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Cardiogenic shock and lower limb Necrosis after delivery: Peripartum cardiomyopathy was incriminated

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ABSTRACT
Peripartum cardiomyopathy is a rare disease accountable for giving a heart failure, which affects women in the last month of pregnancy or within the first 5 postpartum months. Early signs and symptoms of heart failure may not be seen, they are often considered as a normal part of pregnancy. When such symptoms and signs are not diagnosed or managed accurately, the consequences can be deleterious for the patient.We are going to briefly report a case of a 28-years-old woman, without any preexisting structural heart disease. The woman, after 3 months of vaginal delivery following normal pregnancy, was admitted for cardiogenic shock and distal lower Limb Necrosis secondary to peripartum cardiomyopathy. Such complications are uncommon, and the management was not easily accepted by the patient.

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Introduction

Peripartum cardiomyopathy (PPCM) is defined as a congestive heart failure resulting from left ventricular systolic dysfunction in the last month of pregnancy or within the first 5 months after delivery [1].

The true incidence of peripartum cardiomyopathy is unknown, but a significantly higher incidence has been reported in South Africa and Haiti.

The etiology and pathogenesis of PPCM is unknown, but several hypotheses have been proposed over the years [2].

PPCM can be associated with severe complications, including pulmonary edema, cardiogenic shock, arrhythmias, thromboembolic events, and mortality [3].Our case report describes a case of a young woman, who was admitted for chronic lower extremity ischemia and cardiogenic shock after spontaneous delivery. As the cause of the symptoms was diagnosed as peripartum cardiomyopathy.

Case Report

A 28-years-old woman, multipara, without history or evidence of preexisting structural heart disease. After 3 months of vaginal delivery following normal pregnancy of her third healthy child, she developed progressive dyspnea with important edema of the lower limbs which hides a bilateral necrosis of Lower Limb.

Unfortunately, she was admitted to our hospital so late with the signs of cardiogenic shock. On physical examination, the woman was orthopnoeic with sweats. She had a respiratory rate of 26 breaths/min and tachycardia at 150 beats/min with low blood pressure of 80/60 mmHg.

The rest of the examination revealed a Jugular turgescence, Hepatojugular reflux and important lower-limb edema with presence of henna embarrassing inspection.

The EKG showed a sinus tachycardia at 145 beats/min with right bundle branch block and inverted T waves in precordial leads.

Chest X-ray at admission had features of cardiomegaly and pulmonaryedema. Laboratory analysis revealed an inflammatory anemia at 9 g/dl, alteration of liver function tests and a functional renal insufficiency.

Echocardiography findings were consistent with biventricular dilated cardiomyopathy with left ventricular end diastolic dimension(LVEDD) of 61mm, global hypokinesia with severely depressed pump function (left ventricular rejection fraction(LVEF) of 17%), increased left ventricular filling pressure, the cardiac index was 1.5 L/min/m² “Fig. 1, Fig. 2”.

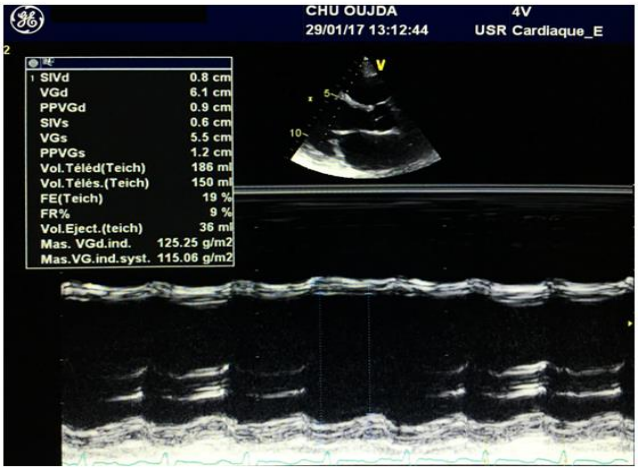

Figure 1. M-mode transthoracic echocardiography on long-axis view showing dilated LF with LVEDD of 61 mm and severe systolic dysfunction.

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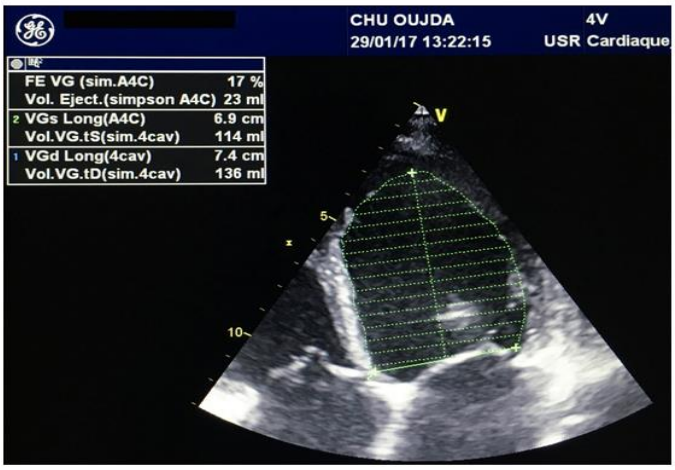

Figure 2.Apical 4-chamber, 2-dimensional echocardiogram showing severe systolic dysfunction at 17%.

The patient benefited from supportive management included propped up position, moist O2 inhalation, inotropic drugs (dobutamine), parenteral diuretics, ivabradine to control tachycardia, blood transfusion and anti-coagulants were also used.

The mechanical cardiac support was not required. Additionally, fluid and salt restriction was instituted. After clinical stability, the beta-blockers and spironolactone were started. The signs of heart failure subsided within 4 weeks.

After regression of edema of the lower limbs, clinical examination showed bilateral necrosis of the toes and the soles of both feet"Fig. 3"

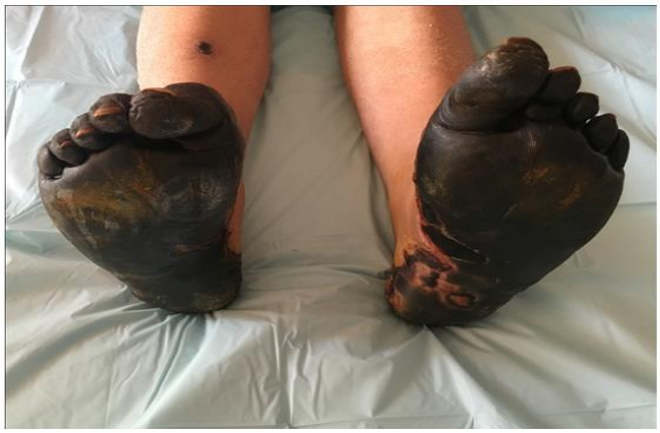

Figure 3. Bilateral necrosis of the toes and the soles of both feet

with the presence of a wound at the dorsal surface and the sole of the right foot"Fig. 4".

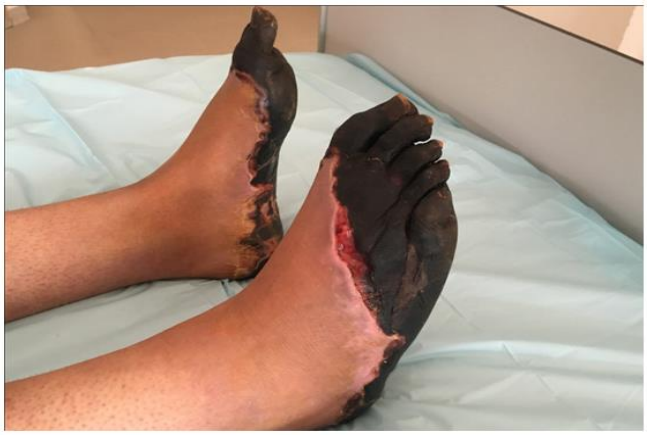

Figure 4.Wound at the dorsal surface and the sole of the right foot

Distal pulses were weak but present. The radiological examinations did not show an arterial disease (Arterial embolism or atherosclerosis) upstream of the necrosis, it was secondary to low cardiac output.

In the absence of viability, the amputation of the toes was indicated with debridement of necrotic wounds.

At the first follow-up,the young woman was clinically stable without significant LVEF improvement. The wound healing is in progress, but she reported symptoms of clinical depression because of the acute onset, severity, and prognostic uncertainty associated with her disease.

Discussion

Peripartum cardiomyopathy was first defined and described in year1971 [4] by the following criteria: (1) development of heart failure (HF) in the last month of pregnancy or within the first 5 postpartum months, (2) absence of a determined etiology, and (3) absence of a demonstrable heart disease before the last month of pregnancy.

The incidence has been reported to vary by geographical location with rates ranging from 1:15,000 pregnancies in united States, to as frequent as 1:299 in a well-studied population in Haiti and 1:100 in a small region in Sub-Saharan Africa [5].

Risk factors include older age, parity, African origin, toxemia or hypertension of pregnancy, use of tocolytics, twin pregnancy, obesity and low socioeconomic status. However 24–37% of cases may occur in young primigravidas. Although the etiology and pathogenesis of PPCM are still unknown, several hypotheses have been proposed, comprising viral myocarditis, apoptosis and prolactin toxicity, auto-immune mechanisms, malnutrition, hormonal changes, a result of complex interactions of pregnancy-associated factors against a susceptible genetic background [6].

Many of the signs and symptoms of PPCM are similar to those of HF caused by other factors. Because normal pregnancy is often associated with signs and symptoms that can resemble those of HF, the diagnosis of PPCM is often missed or delayed [3].

An electrocardiogram usually shows sinus tachycardia and nonspecific ST-segment and T wave changes. LV hypertrophy and conduction abnormalities can also be seen. A chest radiograph commonly demonstrates cardiomegaly, pulmonary venous congestion, and occasionally pulmonary edema and pleural effusion [7].

The gold standard for the diagnosis of PPCM is the echocardiography. Echocardiographic criteria include an ejection fraction less than 45%, LVEDD greater than 2.7 cm/m2, and fractional shortening of less than 30% [8].

Cardiac MRI has been used in a limited number of PPCM patients for the assessment of cardiac function and the detection of mural thrombi or myocardial fibrosis [9].

PPCM can be associated with severe complications, including pulmonary edema, cardiogenic shock, arrhythmias, thromboembolic events, and mortality [3]. Presentation with cardiogenic shock as our case is uncommon, but may require mechanical circulatory support as bridge to recovery or transplantation [10].Distal necrosis of the lower limbs was secondary to low cardiac output.

PPCM is known to have high mortality, ranging from 15 to 50% [11].Overall, cardiac function tends to return to normal in about 23 – 41% of cases [12].

There is still much to learn, but among the greatest advances have been an increased awareness of PPCM; and the application of the combination treatment of b-blockers

(BB) with angiotensin-converting enzyme inhibitors (ACEIs)/ angiotensin releasing hormone blockers (ARBs), as found in class I ("should") recommendations of the American Heart Association and European Society of Cardiology Guidelines for treatment of heart failure with reduced left ventricular ejection fraction (LVEF) [13].

The role of ivabradine has not been tested in a clinical trial in the management of PPCM. Moreover, its role in patients with cardiogenic shock is controversial [14].

In PPCM, anticoagulation is advisable from the time of the diagnosis until LV function recovers (LVEF >35%), or for the treatment of atrial fibrillation, because of the high incidence of thromboembolism associated with the disease [15, 16, 17].

The efficacy and safety of bromocriptine in PPCM patients have been evaluated in only one controlled randomized clinical trial [4]. More information is needed to clearly establish the safety and efficacy of this therapy for the treatment of PPCM [18].

Up to date, research on PPCM has largely focused on survival and little is known about long-term behavioral or psychosocial outcomes. Given the acute onset, severity, and prognostic uncertainty associated with PPCM, women appear to have an inherently higher risk for poor psychological outcomes, depression in particular [19].

Conclusion

As previously demonstrated with the patient in the case report. It is crystal clear that towards the end of pregnancy or after delivery, the possibility of developing cardiomyopathy with high mortality and severe complications does exist despite entering pregnancy without preexisting heart disease. Therefore, making an earlier diagnosis and Aggressive medical is crucial for a good outcome.

Conflict Of Interest

The authors report no relationships that could be construed as a conflict of interest.

Reference

[1]J. Abboud, Y. Murat, C. Chen-Scarabelli. Peripartum cardiomyopathy: a comprehensive review. International Journal of Cardiology 118 (2007) 295–303.

[2]K.M. Karaye, M.Y. Henein. Peripartum cardiomyopathy: A review article. International Journal of Cardiology 164 (2013) 33–38

[3]Goland S, Modi K, Bitar F, et al. Clinical profile and predictors of complications in peripartum cardiomyopathy. J Card Fail 2009;15:645–50.

[4]Demakis JG, Rahimtoola SH. Peripartum cardiomyopathy. Circulation 1971;44:964–8.

[5] Sliwa K, Fett J, Elkayam U. Peripartum cardiomyopathy. Lancet. 2006;368:687-693.

[6]Kamilu M. Karaye, Michael Y. Henein. Peripartum cardiomyopathy: a review article. Int J Cardiol 2013;164:33-38.

[7]Uri Elkayam, Mohamad Barakat. Peripartum Cardiomyopathy. Cardiolgy Clinics 30 (2012) 435–440.

[8] Amos AM, JaberWA, Russell SD. Improved outcomes in peripartumcardiomyopathy with contemporary. Am Heart J 2006;152:509–13.

[9]Elkayam U. Clinical characteristics of peripartum cardiomyopathy in the United States: diagnosis, prognosis and management. J Am Coll Cardiol 2011;58:659–70.

[10]Emmert MY, Prêtre R, Ruschitzka F, Krähenmann F, Falk V, Wilhelm MJ. Peripartum cardiomyopathy with cardiogenic shock: recovery after prolactin inhibition and mechanical support. Ann Thorac Surg. 2011;91:274-276.

[11]Bonow Robert O, Mann Douglas L, Zippes Douglas P, Libby Petar, et al. Braunwald's Heart Disease, Textbook of Cardiovascular Medicine. 9th ed. 2010:1776-1777.

[12] Kamilu M. Karaye, Michael Y. Henein. Peripartum cardiomyopathy: a review article. Int J Cardiol 2013;164:33-38.

[13]James D. Fett, Dennis M. McNamara. Peripartum Cardiomyopathy in 2015. Canadian Journal of Cardiology - (2015) 1-3.

[14]K.U. Mert, G.Ö. Mert, İ. Altun, M. Biteker, Ivabradine in cardiogenic shock: fact or fiction?.Heart Lung 44 (2) (Mar–Apr, 2015) 178.

[15]Pearson GD, Veille JC, Rahimtoola S, et al. Peripartum cardiomyopathy: National Heart, Lung, and Blood Institute and Office of Rare Diseases. JAMA 2000;283:1183–8.

[16]Sliwa K, Hilfiker-Kleiner D, PetrieMC, et al. Current state of knowledge on aetiology, diagnosis, management, and therapy of peripartum cardiomyopathy: a position statement from the Heart Failure Association of the European Society of Cardiology Working Group on peripartum cardiomyopathy. Eur J Heart Fail 2010;12(8):767–78.

[17]Karaye KM, Sa'idu H, Habib AG. Peripartum and other cardiomyopathies in a Nigerian adult population. Int J Cardiol 2011;147(2):342–3.

[18]K. Sliwa, D. Hilfiker-Kleiner, Evaluation of bromocriptine in the treatment of acute severe peripartum cardiomyopathy: a proof-of-concept pilot study, Circulation 121 (13) (Apr 6, 2010) 1465–1473.

[19]Lindsey Rosman. Samuel F. Sears. Depression and health behaviors in women with Peripartum Cardiomyopathy. Heart & Lung (2017) 1-6.