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Case Report

Preeclampsia Complicated with Peripartum Cardiomyopathy: A Case Report

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Abstract

Peripartum cardiomyopathy is a life-threatening and rare clinical syndrome requiring emergent treatment. Preeclampsia is one of the high-risk factors of peripartum cardiomyopathy. Signs of peripartum cardiomyopathy are often confused by other forms of heart failure, and delayed diagnosis will bring fatal complications to the mother and child. In this article, we report a case of a 31-year-old woman who presented as preeclampsia complicated with acute heart failure initially. According to the results of laboratory and echocardiography, we confirmed the diagnosis of perinatal cardiomyopathy. Through timely termination of pregnancy and postoperative heart failure treatment, the woman recovered well. Obstetricians often feel uncertain about how to quickly identify and diagnose peripartum cardiomyopathy, especially when combined with preeclampsia. An in-depth understanding of the different definitions and diagnostic features of these two conditions, as well as accurate characterization of the echocardiography in preeclampsia and peripartum cardiomyopathy, allows clinicians to manage these conditions appropriately.

Keywords: Peripartum cardiomyopathy; B-type natriuretic peptide; Echocardiography; Heart failure; Preeclampsia

Abbreviations

PPCM: Peripartum Cardiomyopathy; HF: Heart Failure; BNP: B-type Natriuretic Peptide; EF: Ejection Fraction

Introduction

Peripartum Cardiomyopathy (PPCM) refers to an idiopathic Heart Failure (HF) of unknown causes, characterized by marked impairment of left ventricular systolic dysfunction that presents in the last month of pregnancy or lasting for 5 months postpartum [1]. Along with increased maternal age, an increase in the rate of multiple pregnancies due to assisted reproductive technology, and increased awareness of the condition, the incidence of PPCM is increasing in America and is estimated to be 1 in 3200 births [2], the incidence of PPCM in Denmark and south Korea is 1 per 10149 deliveries [3] and 1 in 1741 deliveries [4], respectively. By contrast, the highest prevalence is in Haiti which is 1 in 300 deliveries [5], these indicate that the incidence of PPCM varies with ethnic and geographic differences, and PPCM is an uncommon form of cardiovascular disease during pregnancy.

In this report, we describe a case of PPCM, in combination with laboratory and echocardiography examination, the diagnosis of PPCM was confirmed. Through this case report, we also shed light on the diagnosis and differential diagnosis of PPCM.

Case Presentation

A 31-year-old primiparous woman who was admitted to our hospital due to elevated blood pressure and progressive edema. The woman had regular pregnancy examination, with normal range of blood pressure and a negative urine protein. One month ago, there was swelling of both ankles, which were progressive aggravation, the weight gain in the past week was 4kg. At 38 weeks of gestation, the woman showed blood pressure of 157/105mmhg, 24-hour protein quantity was 4921mg, so, she was hospitalized with a diagnosis of severe preeclampsia. Physical examination was as follows: blood pressure: 150/120mmhg, heart rate: 120times/min, edema (+ + + +), shortness of breath, occasional dry cough and orthopnea, pulmonary auscultation revealed bibasilar thick. Emergency biochemistry: normal liver and kidney function. No obvious abnormalities in blood routine and coagulation. B-type Natriuretic Peptide (BNP): >10000pg/ml. Echocardiography: Ejection Fraction (EF): 40% (Figure 1A), left heart enlargement (Figure 1B), normal right heart,

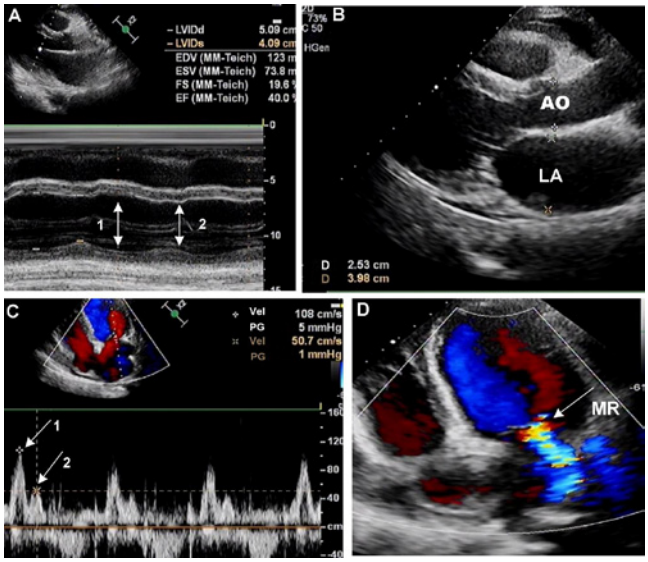

Figure 1: (A) Echocardiography showing: Ejection fraction; (EF): 40%; low cardiac contractility: Diastolic Inner Diameter of Left Ventricular (LVIDd): 5.09cm (arrow 1), systolic Inner Diameter of Left Ventricle (LVIDs): 4.09cm (arrow 2). (B) Evaluation of the Left Atrial (LA) size, the anteroposterior distance of LA is 3.98cm. (C) Evaluation of E peak (arrow 1) and A peak (arrow 2) of mitral valve forward flow. (D) Echocardiography showing regurgitation of mitral valve (MR) (white arrow). Ao: Aorta; EDV: End-Diastolic Volume; ESV: End-Systolic Volume; FS: Fractional Shortening.

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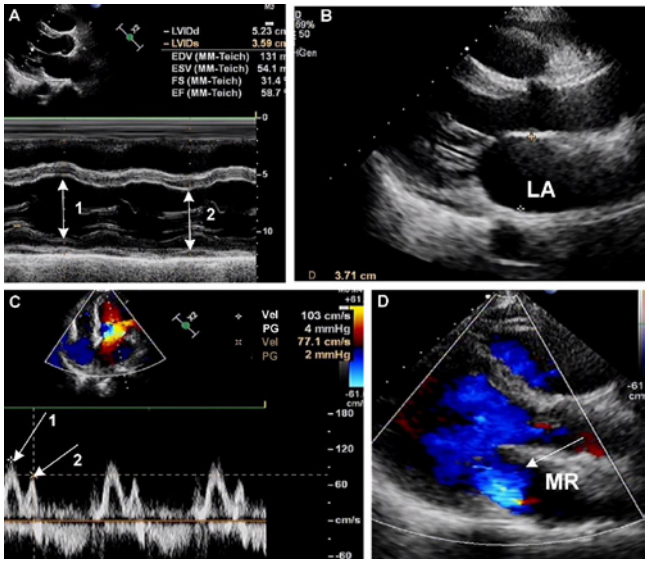

Figure 2: (A) Echocardiography showing: ejection fraction; (EF) 59%, improvement of cardiac contractility: diastolic Inner Diameter of Left Ventricular (LVIDd): 5.23cm (arrow 1), systolic Inner Diameter of Left Ventricle (LVIDs): 3.59cm (arrow 2). (B) Evaluation of the Left Atrial (LA) size, the anteroposterior distance of LA is 3.71cm. (C) Evaluation of E peak (arrow 1) and A peak (arrow 2) of mitral valve forward flow. (D) Echocardiography showing regurgitation of mitral valve (MR) (white arrow). Ao: Aorta; EDV: End-Diastolic Volume; ESV: End-Systolic Volume; FS: Fractional Shortening.

e peak of mitral valve forward flow was more than 2A peak (Figure 1C), in systolic phase, mitral valve and tricuspid valve showed a small amount of regurgitation (Figure 1D), in diastolic phase, aortic valve showed a small amount of regurgitation. Electrocardiogram showed sinus rhythm. The past history and family history of the woman were unremarkable.

In a combination of laboratory and echocardiography examination, the diagnosis of PPCM was made, emergency cesarean section was performed on the woman. During the operation, general anesthesia was performed, the woman was managed with blood pressure from 121/68 to 137/80mmHg. The intraoperative blood loss was 200 ml, and 200ml of crystalloid was administrated. After operation, furosemide was given for volume control, angiotensin receptor antagonist for antihypertensive therapy, and low molecular weight heparin sodium for thromboembolism prevention. BNP was continuously monitored: 5934-2621-1362pg/ml. On the third day postpartum, Echocardiography indicated improvements in cardiac function: EF: 59% (Figure 2A), normal size of left atrial (Figure 2B), E peak of mitral forward flow > A peak (Figure 2C), and mitral valve showed a small amount of regurgitation (Figure 2D). Chest radiograph: the shape and size of the heart were normal, and the texture of both lungs was increased and blurred.

The woman had a satisfactory recovery, 24-hour ambulatory blood pressure monitoring showed an average blood pressure of 132/87mmhg, an average heart rate of 76 beats/min.

Discussion

Relationship between PPCM and preeclampsia

Preeclampsia is strongly associated with PPCM [6], which has been recorded to occur in 30% of PPCM cases [7]. Research reported increased prevalence of hypertensive disease among patients with PPCM [8], and the strength of association is related to the severity of hypertension [9]. The high incidence of preeclampsia in woman with PPCM indicates a shared pathophysiology: Angiogenic imbalance [10], which was characterized by an excess of soluble fms-like tyrosine kinase-1 that is crucial in the mechanism of preeclampsia [11], and soluble fms-like tyrosine kinase-1 is also increased in PPCM [12]. Therefore, preeclampsia is closely related to PPCM, the two conditions have a common etiological pathway.

How to distinguish PPCM from preeclampsia-induced heart failure?

Preeclampsia is a distinct complication of pregnancy that affects 2-5% all pregnancies [13]. As the clinical syndrome of preeclampsia develops, the cardiac output drops dramatically followed by increased systemic vascular resistance, these can lead to diastolic dysfunction, which in turn cause HF [14,15], thus, preeclampsia is a high-risk factor of HF [16], study found that 51.43% of pregnant women with HF was complicated with severe preeclampsia [17]. Therefore, HF related to PPCM and to preeclampsia presents specific diagnostic challenge.

Researchers suggested that there were significant differences in the time of onset of HF, clinical, electrocardiographic and echocardiographic features of hypertension-induced HF of pregnancy compared to PPCM [18].

- PPCM is found to be a postpartum condition, with 53% of PPCM patients presenting within the first month of the puerperium and other 47% presenting within the subsequent four months [18]. Whereas 85% of hypertension-induced HF presented in the late pregnancy due to increased placenta secretes increasing levels of antiangiogenic factors into maternal circulation [19]. This is consistent with other studies that most PPCM women present postpartum [20,21].

- HF in preeclampsia is often associated with abnormalities of hepatic, hematological and renal function, and acute pulmonary edema is the most common cardio-pulmonary complication of preeclampsia. The majority of PPCM women only present with signs and symptoms of HF.

- With regarding to electrocardiogram, PPCM typically shows sinus tachycardia with nonspecific changes [22]. Due to increased systemic vascular resistance of preeclampsia, it was shown on the electrocardiogram as ventricular arrhythmias, in particular ventricular tachycardia.

- Echocardiography is the most important tool for diagnosis and differential diagnosis of PPCM, on echocardiography, diastolic dysfunction with preserved EF, increased filling pressure, increased left ventricular wall thickness and ventricular hypertrophy are present in patients with preeclampsia [23-25]. In contrast, the most important echocardiographic finding of PPCM is left ventricular systolic dysfunction, then of left ventricular and biatrial enlargement, mitral and tricuspid regurgitation.

Meaning of BNP in differential diagnosis of PPCM

BNP is secreted from cardiac myocytes as response to left

ventricle myocyte distension, which is an approved biological marker for the diagnosis and evaluating of non-pregnancy HF [26]. For healthy pregnant women, BNP values remain a relatively stable low level [27], and BNP under 100pg/mL has a good correlation with no adverse cardia events during pregnancy [28]. Research demonstrated that BNP was markedly elevated in PPCM patients [29] and in patients with gestational hypertension-induced HF [30]. However, the threshold of BNP for diagnosis and differential diagnosis of PPCM and hypertension-induced HF is lack, and many factors affect the secretion of BNP, such as neurohormonal factors, ischaemia and anaemia [31], so BNP is helpful in the diagnosis of PPCM but can't differentiate between PPCM and other form of HF.

Conclusion

In summary, enormous contribution has been made in the last decades in our understanding of PPCM. However, there are still many unsolved mysteries, such as specific diagnostic tests and disease-specific markers are lacking. We believe that with appropriate multi-center, international efforts, progress in the next few years will be tremendous.

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