The Internet Journal of Gynecology and Obstetrics

Volume 24, Number 1

Original Article

Case Study

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Internet Journal of Gynecology and Obstetrics

COUNTRY

United States

Universities and research institutions in United States

SUBJECT AREA AND CATEGORY

Medicine
Obstetrics and Gynecology

PUBLISHER

Internet Scientific Publications, LLC

PUBLICATION TYPE

Journals

ISSN

15288439

COVERAGE

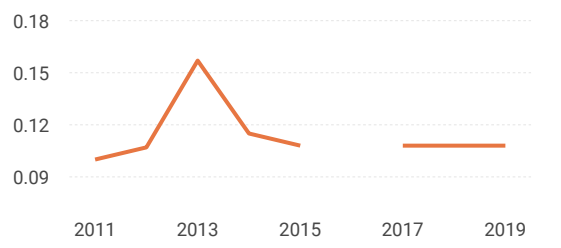
2010-2012, 2016

SCOPE

Information not localized

Quartiles

SJR



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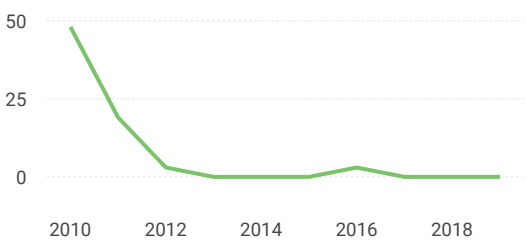

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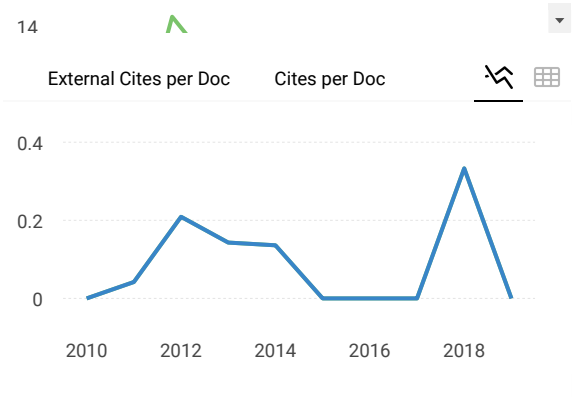

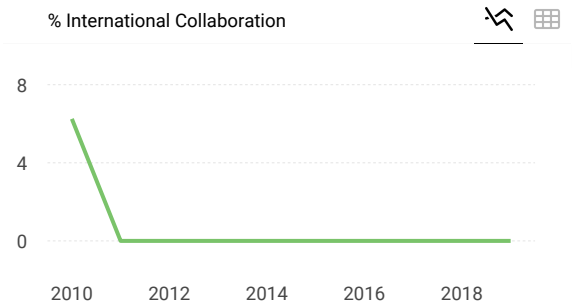

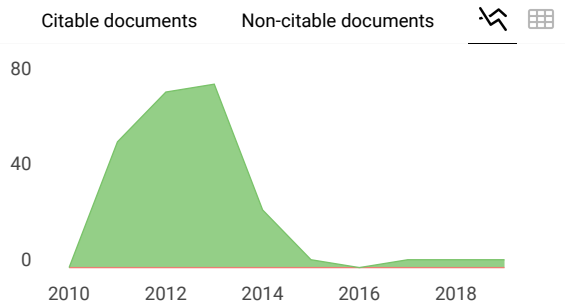

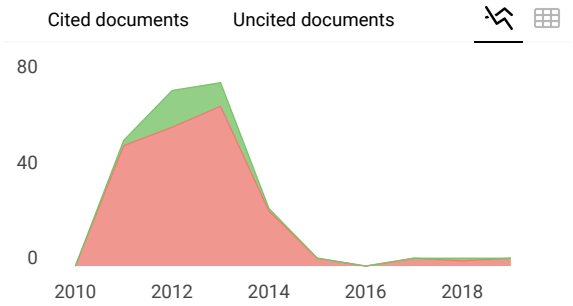

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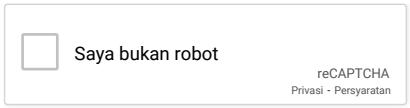

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Peripartum Cardiomyopathy After Cesarean Section In Women With Preeclampsia Related To More Severe Outcome: A Case Series

A Suryawan, B Handono, A Suryawan, T Rahardjo

Citation

A Suryawan, B Handono, A Suryawan, T Rahardjo. *Peripartum Cardiomyopathy After Cesarean Section In Women With Preeclampsia Related To More Severe Outcome: A Case Series*. The Internet Journal of Gynecology and Obstetrics. 2020 Volume 24 Number 1.

DOI: 10.5580/IJGO.54805

Abstract

Background: Peripartum cardiomyopathy (PPCM) is an unusual and uncommon form of non-ischemic dilated cardiomyopathy that presents in the last month of pregnancy or within 5 months after delivery in the absence of other causes. The incidence of PPCM is 1 in 3.000 normal pregnancies with mortality rate as high as 50% to 75%. Etiology of PPCM could be many potential causes, including viral myocarditis, abnormal immune response in pregnancy and abnormal response to the increased hemodynamic burden of pregnancy. The occurrence of PPCM in woman with preeclampsia is a very rare case. This case series present two women with preeclampsia underwent Cesarean Section and suffered by PPCM. The limitation on data and the rarity of similar case cause difficulties in the management.

Method: Case Series

Result: Peripartum Cardiomyopathy related to more severe effect for women with preeclampsia who underwent Cesarian Section and might be influenced by an excess secretion of anti-angionic factor from the placenta. In this case series, one woman survives but the other woman died on the way to the Emergency Department.

Conclusion: Peripartum Cardiomyopathy occurred after Cesarean Section in woman with preeclampsia can be related to severe effect and even death.

INTRODUCTION

Peripartum cardiomyopathy (PPCM) is a very rare form of non-ischemic dilated cardiomyopathy occurred in the puerperium as heart failure secondary to left ventricular systolic and diastolic dysfunction in the absence of another causes of heart failure. Peripartum cardiomyopathy is diagnosed when heart failure develops during the last month of pregnancy or within 5 months after delivery in the absence of other causes. The incidence of PPCM is affected 1 in 3.000 pregnancies, as many as 25% of women with PPCM in developing countries die within 5 years with mortality rate as high as 50% to 75%.[1-7]

The etiology of PPCM remains enigmatic, and many potential causes suspected to have role in this condition, including viral myocarditis, abnormal immune response to pregnancy, abnormal response to the increased hemodynamic burden of pregnancy, hormonal interactions, malnutrition, inflammation, and apoptosis. Some previous studies reported that toxaemina, an older term for preeclampsia, was detected in approximately 22% affected women. Recent researches suggest that PPCM is a vascular disease, triggered by excess anti-angiogenic signaling from the placenta and pituitary during late period of pregnancy. This study showed the possibility of specific relationship between PPCM and preeclampsia, both of them have a similar feature caused by excess secretion of anti-angionic factor from the placenta.[8-12]

CASE REPORT

First Case

A 41 years old female, G2P1A0, 38-39 weeks pregnancy, with chief complaints hypertension and low extremities

edema during the last two weeks. She never experienced the symptoms before, so she was taking no antihypertensive drugs. There were no complaints of headache, nausea or vomiting and upper quadrant abdominal pain.

At presentation in the emergency department, she was 114 kg BW and 167 cm tall with blood pressure 160/100 mmHg. Laboratory finding was anemia with hemoglobin 9.3g/dL, hematocrit 29% and eritrocyte 3.8 x10/mm3. Urinalysis showed protein ++ and ketone ++. Fetal Non Stress Test (NST) showed prolonged fetal bradycardia and late deceleration. She was diagnosed 38-39 weeks pregnancy with severe preeclampsia, anemia and intrauterine fetal distress. She had an emergency Caesarean operation based on the condition. Preoperatively, she has been given oxygen 3 L/minutes, intravenous ringer lactate solution with magnesium sulfate and oral anti hypertension methyldopa 250 mg preoperatively. Blood pressure was slightly increased to 170/110 mmHg before the operation. Post operative vital signs were blood pressure 150/100 mmHg, heart rate 84 x/min, respiratory rate 20 x/min, oxygen saturation 98% with oxygen 3 L/min, and sinus rhythm at electrocardiography (ECG). After the operation, patient fluid level and blood pressure were maintained by ringer lactate and magnesium sulfate.

About 18 hours after operation, the patient reported having dyspnea, fatigue and slightly anxious. The patient was transferred to intensive care unit. She had blood pressure 162/108 mmHg, pulse rate of 139 x/min, respiratory rate 29 x/min and oxygen saturation 91% with oxygen 10 L//min. Physical examination showed GCS 15, no sign of jugular venous distention, S3 heart sound, edema, or hepatosplenomegaly. She was slightly tachypnea but not in any acute respiratory stress. Therapy was started with intravenous furosemide 40 mg twice a day, oral K-l aspartate for potassium replacement, oral antihypertensive treatment with oral angiotensin II receptor inhibitor (ARB) candesartan 8 mg four times a day and spironolactone 100 mg. An intravenous nitroglycerin drip was initiated to decrease afterload and preload. The patient was also given intravenous digoxin 0.75 mg, continued with oral digoxin 0.25 mg. Oral blockers, bisoprolol 2.5 mg, also used to decrease the effect of excessive sympathetic nervous system activation on the heart. The echocardiography showed peripartum cardiomyopathy with left ventricle dilatation, estimated ejection fraction 27.01%, and mild diastolic dysfunction. Chest radiograph findings were cardiomegaly and pulmonary congestion, early pulmonary edema. A cardiology consultation was done and the patient was given appropriate medications for heart failure, which alleviated her symptoms. Her dyspnea and fatigue greatly decreased with diuresis, she was discharged from the hospital three days later.

Second Case

A 38 years old female, G3P1A1, 37-38 weeks pregnancy, with chief complaints hypertension and mild low extremities edema that occurred for one month. Similar to the first patient, she did not take any antihypertensive drugs and never felt headache, nausea or vomiting and upper quadrant abdominal pain.

At presentation in the emergency department, she was 99 kgBW and 150 cm tall with blood pressure 170/100 mmHg. Laboratory findings were anemia with haemoglobin 9.8 gr/dL. Urinalysis showed protein ++ and ketone ++. NST showed prolonged fetal bradycardia and late deceleration. She was diagnosed G3P1A1, 37-38 weeks pregnancy with severe preeclampsia, anemia and intrauterine fetal distress. She had similar treatment as the first patient and underwent emergency operation because of her condition. Blood pressure was 180/110 mmHg before operation. Post-operative vital signs were blood pressure 155/100 mmHg, heart rate 86 x/min, respiratory rate 18 x/min, oxygen saturation 99% with oxygen 3 L/min with sinus rhythm at ECG. The echocardiography showed peripartum cardiomyopathy with left ventricle dilatation, estimated ejection fraction 30%, and mild diastolic dysfunction. Chest radiograph findings were cardiomegaly and early pulmonary edema.

The patient transferred to the ICU for two days, returned to inpatient room at third day and went home at fifth day. Vital signs at the time of discharge were blood pressure 140/95 mmHg, heart rate 86 x/min, respiratory rate 18 x/min and oxygen saturation 99%. After five days at home, the patient reported having severe dyspnea. The patient died on the way to hospital.

DISCUSSION

Peripartum cardiomyopathy is diagnosed when heart failure develops in the last month of pregnancy or within 5 months after delivery in the absence of other causes.[1,2] It occurs in one of every 3.000 pregnancies, as many as 25% of women with PPCM in developing countries died within 5 years with

mortality rate as high 50%-75%.3-7 The etiology of PPCM remains enigmatic, and many potential causes suspected to have contribution in this disease, including viral myocarditis, abnormal immune response to pregnancy, abnormal response to the increased hemodynamic burden of pregnancy, hormonal interactions, malnutrition, inflammation, and apoptosis.[8]

The disease has been reported occurs more frequently in women with preeclampsia and detected in 22% affected women.[9] A fluid overload in pregnancy may cause subclinical heart disease, which becomes overt because the additional stress on the cardiovascular system.[10,11] Cardiac biopsy may demonstrate myofiber hypertrophy or degeneration, fibrosis, interstitial edema and lymphocytic infiltration.[14]

The relationship between preeclampsia and PPCM seems to have a genetic basis. One study found women with preeclampsia are more likely to have protein-altering mutations in genes associated with cardiomyopathy. The mutation occured especially in TTN gene which encodes the sarcomeric protein titin. Mutations triggering cardiomyopathy are prevalent in preeclampsia, idiopathic cardiomyopathy, and peripartum cardiomyopathy. All of them are important risk factors for a widening spectrum of cardiovascular disorders.[15]

The important physiologic changes that peak during the second trimester of pregnancy are blood volume expansion, relative anemia, alterations in vascular resistance, ventricular dilatation, an increase in preload and heart rates. These hemodynamic stressor lead to decompensated heart failure and PPCM must be strongly considered in this condition.[13,14,15]

Women usually present with dyspnea, palpitations, chest discomfort, and sometimes chest or abdominal pain. Physical examination shows increased blood pressure, jugular vein pressure, cardiomegaly, third heart sound, mitral and/or tricuspid regurgitation, pulmonary rales, peripheral edema, hepatomegaly, and less commonly, embolic events, hemoptysis, or ascites.[16,17]

An ECG may demonstrate no abnormalities or may show nonspecific ST segment and T wave changes, evidence of left ventricular hypertrophy (LVH), prolongation of the PR or QRS intervals, low voltage, and occasionally left bundle branch block (LBBB). Chest X-ray reveals typical signs of congestive heart failure, including cardiomegaly, pulmonary venous congestion, interstitial edema, and occasionally pleural effusion. Echocardiography demonstrates dilated cardiomyopathy involving all four chambers, but primarily the left heart. Normal wall thickness, high intra cardiac pressure with low cardiac output and diffuse symmetric hypokinetic are specific characteristics of PPCM.[17,18,19]

The management of PPCM is similar to other non ischemic dilated cardiomyopathies, including therapy for congestive heart failure, bed rest, fluid restriction, and sodium restriction. The most important goal of treatment is afterload reduction, ACEi is the best choice for reduction of resistance (category D in table I), breastfeeding should be discouraged in women with PPCM who require ACEi. Alternatively to ACEi is the newer ARB, but they also contraindicated during pregnancy. Hydralazine (category B) is the drug of choice.[20-23]

Table 1

Categories of all drugs based on the level of safety in pregnancy. Source from Briggs GG.23

Category	Definition
A	Safety has been established using human controlled studies.
B	Presumed safety based on animal studies, but no controlled studies in pregnant women.
C	Uncertain safety; either studies in animals have revealed adverse effects on the fetus and there are no controlled studies in pregnant women, or studies in women and animals are not available. Drugs should be given only if the potential benefit justifies the potential risk for the fetus.
D	Unsafe; there is evidence of human fetal risk, but drug use may be justifiable despite the risk in life threatening clinical situations.
X	Highly unsafe; risk of use outweighs any possible benefit.

Other important aspects of PPCM management are preload reduction with diuretics and oral nitrates and increase inotrophy with digoxin (category C) is believed to be safe in pregnancy and no adverse effect during breast feeding.[24]

 blockers (category C) have recently been recommended for the treatment of congestive heart failure to decrease the effect of excessive sympathetic nervous system activation on the heart. Breast feeding is allowed, but bradycardia in infants is possible.[25]

Thromboembolism may contribute a complicate PPCM. Anticoagulant can be used in selected patients without evidence of thrombi on echocardiography, but on condition that a left ventricular ejection fraction less than 25% or with a previous history of emboli. Thromboembolic complications are treated with subcutaneous heparin (category B) in pregnancy and heparin or warfarin (category D) in the postpartum period. Neither heparin nor warfarin is

secreted into breast milk.[25] Enoxaparin (category B), a low-molecular weight heparin, has also been used in pregnancy, without complications in the mother or fetus.[23]

Cesarean section is recommended in these two cases with severe preeclampsia and fetal distress as indications. The recommended cardiac rehabilitation program are walking or biking approximately 1 mile daily. The prognosis is worse for black women, multiparous women, and those older than 30.27 Death is secondary to progressive heart failure, arrhythmia, or thromboembolism.[23,27,28]

CONCLUSION

Peripartum cardiomyopathy is a disease that is often fatal for the mother, but fetal outcome is quite good. Obstetrician and cardiologist should educate the patient about future pregnancies, breast feeding and contraception. Recovery of left ventricular function within six months is critical for good outcome of future pregnancies. If a patient regains normal ventricular function, another pregnancy may be considered, although there is no guarantee that the condition will not recurrent. Subsequent pregnancy will need a tight monitor to signs and symptoms of heart failure, especially in the late pregnancy and puerperium. A serial echocardiograms may be helpful. If left ventricular dysfunction persists, future pregnancies should be discouraged because it can lead to death secondary to progressive heart failure, arrhythmia, or thromboembolism.

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