CASE REPORT

A Rare Case of Peripartum Cardiomyopathy Manifesting With Thromboembolic and Cardiogenic Shock: A Case Report

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ABSTRACT

Peripartum cardiomyopathy (PPCM) is a rare type of heart failure with an unknown cause. It typically presents with signs and symptoms of congestive heart failure, and rarely with thromboembolic complications and cardiogenic shock, which can lead to life-threatening conditions. This report presents a case of PPCM in a 25-year-old woman who was admitted to our hospital, 15 days after vaginal birth delivery. The patient presented with breathlessness, arm swelling, and coughing up blood. During examination, an intracardiac thrombus was discovered. It migrated and caused embolism, leading to deep vein thrombosis (DVT) and pulmonary edema. The patient experienced cardiogenic shock due to the decreasing heart pumping capability of PPCM.
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INTRODUCTION

Peripartum cardiomyopathy (PPCM) is a rare type of heart failure with an unknown cause. It occurs in approximately 1 in 4000 live births. Typically, it develops during late pregnancy or the early postpartum period. in women who were previously healthy (1). PPCM usually starts with initial symptoms of heart failure and, less commonly, may lead to complications like blood clots and cardiogenic shock. We present a rare case of PPCM in a previously healthy woman who experienced blood clots and cardiogenic shock as her initial symptoms.

CASE REPORT

A 25-year-old woman with para 1, live 1 (P1 L1) was referred to our emergency department presenting with dyspnea and hemoptysis, 15 days after labour by vaginal delivery. The dyspnea had lasted since 8 months of gestation. There was no history of cardiovascular disease. Her vital signs showed unstable hemodynamic with a low blood pressure of 105/70 mmHg on norepinephrine 100 ng/kg/min and dopamine 3 mcg/kg/min, a pulse rate of 96 beats-per-minute, a respiratory rate of 26 breaths-per-minute, and an oxygen saturation of 99% while receiving oxygen through a 6-L nasal cannula. Physical examination revealed a jugular vein pressure elevation and rales auscultated in 1/3 basal of bilateral lungs. Her extremities were non-edematous. Electrocardiography showed sinus tachycardia with a normal axis. Laboratory tests revealed thrombocytopenia. Chest-x-ray showed cardiomegaly. Transthoracic-echocardiography (TTE) on admission showed left ventricular (LV) dilatation with eccentric left ventricular hypertrophy (LVH), ejection fraction (EF) of 41% and intra-cardiac LV thrombus measuring 1.35x0.96 cm (Figure 1). She was diagnosed with peripartum cardiomyopathy (PPCM) with congestive heart failure, cardiogenic shock and LV thrombus. The patient was initially treated with diuretics, anticoagulants and bromocriptine.

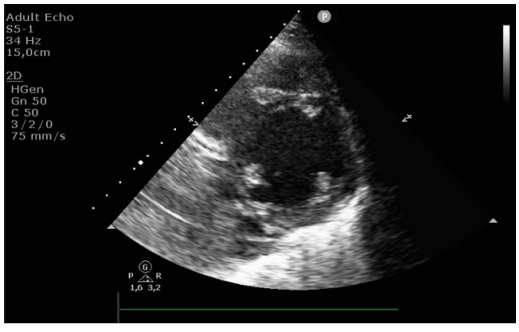

Figure 1: TTE showed LV dilatation with eccentric LVH, an EF of 41% and an intracardiac LV thrombus of 1.35×0.96 cm..

Furthermore, the patient reported pain, redness, and swelling in her right arm. A compression ultrasonography (CUS) was performed on the right axillary vein, which showed negative results due to the presence of a thrombus, most likely a right upper extremity superior deep vein thrombosis (DVT) (Figure 2).

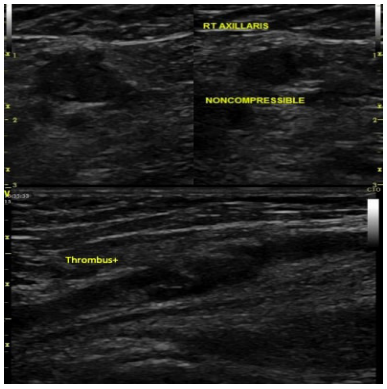

Figure 2: CUS was performed on the right axillary vein, which showed negative results (noncompressible) due to the presence of a thrombus.

On the 10th day of hospitalization, the patient reported worsening breathlessness and hemoptysis. The D-dimer test was elevated (15250 ng/mL), indicating a blood clot problem. A CT-pulmonary-angiography (CTPA) revealed a total occlusion in segments 8, 9 and 10 of the left pulmonary arteries, inferior lobe, due to pulmonary embolism (PE) (Figure 3). Revascularization was performed in this patient using catheter-directed thrombolysis (CDT) with alteplase 10 mg for 15 hours. The evaluation showed restoration of blood flow with a partial thrombus in the left pulmonary artery (Figure 4).

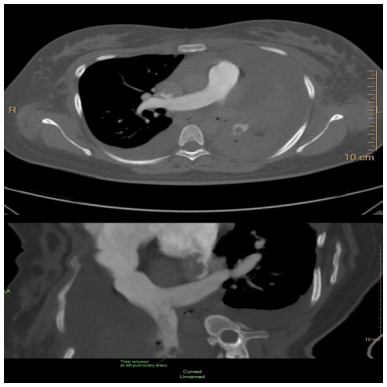

Figure 3: Axial-image from the CTPA showed a total occlusion in segments 8, 9 and 10 of the left pulmonary arteries, inferior lobe.

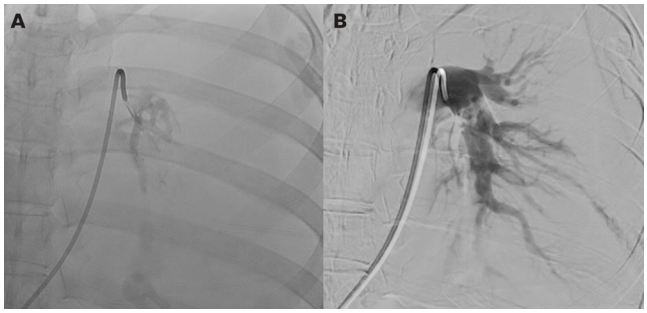

Figure 4: A) CDT was performed with alteplase 10 mg for 15 hours. (B) The blood flow restored with a partial thrombus in the left pulmonary artery.

The significant pleural effusion in the left lung was discovered on day 16 of hospitalization, presumably caused by a PE. A pleural fluid drainage was conducted and 1500cc of serous fluid was effectively removed.

The patient showed clinical improvement and was discharged from the hospital after 20 days of admission. The patient was advised to take rivaroxaban as an anticoagulant. Follow-up TTE and CTPA examinations have been scheduled for 3 months later.

DISCUSSION

PPCM is a rare form of heart failure occurring in late pregnancy or early postpartum, characterized by heart failure development, absence of an alternative cause, and LV systolic dysfunction with an ejection fraction consistently below 45%. The etiology of PPCM is unknown, with suggested factors including African descent, age, pregnancy-related hypertension, multiparity, multiple gestations, obesity, chronic hypertension, and prolonged tocolytic use (1). Thromboembolism rates in PPCM are notably higher than in many other cardiomyopathies, reported at 6.6% in affected U.S. women. Mortality rates range from 7% to 50%, often due to complications such as cardiogenic shock, thromboembolism, and arrhythmias (2). PPCM presents initially with signs and symptoms of heart failure and rarely with thromboembolic complications and cardiogenic shock.

Our patient, presenting with dyspnea persisting since her pregnancy eight months ago and recent postpartum coughing up of blood, arrived at the emergency department. Despite no prior cardiovascular issues, vital signs indicated unstable hemodynamics, with low blood pressure, an elevated heart rate, and other abnormalities. Physical examination revealed jugular vein pressure elevation and lung abnormalities. Diagnostic tests confirmed PPCM, congestive heart failure, cardiogenic shock, and a left ventricle blood clot. Initial treatment involved diuretics, anticoagulants, and bromocriptine. However, the patient's condition deteriorated, revealing a PE on a CT-scan and the CDT addressed this issue. DVT in the right arm led to pleural effusion in the left lung due to the risk of embolization, where a clot traveled to the pulmonary arteries, causing PE. PE increased pulmonary vascular pressure, resulting in fluid leakage into the pleural space. In addition, it is also noteworthy that the pleural effusion may be caused by pulmonary infarction and inflammation. After 20 days of hospitalization, she was discharged with a prescription for rivaroxaban and scheduled follow-up exams.

PPCM presents various complications, including common occurrences of heart failure such as cardiogenic shock, cardiac arrhythmias, and thromboembolic complications, with reported incidences in the U.S. ranging from 6.6% to 50% (3). The elevated risk of

thromboembolism in PPCM is attributed to factors like left ventricular dilatation, endothelial injury, immobility, and postpartum hypercoagulability. The decrease in cardiac output observed in hypokinetic cardiomyopathies, like PPCM, further amplifies the likelihood of thrombosis and embolism types, encompassing DVT, PE, intracardiac thrombi, arterial embolism, and ischemic stroke (4). Management of thromboembolic complications in PPCM aligns with approaches used for other etiologies, as exemplified in a case where catheter-directed thrombolysis with alteplase for 15 hours was employed for revascularization.

Cardiogenic shock in PPCM arises from a compromised ability to pump blood effectively, primarily due to a weakened and dilated left ventricle with reduced EF. This results in diminished oxygen delivery to vital organs. In PPCM, blood clots may form within the weakened left ventricle, potentially causing blockages in other parts of the body, exacerbating the condition and leading to PE. The recommended management for PPCM with severe acute heart failure or cardiogenic shock involves optimizing preload, respiratory support for optimal oxygenation, using inotropes and/or vasopressors, urgent delivery, and considering mechanical circulatory support if necessary. Further research is needed to explore the true incidence of PPCM presenting with thromboembolic events and cardiogenic shock (5). In this case, the patient was treated with norepinephrine 100 ng/kg/min, dopamine 3 mcg/kg/min, and a 6-L nasal cannula, resulting in a blood pressure of 105/70, a respiratory rate of 20 breaths per minute, and an oxygen saturation of 99%.

The recovery of cardiac function from PPCM is greater than that in men and non-peripartum cardiomyopathy, and recovery frequently occurs within 3 to 6 months. Our patient showed clinical improvement and was discharged from the hospital after 20 days of admission.

CONCLUSION

Patients with PPCM may rarely face thromboembolic events and cardiogenic shock. The elevated risk of thromboembolism in PPCM is attributed to factors such as left ventricular dilatation, endothelial injury, immobility, and postpartum hypercoagulability. Cardiogenic shock in PPCM results from the heart's compromised ability to pump blood effectively. Further research is necessary to accurately determine the incidence of PPCM presenting with thromboembolic events and cardiogenic shock.

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