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Dilated Cardiomyopathy Due to Methamphetamine Use in a Pregnant Patient: Case Report

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ABSTRACT

Background: Dilated Cardiomyopathy (DCM) is characterized by dilation of the left ventricle (LV) and, by consequence contractile dysfunction. DCM is caused by multiple etiologies, often the result of myocarditis, alcohol consumption, drugs and metabolic or endocrine alterations.

Case: We report the case of a 36-year-old patient with a 27.6-week pregnancy with a pathological history of Heart Failure (HF) of 4 years, alcoholism of more than 15 years, methamphetamine use of more than 3 years. She presented agitation and class III dyspnea, the fatigue progressed until she presented respiratory distress accompanied by edema of the lower limbs, an echocardiogram was performed, Dilated Cardiomyopathy was diagnosed and therefore primary management was made with levosimendan and diuretic then cesarean surgery was performed.

Conclusion: for this patient the use of levosimendan was beneficial, improving the symptomatology, increasing the cardiac function and making possible the performance of the cesarean delivery. There was no harm for the patient or the baby.

KEYWORDS: "Dilated Cardiomyopathy", "Methamphetamine", "Pregnancy", "Levosimendan"

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INTRODUCTION

The introduction of levosimendan to the market in 2000 has been very beneficial for patients with HF and DCM. The strongest indication for levosimendan is the use in short-term treatment of acutely decompensated chronic heart failure; it has shown superior efficacy compared to dobutamine in decompensated patients that use beta-blockers in their daily management. In context of the safety of this drug, there have been many clinical trials with evidence of being a safer option than traditional agents. [1]

An enlarged LV with a diminished systolic function measured by the left ventricle ejection fraction (LVEF) is what characterizes DCM. The systolic dysfunction is way more notable than the diastolic dysfunction. Although DCM syndrome has multiple causes, it is often the result of myocarditis, alcohol consumption, drugs, and metabolic or endocrine alterations.[2]

Methamphetamine usage can cause direct myocardial damage by increasing the production of free radicals which promotes oxidative stress, altered expression of genes, mitochondrial dysfunction, and lack of control in intracellular calcium, all of this contributes to the contractile dysfunction of the heart by generating deficiency in electromechanical coupling, calcium signaling and by increasing inflammation. Chronically, inflammation and myocyte loss with replacement fibrosis lead to dilation of cardiac cavities with impaired systolic function. [3]

The clinical presentation of DCM is similar to HF and includes symptoms of LV failure, as progressive exertional dyspnea, fatigue, asthenia, adynamia, orthopnea, paroxysmal nocturnal dyspnea and nocturnal cough. While chronic systemic venous congestion induces ascites and peripheral

edema. The symptoms may remain hidden, so the patient may report edema and dyspnea. [4]

The incidence of DCM is 5-8 per 100,000 inhabitants, while the prevalence is 36 per 100,000. DCM is responsible for about 10,000 deaths each year and accounts for between 30% and 40% of HF cases.

Imaging studies are essential to determine the severity of DCM. Chest X-rays show an increase in the size of the cardiac silhouette, interstitial and alveolar edema, pleural effusion, and even redistribution of pulmonary blood flow. [5]

The transthoracic echocardiogram provides a reliable first instance prognosis and helps to identify additional cardiac abnormalities such as valvular diseases, allowing the first therapeutic steps to be initiated. This characteristically shows dilatation of the four chambers, with thinning of the wall, global hypokinesia, and a LVEF of less than 35-40%. Treatment is based on symptoms, it must be non-pharmacological (reduction of water and salt intake, moderate exercise) and pharmacological (Angiotensin-converting Enzyme Inhibitors, Angiotensin Receptor Antagonists, ß-blockers, Mineralocorticoid Receptor Antagonists). The initial treatment is a combination of ACEI and ß-blocker. A mineralocorticoid receptor antagonist is added if symptoms persist and an LVEF of 35% or less, if symptoms continue, defibrillator implantation is recommended. [6]

HEART FAILURE & PREGNANCY

Cardiac adaptation to pregnancy consists of an increase in circulating volume with an increase in heart rate of 10-15 beats per minute with an associated increase of 30% to 50% in cardiac output, this is proportionally associated with gestational age. However, in patients with HF this adaptation is modified, since it is in the third trimester where a greater increase in hemodynamic load is observed, which can lead to cardiac decompensation due to previous myocardial remodeling, with an increase percentage to 18% in maternal-fetal morbidity and mortality, compared to pregnant patients without previous DCM (7%) [7].

Symptoms of HF, cardiogenic shock can appear during peripartum cardiomyopathy, this term denotes HF in the form of DCM, its treatment is based on symptoms and does not differ from the treatment of HF, it has been emphasized in the positive impact of levosimendan in this type of cardiomyopathy. Levosimendan is a positive inotrope, which increases cardiac output (CO), increases stroke volume (SV), decreases pulmonary capillary pressure (PCP), decreases pulmonary vascular resistance (PVR), decreases mean pulmonary arterial pressure (mPAP) and decreases systemic vascular resistance (SVR). [8]

Several factors can worsen the prognosis including advanced NYHA classification, male gender, chronic HF, kidney failure. Progression to HF depends on the ejection fraction and the cause of the disease. Almost 50% of patients die within 5 years. [9]

CASE

A 36-year-old patient with a weight of 105 kg and a height of 157 cm, with a 27.6-week pregnancy due to US, a 4-year history of heart failure secondary to toxins (methamphetamines and alcohol) initially managed with metoprolol, spironolactone, furosemide and enalapril, currently treated with methyldopa, 2 previous pregnancies (1 natural delivery, 1 fetal death at 24 weeks of gestation), occasional alcoholism for 15 years, suspended 6 months before the hospital admission, heavy use of methamphetamines for more than 8 years, allergies, transfusions and previous surgeries denied.

Present condition begins 10 days previous to the hospital admission with fatigue and dyspnea (NYHA III). Fatigue progresses, which prompted the search for medical attention. At the arrival to emergency room, physical examination showed lung fields with right bibasal hypoaeration, pregnant abdomen due to gravid uterus, with a single live product (patient referred the presence of fetal movements), lower limbs with bilateral infracondylar edema ++/+++, capillary refill of 3 seconds. The initial management in hospitalization was with Furosemide 40 mg every 8 hours intravenously, Acetaminophen 1 gram every 6 hours intravenously, Betamethasone sodium phosphate 12 mg every 24 hours intramuscularly, Isosorbide 10 mg every 12 hours orally.

Given the diagnosis of HF and dyspnea, dobutamine was started at a dose of 0.79 mcg/kg/min as well as a 40 mg loop diuretic with a balance in the first hours of -2435 ml. An echocardiogram was performed which reported severely dilated LV. LVEF of 20%. Global and severe hypokinesia pattern. Diastolic dysfunction with restrictive filling pattern. Left and right atrium severely dilated. Severely dilated right ventricle. 18mm TAPSE. Critical mitral regurgitation. Dilated pulmonary veins. Tricuspid valve with severe regurgitation. Maximum IT of 3.80 m/s corresponding to a pressure of 57.76 mmHg. Dilated inferior vena cava without inspiratory collapse. PASP +15 mmHg. Total PASP 72.76 mmHg corresponds to severe pulmonary hypertension.

Global DCM is diagnosed, dilated LV with global hypokinesia with LVEF of 20%, secondary pulmonary hypertension and mitral regurgitation was integrated. Given this, it was decided to add levosimendan at a dose of 0.5 gammas, in the following hours with hemodynamic stability.

Obstetric ultrasound reported a live single fetus of 30.6 weeks of gestation and weight of 1768 grams.

The evolution at 24 hours was satisfactory, with the previously described management, tolerating decubitus, saturation of 96% without supplemental oxygen, blood

pressure of 110/60 mm Hg, heart rate of 100 beats per minute, respiratory rate of 14 breaths per minute. On physical examination, left ventricular gallop and murmur of mitral insufficiency were auscultated, absence of pulmonary rales, and presence of edema of the lower limbs +/+++. At 48 hours of evolution, based on the satisfactory hemodynamic evolution and fetal status, the termination of the pregnancy by cesarean section was decided.

At 72 hours, the patient was stable, with mean arterial pressure of 70 mmHg, without data of respiratory distress and saturating 97% at room air. Levosimendan dosage was gradually increased from 0.06 mcg/kg/min to 0.1 mcg/kg/min.

The patient was transferred to the operating room where a single live product weighing 1,540 grams was obtained. After the cesarean section, arterial hypotension of 95/60 mmHg was reported, for which the infusion of levosimendan is reduced to 0.06 mcg/kg/min and norepinephrine is started at dose of 0.03 µg/kg/min (ranges), then Digoxin 0.5 mg intravenously every 8 hours for 3 doses was started, then 0.125 mg intravenously every 24 hours for treatment of long-term HF.

Weaning and withdrawal of aminergic and inotropic drugs was started and prescribed with regular pharmacological management with ACE inhibitors, beta-blockers, diuretics, as well as digoxin.

At the fifth day of hospitalization, the evolution had been adequate, so she was able to be discharged from the unit with usual medication and admitted to Hospital Materno Infantil for post-cesarean section care.

DISCUSSION

The present case shows a young woman, with a full-term pregnancy who had HF secondary to methamphetamines, this entity is increasingly frequent due to the use of these substances more deeply rooted in the population, consumption has been increasing during the last decade , reporting 15.8% of men and 4.3% in women, being already a public health problem[10], making this a very high risk pregnancy. The management allowed to carry out with good results. Without ceasing to be a challenge the management of similar patients.

It has been seen that most of the causes of MCD are genetic, adding the use of methamphetamines at an early age we will be able to observe in the future an increase in the incidence of MCD in young patients, which must be considered due to the presentation of the symptoms, since the beginning of the picture can go unnoticed as it is non-specific.

HIGHLIGHTS

- Use of Levosimendan in a rare case of a pregnant patient with DCM
- Levosimendan as a pre-surgical measure
- Reassures the safety of the use of levosimendan in the third trimester of pregnancy

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