

## Case Study on Cardiac Decompensation in Patients with Preeclampsia and Peripartum Cardiomyopathy

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### Abstract

In pregnant women, cardiovascular complications are among the causes of maternal death, one of which is peripartum cardiomyopathy (PPCM). The similarity in the mechanism of PPCM and pre-eclampsia makes preeclampsia a risk factor for PPCM. Preeclampsia, together with PPCM, also contributes to the increasing maternal mortality when most of the causes of maternal death are preventable. This case study aimed to provide descriptions as the basis in education, raising public awareness, early detection, and early treatment, especially for patients at risk. Eight PPCM cases with preeclampsia were identified during the period of 2017–2018 in Dr. Hasan Sadikin General Hospital Bandung, Indonesia (RSHS). Cases of PPCM with preeclampsia are rarely reported in RSHS. Out of 8 patients, 7 experienced severe preeclampsia and 1 had impending eclampsia. Diagnosis in PPCM patients requires a complete history taking, physical examination, and other diagnostic tests such as echocardiography. In this study, all patients (100%) complained of dyspnea on exertion as a chief complaint and, based on the New York Heart Association in 1994, 87.5% were included in functional class III–IV. The average ejection fraction was shown to be 34.6%, indicating a value below normal limit.

**Key words:** Cardiac decompensation, peripartum cardiomyopathy, preeclampsia

## Studi Kasus Dekompensasi Jantung pada Pasien Preeklampsia dan Kardiomiopati Peripartum

### Abstrak

Pada wanita hamil, komplikasi kardiovaskular berperan dalam kematian ibu, salah satunya adalah kardiomiopati peripartum (PPCM). Persamaan patogenesis antara PPCM dan pre-eklampsia menjadikan preeklampsia sebagai faktor risiko untuk PPCM. Preeklampsia bersama dengan PPCM berperan dalam peningkatan angka kematian ibu (AKI) ketika sebagian besar penyebab AKI dapat dicegah. Studi kasus ini bertujuan diharapkan untuk memberikan deskripsi sebagai dasar dalam pendidikan, meningkatkan kesadaran masyarakat, deteksi dini, dan perawatan dini, terutama untuk pasien yang berisiko. Pada tahun 2017–2018, terdapat 8 kasus pasien PPCM dengan preeklampsia yang dirawat di RSUP Dr. Hasan Sadikin Bandung (RSHS). Kasus PPCM dengan preeklampsia jarang dilaporkan di RSHS. Dari 8 pasien, 7 pasien mengalami preeklampsia berat dan 1 pasien dengan *impending eclampsia*. Diagnosis pada pasien PPCM memerlukan anamnesis yang lengkap, pemeriksaan fisik, dan pemeriksaan penunjang seperti ekokardiografi. Pada penelitian ini, semua pasien (100%) mengeluhkan sesak nafas sebagai keluhan utama dan berdasar atas klasifikasi *New York Heart Association* tahun 1994, 87,5% termasuk dalam kelas fungsional III–IV. Rata-rata fraksi ejeksi menunjukkan 34,6% yang menandakan nilai di bawah batas normal.

**Kata kunci:** Dekompensasi jantung, kardiomiopati peripartum, preeklampsia

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## Introduction

Peripartum cardiomyopathy (PPCM) is a rare case of heart failure secondary to left ventricular systolic dysfunction that occurs at the end of pregnancy and several months postpartum.<sup>1</sup> The incidence varies between countries around the world. A high incidence of PPCM is observed in Haiti (1 per 300 births) and Nigeria (1 per 100 births) whereas Japan has a much lower incidence rate with 1 per 20.000 births.<sup>2,3</sup> These variabilities are influenced by several risk factors such as race, inadequate nutrition intake, and hypertension status.<sup>2</sup>

The similarity in the mechanism of peripartum cardiomyopathy and preeclampsia, which is one of the hypertensive complications in pregnancy, has been observed. An increase in placental soluble fms-like tyrosine kinase 1 (sFlt-1) in preeclampsia patients is also seen in PPCM patients. Increased sFlt-1 and decreased peroxisome proliferator-activated receptor gamma coactivator 1-alpha (PGC- $\alpha$ ) give rise to angiogenic disorders and account for cardiac dilation.<sup>1,4,5</sup> These resemblances in several mechanisms during the course of the two diseases, such as genetic predisposition (TTN gene) and an increase in antiangiogenic factors, make preeclampsia one of the risk factors of PPCM.<sup>4</sup>

Peripartum cardiomyopathy has a high mortality rate despite the rarity of the disease.<sup>6</sup> In the United States, 26.5% of maternal mortalities are caused by cardiovascular diseases, with PPCM accounts for 23% of deaths during the postpartum period.<sup>7</sup> On the other hand, preeclampsia has high incidence and mortality rates. According to the World Health Organization, the incidence of preeclampsia is seven times higher in developing countries.<sup>8</sup> In Indonesia, preeclampsia and other hypertensive complications in pregnancy are the second leading cause of maternal mortality.<sup>9</sup> Undiagnosed preeclampsia and PPCM that do not receive adequate therapy will lead to maternal mortalities that will eventually increase the maternal mortality ratio (MMR).

Data from the Indonesian Ministry of Health data in 2012 presented that about 52.6% of maternal mortality occurs in 6 large provinces, one of which is West Java Province. In the same year, the Expanding Maternal and Neonatal Survival (EMAS) program has succeeded to decrease the MMR to 305 per 100.000 live births, which is still far below the Sustainable Development Goals' (SDGs) target of reducing MMR to 70 per 100.000 live births in 2030.<sup>10</sup>

Maternal deaths are caused by complications that are actually preventable. Thus, the role of the primary, secondary, and tertiary prevention is essential, especially for the high-risk pregnant women, including those who have a high risk for preeclampsia.

To be able to provide appropriate prevention and treatment for PPCM, a good understanding of the disease is required. Hence, good studies on this condition are very much needed.

Despite the widely available individual studies on preeclampsia and peripartum cardiomyopathy, studies on PPCM with preeclampsia patients are still scarce, especially in Indonesia. There is one study available that described the characteristics of 20 PPCM patients with preeclampsia in 2014–2016 at Dr. Soetomo, Surabaya, Indonesia; nevertheless, data and studies on PPCM with preeclampsia in West Java are still difficult to find.<sup>11</sup> This study aimed to report the rare cases of PPCM and preeclampsia among patients visiting Dr. Hasan Sadikin General Hospital Bandung, as one of the referral hospitals in West Java. It is expected that the results can be used as inputs in medical education, raise public awareness, and enable early detection and immediate treatment, especially for patients at risk.

## Case

A series of PPCM cases were collected retrospectively from the inpatient medical records of Dr. Hasan Sadikin General Hospital Bandung during the period of 2017–2018. Eight cases of PPCM patients with preeclampsia were identified. All patients met the following criteria: (1) no history of previous cardiac structure disorder; (2) diagnosed with preeclampsia; (3) diagnosed with PPCM in peripartum or postpartum period and (4) ejection fraction <45%.

In general, two patients had a sign and symptoms that were similar to the left heart failure, such as dyspnea on exertion, orthopnea, paroxysmal nocturnal disease, coughing with phlegm, and fine crackles. Meanwhile, six other patients had a sign and symptoms similar to the left and right heart failure, with an addition of increased jugular venous pressure and/or lower extremities edema. The presentation of the cases is shown in Table 1. Seven patients were diagnosed with severe preeclampsia and 50% were diagnosed with PPCM in the postpartum period (Table 2). All patients complained of

**Table 1 Patients Characteristics**

Pt. no.	Age and GPA	Type of Preeclampsia	Sign				Echocardiography	
			BP (mmHg)	HR (bpm)	Cardiac Enlarge	Sound	Chamber Dilation	EF (%)
1	34 Y.O, G3P2A0	Severe preeclampsia	160/100	90	+	Murmur	LA-LV	27
2	28 Y.O, G1P0A0	Severe preeclampsia	160/110	120	+	Normal	LV	35
3	19 Y.O, G1P0A0	Severe preeclampsia	160/110	142	-	Normal	-	41
4	36 Y.O, G1P0A0	Severe preeclampsia	160/100	98	+	S3, S4, murmur	LV	36
5	28 Y.O, G2P1A0	Severe preeclampsia	140/90	150	-	S3, S4, murmur	-	32
6	35 Y.O, G4P1A2	Severe preeclampsia	140/90	148	+	Normal	LA-LV	38
7	20 Y.O, G1P0A0	Severe preeclampsia	160/100	114	+	Normal	LA-LV	43
8	21 Y.O, G1P0A0	Impending eclampsia	149/108	110	-	Normal	-	25

Notes: GPA: gravida, para, abortus; Y.O: years old; BP: blood pressure; HR: heart rate; LA: left atrium; LV: left ventricle; EF: ejection fraction

**Table 2 Patient Characteristics**

Characteristics	n (%)
Age	
Mean (Stdev)	26.4 (6.12)
<30 years old	6 (75%)
≥30 years old	2 (25%)
Multiple gestation	
Yes	2 (25%)
No	6 (75%)
Multiparity	
Yes	3 (37.5%)
No	5 (62.5%)
Type of hypertension	
Severe preeclampsia	7 (87.5%)
Impending eclampsia	1 (12.5%)
Time of PPCM diagnosis	
Antepartum	4 (50%)
Postpartum	4 (50%)
NYHA	
I-II	1 (12.5%)
III-IV	7 (87.5%)

Notes: PPCM: peripartum cardiomyopathy; NYHA: New York Heart Association

dyspnea on exertion as their chief complaint. Other signs and symptoms are presented in Table 3.

Of the eight cases, only four cases underwent electrocardiography examinations. All showed sinus tachycardia. In one case, there was also a poor R wave progression and an old anteroseptal myocardial infarction while in two cases, left ventricular hypertrophy and a poor R wave progression were observed. X-ray was only performed in one patient with the result showing right pleural effusion and cardiomegaly without pulmonary congestion. Echocardiography results are presented in Table 4. All patients had an ejection fraction of <45% with an average of 34.6%.

Of these 8 patients, 4 were diagnosed with peripartum cardiomyopathy in the antepartum period and choice of drugs given are presented in table 5 while the medications for patients that were diagnosed during the postpartum period are presented in Table 6.

### Discussion

Peripartum cardiomyopathy (PPCM) is a heart failure secondary to left ventricular systolic dysfunction that occurs at the end of pregnancy and several months post-delivery.<sup>1</sup> A previous

**Table 3 Signs and Symptoms of Heart Failure**

Signs and Symptoms	f (%)
DOE	8 (100%)
Orthopnea	6 (75%)
PND	4 (50%)
Cough with phlegm	3 (37.5%)
Tachycardia	7 (87.5%)
Increased JVP	3 (37.5%)
HJR positive	2 (25%)
Cardiomegaly	6 (75%)
S3 heart sound	2 (25%)
S4 heart sound	2 (25%)
Murmur	3 (37.5%)
Bilateral fine crackling sound	6 (75%)
Lower extremities edema	6 (75%)

Notes: DOE: dyspnea on exertion; PND: paroxysmal nocturnal dyspnea; JVP: jugular venous pressure; HJR: hepatojugular reflux

study defines PPCM as an idiopathic and new-onset heart failure that occurs in a patient at the end of pregnancy or during the first 5 months post-delivery.<sup>4</sup> In this study, 4 patients were diagnosed with PPCM during the postpartum period.

In the United States, cardiovascular diseases remain the major cause (26.5%) of maternal

**Table 5 Medications of Patients Diagnosed in Antepartum**

Medications	f (%)
Pre-delivery	
Methyldopa	3 (75%)
Furosemide	3 (75%)
Bisoprolol	1 (25%)
Nitroglycerin	1 (25%)
Post-delivery	
Methyldopa	1 (25%)
Furosemide	4 (100%)
Bisoprolol	4 (100%)
Nitroglycerin	1 (25%)
Captopril	3 (75%)
Bromocriptine	1 (25%)
Amlodipine	1 (25%)
Ramipril	2 (50%)

**Table 4 Echocardiography Results**

Echocardiography	f (%)
Heart Chamber Dilation	
Left atrium and ventricle	3 (37.5%)
Left ventricle	2 (25%)
No dilation	3 (37.5%)
Ejection Fraction	
Mean (Stdev)	34,6 (6,3)
Global hypokinetic	8 (100%)
Left ventricular systolic dysfunction	7 (87.5%)
Decreased right ventricular contraction	2 (25%)
Pulmonary Hypertension	
Mild	4 (50%)
Low probability	1 (12.5%)
High probability	1 (12.5%)
Heart Valve Disorders	
Trivial MR	1 (12.5%)
Structural TR	1 (12.5%)
Mild MR	3 (37.5%)
Mild TR	1 (12.5%)
Mild PR	1 (12.5%)
Mild AR	1 (12.5%)
Moderate TR	1 (12.5%)

Notes: MR: mitral regurgitation; TR: tricuspid regurgitation; PR: pulmonary regurgitation; AR: aortic regurgitation

deaths, with peripartum cardiomyopathy accounts for 23% of deaths in postpartum.<sup>7</sup> The incidence of PPCM varies across regions with Haiti (1 per 300 birth) and Nigeria (1 per 100 birth) show a high incidence. The incidence of PPCM in Japan is much lower with 1 per 20.000 birth.<sup>3</sup> These variabilities are influenced by

**Table 6 Medications of Patients Diagnosed during Postpartum**

Medications	f (%)
Methyldopa	1 (25%)
Furosemide	4 (100%)
Bisoprolol	4 (100%)
Nitroglycerin	1 (25%)
Captopril	4 (100%)
Bromocriptine	1 (25%)

several risk factors such as race, inadequate nutrition intake and hypertensive complications, such as preeclampsia.<sup>2</sup>

Risk factors for peripartum cardiomyopathy include maternal age  $\geq 30$  years old, black race, multiple gestation, multiparity, hypertension, anemia, autoimmune disease, thyroid dysfunction, malnutrition, smoking, diabetes, and tocolysis use.<sup>1,2,4</sup> In this case study, risk factors were identified as follows: 2 patients were  $\geq 30$  years old, 2 patients had multiple gestation, and 4 patients were multiparity. These are different from the previous studies because the sample size and the study duration were limited in this case study.

Preeclampsia is one of the risk factors for peripartum cardiomyopathy. A study on 535 PPCM patients revealed that 29.3% of PPCM patients were preeclamptic.<sup>12</sup> Another study in Dr. Hasan Sadikin General Hospital Bandung discovered that 26.3% of PPCM patients were preeclamptic.<sup>13</sup> Preeclampsia is classified based on its severity into preeclampsia and severe preeclampsia. In this study, 7 patients were diagnosed with severe preeclampsia and 1 patient was diagnosed with impending eclampsia. This is similar to the previous study, where 18 cases with severe preeclampsia and 1 case with eclampsia were identified.<sup>11</sup>

Preeclampsia and peripartum cardiomyopathy have a similar pathogenesis. Some studies stated that genetic, inflammation, and unbalanced angiogenesis factors influence the correlation between the two diseases.<sup>1,2,4,5</sup> In preeclamptic patients, placental hypoxia accelerates the placental soluble fms-like tyrosine kinase 1 (sFlt-1) secretion, causing vascular endothelial growth factors and placental growth factors fail to bind with their receptor, reflecting their roles in endothelial dysfunction mechanism.<sup>15,16</sup> Increased sFlt-1 level is also seen in PPCM patients.<sup>1,4,5</sup> The loss of peroxisome proliferator-activator receptor gamma coactivator 1-alpha (PGC- $\alpha$ ) and the increase in sFlt-1 cause the heart to be unprotected and also become the reason why PPCM occurs late in pregnancy and during the postpartum period. Changes in the balance of angiogenesis that is caused by preeclampsia increase the risk in PPCM patients with susceptibility to heart problems.<sup>17</sup>

In diagnosing peripartum cardiomyopathy patients, complete history taking is important to exclude other differential diagnoses.<sup>1</sup> In general, patients would experience signs and symptoms of heart failure such as dyspnea on exertion, orthopnea, paroxysmal nocturnal

dyspnea, and lower extremities edema. On physical examination, signs associated with left and right heart failure such as pulmonary rales, increased jugular venous pressure, and edema can be observed.<sup>4</sup> In this study, all patients complained about dyspnea on exertion as their chief complaint.

The postpartum sFlt-1 concentration level relates to the functional class of the New York Heart Association (NYHA) in patients.<sup>2,4</sup> The ninth edition of NYHA has classified heart failure based on its functional capacity. In this case series, 7 patients were classified to NYHA functional class III-IV and 1 patient was classified to NYHA functional class I-II. By location, heart failure can also be classified as left heart failure, right heart failure, or both.<sup>18</sup> In this study, 6 patients had signs and symptoms similar to left and right heart failure while the remaining 2 had left heart failure. This finding is similar to that of the previous study.<sup>4</sup>

Laboratory examination results usually show increasing B-type natriuretic peptide, N-terminal pro-BNP, and troponin levels. However, in patients with preeclampsia without PPCM there is also an increase in atrial natriuretic peptides, brain natriuretic peptides, and cystatin C.<sup>19</sup> Imaging such as cardiovascular magnetic resonance imaging and computed tomography scan can also be performed but endomyocardial biopsy is not permitted to be used to diagnose PPCM patients.<sup>4</sup>

In peripartum cardiomyopathy patients, electrocardiography (ECG) would reveal sinus rhythm and is mostly accompanied by a T wave abnormality.<sup>4</sup> Only four out of eight in this study were examined using ECG. The ECG results were similar to that of the previous study with the majority of patients experienced sinus tachycardia (100%). Some patients also revealed poor R wave progression (75%), left ventricular hypertrophy (50%), and old anteroseptal myocardial infarction.

On radiography examination, peripartum cardiomyopathy patients usually show pulmonary edema and enlarged heart silhouette.<sup>4</sup> In this case series, the chest x-ray was only performed in one patient, showing cardiomegaly, which is similar to the one in the previous study. The patient did not have pulmonary congestion but pleural effusion is one of the signs of cardiac decompensation.

Preeclamptic patients with heart failure manifestation do not always have peripartum cardiomyopathy. Changes in blood vessels in preeclamptic patients will cause an increase

in arterial blood pressure and total vascular resistance. Increased vasoconstriction plays a role in increasing the afterload of the left ventricle and causing a decrease in cardiac output. Thus, patients have impaired left ventricular function that is characterized by concentric hypertrophy and diastolic and systolic dysfunctions.<sup>19</sup>

Echocardiography is the main modality in diagnosing peripartum cardiomyopathy. This examination can distinguish whether preeclampsia patients have PPCM or not. Preeclampsia patients without PPCM usually have preserved ejection fraction.<sup>19</sup> The diagnosis of PPCM is made when there is left ventricular dysfunction with an ejection fraction of <45% during peripartum or early postpartum period and without any history of structural heart disease.<sup>1,4</sup>

All patient's echocardiographic results showed an ejection fraction of <45% with the highest and lowest EF of 41% and 25%, respectively. The mean value of EF was 34.6%, which indicates that patients had left ventricular function that is below the normal range. The lowest EF (25%) belonged to the PPCM patient with impending eclampsia. However, it is unknown whether the severity of preeclampsia affects the low value of left ventricular ejection fraction or not. A study in Barnes-Jewish Hospital to assess left ventricular ejection fraction recovery during a follow-up in PPCM patients with and without preeclampsia shows that the EF in PPCM patients with preeclampsia are more likely to recover to the normal range.<sup>20</sup>

In this present study, 3 patients showed left atrial-ventricular dilatation, 2 patients showed left ventricular dilatation only, and 3 patients did not have any cardiac chamber dilatation. This is similar to the finding in the previous study that stated left ventricular dilatation does not always occur in these patients.<sup>1,4</sup>

Heart failure that occurs in peripartum cardiomyopathy patients can develop rapidly. Thus, the management used for these patients is the same management for heart failure.<sup>1,4</sup> In Dr. Hasan Sadikin General Hospital Bandung, the management for PPCM patients is based on the European Society of Cardiology (ESC) guideline.<sup>13</sup> According to the ESC guideline in 2018, the essential therapy for PPCM management, which is referred to as BOARD, consists of bromocriptine, oral heart failure therapies, anticoagulant, vasoRelaxing agent, and diuretics.<sup>1</sup>

Medication in pregnant and nursing mothers is quite challenging due to the consideration that

must be taken regarding the ability of the drugs to cross the placental barriers and to be excreted with breast milk to avoid any side effects on the fetus and breastfed baby. Medicines that are toxic to the fetus include angiotensin-converting enzyme inhibitor, angiotensin II receptor blocker, angiotensin receptor neprilysin inhibitor, mineralocorticoid receptor blocker, and atenolol. These drugs should not be given to pregnant women.<sup>1,4,13</sup> For patients diagnosed in antepartum, the choice of drugs may include hydralazine, nitrate, and beta 1 selective blockers. Diuretic (loop diuretic and thiazide) administration can be considered when there is an indication of pulmonary edema.<sup>1,4</sup>

Four of the respondents who were diagnosed during pregnancy were given medications that are appropriate and safe for the fetus. Most of them received methyldopa (75%) and furosemide (75%) during the period before delivery. After delivery, all patients were given furosemide and bisoprolol. In patients who were diagnosed during the postpartum period, most were given furosemide, bisoprolol, and captopril. These are in concordance with the findings in a study on PPCM in Dr. Hasan Sadikin General Hospital Bandung during the period of 2011–2013.<sup>13</sup>

Bromocriptine can be used to manage heart failure as it is able to inhibit prolactin so as to suppress lactation.<sup>1,4,13</sup> This is based on the PPCM mechanism that is associated with the catabolism of prolactin.<sup>4,13</sup> The recommended dose is 2.5 mg per day for 1 week and it should be given with heparin because this drug can cause thrombogenesis.<sup>1,4</sup> In this study, two patients were given bromocriptine but only one patient was recorded as being given 2 doses of heparin prophylaxis 500 units subcutaneously.

Among the eight patients, there was one patient who had to be admitted to the high care unit. The patient experienced somnolence, delirium, and severe shortness of breath with a diagnosis of respiratory failure. She was intubated. On the third day, the patient's condition improved and was transferred to the inpatient room.

All patients survived and were eventually discharged from the hospital after the cardiac decompensation had improved, the general condition was stable, and no complaints from the patients. The average length of stay was 9.5 days with 6 days as the shortest and 19 days as the longest. Decreased consciousness due to hypoxic encephalopathy was observed in patients with the longest length of stay.

There is a high number of preeclampsia cases in Dr. Hasan Sadikin General Hospital Bandung, with a total of 767 cases in the period of 2017–2018. However, cases of PPCM and PPCM with preeclampsia are quite rare in this hospital. From 2017 to 2018, 40 PPCM patients were admitted with only eight (20%) of them were diagnosed with preeclampsia. A previous study shows that there are 20 PPCM with preeclampsia cases per 25 PPCM patients.<sup>11</sup> With this reference, it can be stated that preeclampsia in the majority of PPCM patients in this hospital was detected and diagnosed. The incidence of preeclampsia in PPCM of this study is lower than that of the previous study in Surabaya which might be caused by a lack of exploration of the risk factors of patients diagnosed with PPCM. As a result, the diagnosis of preeclampsia is not made in patients with PPCM. Underdiagnosis might also be one of the reasons due to the fact that diagnosing patients with PPCM is quite challenging, especially in the antepartum period. The physiological state in the second trimester of pregnancy, which is the trimester with peak body fluid volume expansion, may complicate the diagnosis of pathologic heart problems because the clinical manifestations of body fluid volume expansion are similar to the manifestation of mild heart failure. In addition, not all patients with signs and symptoms of heart failure were consulted to the cardiologist, meaning that they are never examined for this condition and no echocardiography was performed, leading to underdiagnosis of PPCM among patients with preeclampsia.

This study has limitations because it is a retrospective study using secondary data from the patient's medical records in only one single center. The lack of control population and the inability to demonstrate a causal relationship between preeclampsia and peripartum cardiomyopathy also becomes another limitation of this study.

In conclusion, cases of peripartum cardiomyopathy with preeclampsia are rarely reported in Dr. Hasan Sadikin General Hospital Bandung. In 2017–2018, there are 40 PPCM patients, with only eight (20%) diagnosed with preeclampsia. The diagnosis in PPCM patients requires a complete history taking, physical examination, and other diagnostic tests such as echocardiography. All patients analyzed (100%) complained of dyspnea on exertion as a chief complaint and 87.5% were included in functional class III–IV based on the New York Heart Association in 1994. The average ejection

fraction was 34.6%, which indicates a value below the normal limit.

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