cardioverter-defibrillator was implanted. At 2.5 months follow up, she presented HF NYHA Class II and recovered LV function.

Discussion We present a case of a woman, three months after delivery, who developed a catastrophic manifestation of PPCM. This case raises awareness about atypical presentations of PPCM, whose diagnosis should be considered in the appropriate clinical context, but ultimately, remains a diagnosis of exclusion.

Keywords Peripartum cardiomyopathy • Cardiac arrest • Cardiogenic shock • Acute heart failure • Breastfeeding • Case report

ESC Curriculum 6.4 Acute heart failure • 6.5 Cardiomyopathy • 7.2 Post-cardiac arrest • 7.3 Critically ill cardiac patient • 9.8 Pregnancy with cardiac symptoms or disease

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Learning points

- Peripartum cardiomyopathy usually presents as heart failure with left-ventricle systolic dysfunction with a left-ventricle ejection fraction <45%, not explained by other causes of heart failure, occurring in the last month of pregnancy or in the first months following delivery.
- Peripartum cardiomyopathy conventionally presents with signs and symptoms of heart failure, although catastrophic manifestations such as life-threatening arrhythmias or cardiac arrest may occur.
- An interdisciplinary team is of paramount importance to guide the patient during the post-partum period, including counselling regarding breastfeeding, contraception, and subsequent pregnancies.

Introduction

Peripartum cardiomyopathy (PPCM) is characterized by heart failure (HF) secondary to left-ventricular (LV) systolic dysfunction with an LV ejection fraction (LVEF) <45%, occurring towards the end of pregnancy or in the first months following delivery, and in the absence of other causes of HF.[1,2]

Timeline

Time	Events
3 months prior hospitalization	Delivery Intense fatigue in the post-partum period
Hospitalization	
Day 1	Cardiac arrest in a shockable rhythm 70 min until return of spontaneous circulation Invasive mechanical ventilation and vasopressor support Echocardiogram: dilated left ventricle with global hypokinesis, severe left-ventricle systolic dysfunction, and right ventricle dysfunction Coronary angiogram: normal coronary arteries; additionally, no aortic dissection or acute pulmonary embolism (PE) Ventriculography: no apical ballooning (not suggestive of Takotsubo syndrome) Admitted on cardiac intensive care unit with cardiogenic shock Targeted temperature management Presumed diagnosis: Peripartum cardiomyopathy Bromocriptine 2.5 mg b.i.d. and perfusion of unfractionated heparin
Day 3	Favourable clinical, neurological and haemodynamic evolution Invasive mechanical ventilation and noradrenaline withdrawal
Day 7	Cardiac magnetic resonance: no signs of inflammation or myocardial fibrosis
Day 14	Subcutaneous implantable cardioverter-defibrillator (S-ICD)
Day 15	Discharged with an LVEF of 34% and without major neurological deficits Heart failure optimal medical therapy Bromocriptine 2.5 mg od and low-molecular-weight heparin for 6 weeks Stop breastfeeding and oral contraception containing oestrogens Reconsider subsequent pregnancies Referred for HF, gynaecology and psychology consultation, and cardiac rehabilitation programme
Follow-up	
2.5 months after discharge	NYHA Class II Echocardiogram: normal cardiac chambers size, LVEF of 50% and normal right-ventricle function
1 year after discharge	Asymptomatic, good functional status, returned to work No rehospitalizations

The pathophysiology of PPCM remains unclear, but current evidence points to a major detrimental role of vascular dysfunction, particularly due to systemic angiogenic imbalance, genetic and physiological predisposition.[3,4]

Peripartum cardiomyopathy usually presents with signs and symptoms of HF, although cardiogenic shock, ventricular arrhythmias, and sudden cardiac death may also occur.[1,2] Due to the diverse nature in the aetiology of *de novo* cardiomyopathies associated with pregnancy, PPCM is a diagnosis of exclusion.

Furthermore, although cardiac function is often recovered in PPCM, due to delayed and difficult diagnosis, PPCM has significant morbidity and mortality.[1,2]

Below, we report a case of a 36-year-old woman with a catastrophic presentation of PPCM, 3 months post partum.

Case summary

We present a case of a 36-year-old Caucasian woman who had a history of smoking and had *pectus excavatum*. Three years before hospital admission, she presented three episodes of syncope, of undiagnosed aetiology despite exhaustive study, which included an electrocardiogram (ECG), 24 h Holter, transthoracic echocardiography (TTE), thoracic and cranial computed tomography, and electroencephalogram. There was no reported family history of sudden cardiac death or cardiovascular disease. Her only regular prescribed medication was trazodone. She had two children, one from a recent pregnancy, with both pregnancies uneventful.

She suffered a cardiac arrest (CA) at home, 3 months post partum. Prior to this event, she had complained of post-partum fatigue, without any clear inciting event, such as previous infection or stressful event. The first documented rhythm during CA was a shockable rhythm, and she received advanced life support for 50 min, with the assistance of a mechanical chest compressor (total CA duration of 70 min), until return of spontaneous circulation.

Subsequently, she was admitted in the emergency room under invasive mechanical ventilation and noradrenaline perfusion.

On physical examination, she presented no changes in cardiac or pulmonary auscultation (no cardiac murmurs or pulmonary crepitations), nor peripheral oedema; capillary refill time was prolonged. Her body mass index was normal (22.6 kg/m^2).

The 12-lead ECG showed sinus rhythm, poor anteroseptal R-wave progression, diffuse ST-segment depression, and normal QTc (*Figure 1*). TTE revealed dilated LV with global hypokinesis, severe LV systolic dysfunction, and right ventricle (RV) dysfunction; no significant valvular heart disease (see Supplementary material online, *Video S1*). Arterial blood gases showed severe metabolic acidosis (pH = 6.96) with hyperlactataemia [14 mmol/L, normal range (NR) 0.5–2 mmol/L]. Blood analysis demonstrated mild leucocytosis with neutrophilia, normal C-reactive protein, normal electrolytes, and kidney function (potassium of 3.78 mmol/L, NR 3.5–5 mmol/L; sodium of 137 mmol/L, NR 136–145 mmol/L; magnesium of 1.78 mEq/L, NR 1.3–2.1 mEq/L; ionized calcium of 1.24 mmol/L, NR 1.20–1.40 mmol/L; and creatinine of 0.85 mg/dL, NR 0.51–0.95 mg/dL), mild-elevation of high-sensitivity troponin T (90 ng/L, NR 5–14 ng/L) and myoglobin (862 ng/mL, NR 28–58 ng/mL), and normal NT-pro-BNP (95 pg/mL, NR 0–125 pg/mL).

In the catheterization laboratory, a triple rule out was performed to exclude aortic dissection/regurgitation, coronary artery disease, or acute PE; ventriculography was not suggestive of Takotsubo syndrome (TTS; see Supplementary material online, *Video S2*). She was transferred to the cardiac intensive care unit under invasive mechanical ventilation, noradrenaline perfusion, and with targeted temperature management initiated.

In this clinical context, with other causes of cardiomyopathy unlikely, and because the diagnosis of PPCM was highly probable, we started bromocriptine 2.5 mg bid and perfusion of unfractionated heparin (1000 units/h).

The patient presented favourable clinical, neurological and haemodynamic evolution, allowing withdrawal of invasive mechanical ventilation and noradrenaline at 72 h. At Day 7 post admission, cardiac magnetic resonance (CMR; *Figure 2*) revealed a dilated LV with global LV hypokinesis, an LVEF of 31%, RV dysfunction, and no signs of inflammation or myocardial fibrosis. During hospitalization, serial 12-lead ECGs showed sinus rhythm, poor anteroseptal R-wave progression, and a normal ST-segment. Genetic tests for dilated cardiomyopathy, LV non-compaction cardiomyopathy and channelopathies were negative. After a thorough discussion within the Heart Team, an S-ICD was implanted.

During hospital stay, she had psychological and psychiatric support, gynaecological counselling, and completed Phase I of the cardiac rehabilitation programme.

At Day 15 post admission, she was discharged, with an LVEF of 34% (see Supplementary material online, *Video S1*) and without major neurological deficits. HF therapy at discharge included ramipril 2.5 mg o.d., bisoprolol 2.5 mg o.d., and spironolactone 50 mg o.d. In addition, bromocriptine 2.5 mg o.d. and low-molecular-weight heparin (enoxaparin 60 mg b.i.d.) were prescribed for 6 weeks. Suspension of both breastfeeding and oral contraception containing oestrogens were recommended, along with careful consideration to subsequent pregnancies. She was referred to the HF clinic, gynaecology and psychology consultation, and for continuation of cardiac rehabilitation programme.

At 2.5 months follow-up, she presented HF NYHA Class II and TTE showed normal cardiac chamber size, with significant improvement of LVEF (50%) and normal RV function (see Supplementary material online, *Video 3*). Cardiac rehabilitation programme was followed for 3 months, with an average of 2–3 weekly sessions, and consisted of the combination of an aerobic exercise programme on a treadmill, with the cycle ergometer and Glittre-ADL test (TGlittre) and muscle strength training. There was a significant improvement in functional

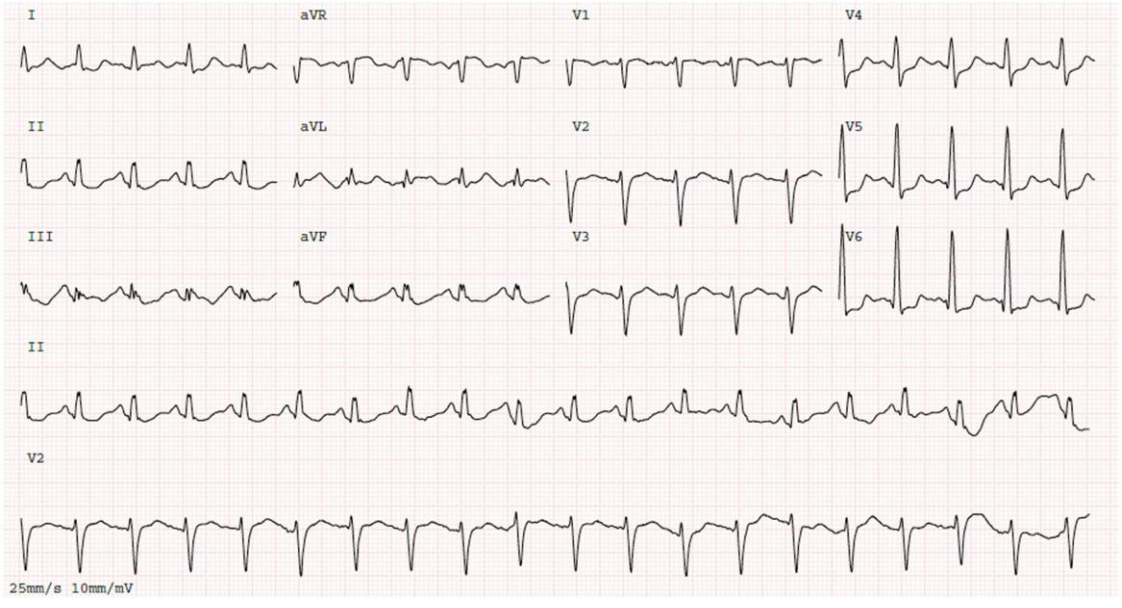

Figure 1 Twelve-lead electrocardiogram at the emergency department showed sinus rhythm, poor anteroseptal R-wave progression, diffuse ST-segment depression, and normal QTc interval.

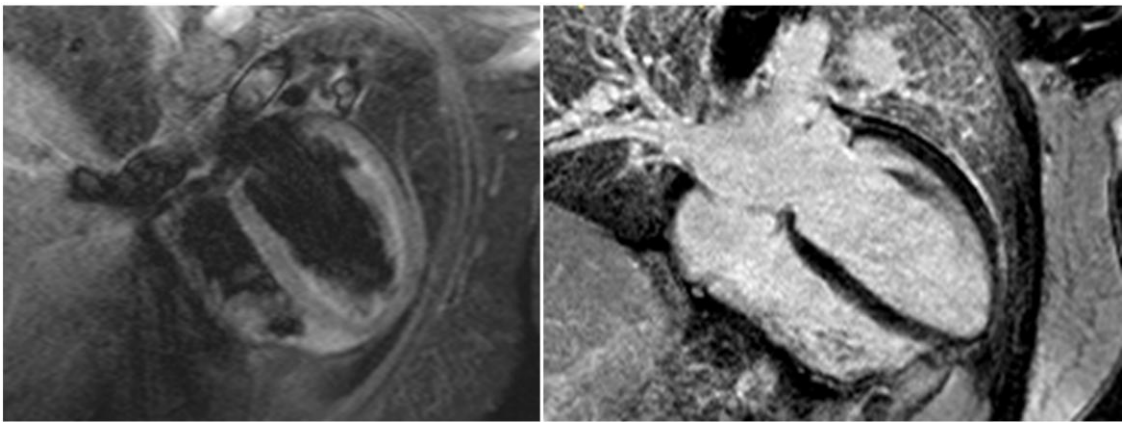

Figure 2 Cardiac magnetic resonance showed no signs of inflammation or myocardial fibrosis. Four-chamber T_2-weighted image (STIR) (left) and four-chamber late gadolinium enhancement (right).

capacity and muscle strength, which translated into an improvement in peak oxygen consumption and exercise tolerance in the cardiopulmonary exercise test. At 1-year follow up, she remained asymptomatic, with good functional status, had returned to work, and had no rehospitalizations. TTE showed normal cardiac chamber size with an LVEF of 50%.

Discussion

We presented a case of a 36-year-old woman, admitted with acute LV dysfunction, without an identifiable cause, 3 months post partum.

In a young woman in the peripartum period with *de novo* LV systolic dysfunction, PPCM should always be considered. According to the 2019 position statement from the HF Association of the European Society of Cardiology Study Group on PPCM,[1] this condition was defined as HF secondary to LV systolic dysfunction with an LVEF <45%, occurring towards the end of pregnancy or in the months following delivery (mostly in the first month), with no other identifiable cause of HF.

In this clinical case, there was no known pre-existing heart disease; thus, all attempts were made to establish a definitive diagnosis of acute LV systolic dysfunction. Predisposing factors[5,6] for PPCM, which include multiple pregnancies, smoking habits, and advanced maternal age (>35 years old), were present in this case. Moreover, while the majority of patients with PPCM present with HF in the early post-partum period,[1] in this case, there was an acute presentation with life-threatening arrhythmias and CA, 3 months post partum. Previous definitions of PPCM included disease presentation up to 5 months post partum.[7,8] There is also an overlap between normal peripartum and HF symptoms, which makes the diagnosis challenging. In this case, the patient had reported post-partum fatigue, and therefore, we cannot exclude the possibility of an initial subacute presentation of PPCM or a first manifestation of a dilated cardiomyopathy (DCM) triggered by the peripartum, which remained undiagnosed and that culminated in a life-threatening CA.

There are no gold-standard criteria that confirm PPCM diagnosis. Nonspecific findings such as the elevation of cardiac troponin or natriuretic peptides may be present in multiple pathologies. In our case, natriuretic peptides levels were normal upon admission, which could be explained by the sudden presentation with CA in the absence of previous known HF. On the other hand, and considering the hypothesis of a subacute presentation of PPCM or a first manifestation of a DCM, the normal levels of natriuretic peptides could be because the patient was already showing recovery of her HF by the time she was assessed. Regardless, the majority of women with PPCM have consistently elevated natriuretic peptides.[1] NT-pro-BNP is a classical, yet unspecific biomarker for HF, it is not suited to differentiate between PPCM and other causes of HF, although it has the potential of being a marker for diagnosis and disease monitoring.[9–11] Also, NT-pro-BNP has a prognostic value in predicting LV recovery of patients with PPCM,[11] with higher baseline levels being predictive of a poor outcome.[12] There was a mild increase in cardiac troponin, representative of acute myocardial injury, caused by the oxygen supply/demand imbalance consequent of the CA. CMR may provide a more accurate evaluation of cardiac structure and function, and despite the absence of a specific PPCM scar pattern, it may be a useful tool for exclusion of other diseases such as myocarditis or TTS which may have a substantial phenotypic overlap with PPCM.[13] Additionally, the presence of late gadolinium enhancement in PPCM was linked to a poor outcome.[14] However, even with the use of CMR, PPCM still remains a diagnosis of exclusion and other causes of LV dysfunction had to be ruled out for a more accurate diagnosis.

Other differential diagnosis such as acute myocardial infarction, aortic dissection, PE, acute myocarditis, and channelopathies became less likely. TTS was considered a major differential diagnosis, but most findings were not compatible with a typical TTS[15,16]: no emotional or physical trigger was found, ECGs did not show diffuse T-wave inversion or prolonged QTc; TTE, ventriculography, and CMR showed a severe LV systolic dysfunction with global hypokinesis, without classical apical ballooning and there was no significant increase in natriuretic peptides upon admission. Furthermore, LV systolic recovery was slower than usual for TTS. On top of it, an InterTAK Diagnostic Score[16] of 25 points made TTS diagnosis even less likely. Nevertheless, we must point out that rare variants of TTS cannot be completely excluded, with a recent history of delivery as a confounding factor.[17]

The management of PPCM patients should include an integrative approach and an interdisciplinary team to guide HF therapy and the post-partum period, including counselling regarding contraception and subsequent pregnancies.[1,18] Optimal medical therapy for HF with reduced LVEF was initiated, although SGLT2 inhibitors were not given as the new HF guidelines[19] supporting their use had not yet been published at the time of this case. Considering the evolution in cardiogenic shock, bromocriptine was also given during the hospital stay and prolonged for another 6 weeks, in combination with anticoagulation (current recommendations suggest that bromocriptine treatment must always be accompanied by anticoagulation with heparin, at least in prophylactic dosages[1,18]).

Given the usual improvement of LV function during HF therapy, early implantation of an ICD is generally not advisable in PPCM patients.[1] Although we hypothesized that PPCM was the most plausible diagnosis, as we stated before, this is a diagnosis of exclusion and there are no unequivocal criteria that could confirm it. The possibility of a first manifestation of a familial DCM triggered by the peripartum presenting with CA was taken into consideration and after careful reflection by the Heart Team, an S-ICD was implanted. In this case, the use of a wearable cardioverter-defibrillator could have been beneficial, until the achievement of a definitive diagnosis or in order to allow more time for a final decision regarding ICD implantation.[1]

We also recommended the patient to suspend breastfeeding. According to the 2018 ESC guidelines[18] for the management of cardiovascular diseases during pregnancy, prevention of lactation might be considered in patients with severe HF due to high metabolic demands of lactation and breastfeeding (Class IIb recommendation), allowing a safe treatment with all established HF drugs.

Long-term outcomes in PPCM vary widely among studies[20,21] and even after full recovery of LVEF, subtle diastolic dysfunction and reduced maximal exercise capacity were observed.[22] TTE at 1-year follow-up showed an LVEF of 50%. However, even though there was a significant improvement in LVEF, we decided to continue with HF therapy to prevent relapse following treatment withdrawal.[23]

Conclusion

This case highlights a catastrophic presentation of PPCM, its challenging diagnosis, particularly because the lack of specific findings to confirm PPCM. Ultimately, PPCM remains a diagnosis of exclusion and a multidisciplinary approach is of paramount importance.

Lead author biography

Mariana Ribeiro Silva, born on 6 March 1992, is a Cardiology resident at Cardiology Department of Centro Hospitalar de Vila Nova de Gaia/Espinho, Portugal. She did her integrated master's in medicine in Faculdade de Medicina da Universidade do Porto, Portugal. In addition to her daily clinical activities, her main area of interest focuses on Acute Heart Failure and Cardiogenic Shock, with particular interest in Cardiac Intensive Care.

Supplementary material

Supplementary material is available at *European Heart Journal – Case Reports* online.

Slide sets: A fully edited slide set detailing this case and suitable for local presentation is available online as Supplementary data.

Consent: The authors confirm that written consent for submission and publication of this case report including the images and associated text have been obtained from the patient in line with COPE guidance.

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