Complete Myocardial Function Recovery with ECMO in a Woman Presenting with Cardiogenic Shock during Peripartum Period

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Abstract: Peripartum cardiomyopathy is a potentially life-threatening cause of heart failure (HF) that affects women toward the end of pregnancy or in months after delivery. Treatment is similar to the treatment for HF with reduced ejection fraction (EF). Most women make full myocardial function recovery within 6 months on conventional HF therapy. In rare instances, catastrophic presentations may occur with hemodynamic instability requiring the use of mechanical support. Because of the small patient population, limited information is available regarding the recovery of myocardial function in women who received mechanical support. We present a case of a woman in her peripartum period who presented with cardiogenic shock and made complete myocardial function recovery after 4 days of extracorporeal membrane oxygenation (ECMO). Our patient's EF at the time of catastrophe was 5–10%, which improved to 60% on day 4 on ECMO. Keywords: cardiomyopathy, postpartum, heart failure, mechanical circulatory support, extracorporeal membrane oxygenation (ECMO).

OVERVIEW

Although heart failure (HF) associated with pregnancy was originally published in the 1800s by Virchow et al. (1,2), it was not until 1971 when the term peripartum cardiomyopathy (PPCM) was introduced in the literature (3). PPCM is defined as development of HF toward the end of pregnancy or in the months after delivery and an ejection fraction (EF) <45% in the absence of previously known structural heart disease (4). The incidence of PPCM in United States is thought to be approximately one in 1,000 to 4,000 live births (5). More than 50% of the cases occur in women aged >30 years and the incidence in United States is higher in black women (5,6). The etiology remains unclear and is thought to be multifactorial. Most women present with dyspnea, cough, orthopnea, paroxysmal nocturnal dyspnea, and pedal edema. Because these symptoms are similar to those observed in normal pregnancy, diagnosis often gets delayed or even missed. In rare instances, patients may present with severe respiratory distress and acute cardiogenic shock necessitating pharmacological and mechanical support.

Treatment of PPCM is similar to the treatment for HF with reduced EF. The goal of medical therapy is focused toward controlling volume status, neutralizing maladaptive neurohormonal responses, and preventing thromboembolic and arrhythmic complications (7). The myocardial dysfunction is improved or normalized in approximately 65 and 45% of women, respectively, by 6 months from diagnosis with medical therapy (8,9). There are very limited studies on when to wean off the HF medications in patients with persistent recovered myocardial function. A stepwise weaning with close clinical and echocardiographic monitoring is currently recommended.

In patients who present with severe myocardial dysfunction and rapid hemodynamic deterioration, inotropes and mechanical circulatory support (i.e., intra-aortic balloon pump, left ventricular and/or biventricular assist devices, extracorporeal membrane oxygenation [ECMO]) have been used successfully. In cases when recovery does not occur, cardiac assist devices may serve as a bridge to therapy or transplantation. However, very limited information is available on the outcomes of women who have
received mechanical circulatory support and they are mostly based on case reports and small case series. Here, we present a patient with PPCM whose myocardial function recovered after 4 days of ECMO.

**DESCRIPTION**

A 25-year-old gravida 5, para 3 female with a recent history of fetal demise 4 months ago presented at the 12th week of pregnancy with acute onset of vomiting pink frothy material and chest discomfort. Initial blood gas showed a pH of 6.88 and a pCO₂ of 103 on 100% non-rebreather. She was emergently intubated. Chest radiograph showed diffuse pulmonary edema (Figure 1). Electrocardiogram showed ST segment elevation in the inferior and anterior leads (Figure 2). Bedside echocardiography showed severely reduced left ventricular function with an EF of <10% and hyperdynamic right ventricular function. Computed tomography scan of the chest was negative for pulmonary embolism. Emergent cardiac catheterization showed clean coronary arteries (Figure 3) and EF of 5–10%. She was started on a dobutamine drip and subsequently became hypotensive and suffered two episodes of cardiac arrest because of pulseless electrical activity. After successful resuscitation, she remained hypotensive while on multiple pressors and was severely hypoxic while on high positive end-expiratory pressure and 100% FiO₂. Venoarterial ECMO via femoral cannulation with flow rate of 3.5–4 L/min was initiated to provide hemodynamic support. She was started on a heparin drip to keep activating clotting time and/or activated partial thromboplastin time level between 160–180 seconds and 45–50 seconds, respectively, for maintenance of

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**Figure 1.** Chest radiograph showing diffuse pulmonary edema.

**Figure 2.** Electrocardiogram showing acute ST-segment elevation in inferior and anterior leads.
a clot-free oxygenator circuit and to stabilize the patient hemostatically. Her mixed venous oxygen saturation was maintained to >60–80%. The dobutamine and epinephrine drips were titrated down with the goal cardiac index of >2.2 L/min/m². The official echocardiogram on day 1 showed severely reduced systolic function with estimated EF in the range of 10–15% (Figures 4A and 4B). Over the subsequent days, the patient improved hemodynamically and was able to be weaned off of the inotropes and pressor support. Repeat echocardiography on day 4 showed an EF of 60% (Figures 4C and 4D). ECMO was able to be weaned and decannulated that day and the patient was extubated the following day. Transvaginal ultrasound showed products of conception in uterus. She underwent dilation and curettage for incomplete abortion on day 7. Follow-up echocardiography on day 14 showed normal systolic function with EF of 65%. She was started on Carvedilol and Lisinopril and was eventually discharged home.
COMMENT

ECMO has shown to serve as a viable treatment for short-term circulatory support in hemodynamically unstable patients with cardiogenic shock from all etiologies. Survival rates for ECMO used for cardiac indication vary from 23 to 71% with lowest for myocarditis and highest in case of cardiac arrest (10). However, there are scarce published data of its use in patient with PPCM. We believe that our case provides a unique example of a patient who made full myocardial function recovery after being on ECMO alone for 4 days.

Our patient was in the 12th week of her pregnancy when she presented to us with cardiogenic shock and severe myocardial dysfunction. She had no known history of structural heart disease. We believe that her cardiomyopathy was related to her history of intrauterine fetal demise that had occurred 4 months before this presentation. Interestingly in the literature, cases of PPCM have only been described in patients who gave live birth. The most widely accepted definition of PPCM does not specify whether the delivery need to be related to live birth or fetal demise. A literature search on PubMed failed to provide any studies specifying whether the delivery related to development of PPCM was a live birth or fetal demise.

Our case serves an example of the vital role that venoarterial ECMO plays in patients with refractory cardiogenic shock who have a high risk of death despite conventional therapy. This case provided a rare example of woman in her peripartum period who made complete myocardial function recovery after being on venoarterial ECMO for only 4 days.

REFERENCES