Peripartum cardiomyopathy — challenges of diagnosis and management. Stay alert and implement BOARD treatment

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A 27-year-old woman at the 27th week of pregnancy presented with preeclampsia (hypertension + proteinuria), which shortly progressed into eclampsia. Antihypertensive medications (labetalol, nifedipine, methyldopa) and intravenous magnesium sulfate were implemented. Four days later, she experienced a hypertensive crisis and loss of consciousness that necessitated an emergency C-section. Due to resistant hypertension and signs of heart failure (HF), she was transferred to the cardiac intensive care unit. Echocardiography showed decreased left ventricular ejection fraction (LVEF, 45%) with impaired global longitudinal strain, pericardial and bilateral pleural effusions (Figure 1). N-terminal pro-B-type natriuretic peptide (NT-proBNP) was increased (8661 pg/ml). Cardiac magnetic resonance (CMR) demonstrated myocardial edema without other signs of myocarditis. Within two days postpartum, the blood pressure and HF symptoms stabilized with β-blockers, furosemide, urapidil, and nitroglycerin. Due to suspected peripartum cardiomyopathy (PPCM), we implemented bromocriptine and anticoagulation along with oral electrolyte supplementation.

Despite the initial improvement, on the 7th day postpartum, the patient experienced cardiac arrest due to ventricular tachycardia (VT) treated with a successful cardiopulmonary resuscitation with defibrillation. Intravenous magnesium supplementation was restarted due to serum magnesium level at the lower limit of the normal range.

Further hospital stay was unremarkable. After one month, CMR revealed no edema or fibrosis, with LVEF of 58% and NT-proBNP of 61 pg/ml. Given all the data and clinical course, the final diagnosis of PPCM was confirmed. A cardioverter-defibrillator was not implanted due to a likely reversible VT cause (electrolyte disturbances and acute phase of cardiomyopathy).

Identifying the etiology of acute HF in a peripartum woman is critical for further management and prenatal counseling. Traditionally, PPCM is a diagnosis of exclusion that meets three criteria: HF towards the end of pregnancy or within months following delivery, LVEF <45%, and the absence of other identifiable causes of HF [1].

In this patient, the hypertensive crisis could have been considered a cause of acute HF, which by definition excludes the diagnosis of PPCM. However, in recent years a link between PPCM and preeclampsia was established. The pathogenesis of PPCM is multifactorial, including genetic predisposition, inflammation, oxidative stress, and hormonal dysregulation. In particular, oxidative stress promotes abnormal cleavage of prolactin into the antiangiogenic subfragment. The damage to the vascular endothelium caused by these prolactin-derived factors can impair cardiomyocyte metabolism and manifest as PPCM [2, 3]. Similarly, preeclampsia is a disease of vascular endothelium with increased levels of antiangiogenic factors produced by the placenta [3]. This preliminary evidence highlights the common etiologies of these two diseases.

According to the European Society of Cardiology EURObservational Research Programme registry, preeclampsia coexisted in

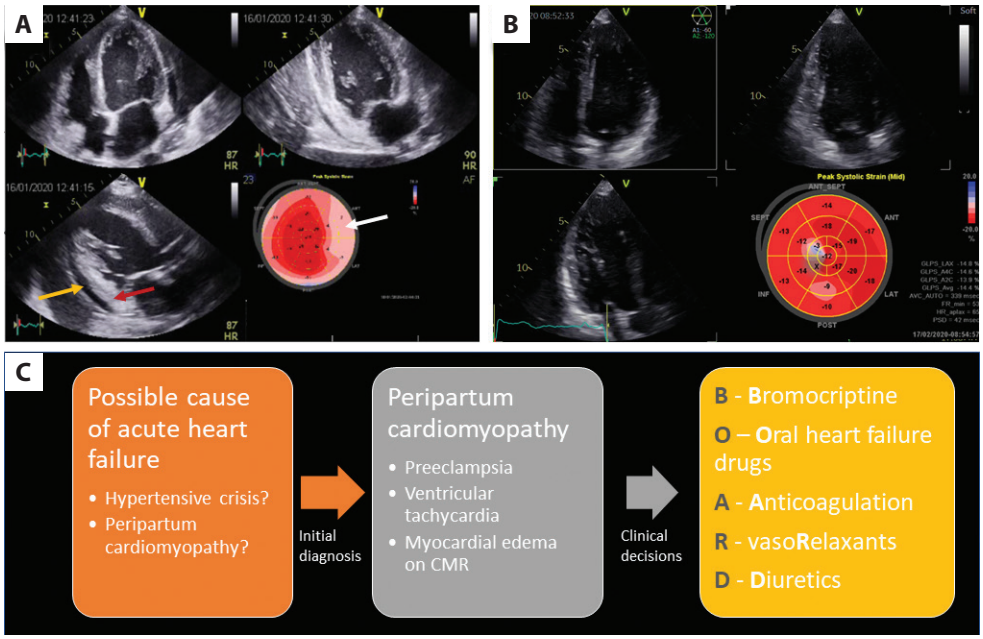

Figure 1. A. Initial echocardiography with impaired regional systolic function and global longitudinal strain of –13.8% (the white arrow — impaired global longitudinal strain of the lateral wall; the yellow arrow — pericardial effusion; the red arrow — the thickened edematous inferolateral wall). **B.** Follow-up echocardiography with improved regional function and global longitudinal strain of –14.4%. **C.** Suggested diagnostic workflow and clinical management of peripartum cardiomyopathy including the BOARD algorithm

Abbreviations: CMR, cardiac magnetic resonance

25% of women with PPCM [4]. Arrhythmias were reported in 19% of women with PPCM, of whom 7% had ventricular tachycardia or a cardiac arrest; the mortality rate reached 6% [5]. Therefore, implementing therapy according to the BOARD algorithm (Bromocriptine, Oral heart failure drugs, Anticoagulation, vasoRelaxants, Diuretics) is highly recommended [2, 4] (Figure 1C).

Preeclampsia and eclampsia (inevitably associated with arterial hypertension) are the conditions that support, rather than exclude, the diagnosis of PPCM. Careful monitoring and implementation of BOARD algorithm treatment are crucial to avoid life-threatening complications.

Article information

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