

Cardiology-2020: Peripartum Cardiomyopathy: Four Case Reports with Different Outcomes - Marzia Cottini - Cardiac Surgery Unit, San Camillo Hospital, Rome, Italy

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Introduction:

Peripartum cardiomyopathy (PC) represents a rare, life-threatening condition of the late pregnancy or early puerperium, being its diagnosis usually suspected when a left ventricular systolic dysfunction and symptoms of heart failure occur between the last month of pregnancy and the first five months of postpartum. Notwithstanding age over 30 years, multiparity, twin pregnancies, history of hypertension as well as black race are all factors thought to increase the risk for developing this condition, for none of them a certain role has been yet demonstrated. The etiopathogenesis of PC was described the mice as increase of oxidative stress causing the augmentation of oxydative LDL and deletion of gene STAT 3 (the protective gene against oxydative distress. This STAT 3 arrangement put at high risk to PC by increasing of Cathepsin D and expression of activated prolactin (aPRO). The aPRO provoked the pro-apoptotic and antiangiogenesis of prolactin that got involved in cardiac cells.

Case Presenatation I

A 30-years-old, primiparous, Caucasian female admitted for cardiogenic shock the Emergency Department (ED) one day after giving birth (caesarean section). The patient was in relatively good health until the day of delivery, when she developed rapidly progressive dyspnea (NYHA functional class IV), supraventricular tachycardia (heart rate 130 bpm), it was marked hypotension. Upon arrival, the patient was immediately intubated and transferred to our Intensive Care Unit (ICU) for treatment prosecution. At the admission, since that the patient remained in shock with severe lactate acidosis despite administration of noradrenaline, dobutamine, and levosimendan, an intra-aortic balloon pump (IABP) was percutaneously inserted. Moreover, on the basis of a high circulating plasma levels of prolactin, and also by considering the early timing postpartum (just one day), a dopamine D2 receptor agonist was promptly

administrated (cabergoline 1 mg, single dose). Finally, the patient underwent also an endomyocardial biopsy in order to exclude a myocarditis etiology. At the third day from the admission in ICU, a respiratory and metabolic improvement was observed and the invasive ventilation was then interrupted. The fourth day it was possible to remove the IABP.

Case Presentation II

A 32-years-old, primiparous, Caucasian female referred to our attention from another hospital where she was hospitalized for acute heart failure 66 days after giving birth (spontaneous full-term delivery). The patient had Marfan Syndrome and she was in relatively good health until the 50 days of delivery, when she developed progressive dyspnea (NYHA functional class IIIb), supraventricular tachycardia, and fatigue. At the admission, a therapeutic approach with dobutamine, diuretics, ACE-inhibitors, and low beta-blockers dose was started. Conversely, given the low circulating plasma level of prolactin (0.4 ng/ mL) and delayed appearance of the clinical picture, cabergoline was not considered useful and, accordingly, not administrated. Six days after, not available withstanding a slight but significant clinical improvement, due to the evidence of repeated episodes of non-sustained ventricular tachycardia, the patient was transferred to our ICU as a precautionary measure.

Case Presentation III

A 25-years-old, primiparous, Caucasian female admitted for acute decompensate heart failure to the Emergency Department (ED) 60 days after giving birth (spontaneous full-term delivery). The patient was in relatively good health until the 50 days of delivery, when she developed progressive dyspnea (NYHA functional class IIIb), and fatigue. Upon arrival, the patient was promptly transferred to the Intensive Coronary Unit where she received standard therapy for acute heart failure, including inotrope, vasodilators and diuretics and, due to the echocardiographic evidence of

left ventricular thrombosis (also confirmed by cardiac resonance imaging), anticoagulation with continuous heparin infusion was started. During the first month of hospitalization, seriate echocardiographic assessments not show any significant improvement in left ventricular systolic function (LVEF from 15% to 20%). Moreover, on the basis of repeated sustained ventricular arrhythmias and one ventricular fibrillation treated with transthoracic DC shock at 360 Joule, the patient received an implantable cardioverter defibrillator as secondary prophylaxis of sudden arrhythmic death. Thereafter, she was referred to our department for treatment prosecution and, mainly, to evaluate patients eligibility for heart transplantation. At the admission, the patient was haemodynamically unstable with supraventricular tachycardia (heart rate equal to 110 bpm), marked hypotension, and severe symptoms of heart failure (NYHA IV).

Case Presentataion IV

A 28-years-old, primiparous, Caucasian female admitted for heart failure to the Emergency Department (ED) 58 days after giving birth (spontaneous full-term delivery). The patient had no clinical history of cardiac disease and other comorbidities. She had developed a respiratory distress and then progressive dyspnea (NYHA functional class IV) and fatigue. She started to receive standard therapy for acute heart failure, including inotrope, vasodilators and diuretics. The echocardiogram evidenced left ventricular ipokinesis and akinesis, low ejection fraction (LVEF <20%), and increase dimension and volume of LV. The anticoagulation oral therapy combined with continuous heparin infusion was started immediately. Meanwhile the seriate echocardiographic assessments documented the functional restore of the heart, she had repeated sustained ventricular arrhythmias (treated with transthoracic DC shock at 360 Joule and anti-arrhythmic drugs), hence the patient received an implantable cardioverter defibrillator as secondary prophylaxis of sudden arrhythmic death. She was discharged 4 weeks after and she patient is still followed by dedicated ambulatory, Nowadays she is completely asymptomatic (NYHA I) and her echocardiographic parameters, as well as her laboratoratoristic data, are all into the normal range.

Discussion:

The current brief report described cases all pertaining to young, Caucasian, primiparous, female patients without any concomitant comorbidities with an overlapping clinical picture of decompensated acute heart failure.

According to our experience, the PC was developed less than 10% before partum and the most of them after delivery (Days after delivery: 55 ± 32). The diagnostic suspicion of PC needed immediately clinical and therapeutic managements because of the rapid and uncontrolled hemodynamic worsening. Also according to Donfrancesco et al., we combined Levosimendan to Carbergoline to functional cardiac recovery monitoring by BNP and PRL daily dosages. Case 2 was admitted and treated rapidly but she had not a complete healing.

Case 3 was more complex because she had coagulative disorders causing multiple peripheral and cardiac intracavitary thrombosis. She was treated with specific anticoagulation and mechanical/medical support but without results. Therefore, she was transplanted and she was followed by our dedicated ambulatory.

Case 4 responded optimally and immediately to the medical support and the seriate echocardiogram documented a complete restore of the cardiac functionality. Recently, she completed her second pregnancy without any major and cardiac complications, Analyzing extensively the four cases, the only differential parameter is leukocytosis: the three cases had neutrophilia instead the case 4 had eosinophilia. At the moment we have not had enough informations and data to explain this unusual type of leukocytosis in our patient but the etiopathogenesis of PC could play a central role. This could be a future step of our research.

Conclusion:

The peripartum cardiomyopathy is a cause of heart failure after partum requiring rapid and complex medical and mechanical supports. In the most of the clinical cases, the appropriate therapy could restore cardiac functionality completely. However the misunderstood of PM could cause life-threatening complications in patient and its rapid and uncontrolled hemodynamic worsening could require heart

transplantation also. A rapid diagnosis and advanced heart failure therapy could reduce PC complications and could restore the cardiac function in order to a good quality of life and in few case (like Case 4) a possibility to another secure pregnancy.

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