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CASE REPORT
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Successful Management of Peripartum Cardiomyopathy in a 29-year-old Woman

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ABSTRACT

Introduction: Peripartum Cardiomyopathy (PPCM) is defined as an idiopathic cardiomyopathy due to left ventricle (LV) systolic dysfunction without any other cause of heart failure. It occurs in the last month of pregnancy or within 5 months after delivery. The diagnosis is commonly delayed due to resemblance of symptoms with the normal pregnancy or postpartum condition. Here, we highlight the importance of recognizing the early symptoms of PPCM.
Case Presentation: A 29-year-old woman was admitted to emergency department with manifestation of heart failure that had lasted for 2 days. There was a history of delivery about 40 days ago. Physical examination showed sign of shock, regular heart rhythm, rhonchi in bilateral basal of lung, mild hepatomegaly, and generalized edema. Laboratory result demonstrated elevation of liver transaminase levels. Chest X-Ray had feature of cardiomegaly while echocardiography finding showed reduction of LV systolic function, global hypokinetic, dilatation of LV, and minimal pericardial effusion. The patient was managed with optimal preload reduction. Afterwards, aldosterone antagonist, anticoagulant, and bromocriptine were given immediately. Clinical symptoms improved and the patient was discharged on the 4th day of hospitalization with the planning of echocardiography re-evaluation regularly.
Discussion: Common misdiagnosed or delayed PPCM would be potentially lethal for mother and the baby. Hence, the importance of high clinical suspicion regarding PPCM symptoms must be highlighted for prompt treatment. BOARD (Bromocriptine, Oral heart failure drugs, Anticoagulation, Relaxant, and Diuretic) regiment is considered to be a promising therapy in PPCM.
Conclusion: Early recognition and management should be addressed properly to achieve favorable outcome.

INTRODUCTION

Peripartum cardiomyopathy (PPCM) is a rare yet life threatening disease identified by left ventricular systolic dysfunction (Douglass & Blauwet, 2021). It affected thousands of women with 1 per 3000-1 per 4000 live births each year. The recognizable risk factors include maternal age >30 years old, black race, multigestational pregnancy, multiparity, pre-eclampsia, gestational hypertension, history of smoking, and malnutrition. In spite of high mortality rate, this disease is commonly undetected due to the similarity of the symptoms with normal state of peripartum period or pregnancy related diseases such as pulmonary emboli and eclampsia or hemolysis,

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elevated liver enzymes, and low platelets (HELLP) syndrome. (Arany & Elkayam, 2016; Dash et al., 2022; Honigberg & Givertz, 2019; Wang, 2009).

Globally, Nigeria has the highest PPCM incidence reported with average of 1 per 100 live births, followed by Haiti (1 per 300 live births), Pakistan (1 per 840), and South Africa (1 per 1000) (Hoes et al., 2022; Honigberg & Givertz, 2019).

Based on a study, the increasing incidence of PPCM was equal to the age increment with 1 per 1200 live births among people aged 20-29 years, 1 per 790 live births among those aged 30-39 years, and 1 per 270 live births among those aged 40-54 years (Honigberg & Givertz, 2019). Genetic also plays an important role of PPCM development with cardiac myosin heavy chain (MYH) along with titin (TTN) and sodium channel gene 5 (SCN5) as the most impacted ones (Dash et al., 2022). There are two forms associated with PPCM etiology, the primary one occurs with unknown specific cause, whereas the secondary form occurs due to preexisting systemic disease, such as autoimmune disorder or alcohol consumption. Other causes may include abundance prolactin excretion, selenium deficiencies, zinc deficiency, prolonged use of tocolysis, viral myocarditis, stress-activated cytokines, abnormal response to pregnancy, and failure of response to hemodynamic stress of pregnancy, genetic predisposition, prolactin, and placental angiogenic factors (Honigberg & Givertz, 2019; Okeke et al., 2013). Other associated risk factors are anaemia, asthma, diabetes mellitus, obesity, and substance abuse (Arany & Elkayam, 2016).

Here, we report a rare case of successfully treated PPCM by multidisciplinary team. The purpose of this case report is to highlight the importance of early diagnosis and management considering the atypical symptoms of the disease. It apparently is a major public health problem due to high risk of recurrence in further pregnancies which can be a life changing point for young primiparous women. This article also provides the detail explanation of PPCM pathogenesis, diagnosis, and therapy in order that delayed diagnosis could be avoided which consequently reducing the PPCM morbidity and mortality rate.

CASE PRESENTATION

A 29-year-old woman was admitted to the emergency department with presentation shortness of breath that lasted for 2 days and aggravated 2 hours before admission. The patient had previous history of caesarean section 40 days ago. This was the first pregnancy, single gestational status, and without any history of abortion. Upon admission, the patient also complained additional symptoms such as palpitation, cold sweat, fatigue, epigastric pain, abdominal discomfort, bloated, restlessness, loss of appetite, history of vomitus and diarrhea 2 days before. There were no any symptoms of chest pain nor unconsciousness. History of diabetes mellitus, hypertension, heart disease, asthma and other comorbidities were also denied. The patient was obese and physical examination showed compos-mentis, Glasgow Coma Scale (GCS) was 15, respiration rate (RR) was 28 times per minute (tpm), pulse rate of 133 beats per minute, blood pressure (BP) of 70/50 mmHg, temperature (T) of 36 °C, and oxygen saturation (SpO2) of 95%. Jugular venous pressure (JVP) was R + 5 cmH20. Apex cordis was palpable at intercostal (ICS) VI midclavicular (MCL) sinistra and systolic murmur grade IV/VI was detected at the apex. There was also rales in the bilateral basal of the lung and increased bowel sound but no sign of dehydration was detected. Pallorness, mild hepatomegaly, ascites, and generalized edema were also discovered. An electrocardiogram (ECG) was performed and it revealed sinus tachycardia along with poor R wave progression along and low QRS voltage as seen in Figure 1 a. Afterwards, laboratory analysis, chest x-ray, and echocardiography were also performed.

Table 1. Laboratory Result

Laboratory Test	Result	Normal Value
Complete Blood Count		
Hemoglobin (g/dL)	11.7	13.0-17.0
Eryhtrocyte (10^6/uL)	4.06	4.50-5.50
Hematocrite (%)	34.2	40-50
MCV (fL)	84.2	80-96
MCH (pg)	28.8	27-32
MCHC (g/dL)	34.2	33-36
Leucocyte (10^3/uL)	7.7	4.0-10.0
Thrombocyte (10^3/uL)	334	150-400
Chemical Test		
Random Blood Glucose (mg/dL)	105	70-160
Ureum (mg/dL)	28	10-59
Creatinine (mg/dL)	0.7	<1.17
Electrolyte		
Natrium (mmol/L)	127	135-148
Kalium (mmol/L)	4.1	3.5-5.3
Chloride (mmol/L)	105	98-107
SGOT (U/L)	549	<35
SGPT (U/L)	386	<45
Albumin (g/dL)	3.7	3.4-4.8
Urine Analysis		
Macroscopic		
Color	Yellow	Yellow
Clarity	Hazy	Clear
Specify Gravity	1.030	1.000-1.030
Erythrocytes (RBC/uL)	Negative	Negative
Leucocyte Esterase (WBC/uL)	Negative	Negative
PH	5.5	5.0-8.0
Protein (mg/dL)	Positive (++)	Negative
Glucose (mg/dL)	Negative	Negative
Keton urine (mg/dL)	Positive (+)	Negative
Bilirubin	Negative	Negative
Nitrit	Positive	Negative
Microscopic		
Leucocytes (per hpf)	0-2	0-5
Erithrocytes (per hpf)	0-2	0-3
Epithel (per hpf)	3-5	0-1

Laboratory result demonstrated a decreased value of hemoglobin, hematocrit, and erythrocyte. Hyponatremia was detected but kalium, ureum, creatinine, and albumin were in the normal levels. Liver function test showed significant increase of serum glutamic oxaloacetic transaminase (SGOT) and serum glutamic-pyruvic transaminase (SGPT). Urinalysis findings showed the presence of positive protein and nitrite (Table 1).

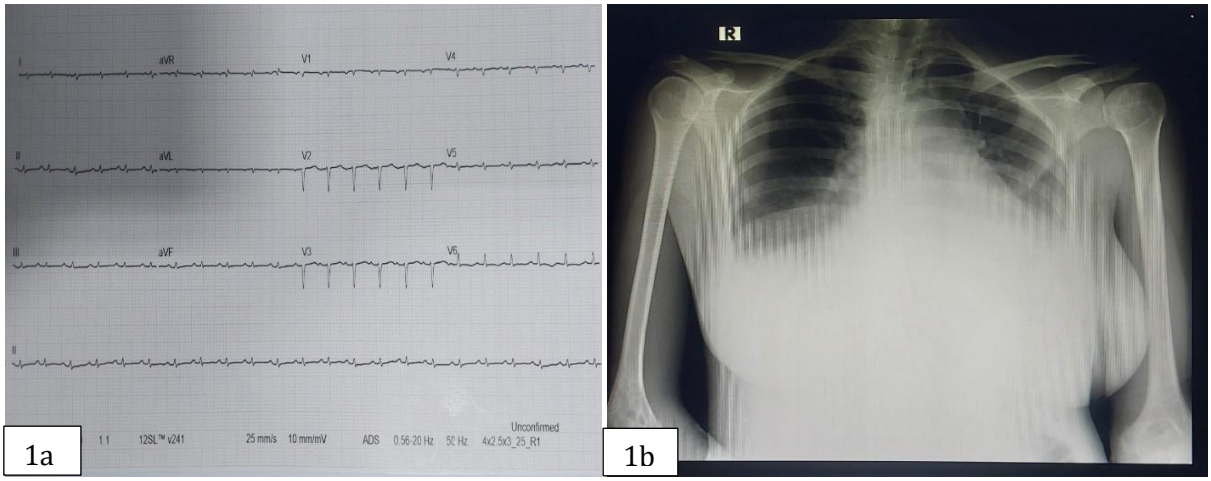

Figure 1. (a) Electrocardiography (b) Chest X ray

Chest X-Ray revealed the feature of cardiomegaly and pulmonal congestive (Fig. 1b). Urology ultrasonography (USG) was also performed, revealing significant ascites with normal result of urinary tract structure and functions (Fig. 2a, 2b).

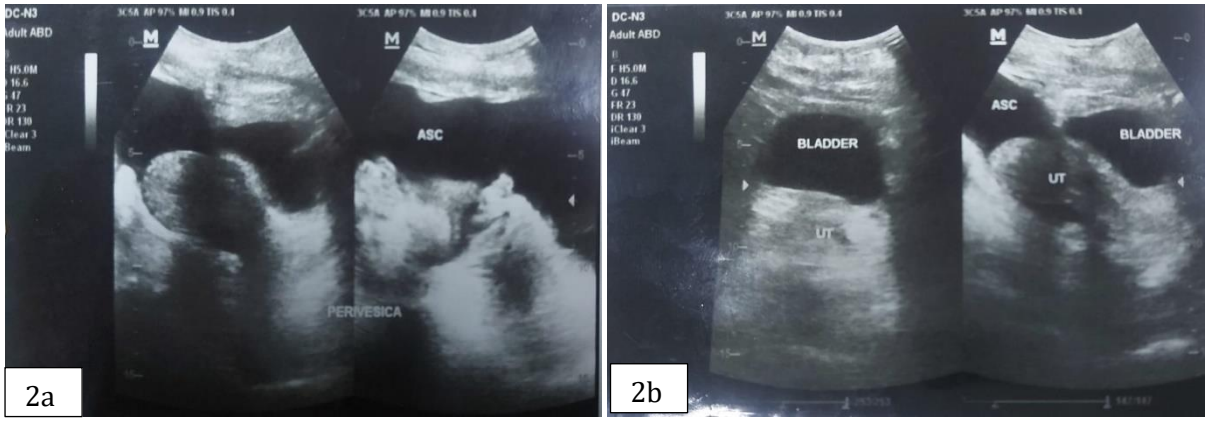

Figure 2. Urology USG (a) ascites (b) ascites with normal bladder structure

The reduction of left ventricle (LV) systolic function with ejection fraction (EF) of 14.49% fractional shortening (FS) of 6.41%% along with mild mitral regurgitation were observed on the echocardiography findings. Eccentric left ventricle hypertrophy and dilatation of LV were also present. Diastolic LV function was pseudo-normal with end-diastolic volume of LV was 86.20 ml and left ventricular internal end diastolic diameter was 43.7 mm. Global hypokinetic and minimal pericardial effusion with diameter of 6-13 mm surrounding the heart were also found (Fig. 3a, 3b).

In the emergency department, the patient was initially treated with fluid management of Ringer Lactate 500 cc (extra), Intravenous (IV) ceftriaxone 1 gr per 12 hours, IV Furosemide 40 mg per 12 hours after vital sign was stabilized, IV pantoprazole 40 mg per 24 hours, and IV metoclopramide 10 mg per 8 hours. Then, the patient was admitted to Intensive Care Unit (ICU) and treated with optimized heart failure therapy. In addition to previous treatments, IV mecobalamin 500 mcg per 12 hours, sucralfate syrup CII t.i.d, mineralocorticoid receptor antagonist (MRA) agent spironolactone 50 mg q.d., potassium chloride KSR 600 mg b.i.d were administered during ICU treatment. Then, on the 2nd day of admission and after the improvement of congestion, digoxin 0.25 mg q.d., rivaroxaban 10 mg q.d. for 7 days, bromocriptine 2.5 mg q.d.

for 7 days, atorvastatin 20 mg q.d.,1 tablet of liverprime t.i.d and 1 tablet of curcuma t.i.d were also administered. The patient was shifted out of ICU in a stable condition after 48 hours since being admitted to the emergency department. In general ward, furosemide IV is tapered to 20 mg per 12 hours while other drugs still continued. Ultimately, the patient was considered in a good condition, can be treated as an outpatient, and discharged on the 4th day of hospitalization with routine consume of medication and the planning of echocardiography re-evaluation regularly. During discharge, furosemide 40 mg q.d, spironolactone 25 mg q.d, rivaroxaban 10 mg q.d, digoxin 0.25 mg q.d, bromocriptine 2.5 mg q.d, 1 tablet of curcuma t.i.d, and 1 tablet of liverprime t.i.d. were prescribed.

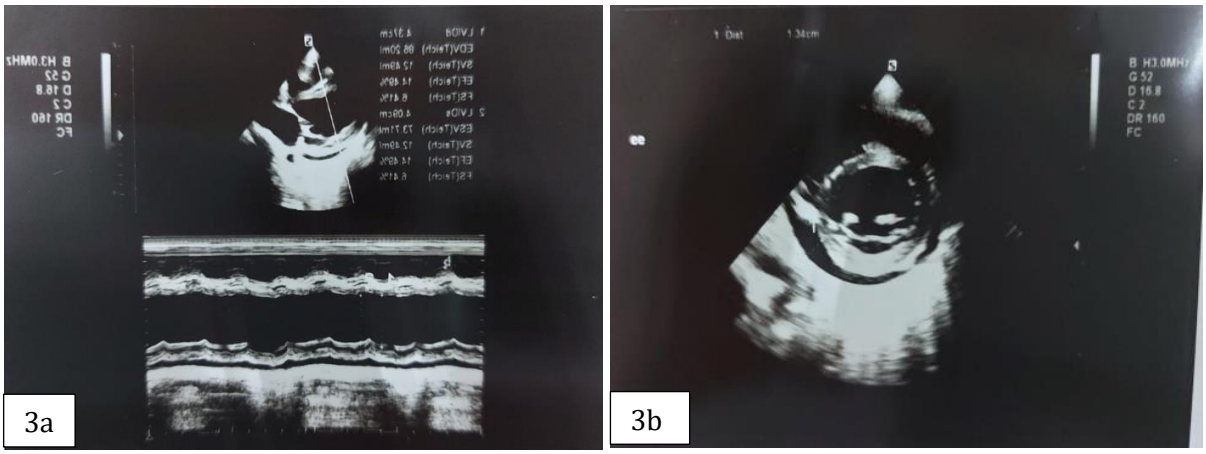

Figure 3. Echocardiography (a) reduced LV systolic function (b) minimal pericardial effusion

On the 2nd day of admission, the patient underwent laboratory test for the evaluation of therapy. Natrium level increased to normal while SGOT and SGPT levels decreased gradually on the 3rd day of admission and returned to normal levels on 12th day of follow-up (Table 2).

Table 2. Follow-up Laboratory Test Result

Laboratory Test	Follow-up Laboratory Result			Normal Value
	2nd day	3rd day	12th day	
Electrolyte				
Natrium (mmol/L)	140	-	-	135-148
Kalium (mmol/L)	3.6	-	-	3.5-5.3
Chloride (mmol/L)	101	-	-	98-107
Liver Function Test				
SGOT (U/L)	-	139	30	<35
SGPT (U/L)	-	215	61	<45
Bilirubin Total (mg/dL)	-	-	0.8	<1.1
Bilirubin Direct (mg/dL)	-	-	0.44	<0.3

At 2nd month of follow up, EF improved significantly reaching 51% with global normokinetic, normal RV systolic function, and pseudonormal LV diastolic function. Dysfunction of valves were not detetced (mitral, tricuspid, aorta, and pulmonal valves were normal) (Fig. 4a). The patient was then prescribed with furosemide 40 mg q.d, spironolactone 25 mg q.d, bisoprolol 1.25 mg

q.d, and candesartan 8 mg q.d. Last, at 6th month of follow-up, LVEF was finally normal (53.42%). Right ventricle (RV) and dyastolic function were also normal. End-diastolic volume of LV and left ventricular internal end diastolic diamete reduced to 66.07 ml and 39.0 mm respectively since first echocardiography. Valves function were normal and no evidence of LVH (Fig. 4b). Then, the patient was prescribed with bisoprolol 2.5 mg q.d for 7 days and suggested for undergoing annual control and evaluation with cardiologist.

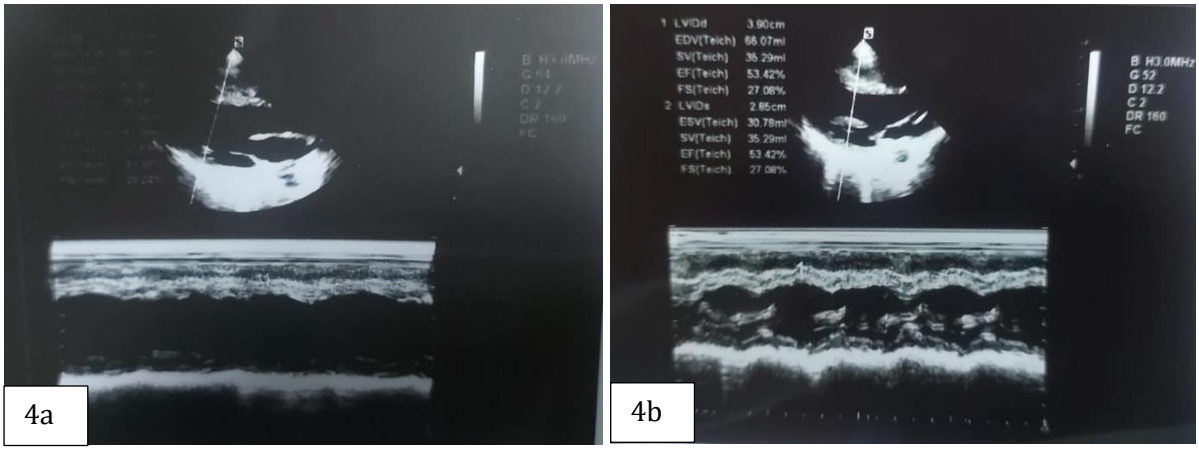

Figure 4. follow-up Echocardiography (a) 2nd month (b) 6th month

DISCUSSION

Peripartum cardiomyopathy refers to the development of heart failure characterized by left ventricular systolic dysfunction in the absence of other identifiable causes of cardiac failure which occur towards the end of pregnancy or 5 months after delivery (Douglass & Blauwet, 2021) along with several echocardiographic findings including left ventricular ejection fraction ≤45%, M-mode fractional shortening ≤30%, and left ventricular end-diastolic dimension ≥2.7 cm/m2 (Karafiatova et al., 2017). The details of this case reported here support this trend; the woman is a young primigravida with early symptoms hardly recognized due to overlapping sign and symptoms between women with normal pregnancy (Wang, 2009). This is due to physiological changes which occur during pregnancy also causing mild ventricular dilatation, increased blood volume, and cardiac output (Sudarman, 2018). Several possible causes of PPCM have been proposed yet unable to be explained by sole ethology and considered to be multifactorial including abnormal immune response to pregnancy, myocarditis, maladaptive response to hemodynamic stresses of pregnancy, prolonged tocolysis, and stress-activated cytokines, hyperexcretion of prolactin, selenium deficiencies, malnutrition, genetic, vascular hormonal imbalance (Karafiatova et al., 2017; Kulkarni et al., 2021; Okeke et al., 2013).

The increase stressors during last semester of pregnancy might disrupt the adaptation of cardiovascular system resulting into heart problems. For instance, high levels of progesterone and fibroblast growth factor 21 (FGF21), fluctuations of prolactin-derived vasoinhibits could dysregulate the metabolic balance of heart and angiogenic balance. Thus, impairment of vascular function occurs eventually inducing cardiac metabolic stress (Hoes et al., 2022). Other multiple pathomechanisms inducing PPCM are inflammation and autoimmune reactions proven by increasing level of circulating cytokines such as tumour necrosis factor-a (TNFa), Interleukin-6 (IL-6), C-reactive protein (CRP), and interferon-gamma (IFNc). Imbalance of oxidative stress and then production of the anti-angiogenic 16-kDa prolactin, disruptions of neurohormonal signalling, elevation of depression-associated microRNA-30e which impairs the function of

serotonin receptor leading to lower circulating levels of serotonin had also been linked to the incidence of PPCM. The elevation of soluble fms-like tyrosine kinase-1 (sFlt-1) also has a role for the development of PPCM (Dash et al., 2022; Sliwa et al., 2021)

According to this case, the patient demonstrated significant heart failure symptoms and blood test result revealed normal finding of leucocyte level, but there was significant decreased of natrium level and raised levels of SGOT and SGPT without hypoalbuminemia. Other supportive findings were cardiomegaly and congestive pulmonal from thorax x-ray, low QRS voltage with poor R wave progression based on ECG, and low LVEF based on echocardiography. The course of these findings were in line with a diagnostic pathway in patients with PPCM.

Laboratory test is used to differentiate disease with similar symptoms to pulmonary edema in PPCM such as non-cardiogenic pulmonary edema, preeclampsia, pyelonephritis with serum albumin levels might decrease in the latter (Kulkarni et al., 2021). Severe congestive heart failure resulted in significant increased levels of SGOT and SGPT. Venous congestion due to increased central venous pressure in congestive hepatopathy along with low cardiac output and hypoperfusion could lead to hepatocellular damage or necrosis eventually increasing SGOT and/or SGPT levels. Transaminase in acute cardiogenic liver injury (ACLI) could sharply elevate to the peak levels in 1-3 days after heart failure onset and usually return to normal levels at 7-10 days after normal haemodynamic status (Çaıli et al., 2015) (Alvarez & Mukherjee, 2011) (S et al., 2019) (Liang et al., 2021).

A significant amount of ascites was found during USG urology which indicated fluid overload in this patient and potentially causing decreased level of natrium. Hyponatremia pathogenesis in heart failure could be multifactorial such as medication effect, increased activity of renin-angiotensin-aldosteron system (RAAS), and arginine vasopressin (AVP) leading to water reabsorption and dilution state of hyponatremia. Other underlying pathomechanism including thirst stimulation due to reduced cardiac output and activation of angiotensin II, water retention due to reduced glomerular filtration rate (GFR) and impairment of renal excretion. In a study, hyponatremia was mostly found in patients with mild-moderate heart failure. History of vomiting and loop diuretic therapy, salt restricted diets were also associated with depletional hyponatremia. Gastorintestinal problems in this patient along with congestive state might induce hyponatremia (Şorodoc et al., 2023) (Rodriguez et al., 2019) (Saepudin et al., 2015) (Adrogué, 2017) (Nankabirwa et al. 2016).

Echocardiography is an important diagnostic tool for confirming the presence of PPCM and determining its severity. Systolic dysfunction, globally decreased contractility along with global dilatation, enlargement of LV end diastolic and end systolic volumes were commonly found in PPCM patients (Davis et al., 2020; Johnson-Coyle et al., 2012; Sliwa et al., 2021; Wang, 2009). In this patient, during emergency department, oxygen via nasal canule (4 lpm oxygen) was administrated which subsequently rose the saturation to 100%. Initially, patient was treated with ringer lactate solution 500 cc rapidly for managing hypotension due to diarrhea. Fluid overload in this patient could be managed using diuretic agents (furosemide) for reducing preload after blood pressure was stabilized. This patient also received bromocriptine, anticoagulant (rivaroxaban), and digoxin. These regiment were appropriate with BOARD (Bromocriptine, oral heart failure drugs, anticoagulation, relaxants for systolic blood pressure more than 100 mmHg, diuretics) regiment. (Arrigo, Blet, & Mebazaa Alexandre, 2017; Bauersachs et al., 2019; Sitio et al., 2021) (Arrigo, Blet, & Mebazaa, 2017) (Kim & Shin, 2017) (Mujkanovic & Qayyum, 2022)

The patient was transferred to the intensive care unit (ICU) for further management. Management of severe stage with unstable haemodynamic status is different from those with stable conditions. Cardiogenic shock could be managed with inotropes, and is considered for

mechanical circulatory support (MCS), ventricular assist devices (VAD), and transplantation (Bauersachs et al., 2019). MCS should be considered in PPCM with acute cardiogenic shock compared to high dose inotropes due to recent reports of its detrimental effects. However this experience is still limited in also require further explorations (Horn et al., 2017). Generally, the aim of treatment in this case is to control volume status with fluid and salt restriction, (diuretic and nitrates) (Arany & Elkayam, 2016; Kulkarni et al., 2021), neuro-humoral inhibition with heart failure drugs (digoxin, beta blockers, mineralcorticoid receptor antagonists, ACE inhibitors or angiotensin receptor blockers), preventing complication of thromboembolic, arrythmia, for alleviating symptoms and improving outcome (Kumari et al., 2012). Digoxin can be considered in PPCM patient with low ejection fraction and it has minimal risk of fetal harm and also compatible with breastfeeding (Kim & Shin, 2017) (Arrigo, Blet, & Mebazaa Alexandre, 2017; Karaye, 2016). Beta blockers should be avoided in the early stage of acute decompensated heart failure (HF) due to its ability to reduce LV contraction and systemic perfusion, but in patient with stable systolic HF, beta blocker selective-I is recommended at least 6 months after full recovery with LVEF>50% and it is also compatible during pregnancy and lactation (Kim & Shin, 2017) (Carlson et al., 2023) (Mujkanovic & Qayyum, 2022).

Bromocriptine an ergot alkaloid and dopamine 2D agonist/antagonist that blocks prolactin secretion, could also be beneficial for treatment of acute heart failure in PPCM (class IIb recommendation) (Bauersachs et al., 2019; Karaye, 2016; Okeke et al., 2013) (Haghikia et al., 2013) (Sliwa et al., 2010) (Horn et al., 2017) (De Jong et al., 2009).It deters the secretion of prolactin thereby stopping the lactation which will improve LVEF and reduce mortality. This is due to the association of 16kDa prolactin fragment with the occurrence of PPCM (Douglass & Blauwet, 2021; Honigberg & Givertz, 2019; Sitio et al., 2021; Sreelatha et al., 2013). Based on study, patients given a dose of 2.5 mg of bromocriptine b.i.d for 2 weeks and then 2.5 mg b.i.d for 4 weeks had better survival and higher LVEF recovery rate than patient receiving standard treatment only (Bhattacharyya A, 2012). In another study, bromocriptine is highly recommended in patients with cardiogenic shock or severe LV dysfunction (EF<25%) (Carlson et al., 2023). A multicenter randomized controlled trial demonstrated higher rate of left ventricular recovery and low morbitidy or mortality in patients treated with 1 or 8 weeks bromocriptine as additional drugs in combination with standard therapy (Hilfiker-Kleiner et al., 2017).

In a case study, an enhancement expression of cathepsin D was found in heart sample of PPCM patient compared to non-heart failure samples during endomyocardial biopsy. Prolactin level decreased to 10.1 ng/ml after 1st day of 5 mg bromocriptine daily administration and continually decreased to 1.5-3.5 ng/ml by the administration of 10 mg bromocriptine per day. LV function gradually improved and bromocriptine was given for 8 weeks (Horn et al., 2017). Based on a review, dosage of bromocriptine depends on the disease severity. Patients treated with bromocriptine had significant LVEF improvement compared with standard guideline medical therapy alone group. LV function recovered better in patients (23 ± 10% at baseline to 55 ± 12%) treated with additional bromocriptine compared to only standard medical drugs (30 ± 12% at baseline to 45 ± 13%) at 6 months of follow-up. However, no significant association between bromocriptine treatment with the occurrence of all cause mortality and heart failure. mortality (Kumar et al., 2023) (Tremblay-Gravel et al., 2019). Yet, in another report, direct effect of bromocriptine on the improvement of left ventricular systolic function remains unclear and need further research (Badianyama et al., 2024). Based on a systematic review involving 263 patients, bromocriptine and standard HF treatment caused a remarkable increment of LVEF approximately 11.37% and reduced PPCM associated mortality compared to only standard therapy. Bromocriptine was reported to be safe, highly tolerable, and no thromboembolism

events were detected during treatment in all patients (Badianyama et al., 2024)(Haghikia et al., 2015).

In contrast with aforementioned statement, the use of bromocriptine remains controversial due to raising concerns regarding side effects. Bromocriptine is an ergot alkaloid derivative which is known for its potential effect for causing vasoconstriction, hypercoagulability, and thromboembolic (TE) events, such as seizure, stroke, coronary vasospasm or thrombosis prominently at higher dose. Thus, bromocriptine should be administered along with anticoagulants (IIa C) at least at prophylactic dose to minimize the risks especially in patient with LVEF<35%. (Badianyama et al., 2024) (Honigberg & Givertz, 2019; Sitio et al., 2021) (Bauersachs et al., 2019)(Arany & Elkayam, 2016; Arrigo, Blet, & Mebazaa Alexandre, 2017; Douglass & Blauwet, 2021; Honigberg & Givertz, 2019; Kulkarni et al., 2021; Sreelatha et al., 2013) (Kido & Guglin, 2019) (Radakrishnan et al., 2024). Currenty, bromocriptine is first choice drug for suppressing prolactin production, but cabergoline, another dopamine D2 receptor (D2R) agonist might be promising as alternative drug. However experimental data of carbegoline had been reported in only a few PPCM cases and need to be validated in more study trials (Pfeffer et al., 2023)(Caruso et al, 2021).

This patient also received rivaroxaban 10 mg q.d. Pregnant women are more susceptible to TE events due to the increased levels of coagulation factors VII, VIII, X, and plasma fibrinogen, von Willebrand, decreased protein C and S activity and fibrinolysis especially in the late gestation and within first 30 days and up 4-6 weeks (Radakrishnan et al., 2024) and in another report up to 6-8 weeks (Mujkanovic & Qayyum, 2022). Post operative status after caesarian section has been associated with higher risk TE events along with endothelial injury, immobility, and dilatation of ventricle as other correlated contributors. This hypercoagulable state along with depressed LV function could lead to the development of LV thrombus, venous and systemic embolism (Mujkanovic & Qayyum, 2022). Hence, prophylactic anticoagulant should immediately initiated in the presence of LV thrombus and in some high risk women. Due to unsafe effect on fetal and increased risk of bleeding in subsequent pregnancy (SSP) along with compliance issue, long term prophylaxis anticoagulant therapy is unlikely to be effective (Fu et al., 2023) (Radakrishnan et al., 2024). Thus, unfractioned or low molecular weight heparin (LMWH) during antepartum or heparin/LMWH or warfarin during post partum period are also suggested in a patient with LVEF<30%. Both warfarin and low-molecular heparin (LMWH) are safe to be used during postpartum including lactation (Mujkanovic & Qayyum, 2022) (Arany & Elkayam, 2016; Bhattacharyya A, 2012; Davis et al., 2020). American Heart Association (AHA) had recently proposed the use of anticoagulation in PPCM women with LVEF<30% whereas heart failure association (HFA) of the ESC study group recommended if LVEF is less than 35% (Mujkanovic & Qayyum, 2022). Yet in another report, anticoagulant should be continued for the treatment of atrial fibrillation or until left ventricular function reaches normal value based on echocardiography (LVEF > 35%) or up to 6-8 weeks of postpartum period (Karaye, 2016; Sitio et al., 2021).

Timing of recovery varies widely with 22% women recovering fully (LVEF 50% or more or improvement greater than 20%) within 2 weeks (Bhattacharyya A, 2012), 30.1% recovered in a year, and 13.8% between 1-10 years. Some other studies showed different range of recovery approximately 3 month-8 years after diagnosis, usually within 3-6 months postpartum (Douglass & Blauwet, 2021). However predictors of poorer outcome include delayed diagnosis, maternal age, multiparity (more than 4), black ethnicity, higher NYHA class, multiparity, a LV thrombus, LV dilatation, RV dysfunction, obesity, other comorbidities, diastolic diameter of more than 60 mm, LVEF < 30% at the time of initial diagnosis, noncompliance to medication, high baseline troponin

levels, Long QTc interval, tachycardia at baseline, using of mechanical support, and the need of transplantation (Bauersachs et al., 2019; Bhattacharyya A, 2012; Davis et al., 2020; Douglass & Blauwet, 2021; Taryètba André et al., 2020).

In an examination during outpatient clinic visit, this patient was in stable condition with NYHA class I-II and follow up echocardiography after 2 months and 6 months revealed significant improvement of EF (53.42%) and none of the valve dysfunctions and LVH were detected. LVEF > 50%, deemed as full recovery of normal LV systolic function, was reported in 23% to 72% of PPCM patients. In this patient, single heart failure therapy with bisoprolol 2.5 mg once a day was still continued until further evaluation. The optimal duration of medical treatment depends on LV function. Standard heart failure treatment with beta blocker and ACEIs/ARBs should be continued at least for 6-24 months or at least a year in low risk patient after full recovery or more in patient with LV systolic dysfunction, whereas it is highly recommended to continue therapy for at least 6 months after full LV recovery in patients with in normalized LV function. Therefore, gradual withdraw of drugs in a stepwise approach should be implemented under vigil observations with serial laboratory and imaging examination including serial echocardiography at the time of discharge, 6 weeks, every 6 months after LVEF is more than 50%, and every year up to 10 years in a stabilized LV function recovery (Karafiatova et al., 2017; Sitio et al., 2021). The drug discontinuation is allowed if treatment in LV recovery function confirmed by transthoracic echocardiography and cardiovascular magnetic resonance (CMR) imaging, all recovered patients are highly suggested for annual clinic visit with cardiologist (Carlson et al., 2023) (Ávila & Carvalho, 2023). (Kido & Guglin, 2019) (Zhu et al., 2021) (Radakrishnan et al., 2024) (Bauersachs et al., 2019) (Arany & Elkayam, 2016).

In a study, more than two thirds patients still received heart failure drug therapy after 5 years of first diagnosis. High rate of relapse is found in dilated cardiomyopathy and complete cardiac recovery after withdrawal of drugs. Based on a cohort in 66 PPCM patients, 86% of them received additional drug treatment with bromocriptine and anticoagulant beside other heart failure therapy. At 5 year of follow-up, 72% patients had full recovery with LVEF>50% with 5% had not recovered. However, other comorbidities have developed over the years including arterial hypertension, arrythmias, paroxysmal supraventricular tachycardia, ventricular tachycardia, or ventricular fibrillation. Mean LVEF of the patients was 55±7% with 70% were still receiving at least one heart failure drug (Moulig et al., 2019).

In this patient, this is the first pregnancy of first child. Thus, SSP is still highly considered in the future. Recommendations regarding the safety SSP are remain inconclusive, but according to a report, recovered LVEF more than 50%, close monitoring and evaluation during pregnancy, and dopamine D2 receptor agonist treatment after delivery would likely minimized the development of cardiomyopathy in the next pregnancy. Of 12 PPCM patients going for preconception evaluation (PE), 6 patients were at higher risk for developing PPCM. Four women were given heart failure drugs e.g beta blocker, hydralazine, and digoxin during cardiology visit. The initiation of digoxin was associated with increased EF, stable EF, and 33% subclinical improvement with no case of EF reduction detected. Hence, digoxin deemed as drug improving heart function, lowering risk of maternal and fetal morbidity. (Nativi-Nicolau et al., 2019)

In contrast, in another report, SSP is not suggested in women with persistent heart failure due to the likelihood of untolerated cardiovascular workload during the pregnancy. Although cardiac function has improved, the contractile of left ventricular is still disrupted and the recurrence rate is greater than 30% (Johnson-Coyle et al., 2012; Kumari et al., 2012; Wang, 2009). Decreased LVEF during SSP had been reported in 20% of cases, but with favorable outcome both for

maternal and fetal. However, a study reported a high incidence of mortality in SSP after woman recovering from PPCM (Mujkanovic & Qayyum, 2022).

CONCLUSION

We present a serious case of PPCM which can be highly threatening for pregnant or postpartum women. Echocardiography is valuable tool for screening, diagnosing and evaluating therapy, wherein the patient was successfully treated via a multidisciplinary approach involving emergent medical therapies especially using BOARD regimen. This is shown by significant clinical and LVEF improvement on the 6th months of follow-up and continually suggested to visit cardiologist yearly. However, therapy also need to consider fetal and maternal safety including compability during lactation. SSP should be cautiously considered and consulted with the experts to reduce the probability of disease relapse. Due to its high mortality, frequent delayed in diagnosis, and the similarity of symptoms with the normal pregnancy or postpartum condition, early diagnosis and treatment are crucial for reducing morbidity and mortality of PPCM.

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