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Short Communication

Intensive Care Strategies For Acute Heart Failure In Peripartum Cardiomyopathy: A Multimodal Approach

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Abstract

Summary. This article addresses the comprehensive management of emergency conditions arising from peripartum cardiomyopathy (PPCM), one of the most severe complications of the gestational period. It provides a detailed analysis of the clinical presentation, contemporary diagnostic criteria, and differential diagnostic aspects of acute heart failure. The study emphasizes the significance of timely and adequate assessment of the severity of cardiopulmonary failure within intensive care settings, along with the verification of the diagnosis through instrumental investigations. Furthermore, the article analyzes clinical protocols for the multidisciplinary management of patients with PPCM. Emergency therapeutic strategies for hemodynamically unstable patients are reviewed, specifically evaluating the role and efficacy of various pharmacological agents, including catecholamines, diuretics, levosimendan, bromocriptine, and anticoagulants.

Keywords: severe peripartum cardiomyopathy; differential diagnosis; acute heart failure; intensive care and resuscitation; pharmacotherapy; inotropic support

Introduction:

According to the definition proposed by the European Society of Cardiology (ESC) Working Group, peripartum cardiomyopathy (PPCM) is an idiopathic cardiomyopathy characterized by heart failure secondary to left ventricular (LV) systolic dysfunction that develops toward the end of pregnancy or within several months postpartum, in the absence of any other identifiable cause of heart failure [1]. In patients with PPCM, left ventricular dimensions may remain within normal limits; however, the left ventricular ejection fraction (LVEF) is almost invariably reduced to below 45%.

The prevalence of this cardiomyopathy varies considerably depending on geographic region. In the United States, the incidence of PPCM ranges from 1 case per 968 to 4,350 live births [2,4]. Outside the United States, the epidemiology of this condition remains insufficiently documented [7]. According to reports from African and Asian countries, the incidence is estimated at approximately 1 case per 1,000 live births [9,12]. In Haiti, PPCM occurs in approximately 1 out of every 300 deliveries, which has been attributed to racial differences, nutritional deficiencies, and the high prevalence of preeclampsia [7]. The highest reported incidence has been documented in Northern Nigeria, reaching 1 case per 100 deliveries [13]. Analysis of data from the Republic of Uzbekistan suggests that the actual number of women affected by PPCM may be substantially higher (Abdullayev T.A., Tsoy I.S., 2023).

The clinical course of PPCM ranges from mild manifestations to severe heart failure, including the development of pulmonary edema and cardiogenic shock (CS). These conditions pose a significant threat to both maternal and fetal survival. Heart failure may progress rapidly, often worsening within days after the initial symptoms appear. Furthermore, the clinical course of PPCM is frequently complicated by various cardiac arrhythmias. In combination with structural and functional left ventricular abnormalities, these rhythm disturbances substantially increase the risk of sudden cardiac death (SCD).

According to a large nationwide study conducted in the United States involving more than 34,000 patients with PPCM, severe adverse maternal outcomes developed in approximately 13% of cases, including in-hospital mortality, cardiac arrest, acute pulmonary edema, thromboembolic complications, the need for heart transplantation, and implantation of cardioverter-defibrillators or permanent pacemakers [11]. The incidence of cardiogenic shock in PPCM increased from 1.0% in 2014 to 4.0% in 2021.

The primary objective of the present review is to highlight contemporary approaches in intensive care and resuscitation management of patients with PPCM complicated by life-threatening conditions. The general principles of treatment for PPCM patients presented herein are based on the practical recommendations for the management of severe PPCM developed in 2019 by the ESC Heart Failure Association Study Group on Peripartum Cardiomyopathy [14]. Due to the extensive scope of the material, the review is divided into two parts. The first part discusses general concepts of PPCM, clinical manifestations of acute heart failure (AHF) in PPCM, patient assessment, and general principles of management. The second part addresses modern treatment strategies for severe PPCM, mechanical circulatory support devices (MCSD), outcomes of heart transplantation, approaches to managing cardiac arrhythmias, prevention of sudden cardiac death, and specific considerations regarding obstetric management in this pathology.

Acute Heart Failure

Acute heart failure (AHF) as a complication of peripartum cardiomyopathy represents a highly challenging clinical condition associated with substantial mortality [10,12]. In the early stages of severe acute PPCM, administration of inotropic agents and mechanical circulatory support devices is frequently required. Following initial stabilization, recovery of left ventricular function is observed in many patients [11–13]. Nevertheless, in some individuals, despite aggressive and intensive therapy, long-term mechanical circulatory support or heart transplantation may become necessary. The degree and timing of left ventricular functional recovery remain unpredictable, and during the first months after diagnosis patients remain at increased risk of ventricular tachyarrhythmias, cardiac arrest, and sudden cardiac death [14]. In addition, severe reduction in left ventricular ejection fraction is frequently complicated by intracardiac thrombosis, which constitutes one of the major causes of thromboembolic events [14–18].

Clinical Manifestations of Severe Peripartum Cardiomyopathy

Most patients with severe peripartum cardiomyopathy present with symptoms characteristic of acute heart failure. These manifestations are associated with venous congestion and impaired cardiac output. Since the early signs and symptoms of acute heart failure in PPCM often mimic physiological changes occurring during pregnancy and the postpartum period, establishing an accurate diagnosis requires considerable clinical expertise and vigilance. PPCM should be suspected in patients presenting with paroxysmal nocturnal dyspnea, nocturnal cough, hemoptysis, chest pain, and hepatomegaly [12].

Differential diagnosis of severe PPCM should include myocarditis, previously existing cardiomyopathies, acquired or congenital heart defects, myocardial infarction, pulmonary embolism, and amniotic fluid embolism [9].

Assessment of Severe Peripartum Cardiomyopathy

As in all cases of acute heart failure, the initial assessment of patients with severe PPCM consists of two major components: evaluation of the presence and severity of cardiopulmonary failure and confirmation of the diagnosis using additional diagnostic modalities.

Assessment of cardiopulmonary failure is of paramount importance because it directly determines further therapeutic strategies. Patients should be admitted to the intensive care unit when the following criteria are present, as these indicate the existence and severity of cardiopulmonary insufficiency [9]:

- Hemodynamic instability (systolic arterial pressure <90 mmHg, heart rate >130 beats/min or <45 beats/min);
- Respiratory failure (respiratory rate >25/min, oxygen saturation [SpO_2] <90%);
- Signs of tissue hypoperfusion associated with impaired cellular oxygen metabolism, including elevated serum lactate (>2.0 mmol/L), central venous oxygen saturation <60%, altered mental status, cold clammy skin, and oliguria (<0.5 mL/kg/h) [23].

Management of Patients with Severe Peripartum Cardiomyopathy

The principal therapeutic measures in patients with PPCM complicated by acute heart failure include the following [20]:

- a multidisciplinary management approach prioritizing both maternal and fetal well-being;
- exclusion of embryotoxic medications during pregnancy, including angiotensin-converting enzyme inhibitors (ACE inhibitors) and mineralocorticoid receptor antagonists;
- avoidance of the aforementioned drug classes during breastfeeding;
- consideration of bromocriptine as adjunctive therapy in severe heart failure (2.5 mg twice daily for 2 weeks, followed by 2.5 mg once daily for 6 weeks according to standard recommendations);
- administration of anticoagulant therapy with heparin for prevention of thromboembolic complications in patients with LVEF ≤35% or those receiving bromocriptine, provided no contraindications exist;
- consideration of levosimendan rather than catecholamines as the first-line inotropic agent in cardiogenic shock (0.1 μg/kg/min for 24 hours);
- urgent transfer of the patient to an intensive care

unit;
- early evaluation of the need for mechanical circulatory support;
- implementation of preventive measures against sudden cardiac death, including urgent consideration of a wearable cardioverter-defibrillator in patients with LVEF ≤35%.

Because robust evidence-based data regarding PPCM treatment remain limited, the initial management of these patients generally follows the principles applied in acute heart failure of other etiologies [30]. In clinical practice, multidisciplinary treatment protocols are commonly used because of their convenience and informational value [12,24]. Figure 2 summarizes the recommended therapeutic algorithm for patients with acute PPCM [15,17]. It should be emphasized that the management of severe PPCM differs substantially from treatment strategies used in hemodynamically stable patients [24,26,28].

Initial Management of Patients with Cardiopulmonary Failure and/or Hemodynamic Instability in Severe PPCM

Patients presenting with signs of cardiopulmonary insufficiency and/or cardiogenic shock require immediate emergency medical care in an intensive care or resuscitation setting. Emergency management includes five major objectives: optimization of preload and oxygenation, restoration of hemodynamic stability using inotropes and/or vasopressors, urgent delivery when indicated, consideration of bromocriptine therapy (2.5 mg twice daily for 2 weeks followed by 2.5 mg once daily for 6 weeks), and prevention of thromboembolic complications.

Optimization of preload depends on the clinical situation and may involve either infusion therapy or administration of diuretics. In the absence of clear signs of fluid overload, particularly in patients with intravascular volume depletion secondary to significant blood loss during delivery or excessive diuretic therapy, a fluid challenge test is recommended (250–500 mL administered over 15–30 minutes). Intravenous vasodilators such as nitrates may be administered in patients with systolic blood pressure >110 mmHg.

The goal of oxygenation therapy is to achieve peripheral oxygen saturation (SpO_2) >95%. Non-invasive ventilation reduces respiratory insufficiency and may prevent progression to invasive mechanical ventilation. Endotracheal intubation and mechanical ventilation should be considered in patients with altered mental status or persistent hypoxemia.

In the presence of cardiogenic shock, rapid restoration of hemodynamic stability is essential to prevent irreversible organ damage. Although inotropic agents and vasoconstrictors may be required, catecholamines can produce adverse effects in severe heart failure and cardiogenic shock [29]. Experimental data and clinical observations suggest that catecholamines may be less effective in PPCM due to underlying metabolic disturbances [27]. Furthermore, because pregnancy itself is associated with systemic vasoconstriction, vasopressor administration may be less appropriate [11]. Therefore, catecholamines should be avoided whenever possible and reserved only for critically necessary situations.

Unlike dobutamine and norepinephrine, levosimendan does not increase myocardial oxygen demand and is therefore considered a preferable inotropic agent. It is administered as a continuous infusion at 0.1 μg/kg/min for 24 hours without a loading dose [18]. A small clinical study involving 28 patients demonstrated that levosimendan contributed to rapid hemodynamic stabilization and significant reduction of pulmonary congestion in PPCM [27]. However, its efficacy in acute heart failure associated with PPCM has not yet been definitively established because randomized clinical trials are lacking [12]. When levosimendan is unavailable, alternative inotropic agents such as milrinone or β_1-adrenergic agonists (dopamine or dobutamine) may be used [5].

Adjunctive therapy with bromocriptine, a prolactin secretion inhibitor, has demonstrated promising results in experimental and clinical studies [18,23]. The standard initial dose is 2.5 mg twice daily, although higher doses may occasionally be used to achieve greater suppression of prolactin levels [19].

Because thromboembolic complications have been reported during bromocriptine therapy, particularly at higher doses, its administration should always be combined with prophylactic anticoagulation using heparin [14,24]. Anticoagulant therapy is indicated in all patients with acute PPCM and significantly reduced left ventricular systolic function (LVEF ≤35%). Reduced LVEF together with the procoagulant state associated with pregnancy markedly increases the risk of thromboembolic events. Additionally, atrial fibrillation and intracardiac thrombi constitute independent indications for anticoagulant therapy [6,30].

In summary, low-molecular-weight heparins and unfractionated heparin remain the preferred anticoagulants in the intensive care management of pregnant women with PPCM [16]. In patients whose hemodynamic instability persists despite intensive treatment, urgent delivery should be performed regardless of gestational age. In such cases, cesarean section under combined spinal-epidural anesthesia with participation of a multidisciplinary team is recommended.

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