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A Pregnant Patient with Aortic Regurgitation and Symptoms of Acute Heart Failure Caused by Peripartum Cardiomyopathy: A Case Report

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Abstract

Introduction: Knowledge of the risks of pregnancy with heart disease is important because the maternal mortality is much higher than the average. Peripartum cardiomyopathy (PPCM) is rare but it is one of major causes of maternal death. We experienced a pregnant patient with severe aortic regurgitation (AR) presented symptoms of acute heart failure. Her heart failure was not better after an emergency cesarean section and aortic valve replacement (AVR) therefore we think that PPCM caused her heart failure. **Case presentation:** A 35-year-old woman diagnosed as having severe AR became pregnant. No changes in the AR were apparent during pregnancy. However, the patient developed symptoms of acute heart failure at 37 weeks of gestation, and an emergency cesarean section was performed under general anesthesia. Her hemodynamic status worsened after the cesarean section, and AVR was performed. She was supported with percutaneous cardiopulmonary support (PCPS) after the operation. As recovery seemed to take longer than usual, we decided to implant a ventricular assist device (VAD). Her condition improved after VAD placement, but then she died from a cerebral infarction. In this case, the heart failure was an acute-onset even though AR was stable before and after the pregnancy, and the heart failure did not improve after AVR. Therefore, we concluded that PPCM, rather than AR caused her heart failure. **Conclusions:** We encountered a case of a pregnant patient with severe AR who presented with symptoms of acute heart failure caused by PPCM. The effect of AR to her heart failure could not be easily denied. This delayed the diagnosis of PPCM, which in turn delayed our decision to use a VAD. Therefore, PPCM should be considered when pregnant patients with heart disease present symptoms of heart failure.

Keywords

Aortic Regurgitation, Peripartum Cardiomyopathy, Acute Heart Failure,

Pregnancy in Patients with Heart Disease

1. Introduction

Knowledge of the risks of pregnancy with heart disease is important because maternal mortality is reportedly much higher than the average mortality in women with heart disease [1] [2]. Most pregnant patients with heart disease have congenital heart disease (66%), followed by valvular heart disease (26%), cardiomyopathy (7%), and ischemic heart disease (2%) [2]. Peripartum cardiomyopathy (PPCM) is a kind of cardiomyopathy occurring during the peripartum period. PPCM rarely occurs during pregnancy, but a pregnancy with a high risk of adverse outcomes for both the mother and baby is difficult to manage [2]. One of the diagnostic criteria of the National Institutes of Health for PPCM that was published in 2000 is "no evidence of preexisting heart disease prior to peripartum symptom onset" [3]. This shows that PPCM is not a well-known complication in pregnant women with heart disease. If pregnant women with heart disease present symptoms of heart failure, the heart disease is easily thought to be the cause of the symptom. However, we had a case of a pregnant patient with severe aortic regurgitation (AR) presented symptoms of acute heart failure. Her heart failure was not better after an emergency cesarean section and aortic valve replacement (AVR) therefore we suspected that PPCM caused her heart failure.

2. Case Presentation

A 35-year-old woman was diagnosed as having severe AR. She became pregnant 4 months after the diagnosis and was gravida 0, para 0, weighing 66 kg. She had no other significant family or personal history. Transthoracic echocardiography (TTE) before the pregnancy showed a slightly decreased left ventricular ejection fraction (LVEF) of 51%, and a dilated left ventricular diastolic diameter (LVDd) of 65 mm. No changes in TTE findings were observed, and no symptoms developed during the pregnancy. She was diagnosed as having pregnancy-induced hypertension at 34 weeks of gestation. At 37 weeks of gestation, she presented with symptoms of acute heart failure (New York Heart Association class IV), including dyspnea, anasarca, and a 1.8 kg increase in body weight (to 80.8 kg) in a week. Chest radiography revealed congestion in both hilar regions and lung fields (**Figure 1**). Electrocardiography revealed a heart rate of 140/min, sinus rhythm, and no ST-T wave change. TTE revealed a remarkable decline in LVEF with no asynergy, but no changes were found in the degree of AR or left ventricular dilatation (**Figure 2**). The TTE data are shown in **Table 1**. An emergency cesarean section was performed under general anesthesia.

During administration of anesthesia for emergency cesarean, she could not lie supine because of dyspnea, and 100 mg of thiopental was administered before transfer to the operating table. Rapid sequence intubation was smoothly performed by using 250 mg of thiopental and 50 mg of rocuronium, and the opera-

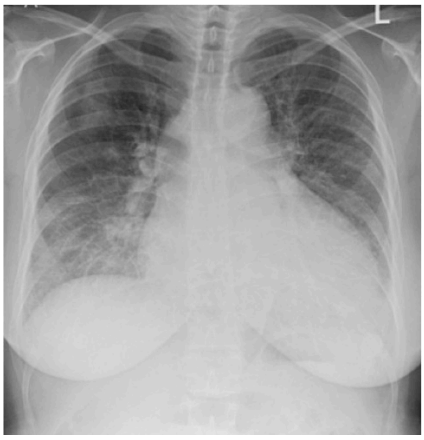

Figure 1. Chest radiograph on admission. Chest radiograph showing congestion of both hilar regions and lung fields.

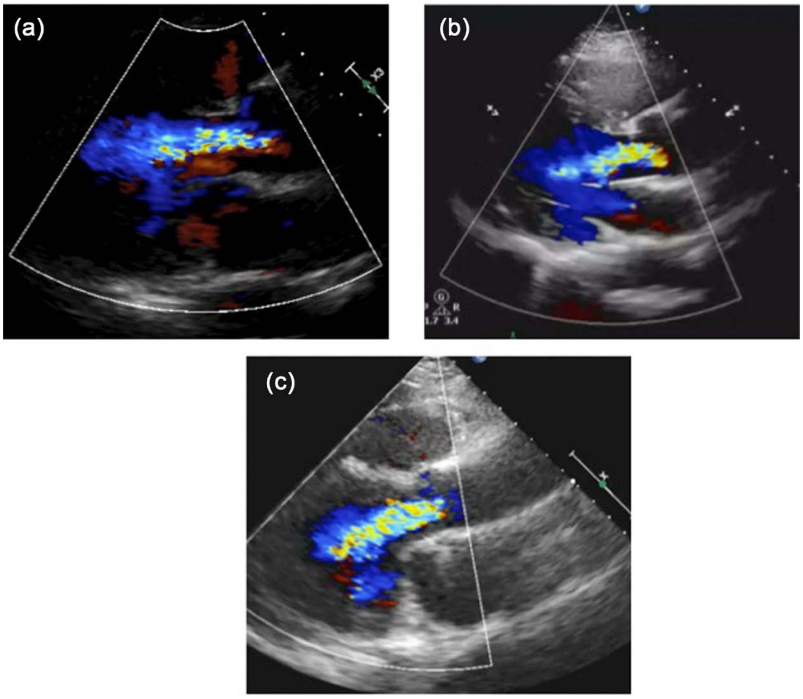

Figure 2. TTE data before and after the pregnancy and heart failure. (a) TTE before the gestation, (b) TTE at 20 weeks of gestation, and (c) TTE at 37 weeks of gestation (on admission); No changes were found in the degree of AR and left ventricular dilatation in (a), (b), and (c). A remarkable decline in LVEF was only found in (c).

Table 1. TTE data before and after the pregnancy and heart failure.

	LVEF (%)	LVDd/Ds (mm)	PHT (seconds)	Vena Contracta (mm)	RV (ml)	RF (%)
A	51	65/48	296	4.4	–	–
B	50	60/44	305	6.3	42	41
C	24	66/58	125	5.7	37	50

A: TTE before the gestation, B: TTE at 20 weeks of gestation, C: TTE at 37 weeks of gestation (on admission); RV and RF were not measured in "A". PHT in "C" was for reference purpose only because of tachycardia. LVDs: left ventricular systolic diameter, PHT: pressure half time, RV: regurgitant volume, RF: regurgitant fraction.

tion was promptly started. The baby was born 1 minute after the operation was started (Apgar scores were 1/6/10 at 1 min/5 min/20 min). Anesthesia was maintained with 0.8% sevoflurane before the delivery, and 200 μg of fentanyl and 150 mg of thiopental were given after the delivery. After the operation, we performed central venous catheterization and transferred her to the intensive care unit (ICU) with intubation. The anesthetic duration was 100 min, and blood loss including amniotic fluid was 1186 ml, so blood transfusion was not performed.

We started administration of dobutamine (5 μg/kg/min), norepinephrine (0.03 μg/kg/min), and carperitide (0.02 μg/kg/min). Extubation was performed in the ICU 3 days after admission. Her activities of daily living were carefully advanced to meeting with her family and nursing. Catecholamine doses were carefully reduced (dobtamin 5 to 3 μg/kg/min and norepinephrine 0.03 μg/kg/min to off). Symptoms of heart failure appeared again at 6 days after admission. Thus, we performed reintubation and pulmonary artery (PA) catheterization (PA pressure, 52/32 mm Hg) and started continuous hemodiafiltration (CHDF) for renal failure at 7 days after admission. We assumed that the decreased LVEF caused the symptoms of heart failure but could not find the cause of the notable decrease in LVEF. Moreover, we did not find any changes in the severity of the AR. However, as severe AR could adversely affect her heart failure, we performed aortic valve replacement (AVR) 8 days after admission.

Her hemodynamic status was maintained with dobutamine 5 μg/kg/min and norepinephrine 0.03 μg/kg/min before anesthesia induction for AVR. Induction was slowly performed with 1% sevoflurane and vecuronium 10 mg, and anesthesia was maintained with 1% sevoflurane before starting cardiopulmonary bypass (CPB). Anesthesia was maintained with propofol 300 mg/hour during CPB. Operative findings showed a tricuspid aortic valve with cusp degeneration. The aortic wall was thick and calcific in spite of her age, and aortitis was suspected (Figure 3). Pathological examination also revealed the evidence of inflammation in the aortic wall but no evidence of inflammation in the myocardium (Figure 4). Aortitis was strongly suspected. A biological valve was implanted. Her hemodynamic status was maintained with dobutamine 2.5 μg/kg/min and epinephrine 0.1 μg/kg/min after AVR. The anesthetic duration was 230 min, and

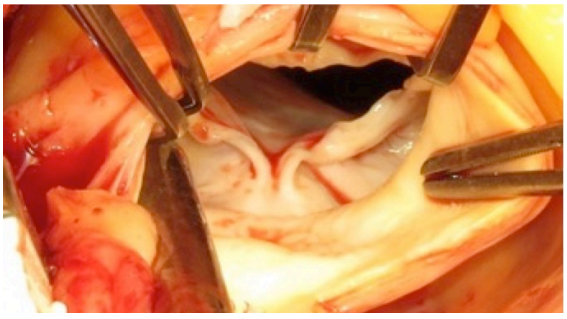

Figure 3. Operative findings. The aortic valve was triplet, and the valve cusp was degenerated. The aortic wall was thick and calcific. Pathological examination revealed no evidence of inflammation in the myocardium, aortic wall, or aortic valve.

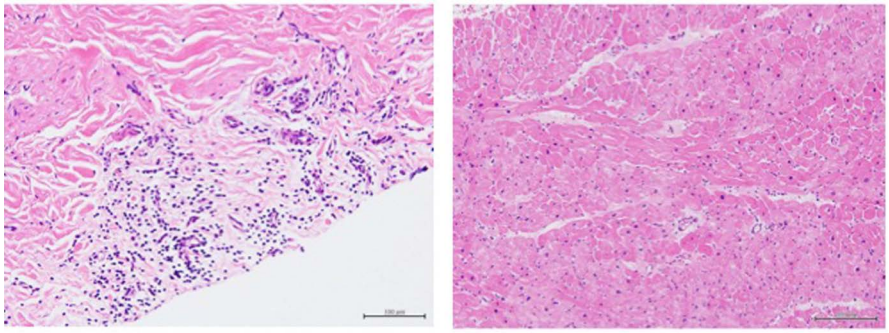

Figure 4. Pathological findings. (a) Evidence of inflammation in the aortic wall, (b) No evidence of inflammation in the myocardium.

blood loss was 60 ml, so blood transfusion was not required.

Her heart failure did not improve after AVR. LVEF remained at 20%, but laboratory data (creatine kinase 87 to 477 IU/L and lactate 1.3 to 10.1 mmol/L) indicated worsening. To stabilize her hemodynamic status, intra-aortic balloon pumping was started 9 days after admission and percutaneous cardiopulmonary support (PCPS) was inserted at 10 days after admission. Based on the operative findings, aortitis was suspected. Steroid pulse therapy was initiated (methylprednisolone 2000 mg × 1 day, 1000 mg × 2 days, 125 mg × 3 days, and 62.5 mg × 2 days). PA pressure was improved to 18/12 mm Hg, and her heart movement did not worsen after support with PCPS. We tried to wean her from PCPS, carefully reducing the flow from 4.3 to 1.5 L/min). No changes in laboratory data and heart movement were observed; thus, she was weaned from PCPS with dobtamin (5 μg/kg/min) and norepinephrine (0.01 μg/kg/min) at 14 days after admission. However, laboratory data (creatine kinase 240 to 14,272 IU/L and lactate 1.6 to 5.7 mmol/L) and central venous pressure (13 to 18 mmHg) showed worsened hemodynamic status. We started milrinone therapy at 0.125 μg/kg/min, and PCPS was reinserted at 16 days after admission. We decided to implant a ventricular assist device (VAD) at 17 days after admission because her recovery from heart failure seemed delayed.

For VAD implantation, her hemodynamic status was maintained with dobutamine (5 μg/kg/min). Anesthesia induction was slowly performed with 2% sevoflurane and vecuronium 8 mg and maintained with 2% sevoflurane before starting CPB. Anesthesia was maintained with propofol 200 - 300 mg/hour during CPB. After left VAD (LVAD) implantation, we tried to wean her from CPB. Right heart failure prevented the weaning of CPB, so we decided to implant a right VAD (RVAD). After implanting RVAD and LVAD, we could wean the patient from CPB. The anesthetic duration was 525 min, blood loss was 580 ml, and 16 units of packed red blood cells were transfused.

We started administration of NO (40 ppm) and prostaglandin (0.016 μg/kg/min). Her heart failure improved with VAD support. We started administration of carvedilol (started with 0.625 mg and then up to 10 mg) and olprinone (0.03 μg/kg/min) at 23 days after admission. The patient was weaned from

CHDF at 24 days after admission and from NO at 32 days after admission. RVAD was weaned at 40 days after admission (LVEF improved to 35.5%). However, she died from extensive cerebral infarction at 65 days after admission. A thrombus might have developed despite a prothrombin time-international normalized ratio of about 4.

3. Discussion

The number of pregnant patients with heart disease is increasing [1]. In women with heart disease, maternal mortality is reportedly 1%, which is much higher than the average of 0.07% [2]. Therefore, understanding the management of pregnancy in patients with heart disease is important. Most such patients have congenital heart disease (66%), followed by valvular heart disease (26%), cardiomyopathy (7%), and ischemic heart disease (2%) [2]. In pregnant patients with valvular heart disease, only 8.8% have AR [2]. Increasing heart rate and circulating plasma volume, and decreasing peripheral vascular resistance characterize hemodynamics in pregnant women. These changes are beneficial in AR by maintaining cardiac output and decreasing regurgitant volume. Therefore, women with asymptomatic AR have few risks in their pregnancies [4] [5]. AVR is not recommended to pregnant women with asymptomatic AR. AVR is recommended in women with symptomatic AR, low LVEF, and pulmonary hypertension before pregnancy. AVR during pregnancy is only recommended in AR with refractory heart failure [4] [5].

In this case, the patient had severe asymptomatic AR before she became pregnant. AVR was not needed before and after her pregnancy according to the criteria. No symptoms of heart failure were observed until 37 weeks of gestation, at which time her degree of AR had not changed. It was difficult to conclude that the AR caused the acute heart failure. We started to consider the possibility of PPCM. However, we could not completely deny the adverse effect of AR to the heart failure. We reviewed this case as follows: 1) AR was stable before and after the pregnancy; 2) her heart failure was acute, not chronic; 3) the main cause of the heart failure was the decreased LVEF; 4) if heart failure was caused by acute exacerbation of AR, the degree of AR should have worsened, but was not; 5) the heart failure did not improve after AVR until support with a VAD; 6) no other diseases were found (including ischemic heart disease, autoimmune heart disease, and inflammatory heart disease). Therefore, we determined that PPCM played a role in this case rather than AR.

PPCM is an idiopathic cardiomyopathy presenting with heart failure secondary to LV systolic dysfunction toward the end of pregnancy or in the months following delivery. It is a diagnosis of exclusion when no other cause of heart failure is found. The LV may not be dilated, but the LVEF is nearly always reduced below 45% [6]. PPCM is rare but a major cause of maternal death, accounting for 4% - 25% of cases [7] [8] [9]. Recent reports suggested prevalence of PPCM in the United States of 1 per 2000 live births and 1 in 20,000 live births in Japan [7] [8]. Risk factors associated with PPCM include age, gravidity, Afri-

can origin, twin pregnancy, smoking, obesity, hypertensive disorder, and pregnancy-induced hypertension. Hypertensive disease and pregnancy-induced hypertension are major complications of PPCM, with an incidence rate of approximately 40% [8] [9]. The cause and mechanism of the pathogenesis of PPCM remain unknown; however, many hypotheses have been proposed, including nutritional disorders and viral infectious agents. No specific treatment has been found to alter the morbidity of PPCM significantly, and only supportive treatment of heart failure is performed [7] [8] [9] [10].

In this case, we initially ruled out PPCM because she had severe AR before pregnancy. Our treatment strategy was stepwise for AR but it was ineffective. Only a few cases of PPCM complicating heart disease in pregnancy have been reported. The reports suggested that PPCM should be diagnosed in pregnant women with heart disease if they show symptoms of heart failure not caused by a preexisting heart disease [11] [12] [13]. Therefore, our treatment strategy should have been maximum for PPCM. For example, if we had decided to implant a VAD before using PCPS, she might have survived. Another aspect of this case could have been managed better. The cesarean section performed at 37 weeks of gestation should have been performed after 34 weeks, when she was diagnosed with pregnancy-induced hypertension.

4. Conclusion

We had a case of a pregnant patient with severe AR who presented with symptoms of acute heart failure caused by PPCM. The effect of the severe AR to her heart failure could not be easily denied. This delayed the diagnosis of PPCM, which in turn delayed our decision to use a VAD. We should have used a VAD earlier. If pregnant patients with a heart disease present symptoms of heart failure, PPCM should be considered and the treatment strategy should be maximum including a VAD.

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